73 Functional disorders of the intestine

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ACUTE ADYNAMIC NEUROMUSCULAR STATES OF THE SMALL I

ACUTE ADYNAMIC NEUROMUSCULAR STATES OF THE SMALL INTESTINE WITH DILATATION: ILEUS
Definition

Ileus can be defined as: a disruption of the normal propulsive ability of the intestine due to a malfunction of contractile activity in the absence of mechanical obstruction. This definition excepts certain older terms such as ‘meconium ileus’ and ‘gallstone ileus’ (see Chapters 17 and 78) that persist in usage, although they are technically misnomers (i.e. there is mechanical obstruction). The term ‘paralytic ileus’, although descriptive for the student, is outdated and not entirely correct since studies show that motor activity is not abolished but rather dysregulated.

APPLIED ANATOMY AND PHYSIOLOGY

The intestine must subserve basic functions of moving contents from proximal to distal in a rhythmical fashion to allow mixing, digestion and absorption of contents. The motility of the intestine has been studied for more than a century and all readers should know of the seminal experiments of Bayliss and Starling in their 1899 paper ‘The movements and innervation of the small intestine’, which led to adoption of the term ‘peri stalsis’. In the small intestine, fasting motility can be described by the three phases of the migrating motor complex (MMC), with fed activity resembling phase II. Colonic motility is muc Sir William Bayliss , 1860–1924, physiologist, and Ernest Henry Starling medicine also included Starling’s principle (capillary pressures) and filling of the heart (Frank–Starling law). Santiago Ramon y Cajal , 1852–1934, Spanish neuroscientist, pathologist and Nobel prize winner (1906) for studies of cellular ana more complicated and still poorly understood with some features akin to the MMC but also specific phenomena such as retrograde movements (presumed to allow greater resident time and therefor e fluid and electrolyte absorption). The main characteristics of intestinal motility are shown in Table 73.1 . The intestine, like the heart, is autonomous in generating its own rhythmical electrical, and therefore local motor, activity by intrinsic pacemaker activity generated by small fibroblast - like cells called the interstitial cells of Cajal. These cells, whic h are mainly resident within the muscularis propria, have several - key functions , including setting the membrane potential of smooth muscle cells so that they are primed to contract and connecting smooth muscle cells electrically so that synchronous h , 1866–1927, University College London, London, UK. Starling’s contributions to tomy of the nervous system.

TABLE 73.1 Contractile activity of the intestine. Region Broad category Small intestine Phase I Quiescence (40–60% of total time) Phase II High-frequency contractions allowing mixing and absorption (20–30% of total time) Phase III High-amplitude propagated activity (5–10 minutes) Large intestine Phasic contractions Low-amplitude propagated pressure waves High-amplitude propagated pressure waves Retrograde pressure waves Simultaneous pressure waves Periodic colonic and rectal motor activity (localised bursts) Tonic contractions Sustained activity responsible for tone a Most akin to phase III of the migrating motor complex and responsible for mass movements of faecal content. The management of common chronic disorders that • present to surgeons such as chronic constipation and irritable bowel syndrome The existence of several rare neuromuscular diseases that • may affect the intestine The limited role of surgery in the treatment of most of • these disorders a

a ff ected by a hierarchy of external control systems but mainly by the enteric nervous system (ENS) via the myenteric plexus. The myenteric plexus is one of the two intramural plexuses of the

ENS (the other being the submucosal plexus). The former has the major role in motor functions while the latter has roles in sensing, mucosal blood flow regulation and secretion. Both are composed of small groups of enteric neurones that congregate with glial cells to form ganglia, these being Summary box 73.1 Regulation of intestinal contractile activity /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF The rectum constitutes a final and specialised end to the intestine. Its role is mainly for temporary storage of fae ces prior to defecation. This role permits both further water absorption and the ability of higher mammals to socially defe cate (an ability shared with small rodents as well as many larger species). To this end, the wall of the rectum is specialised in terms of compliance and of having nerve endings that provide conscious perception of filling. In concert with the upper anal The ENS has a neurochemical complexity and number of neurones (five times the number in the spinal cord) that has led to it being called the ‘little brain’ (Summary box 73.1). Thus, although higher control mechanisms including the autonomic nervous system (ANS) and brain allow the intestinal motility to respond to wider environmental cues, e.g. waking, exercise, the smell and taste of food and stress, the intestine can initiate and sustain peristalsis without any external inputs. canal, the rectum is also capable of distinguishing solid, liquid and g as by the ‘sampling’ reflex. Together the act of defecation requires complex neuromuscular functions and it is no sur - prise that it goes wrong with su ffi cient regularity to cause much human miser y in the form of constipation and incontinence (see Chapter 80).

Longitudinal muscle Mucosa Figure 73.1 Schematic diagram of the enteric nervous system. SMP , submucosal plexus. (Reproduced by permission from Springer Nature. Furness JB. The enteric nervous system and neurogastroenterology. Hepatol 2012; 9 : 286–94. © 2012.) Myogenic control mechanisms Interstitial cells of Cajal generating slow wave activity Neurogenic control mechanisms ENS (a variety of cells in myenteric and submucosal ganglia) ANS (sympathetic and parasympathetic mainly via ENS ganglia) Central nervous system (CNS) (brain–gut interactions) Chemical control mechanisms Local paracrine (especially from mucosal enteroendocrine cells) Endocrine Myenteric plexus Circular muscle Deep muscular plexus Outer SMP Inner SMP Submucosal artery Muscularis mucosae Nat Rev Gastroenterol

CHRONIC IMPAIRMENT OF INTESTINAL MOTILITY WITH DIL

CHRONIC IMPAIRMENT OF INTESTINAL MOTILITY WITH DILATATION OF THE SMALL INTESTINE: INTESTINAL PSEUDO OBSTRUCTION Definition

Intestinal pseudo-obstruction (IPO) is defined as: A clinical syndrome caused by severe impairment of intestinal motility leading to small intestinal dilatation in the absence of a mechanical cause. The term ‘chronic’ is sometimes added for clarity . CHRONIC IMPAIRMENT OF INTESTINAL MOTILITY WITH DILATATION OF THE LARGE INTESTINE: MEGACOLON AND MEGARECTUM Definition

Chronic dilatation in the absence of a mechanically obstruct - ing cause can be focused in the colon (megacolon) or rectum (megarectum), although in practice these commonly overlap (Figure 73.8). Megacolon may also accompany some forms of IPO in patients found to have chronic small and large intestinal dilatation. Toxic megacolon refers to an acute condition in which acute inflammation leads to a loss of compliance and rapid dilatation (it has nothing in common other than the name).

CHRONIC IMPAIRMENT OF INTESTINAL MOTILITY WITHOUT

CHRONIC IMPAIRMENT OF INTESTINAL MOTILITY WITHOUT DILATATION

Constipation and IBS are very common conditions and collec tively represent about a third of patients presenting to the aver age colorectal clinic in the Western world. They are presented here as separate entities to reflect the general approach of most physicians; however, the reader should be aware that ther very considerable overlap, especially between constipation and the constipation-predominant form of IBS (C-IBS). Patients can fulfil the criteria for both diagnoses concurrently or move between diagnoses over time.

Causes and risk factors

Causes and risk factors

The risk factors for ileus are listed in Summary box 73.3 Postoperative ileus (POI) occurs in 10–20% of patients under going elective major abdominal surgery and is usually defined by a failure to tolerate oral intake or pass stool 72 hours after surgery . Summary box 73.3 Risk factors for ileus /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Recent surgery: POI Local in /f_l ammation (peritonitis, severe acute pancreatitis) Systemic in /f_l ammation by any cause, e.g. sepsis, trauma Electrolyte disturbance (especially hypokalaemia and hypercalcaemia) Acute endocrine disturbance (hypothyroidism, diabetic ketoacidosis) Medications, e.g. opioids Acute CNS disease (especially high spinal transections) Intestinal ischaemia (mesenteric vascular disease)

Causes of megacolon and megarectum

Causes of megacolon and megarectum

Primary and secondary causes (Table 73.4) vary between megarectum and megacolon. The most common disease to use the term megacolon is Hirschsprung’s disease (occurring in 1 in 5000 live births) (see Chapter 17). Actually , in this instance, it can be argued that the so-called ‘congenital megacolon’ does in fact reflect a degree of distal obstruction from the distal contracted aganglionic segment. This leads to the absence of passage of meconium at birth and is generally incompatible with life without urgent surgery . Adult Hirschsprung’s disease is a very rare disease and leads to a megarectum because the a ff ected segment is ‘ultrashort’, a ff ecting only the transition zone of the anus. Histologically , this is very di ffi cult to diagnose with certainty and some challenge its existence at all. -

Megarectum Megacolon Figure 73.8 Schematic drawing of the distribution of bowel dilatation in megacolon and megarectum.

More common causes of megacolon include extreme senil ity and CNS neurodegenerative disease, resembling an attenu ated form of ACPO. Others are denoted ‘idiopathic’ to reflect that no cause is established; this group, who are predominantly female, phenotypically resemble a severe form of slo w-transit constipation (see Constipation). All are rare. Patients with megarectum are usually divided into two groups by clinicians. The first are those who have had previ ous surgery for Hirsc hsprung’s disease or anorectal malforma tions in whom ongoing problems are common – due perhaps to surgical reconstruction or an as yet undetermined neuro muscular disease. The second, pr edominantly male, group are sometimes described as ‘idiopathic’; however, nearly all, if assessed carefully , will have some form of psychobeha disorder. The pathogenesis is considered to be stool withhold ing in infancy or childhood, leading to chronic distension and loss of compliance.

Megacolon Primary Congenital Classic (rectosigmoid) Hirschsprung’s disease Rare early-onset (some genetic) myopathies and neuropathies Acquired Rare late-onset (some genetic mitochondrial) myopathies and neuropathies Unknown (termed ‘idiopathic’) Secondary Genetic Muscular dystrophy and other rare genetic muscle diseases MEN type 2B with ganglioneuromatosis Rare genetic autonomic neuropathies Acquired CNS diseases, including senility, Parkinson’s, dementias, amyloid and spinal cord injury Connective tissue disease, especially scler Infections: Chagas’ disease (South American trypanosomiasis) Autonomic neuropathies secondary to diabetes and paraneoplasia Megarectum Primary Congenital Ultrashort-segment Hirschsprung’ Inadequately resected Hirschsprung’ Anorectal malformations (post reconstruction) Secondary Congenital Severe psychobehavioural + cognitive impairment (+ genetic) Acquired Later-onset behavioural (autistic spectrum) disorders Sexual abuse; neglect; parental negativism CNS, central nervous

system; MEN, multiple endocrine neoplasia.

Causes

Causes

IPO is a rare disease. Approximately half of cases arise shortly after birth or in infancy , caused by a number of very rare enteric neuropathies and myopathies, including genetic and familial, inflammatory and degenerative forms. Other cases arise later in life when a secondary aetiology is more common. In some patients, a cause is not found and these are termed idiopathic. The full list of causes is given in Summary box 73.5 . Summary box 73.5 Causes of intestinal pseudo-obstruction /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Carlos Justiniano Ribeiro Chagas , 1879–1934, Director of the Oswaldo Cruz Institute and Professor of Tropical Medicine, University of Rio de Janeiro, Brazil. - IPO presents clinically with the symptoms and signs of small bowel obstruction with pain, distension and vomiting. After clinical evaluation and plain radiology , a degree of suspicion is helpful to avoid unnecessary and potentially harmful surgery . Such suspicion is merited when there is no obvious cause for - mechanical obstruction, i.e. no known bowel disease, previous surgery or hernia, and on the length of history . Here, knowing the list of secondary causes becomes helpful. For instance, in someone who is a smoker with finger clubbing, a small cell carcinoma of the lung may be the cause of paraneoplastic pseudo-obstruction; alternatively , the patient may have clinical signs of scleroderma. Axial imaging is essential to exclude mechanical obstruction. Adjunctive blood and imaging tests may help define a cause and these can include MRI of the brain and skeletal muscle biopsy for rare diagnoses such as mitochondrial myopathies. Primary neuropathies and myopathies can be diagnosed histologically , but this requires full-thickness tissue and a vari - - ety of special stains (available only in specialist centres). Since laparotomy and bowel resections are best a voided, a laparo - scopic or minilaparotomy full-thickness biopsy may be war - ranted for diagnosis (Figures 73.6 and 73.7).

Primary Several very rare enteric myopathies and neuropathies Unknown (termed ‘idiopathic’) Secondary Connective tissue disease, especially scleroderma Radiation injury Amyloidosis Autonomic neuropathies including diabetes and paraneoplasia Infections: Chagas’ disease (South American trypanosomiasis)

Clinical features

Clinical features

Symptoms include abdominal distension and vomiting akin to mechanical small bowel obstruction (see Chapter 78); however, colicky pain is less of a feature. On examination, other than evidence of the cause, e.g. recent surgery , the abdomen will be distended, tympanic and have reduced or absent bowel sounds. . Clinical features

Symptoms include abdominal distension, absolute constipa - tion and, as a later feature, vomiting akin to mechanical large), the bowel obstruction (see Chapter 78); however, colicky pain is less of a feature. The histor y is very important to establish risk factors, some of which may be modifiable. On abdominal examination, the abdomen is usually grossly distended and tympanic. In uncomplicated cases, the abdomen should not be tender. Tenderness and especially any evidence of peritonism indicate that massive colonic dilatation may have led to isch - aemia with/without perforation – a surgical emergency . Such complications occur in 3–15% of patients with advanced age and increased caecal diameter, with a delay in decompression increasing risk. Diagnosis relies upon accurate clinical observation and plain abdominal radiography showing deg rees of colonic dila - tation, mainly involving the proximal colon. CT is however the definitive in vestigation (Figure 73.5) to di ff erentiate mechan - ical fr om pseudo-obstruction, to provide a caecal diameter and to show any evidence of complications (e.g. perforation). A CT scan will also di ff erentiate pseudomembranous colitis with toxic dilatation, which is a further di ff erential diagnosis in hospitalised or institutionalised patients due to Clostridium di ffi cile infection.

Constipation

Constipation

Definitions ‘Constipation’ is not a disease but rather a term often used by patients to describe dissatisfaction with their bowel function or their ability to defecate. As such it means di ff erent things to di ff erent patients (and di ff erent doctors) and can describe symptoms that directly relate to defecation, e.g. straining, or those considered consequent in the abdomen, e.g. pain and bloating. More formal definitions such as that of the American College of Gastroenterologists – ‘unsatisfactory defecation, for at least 3 months’ – cover most symptoms and introduce a time criterion to exclude patients with transient symptoms (sometimes called ‘simple constipation’). Stricter definitions of ‘c hronic constipation’ include a measure of resistance to treat - ment – ‘unsatisfactory defecation characterized by infrequent stools, di ffi cult stool passage or both for at least 6 months where this has proven unresponsive to lifestyle alterations and basic laxative therapy’. Epidemiology - Self-reported constipation is very common, with a worldwide - prevalence of about 10% (making it one of the commonest ailments in humans). Fortunately , patients with chronic constipation (based on 6 months of symptoms and failure of e is at least two laxatives) are much less common (approximately 0.5%). Most studies report a higher prevalence of self-reported constipation in women than in men with a ratio of 2:1. The ratio is much higher for chronic constipation at approximately 9:1 female to male. Risk factors The vast majority of patients with chronic constipation lack a single unifying cause for their problems. The main associated medical conditions and diseases within the gastrointestinal tract itself are listed in Table 73.5 . Diagnosis Clinical history A thorough history will determine whether constipation represents a new complaint, i.e. one indicative of a change

TABLE 73.5 Risk factors for constipation. Gastrointestinal causes Mechanical obstruction Benign and malignant strictures Functional obstruction Pelvic organ prolapse syndromes (dynamic obstruction at the level of the anorectum) Megarectum Anal pain, e.g. chronic /f_i ssure Medical causes Metabolic disorders Hypercalcaemia, uraemia, hypokalaemia, hypomagnesaemia Endocrine disorders Hypothyroidism, diabetes, pregnancy Degenerative CNS diseases, e.g. multiple sclerosis, Parkinson’s, cerebrovascular disease, Neurological disorders spinal or pelvic nerve lesions, autonomic neuropathies, cognitive impairment Drugs Opioids Anticholinergics Calcium channel blockers Psychological Severe endogenous depression Eating disorders Cognitive behavioural disorders Other Connective tissue diseases Joint hypermobility Causes of immobility, e.g. degenerative joint disease CNS, central nervous system.

the frequency and consistency of bowel movements and the progress of such changes over time (as well as other alarm symptoms such as rectal bleeding and weight loss). With additional information regarding family history , previous colon cancer screening and other gastrointestinal investigations, an informed decision can be made whether intraluminal investigation of the colon is required. Other organic causes of constipation may be deduced by appropriate history taking and biochemical investigation. With the exclusion of treatable secondary causes, if the history is short

and multiple previous therapies have not already been tried, the patient may be first considered to have ‘simple’ constipation that can be managed with reassurance and lifestyle advice (fibre, fluids and exercise) with/without simple laxative therapy . In patients with chronic symptoms, after exclusion of a sec ondary cause, the focus should shift to the investigation and management of chronic constipation. Many patients may attribute the start of symptoms to a major life e vent. Common among these are hysterectomy and childbirth, other abdomi nal surgeries or trauma. Constipation can also be associated with previous abuse and it may sometimes be necessary to tact fully seek a history of physical or sexual abuse. Other patients will have no such triggers , having had symptoms from child hood and on occasion fr om infancy . Such patients are overwhelmingly female (>95%) and on investigation are often found to have generalised slow-transit constipation as opposed to other pathophysiological findings (this group , who represent 5–10% of patients with chronic constipation, are variably referred to in the literature are ‘idiopathic slow-transit consti pation’ or ‘colonic inertia’). It is helpful to systematically docu ment the main symptoms that in the patient’s mind constitute a problem since this has some bearing on treatment decisions and subsequent monitoring of e ff ectiveness. Several questions form detailed scoring systems to systematically facilitate this in a research context. How ever, in routine practice it is su ffi cient to list in the patient’s record the presence or absence of several common symptoms (Summary box 73.7). The presence of prolapse symptoms reflects the overlap between diagnoses in patients with pelvic floor disorders (see Chapter 80 remaining history should document prescribed and self- administered laxatives (and therapeutic benefit thereof) and also gain an impression of the quality of diet in respect of fibre and fluid intake. Clinical examination Poor nutritional status should prompt a search for a secondary cause, including occult carcinoma, more widespread intestinal motility disorders such as IPO (see Chronic impairment of intestinal motility with dilatation of the small intestine: intestinal pseudo-obstruction) and eating disorders. An abdominal examination should be conducted to look for scars, any significant abdominal distension, tenderness or masses. Bloating is a common and expected finding with chronic constipation, but significant distension, tenderness or masses should prompt a full investigation. All patients presenting with constipation should undergo a rectal examination. The perineum and anus should be examined for evidence of faecal incontinence that may indicate Symptoms to directly question in patients with constipation /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF - /uni25CF /uni25CF - - impaction and overflow . Some degree of faecal incontinence and chronic constipation coexists in 40% of patients; marked soiling of the underwear is especially associated with the rarer diagnosis of megarectum. Scarring, e.g. from episiotomy , sen - tinel pile formation secondary to an underlying anal fissure, external haemorrhoids or prolapse, may also be present. The - degree of perineal descent on straining, indicative of pelvic - floor weakness , should also be determined visually (>3 /uni00A0 cm is usually considered abnormal). A digital rectal examination will diagnose impaction, gain a rough measure of anal tone at rest and on squeeze and ascertain obvious sphincter defects. An e ff ort should be made to look for any anterior defect in the rectovaginal septum leading to a rectocele. Anoscopy and proctoscopy should be performed if there is any history of rectal bleeding and may indicate fissure or internal piles. A urogynaecological examination is desirable in all patients with). The suspected pelvic multi-organ prolapse. Investigations While findings from history or physical examination may indicate a secondary cause of constipation, making further investigation mandatory , it is also typical practice in patients with chronic constipation to exclude certain secondary causes by investigation even though the diagnostic utility of such investigations is acknowledged to be low (the commonest undiagnosed systemic disease is hypothyroidism). Thus,

serum electrolyte, creatinine, calcium, glucose, haemoglobin levels and thyroid function tests are usually performed. The approach taken to structural investigation of the colon when patients have no suspected intraluminal pathology varies on the basis of available resource and may include colonoscopy . In patients with chronic constipation in whom basic laxatives have failed, further specialist investigative tests may be warranted. Colonic transit can be investigated by a radio-opaque marker study (Figure 73.2). In addition, rectal

Abdominal symptoms Abdominal pain Bloating Defecatory symptoms Frequency of spontaneous or assisted bowel opening Painful defecation Stool consistency (can use Bristol stool scale) Digitation (vaginal or anal) Straining Incomplete/unsuccessful evacuation Leakage/incontinence Prolapse Other pelvic symptoms Vaginal bulging or prolapse Urinary incontinence

if the patient has a functional or dynamic structural cause of evacuation disorder. Problems such as dyssynergic defecation (functional) and intussusception/rectocele (structural) may occur in isolation or coexist with transit disturbances (Figure 73.10). Management The treatment of chronic constipation follows a stepwise progression from lifestyle changes through potentially to major surgery in a small minority of patients. Table 73.6 lists the main available approaches, noting where some apply only to certain diagnoses derived from the results of specialist tests of colonic and anorectal function. Figure 73.11 provides a basic algorithm to accompany Table 73.6 . symptoms for many years and will have tried a number of rem - edies and prescribed laxatives. They will also usually have tried to address lifestyle modifications. Before resorting to specialist tests, it is possible to try and rationalise laxative therapy and provide a programme of n urse-led behavioural interventions. In regard to laxatives, current advice is to stop current laxatives (unless these are working well) and then titrate an oral osmotic laxative, e.g. polyethylene glycol (PEG), until the stool form is soft or liquid. If this is insu ffi cient then a stimulant laxative such as bisacodyl may be added. If symptoms of obstructed defecation predominate then rectal laxatives in the form of suppositories or enemas may be tried with or without contin - uation of oral laxatives. The failure of such drugs should then prompt a trial of one of the newer prokinetic or secretagogue

Figure 73.10 Schematic overview of pathophysiology of chronic constipation. DD, defecation disorder; STC, slow-transit constipation. TABLE 73.6 Treatment options in patients with chronic constipation. Lifestyle Increase /f_l uid intake Dietary modi /f_i cation, e.g. increased /f_i bre Increase exercise Reduce body mass (pelvic /f_l oor prolapse syndromes) Drugs Oral laxatives (favoured for slow transit) Rectal laxatives (favoured for rectal evacuation disorders) Prokinetics, e.g. prucalopride Secretagogues, e.g. linaclotide Behavioural therapies Habit training Habit training with direct visual biofeedback (favoured for dyssynergic defecation) Pelvic /f_l oor muscle training (favoured for pelvic /f_l oor prolapse syndromes) Transanal irrigation High- or low-volume systems available Surgery See Summary box 73.8 15% normal 5% STC 45% mixed STC and DDs 40% DDs Structural Functional

drugs. These drugs are successful in a proportion of patients but do have some unwanted side e ff ects (that the patient should be warned about). All drugs should be tried daily for a mini mum of 4 weeks before concluding that they are ine ff ective and the reactionary use of laxatives, i.e. in response to being con stipated, rather than their preventative use, should be str discouraged. The most common form of behavioural intervention is often described by the term ‘habit training’. This

involves optimising dietary patterns to maximise gastrocolic response and the morning clustering of colonic high-amplitude prop aga ted contractions that propel contents towards the rectum for subsequent evacuation. Dietary advice to optimise intake of liquid and fibre is given as well as advice about frequency and length of toilet visits and posture (Figure 73.12). Patients ar e also instructed on basic gut anatomy and function and gain an appreciation of how psychological and social stresses may influence gut functioning. Simple pelvic floor and balloon expulsion exercises are often included. Such appointments also μ - - ongly -

modi /f_i cation and basic pharmacological treatment Review lifestyle modi /f_i cation (/f_i bre, /f_l uid, exercise) Rational laxative use (PEG, stimulant laxatives) Prokinetics if naive (prucalopride 1–2 mg daily or linaclotide 290 Response Anorectal function Abnormal Dyssynergic Other evacuation defecation disorder defecography, rectal No sensory testing and response Direct visual anorectal manometry) biofeedback

No response Transanal irrigation Response 4 initiated high volume Figure 73.11 Algorithm of chronic constipation management. MDT, multidisciplinary team; PEG, polyethylene glycol. 1, alarm features excluded and secondary causes treated appropriately; 2, in constipation-predominant irritable bowel syndrome, consider antispasmodics or neuromod ulators in case constipation improves but abdominal pain persists and is dominant symptom; 3, examples of overt prolapse include anterior (stage 3

cystocele), middle (stage 3 rectocele, uterovaginal prolapse) and posterior compartments (grade IV/V intussusception); 4, unless patient preference for low volume or speci /f_i c contraindications to high volume; 5, may reduce speci /f_i c symptoms but not have overall effect on quality of life; 6, common adjuncts include sacrocolpopexy, hysterectomy, transvaginal tape and cystocele repair. 1 2 Response g or other secretagogues) No response Obvious clinical evidence of Habit training 3 overt pelvic

organ prolapse No response Colonic/whole gut transit Normal +/– defecography testing (balloon +/– adjunctive tests, e.g. urodynamics expulsion test, Abnormal Abnormal Re-evaluation of MDT meeting to symptom–investigation discuss surgical correlation to focus on further options pharmacology or other untried interventions Other surgical targets Posterior compartment prolapse and procedures syndrome with high +/– grade intussusception retrocele Consider laparoscopic

ventral 5 rectopexy or alternative, 6 +/– e.g. STARR adjuncts

Figure 73.12 Correct posture for defecation.

therapy . If this fails, there may be recourse to the specialist tests to assess colonic transit and also anorectal function (see Chapter 80). Armed with the results of these tests, the patient may have a more targeted approach relative to their observ ed pathophys iology . One example of this approach is for patients with a con dition termed ‘dyssynergic defecation’, where there is a failure to relax, or even paradoxical contraction of the pelvic floor muscles (especially puborectalis) during defecatory e ff orts. In such patients, instrument-based biofeedback learning tech niques pro vide direct visual computer-based biofeedback of pelvic floor activity . The aim is to retrain the patient to appro priately contract abdominal and relax pelvic floor m during defecation with the patient receiving feedback of anal and pelvic floor muscle activity as recorded by surface electro myographic anal pressur e sensors or digital examination by the therapist. Transanal irrigation (TAI) may be used for any patient with an evacuation disorder when habit training and/or biofeedback have failed. A number of devices are available that administer a low (appro ximately 50–100 /uni00A0 mL) or high volume (approximately 500 /uni00A0 mL) of irrigant fluid into the rectum. The patient sits on the toilet to evacuate the fluid and faecal material. Some patients with chronic refractory symptoms may seek a surgical solution to their problem. Surgical procedures can be broadly divided into those addressing dynamic structural problems of the pelvic floor (prolapse pr ocedures), those that seek specifically to address slow-transit constipation and those that may have a role for both (Summary box 73.8). Summary box 73.8 Surgical options in patients with chronic constipation /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF All surgery should be undertaken in the knowledge that none of the above-listed operations is perfect. All represent a trade-o ff between benefits and short-term harms and poor Edmond Delorme , 1847–1929, French military surgeon and Professor of Surgery , V al-de-Grace Military Hospital, Paris, France. Peter Graham Chait , contemporary , radiologist, Toronto, Canada. essential requirements before surgery is undertaken: /uni25CF pathophysiological findings from specialist tests concur with the symptomatology and findings on clinical exam - ination; - /uni25CF conservative (non-surgical) treatment options have been - tried; /uni25CF the patient’s case has been reviewed at a multidisciplinary team (MDT) meeting and surgery recommended; /uni25CF the patient has been consented in the very clear knowledge - of the range of possible outcomes; /uni25CF surgery is undertaken in a centre with expertise in manag - - ing functional conditions. uscles The range of procedures for rectal prolapse are covered in - detail in Chapter 79 . Those primarily targeting the intestine are covered briefly here. Colectomy Colectomy is a radical and clearly irreversible final solution for patients with refractory slow-transit constipation. Its use should be very highly selective, not least because it is not actually a solution for many patients even when the surgery itself passes without complication. Removal of the whole colon with ileorectal anastomosis (as performed for inflam - matory bowel disease) is best studied; subtotal resections with ileosigmoid or caecorectal anastomosis are alternatives. Outcomes vary greatly and are often compromised by ear ly problems of ileus and a higher than expected rate of adhesional small

bowel obstruction. Later problems include ongoing constipation and obstructive symptoms, diarrhoea and urgency , abdominal pain and bloating. Embarking on this procedure requires very careful MDT review , documen - tation of generalised slow-transit constipation and exclusion of a long list of relative contraindications. Stoma A stoma may be used as a definitive procedure, as a guide to further treatment or as salvage from a failed or complicated prior surgical intervention. There are few published data to support evidence-based use; however, an ileostomy may be employed as a guide to colectomy with subsequent resection avoided if ileostomy output is unsatisfactorily high or symp - toms such as pain and bloating are untouched by diversion. As a definitive procedure, there is little evidence in adults to guide the choice of ileostomy or colostomy; however, it is generally considered that slow-transit constipa tion is unsatisfactorily treated by colostomy . Anterograde colonic enema procedures The formation of a conduit to introduce irrigant into the colon is best established in children and in patients with neurological disease. A variety of methods have been proposed to access the caecum either directly , e.g. with a Chait tube caecostomy , or indirectly via the appendix (appendicostomy). The latter is almost certainly preferable although only possible when the

Prolapse procedures for dynamic structural causes of ob structed defecation Hitching procedures, e.g. rectopexy Rectal wall excisional procedures, e.g. stapled transanal rectal resection (STARR) Rectovaginal reinforcement procedures, e.g. posterior vaginal repair, intra-anal Delorme’s procedure Procedures for slow-

transit constipation Colectomy and ileorectal anastomosis Other variants of subtotal colectomy Procedures for refractory chronic constipation in general Stoma: ileostomy or colostomy ACE procedures Neuromodulation

The appendix can be reversed (Malone anterograde continent enema technique) or used in its native orientation (much simpler). Outcomes in adults with chronic constipation are variable but generally this is a good option in patients consid ering colectomy or stoma as the only alternative. Neuromodulation The attraction of being able to treat chronic constipation with a minimally invasive and safe approach such as sacral neuromodulation is supported by research data showing that stimulation improves motility and also some observational data. It is now clear from randomised trials that it has no role for slow-transit constipation but it may yet have a place in modifying anorectal function in some patients with severe functional syndromes leading to obstructed defecation (as it does for the bladder).

Diagnosis and management

Diagnosis and management

Megarectum may present with a mass the size of a full-term baby (Figure 73.9) but diagnosis is mainly radiological. The mainstay of management of both (in brief) requires getting the rectum empty . In some patients with megarectum this may require manual disimpaction under anaesthesia. Thereafter, high doses of regular osmotic and simulant laxatives orally as well as regular enemas (or high-volume transanal irrigation (TAI); see Constipation) are required to keep it empty . Prokinetics may also have a role. Compliance with medication is often an issue in young patients with psychobehavioural problems. Surgery has an important role in patients who fail medical management. Colectomy or subtotal colectomy is generally required for megacolon. A variety of options exist - - - - - vioural - for megarectum. A first step may be an anterograde colonic enema (ACE) procedure (see Constipation). If this fails, defin - itive surgery includes pull-through procedures, low anterior resection, r estorative proctocolectomy and rectum-reducing procedures, e.g. vertical reduction rectoplasty . All should be undertaken with covering loop ileostomy and many advocate performing an ileostomy for 6 months to 1 year prior to surgery . This allows the rectum to shrink and reduce in vascularity , making eventual surgery safer; some patients may also simply

oderma s disease (congenital megarectum) s disease (post reconstruction) Figure 73.9 Plain abdominal radiograph of a teenage male with megarectum.

The hazard of operating on a rectum that occupies the whole pelvis with serosal veins that sometimes resemble the iliac veins cannot be underestimated and surgery should be performed in specialist centres.

Diagnosis

Diagnosis

- CT scanning is frequently required to exclude both mechanical obstruction and any local driver of ileus in the peritoneum such as inflammation or infection (Figure 73.4). In instances of POI this is required to exclude local complications of surgery . Blood tests should be used to detect any drivers of ileus such as metabolic abnormalities (especially hypokalaemia).

FURTHER READING

FURTHER READING

Bharucha A, Knowles CH. Chronic constipation. In: Sagar PM, Hill AG, Knowles CH eas et al. (eds). Keighley & Williams’ surgery of the anus, rectum and colon , 4th edn. Boca Raton, FL: Taylor & Francis, 2019: 305–46. Enck P , Aziz Q, Barbara G et al . Irritable bowel syndrome. Nat Rev Dis Primers 2016; 2 : 16014. van Bree SHW , Nemethova A, Cailotto C et al . New therapeutic strat - egies for postoperative ileus. Nat Rev Gastroenterol Hepatol 2012; 9 : 675–83.

Introduction

Introduction

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Irritable bowel syndrome

Irritable bowel syndrome

Surgery has no role in treating IBS. Nevertheless, patients with chronic abdominal pain and a change in their bowels are very common in surgical clinics and all surgeons should at least have a passing familiarity with a disorder that is a source of misery to millions of people worldwide. Definitions IBS is a functional bowel disorder characterised by abdominal pain or discomfort, stool irregularities and bloating. The term replaced nineteenth century descriptions such as ‘irritable’ or ‘spastic’ colon in 1979 to reflect the fact that the colon is not the only site of the problem. Diagnostic criteria have evolved to the now used Rome IV Foundation definition: Recurrent abdominal pain on average at least 1 day/week in the last 3 months, associated with two or more of three criteria: (1). related to defecation; (2). associated with a change in the frequency of stool and (3). associated with a change in the form (appearance) of stool. These criteria should be fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis. The change in frequency and form of the stool dictates subdivisions of IBS into constipation-predominant (IBS-C), diarrhoea-predominant (IBS-D) and mixed (IBS-M) subclasses (Figur e 73.13). Aetiology and risk factors Regardless of exact definition, the cardinal symptoms of chronic abdominal pain and ‘deranged digestion’ favour a ‘biopsychosocial model’ (Figure 73.14) that encompasses the role of stressful life events and brain–gut interactions in symp tom generation. Several common life events are particularly well documented; these include postinfective IBS, where a seemingly discrete attack of gastroenteritis (viral, bacterial or otherwise) is follow ed by chronic ongoing symptoms; physical and/or sexual abuse (and neglect); surgery; and trauma. One Patrick Malone , contemporary , surgeon, Southampton, UK. - implication of the model is that IBS has much overlap with other medical conditions that have similar or nearly identical biopsychosocial determinants (Summary box 73.9). Summary box 73.9 IBS-associated comorbidities /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF

Abdominal pain Constipation IBS-C IBS-M Diarrhoea IBS-D IBS-U Figure 73.13 Irritable bowel syndrome (IBS) subtypes according to the Rome criteria. All patients have abdominal pain but subtypes vary according to bowel form at presentation such as to meet criteria for IBS with constipation (IBS-C), IBS with diarrhoea (IBS-D), mixed-type IBS (IBS-M) and unsubtyped (IBS-U). General Fibromyalgia syndrome Chronic fatigue syndrome Chronic pelvic pain, chronic prostatitis and bladder pain syndromes Chronic back pain Migraines Depression Anxiety Somatisation Sleep disturbance a Gastrointestinal disorders Eating disorders Dyssynergic defecation Levator ani syndrome and proctalgia fugax Food intolerances a The overlap of IBS with other Rome-de /f_i ned functional gastro- intestinal disorders should also be noted, including functional dyspepsia and functional constipation.

Diagnosis Clinical history Besides symptoms required by the diagnostic criteria, other symptoms may be present. Common associated symptoms include bloating (very common), straining at defecation, excessive flatulence and postprandial indigestion. A history of precipitating events (as

per the model) and of comorbidities (Summary box 73.7) should also be sought to support the diagnosis. The patient may also have a history of multiple operations, which on reflection may have been directed to chronic abdominal pain, e.g. appendicectomy , cholecystectomy or hysterectomy . Clinical examination Physical examination helps to reassure patients and also to exclude another organic cause for symptoms. However, abdominal examination rarely discloses a specific diagnosis (abdominal tenderness is often present but non-specific); the absence of objective findings supports a diagnosis of IBS. A digital rectal examination may identify patients with dyssyner - gic defecation and other causes of constipation. Investigations There are no valid laboratory biomarkers of IBS. Routine blood panels, including inflammatory markers, are generally

Early life • Genetics • Epigenetics Local environmental factors • %iet • Acute infections • Surgery Figure 73.14 Biopsychosocial model of irritable bowel syndrome (IBS). The scheme is a conceptualisation of the pathogenesis and clinical expression of IBS showing interrelationships between various risk factors and changes in physiology. TABLE 73.7 Treatments for irritable bowel syndrome. Nutrition Increased (constipation) or reduced (bloating) /f_i bre Gluten-free diet (especially if equivocal diagnosis of coeliac disease) FODMAP diet Probiotics Consider dietary supplements, prebiotics Drugs Antispasmodics: peppermint oil, hyoscine butylbromide (Buscopan) Laxatives, e.g. stool softeners, osmotic and stimulant (avoid lactulose because of bloating and pain) Antidiarrhoeals: loperamide (/uni03BC -opioid receptor agonist); 5-HT Motility accelerants, e.g. linaclotide (guanylyl cyclase C agonist), prucalopride (5-HT Low-dose antidepressants: tricyclics and selective serotonin reuptake inhibitors Manipulation of the microbiota by non-absorbable antibiotics, e.g. rifaximin Neuromodulators, e.g. gabapentin and pregabalin Psychotherapy Cognitive–behavioural therapy Gut-directed hypnosis Guided self-help interventions 5-HT, 5-hydroxytryptamine; FODMAP , fermentable oligosaccharides, disaccharides, monosaccharides and polyols. Psychosocial factors • Life stress • Psychological state and trait • Coping • Social support IBS Brain-gut axis • Symptoms • #ehaviour Physiology • Motility • Sensation • Permeability • Inflammation • Altered flora receptor antagonists, such as alosetron, ondansetron 3 receptor agonist) 4

disease, e.g. cancer, inflammatory bowel disease or diverticu lar disease. Specific tests include serological tests for coeliac disease, faecal calpr otectin and stool microbiology in cases of diarrhoea predominance. Invasive procedures are generally not warranted unless alarm features ar e present that mandate endoscopy . That noted, it is quite common practice to perform colonoscopy , not least to reassure the patient that their chronic symptoms do not have an organic basis. In patients with IBS-D, colonoscopy with random biopsies is warranted to exclude microscopic colitis. Other tests that may be relevant include 75 (75Se-homocholic acid taurine [SeHCAT] test or serum serum 7- α -hydroxy-4-cholesten-3-one (C4) levels) for bile salt malabsorption, breath testing for carbohydrate malabsorption, gastrointestinal physiology for constipation and upper gastro intestinal endoscopy for associated dyspeptic symptoms. Management Only a fraction of patients with IBS-like symptoms seek medi cal care and most will initially consult primary care physicians for their symptoms. The factors that drive this consultation are symptom sev erity , especially pain, and concerns that symptoms might indicate an underlying severe disease, e.g. cancer. There fore, in many cases, the doctor’s role is to exclude diseases that can mimic IBS symptoms by relevant investigations such as endoscopy . When a positive diagnosis of IBS has been made, man agement requir es an integrated approach, including educa tion, reassurance, dietary alterations, pharmacotherapy and behavioural or psychological

interventions/support. The initial treatment strategy should be based on predominant symptoms and includes antispasmodics for abdominal pain, antidiarrhoeals for IBS-D and laxatives for IBS-C, wher nutritional interventions and psychotherapy can be used in all subtypes. Table 73.7 provides a list of potential management strategies for IBS. This list is not all encompassing, nor does it provide weighting to one treatment over another in terms of e ff ectiveness in clinical trials. Some treatments are popular, e.g. low-dose antidepressants but such use is o ff -label; others may - there is no trial evidence. A key point in the management of IBS rests with nota - ble exclusions from Table 73.7 . Thus the table makes no ref - erence to standard analgesics and surgery . Opioid analgesia should be avoided in IBS because the further disturbance to motility worsens the prognosis and in extreme use can lead to narcotic bowel syndrome (an opioid-induced state of hyper - algesia whose main driver is the activa tion of glial cells). Sur - gery has a well-documented association with symptom onset of IBS (cholecystectomy , appendicectomy , hysterectomy and back surgery); further surgery leads not only to greater poten - tial visceral sensitisa tion (via injury) but also serves to confuse subsequent diagnosis, e.g. adhesional versus functional cause - for symptoms. There is also a body of evidence to suggest that surgery perpetua tes a search for an ‘organic’ diagnosis that hinders patient acceptance and adaption to their chronic prob - lem. For the surgeon, the key is to exclude any surgical cause - of pain and then prevent further harm by avoiding surgery .

Learning objectives

Learning objectives

To recognise and understand: The spectrum of intestinal disorders resulting from • abnormal neuromuscular functions The management of relatively common acute motility • disturbances

Management

Management

Ileus may be managed by nasogastric drainage and restriction of oral intake until there is evidence of improvement. Support - ive care such as attention to fluid and electrolyte balance and nutrition is also important, especially if ileus persists. Underlying drivers of ileus, e.g. abscess or peritonitis, should be managed on their merits. Reg rettably , despite improved knowledge of the pathophysiology , specific drugs aimed at blocking inflammation or stimulating local neuromuscular function, e.g. prokinetics, have not proved su ffi ciently e ff ective yet to be adopted for routine use. In patients with POI, if prolonged, CT scanning is the most e ff ective investigation; it will demonstrate any intra-abdominal sepsis or mechanical obstruction and therefore guide an y requirement for laparotomy . Otherwise the decision to take a patient back to theatre in these circumstances is always di ffi cult. The need for a laparotomy becomes increasingly likely the longer the bowel inactivity persists, particularly if it lasts for more than 7 days or if bowel activity recommences following surgery and then stops again.

Inhibitory spinal (adrenergic) re /f_l exes Acute stress response HPA axis activation releases catecholamines Mast cell Bowel activation handling Prolonged in /f_l ammatory response Figure 73.3 Pathophysiology of postoperative ileus. HPA, hypothalamic–pituitary–adrenal axis. (a) Figure 73.4 Computed tomography abdomen scout /f_i lm (a) and representative coronal image dilatation of the small intestine (ileus) secondary to a driving in /f_l ammatory focus (pelvic collection, arrow) (courtesy of Dr Arman Parsai, Barts Health NHS Trust, London, UK). Prolonged Immediate local and decrease or distant abolition of decrease or motility abolition of motility Macrophage and neutrophil migration and activation Inhibitory Increased Afferent spinal mucosal sensitisation (adrenergic) permeability re /f_l exes Bacterial translocation (b) (b) of a 22-year-old woman showing widespread

Management

The management of ACPO depends on whether complica - tions are evident or considered imminent. In patients with clinical and radiological features of caecal ischaemia or perforation, emergency surgery will be required and usually necessitates a subtotal colectomy and end ileostomy (with high levels of morbidity and mortality). The majority of patients can however follow a more stepwise approach, starting with conservative measures (Table 73.3). Clearly the underlying cause where relevant, e.g. UTI, respiratory tract infection or myocardial infarction, should also be managed in parallel. It is reasonable to wait before progressing from one stage to the next but caecal diameters of 12 /uni00A0 cm or above warrant rapid decompression to reduce perforation risk. The decision of whether to use intravenous neostigmine is di ffi cult and is usually reserved for patients in whom supportive measures and colonic decompression have failed. Treatment is tion, brady - associated with profound autonomic e ff ects (saliva cardia, bronchospasm and hypotension) as well as abdominal cramps, followed often by a massive evacuation of flatus and faeces. Cardiac monitoring and a health professional compe - tent in the

emergency administration of resuscitative drugs (especially atropine) are essential. Contraindications to the use of neostigmine include renal insu ffi ciency , recent myocardial infarct, arrhythmias and asthma. Surgery is associated with high morbidity and mortality should be reserved for those with impending perforation and e failed or perforation has occurred. when other treatments hav

(c) TABLE 73.3 Management of acute colonic pseudo- obstruction. Reversal of risk Correct /f_l uid and electrolyte imbalances factors Stop or reduce offending drugs, e.g. opioids, anticholinergics, calcium channel blockers (where possible) Empty the rectum by enemas and/or /f_l atus tube Endoscopic Colonoscopy +/– /f_l atus tube decompression Pharmacological Intravenous neostigmine unless decompression contraindicated (risk of arrhythmia and a

bronchospasm) Surgery Subtotal colectomy (usually with ileostomy) Venting stoma, e.g. caecostomy, in very un /f_i t patients a Requires high-dependency unit-level monitoring and support on hand for cardiorespiratory complications. (d) Figure 73.5 Scout /f_i lm (a) and representative coronal computed tomography image (b) of a patient with acute colonic pseudo-obstruction. The entire colon and rectum is variably distended with /f_l uid and gas. (c) Plain abdominal radio

graph (courtesy of James Hill) and (d) intraoperative photograph of the colon during sur gery for acute colonic pseudo-obstruction (courtesy of James Hill).

ACPO is a life-threatening condition in which prompt diag nosis and appropriate management can limit the occurrence of complications (e.g. ischaemia or perforation). Such complications occur in about 5–10% of patients and require emergency surgery with mortality ra tes between 30% and 60%. Recurrence is an issue in some patients with unmodi fiable risk factors, e.g. senility and neurological disease. Such patients should have chronic modification of polypharmacy to avoid o ff ending drugs and keep the rectum empty by regular enemas. Prokinetic medications, suc h as those used for chronic constipation, may have a role in such patients, although none are licensed for this indication. Management

The main lines of management are shown in Summary box 73.6 , noting that for most patients there is no cure. Surgery , with the exception of placing feeding tubes or formation of a venting stoma, is impotent for a condition that is a di ff use neuromuscular disease. Further, surgery worsens the prognosis by adding the risk of adhesions into the diagnosis and, if resections or complications occur, speeding the patient towards intestinal failure. Small bowel (or multivisceral) transplantation is an option in selected patients.

Figure 73.6 Intestinal pseudo-obstruction in a young male patient. A full-thickness biopsy was undertaken from the proximal jejunum at minilaparotomy.

Summary box 73.6 Management of intestinal pseudo-obstruction /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

(b) Figure 73.7 Two examples of myopathy: (a) hollow visceral myopathy (note the vacuolation of the smooth muscle, arrows); (b) extra muscle layer in the muscularis propria (arrows). Nutrition (enteral/parenteral) Analgesia (but try to avoid opioids) Prokinetics (generally disappointing) Antibiotics (overgrowth) Immunotherapy – speci /f_i c in /f_l ammatory cases (limited data) Psychological support, including speci /f_i c patient support groups Palliative care Surgery (very selected cases)

NEUROMUSCULAR STATES OF THE LARGE INTESTINE WITH D

NEUROMUSCULAR STATES OF THE LARGE INTESTINE WITH DILATATION: ACUTE COLONIC PSEUDO-OBSTRUCTION Definition

The term acute colonic pseudo-obstruction (ACPO) is defined /uni00A0 as: Acute massive dilatation of the colon with obstructive symptoms but in the absence of mechanical obstruction. ACPO was first described by Sir William Ogilvie, who in 1948 recognised this syndrome in two patients with sudden onset of abdominal pain, constipation and large bowel dilatation (hence the eponym Ogilvie’s syndr ome). It is one of the three common diagnoses in patients evaluated for a clinical presentation of large bowel obstruction (see Chapter 78 other two being colorectal cancer and volvulus (remember the three Ts: tumour, torsion and ‘tired out’). Toxic megacolon (see Chapter 75), although conveniently being a fourth ‘T’, should be considered as a di ff erent condition entirely although the end point is also one of acute dilatation.

Pathophysiology

Pathophysiology

Classic teaching points to a reflex inhibition of intestinal motility caused by deranged ANS inputs. This teaching, which fits nicely with basic ‘fight and flight’ concepts of increased sympathetic signalling and parasympathetic withdrawal during trauma (including surgery), has been superseded by the concept of a two-phase response. First, an immediate stress response, mediated by spinal reflexes and activation of the hypothalamic–pituitary–adrenal axis (HPA) axis, leads to a decrease or abolition of motility . This is then followed very rapidly by evolution of a more prolonged inflammatory response in the bowel wall itself, mediated first by mast cell activation and thence recruitment and activation of macro - phages and neutrophils (Figure 73.3). These lead to inhibition of enteric neuronal and smooth muscle function as well as further e ff ects on spinal reflexes.

Nomenclature Ileus (including postoperative ileus) Acute colonic pseudo-obstruction Intestinal pseudo-obstruction Megacolon Constipation and irritable bowel syndrome

Pathophysiology

This is poorly understood. It can however be appreciated that, like ileus, risk factors reflect both ‘imbalanced’ extrinsic autonomic innervation and an ‘inflammatory’ state. Evidence to support the former is provided by the response to anticho - linesterase pharmacological therapy .

Prevention

Prevention

Minimally invasive surgical approaches have reduced risks of POI for many operations. The enhanced recovery programme (see Chapter 74) seeks to further reduce risk of POI by avoidance of opioid-containing drugs and suppression of the inflammatory response.

Prognosis

Prognosis

Prognosis is poor – sometimes considered the ‘motor neurone disease’ of the gut. Infantile forms have a mortality of approx imately 50%. This is generally lower in adult forms depending on cause and avoiding repeated surgery and overuse of opioid Harald Hirschsprung , 1830–1916, physician, The Queen Louise Hospital for Children, Copenhagen, Denmark, described congenital megacolon in 1887. ity and progression to type II/III intestinal failure with the need f or lifelong parenteral nutrition.

Risk factors

Risk factors

In Ogilvie’s original report, the clinical picture was associated with a retroperitoneal neoplasm infiltrating and destroying prevertebral ganglia. This is actually a very rare cause. The main risks are shown in Summary box 73.4 . Summary box 73.4 Risk factors for acute colonic pseudo-obstruction /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Sir William Heneage Ogilvie , 1887–1973, surgeon, Royal Army Medical Corps (First World War), Oxford, and Guy’s Hospital, London, UK. James Parkinson , 1755–1824, general practitioner of Shoreditch, London, UK, published ‘ An essay on the shaking palsy’ in 1817. have a high background risk and a small acute event (e.g. the elderly patient with Parkinson’s disease and a urinary tract infection [UTI]) – the colon has little ‘reserve’ and a small insult tips the balance into one of progressive abolition of motility and tone with consequent gaseous dilatation; and those with little background risk and a large acute event, e.g. major surgery/trauma.

Frailty and senility Neurological Neurodegenerative diseases Stroke Spinal cord injury Retroperitoneum tumour in /f_i ltration Trauma/surgical Major orthopaedic injuries or surgery, e.g. vertebral, pelvic and femoral Major gynaecological surgery Obstetrics, including caesarean section Systemic in /f_l ammation by any cause, e.g. sepsis, trauma, especially with multiorgan failure Localised infective conditions, e.g. respiratory, urinary Myocardial infarction Metabolic and electrolyte disturbances Medications, e.g. opioids and any with anticholinergic actions (e.g. psychiatric and Parkinson’s), calcium channel antagonists

SCOPE OF DISEASE

SCOPE OF DISEASE

- A functional diagnosis is usually made when routine investigations - fail to find an easy explanation (e.g. a structural or biochemical - cause) for a combination of typical symptoms. For instance, in a patient with lower abdominal pain, constipation and bloating if routine investigation finds a morphological abnormality , e.g. sigmoid diverticulosis, then the patient will be given a diagnosis of diverticular disease. However, if all usual tests, including colonoscopy , yield no findings then the same patient might be described as having IBS – a functional intestinal disorder. Like much of medicine, there are however grey areas. Further, understanding is not aided by historic nomenclature where - terms such as pseudo-obstruction describe di ff erent entities in the small and large intestine (Table 73.2). This chapter consid - ers the main disorders using this classification with a focus on those most pertinent to the surgical reader.

History of onset Visceral diameter Region predominantly affected Acute Dilated Small intestine Large intestine Chronic Dilated Small intestine Large intestine Chronic Normal Intestine

SUMMARY

SUMMARY

- Functional intestinal disorders range from the very common – constipation and IBS – through to the very rare, e.g. various genetic and familial neuropathies and myopathies causing IPO. The surgeon will almost certainly encounter acute problems - such as POI and ACPO. This chapter provides an o verview that - can be supplemented by the recommended further reading.

TESTS OF INTESTINAL FUNCTION

TESTS OF INTESTINAL FUNCTION

Subsequent chapters address diagnostic tests specific to the rectum (see Chapter 79) and anus (see Chapter 80). Here the focus is on tests that may be relevant to studying the motility of the small intestine and colon. A general proviso in reading this section is that our current ability to understand the physiology of the intestine in humans is limited by both access and under - standing. In general, we measure what can be measured and all tests have inherent limitations to interpretation. Summary box 73.2 provides an overview of all tests, denoting those that hav e general clinical application versus those that are the preserve of highly specialised units or research studies. - Summary box 73.2 makes clear that few tests are in general use. Small bowel contrast studies, e.g. barium follow- - through, although available, hav e poor sensitivity for detecting much other than visceral distension (superseded by axial imaging with computed tomography [CT] or MRI) or grossly retarded transit. Breath hydrogen testing assesses the presence Tests of small intestinal function /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Tests of colonic function /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF of carbohydrate malabsorption and is an indirect measure of transit because stagnated content allows some degree of bacterial overgrowth and fermentation products (hydrogen, methane and carbon dioxide). Although frequently used in patients with unexplained chronic abdominal symptoms such as irritable bowel syndrome (IBS), its utility in reliably measuring transit or detecting bacterial overgrowth is limited by issues of reproducibility . The wireless motility capsule measures pH, temperature and pressure as it traverses the whole gastrointestinal tract; changes in these variables can be used to determine timings as it migrates from stomach to small bowel and large bowel. While it o ff ers a number of advantages over and above current techniques, especially with respect to patient tolerability , safety and standardisation, it is not widely available owing to cost. Prolonged measurement of small bowel contractile activ ity can be performed using multichannel pressure recordings called manometry that show phases of the MMC. Some find ings may be indicative of underlying small bowel neuromuscu lar diseases such as myopa thies and neuropathies (see Chronic impairment of intestinal motility with dilatation of the small intestine: intestinal pseudo-obstruction) but these findings have issues of specificity and the technology itself is only available in a small number of centres worldwide. Dynamic MRI (long sequences of image acquisition with computer analysis) is currently a research tool but may well represent the future. The radio-opaque marker study is the mainstay of evalua tion of colonic transit. Though variations in technique exist in terms of the number of markers, interval to radiograph and definition of slow transit, the basic premise is that a number of markers (small pieces of plastic tubing, prepackaged in gelatin capsules) are ingested and an abdominal radiograph (which includes the pelvis) taken at an interval. The patient abstains from laxatives for the duration of the study . In patients with sig - nificant numbers of retained markers (based on control data), slow-transit constipation is

diagnosed (Figure 73.2). Other studies of colonic transit, e.g. isotope scintigraphy and direct measurements of colonic contractile activity , are restricted to a very small n umber of specialist centres worldwide.

Transit a Small bowel barium contrast study Breath hydrogen small bowel transit tests (lactulose or lactose 13 a C-ureide) b Wireless motility capsule small bowel transit study Contractile activity Antroduodenal manometry (ideally prolonged [24 hours] ambulatory study) Dynamic magnetic resonance imaging (MRI) studies Transit a Radio-opaque marker studies Isotope scintigraphy b Wireless motility capsule whole-gut transit study Contractile activity Colonic manometry Dynamic MRI studies a Denotes general availability. b Adopted by some highly funded health systems. Figure 73.2 Radio-opaque marker transit study in a woman. All 50 markers are retained, indicating slow-transit constipation.