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Diseases of the colon and rectum

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This tutorial is devoted to Diseases of the colon and rectum. Differential diagnosis of jaundice. The authors present current data on the etiology, pathogenesis, clinic, diagnosis, preoperative and intraoperative tactics of management and treatment of patients with R. the views on controversial and unresolved issues relating to the current state of this problem are Reflected.

The manual is developed in accordance with the requirements of the Federal state educational institution, is intended for senior students of medical Universities and faculties enrolled in the specialty 31.05.01 Medical business.

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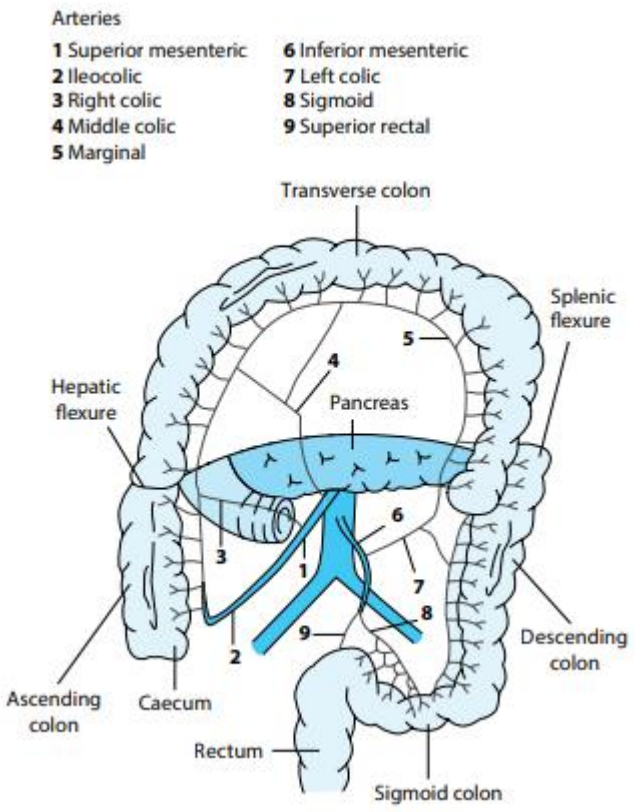
Contests:

Anatomy

The large intestine consists of the caecum, vermiform appendix, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid colon, rectum and anal canal. The anatomy of the appendix and anal canal is dealt with separately in the appropriate chapters. Originally a mid-line structure, the large intestine undergoes rotation during embryological development and as a result the ascending colon and the descending colon are essentially retroperitoneal structures. However, the degree to which the large intestine has a mesentery is highly variable as is its total length that averages 1.5 m. The whole of the large intestine is capable of considerable distension, although in an adult living in the Western world the left side of the colon tends to be less distensible than the right owing to muscular hypertrophy. The caecum lies in the right iliac fossa and is approximately 7 cm in length and width. Proximally it becomes the ascending colon at its junction with the terminal ileum. The caecum lies on the iliac and psoas muscles and on the genitofemoral, femoral and lateral cutaneous nerves. It also lies anterior to the testicular or ovarian vessels and the ureter. The exact position of the caecum is variable and it may extend into the true pelvis. The caecum is almost completely surrounded by peritoneum but it is often attached to the iliac fossa medially and laterally. This can produce a retrocaecal

peritoneal recess which may extend upwards posterior to the ascending colon. The ileocaecal junction is extremely variable in appearance. In most circumstances the ileum enters obliquely into the large bowel through a horizontal slit and is partly invaginated into the caecum to form a fold (the ileocaecal valve). Reflux of caecal contents into the small bowel is prevented by contraction of the circular muscle of the ileum which leads to closure of the ileocaecal valve. However, the muscle in the valve is poorly developed and the ileocaecal valve is frequently incompetent. The ascending colon varies from about 10 to 20 cm in length. It lies on the iliac muscle, the iliac crest and quadratus lumborum, crossing the lateral cutaneous nerve of the thigh, the ilioinguinal and iliohypogastric nerves. It ends at the hepatic flexure where the large bowel turns to the left on the lower portion of the right kidney inferior to the liver. Under most circumstances peritoneum covers the front and the sides of the ascending colon and fixes it firmly to the posterior abdominal wall, but occasionally there is a short mesentery. The transverse colon is the longest section of the colon varying from 40 to 70cm in length. It extends from the hepatic flexure to the splenic flexure and forms a dependent loop between both of these points. The lowest point of the transverse colon may reach below the umbilicus, although it is usually just superior to it. The transverse colon is suspended by the transverse mesocolon which is fused to the posterior surface of the greater omentum. This transverse mesocolon is attached to the descending part of the duodenum, to the head and the lower aspect of the body of the pancreas and to the anterior surface of the left kidney. It contains both the middle colic vessels and branches of the right and left colic vessels with associated nerves and lymphatics. Thus the transverse colon starts immediately anterior to the descending part of the duodenum and the head of the pancreas, descends anterior to the small intestine and ascends to the splenic flexure. At this point it is anterior to the left part of the left kidney and

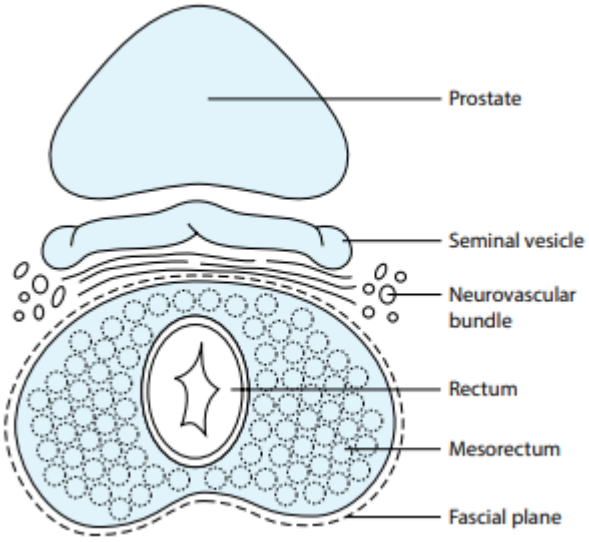


immediately below the spleen. The splenic flexure is attached to the diaphragm by peritoneum (phrenicocolic ligament) and can be extremely close to the spleen. At this point the greater omentum frequently has attachments to the spleen and is closely associated with the colon. Traction on these splenic attachments may cause splenic bleeding in the course of mobilization of the splenic flexure. The descending colon extends from the splenic flexure to the rim of the true pelvis close to the inguinal ligament. The descending colon is attached by peritoneum to the posterior abdominal wall in the left paravertebral gutter and iliac fossa. Superiorly it is anterior to the lateral surface of the left kidney and medial to the diaphragm, and then it lies on the same muscles and nerves as the ascending colon. At the anterior superior iliac spine, the descending colon turns medially, superior to the inguinal ligament, and lies on the femoral nerve, psoas muscle and the genital vessels, and becomes the sigmoid colon immediately anterior to the external iliac vessels. The sigmoid colon is the most variable part of the colon in terms of its length (50–80cm) and its mobility. It extends from the end of the descending colon to the rim of the true pelvis where it becomes the rectum. It has a long mesentery (the sigmoid mesocolon) which has quite a short base starting at the end of the descending colon and ascending on the external iliac vessels to the mid - point of the common iliac artery. At this point it turns downwards and to the right to the rim of the true pelvis. The mesocolon contains the sigmoid branches of the inferior mesenteric artery and associated nerves and lymphatics. Under normal circumstances the sigmoid colon lies entirely in the left iliac fossa and the true pelvis but it may also extend across to the right iliac fossa. The rectum is that part of the large bowel which lies in the true pelvis at the point where the sigmoid mesocolon ends. Again, this is highly variable in length depending very much on the build of the individual but it is said to be 15 cm long as measured by a rigid sigmoidoscope. It follows the curve of the sacrum and the coccyx and then runs anteriorly and inferiorly to the central perineal tendon lying on the anococcygeal ligament and the levator ani muscles. It then ends by turning posteriorly and inferiorly as the anal canal, immediately posterior to the central perineal tendon and to the apex of the prostate in the male. The lowest part of the rectum is more capacious than the rest and is known as the ampulla. The rectum is not straight; in the sagittal plane it follows the curve of the sacrum and coccyx and in the coronal plane it is S shaped. This gives rise to prominent folds within the lumen of the rectum known as the valves of Houston. The front and the sides of the upper third of the rectum are covered with peritoneum but this

gradually moves anteriorly and turns off the front of the rectum at the junction between its middle and lower thirds. This forms the rectouterine or rectovesical pouch by passing upwards on the back of the posterior fornix of the vagina or the back of the bladder respectively in the female and the male. In its lower third, the rectum lies behind the base of the bladder, the seminal vesicles and the prostate in the male and behind the vagina in the female. In both sexes the rectum and its surrounding areolar tissue is separated from the anterior structures by a fascial layer known as Denonvilliers' fascia. Posteriorly the rectum is separated from the sacrum and the coccyx and anococcygeal ligament and the muscles attached to these (piriformis and levator ani) by a layer of pelvic fascia. This fascia is known as Waldeyer's fascia. In its upper two-thirds the actual muscular wall of the rectum is separated from the pelvic fascia by a posterior cushion of areolar tissue which becomes circumferential below the rectouterine or rectovesical pouch. This carries the blood supply to the rectum and its lymphatic drainage and is known as the mesorectum. Inferiorly and posteriorly the mesorectum has a bilobed structure.

Taeniae coli

The taeniae coli are three ribbon-like thickenings of the otherwise thin longitudinal muscle of the large bowel which arise from the longitudinal muscle at the root of the vermiform appendix and end by spreading out at the end of the sigmoid colon to become continuous with the thicker longitudinal muscle of the rectum. These three taeniae are spaced out uniformly around the circumference of the colon and between them the wall of the colon bulges outwards forming pouches or sacculations. In the ascending colon and descending colon, the taeniae are anterior, posteromedial and posterolateral, whereas in the transverse colon the positions become posterior, superior and anterior.



Blood supply of the large intestine

The most important vessels involved in the blood supply of the colon are the superior mesenteric artery, the inferior mesenteric artery and the marginal artery, which supplies an anastomosis between these two vessels . The superior mesenteric artery originates from the aorta just below the coeliac axis and passes posterior to the pancreas. Its terminal branch which supplies the caecum is known as the ileocolic artery. Its other named main branches which supply the colon are the right colic artery, which supplies the ascending colon, and the middle colic artery, which runs in the transverse mesocolon to supply the transverse colon. The inferior mesenteric artery arises from the aorta just inferior to the third part of the duodenum and descends on the left side of the aorta posterior to the peritoneum. Its first branch is the left colic artery which passes to the left and divides into ascending and descending branches. The ascending branch supplies the left side of the transverse colon and the splenic flexure, whereas the descending branch supplies the descending colon. The inferior mesenteric artery terminates at the base of the sigmoid mesocolon where it divides into sigmoid branches supplying the sigmoid colon and the superior rectal artery, which descends in the mesorectum to supply the upper part of the rectum. The marginal artery runs along the ascending, transverse and descending colons, receiving branches from the other colic arteries so that under normal circumstances ligation of any one of the main colic arteries would not result in ischaemia of any part of the colon. It must be remembered, however, that the marginal artery can become quite tenuous at the splenic flexure and continuity of the blood supply may be maintained by the ascending and descending branches of the left colic artery. It is therefore imperative that these two vessels are preserved when dividing either the left colic artery or the inferior mesenteric trunk during mobilization of the colon. As mentioned above, the upper part of the rectum derives its blood supply from the superior rectal artery which is a terminal branch of the inferior mesenteric artery, but the lower rectum receives blood from the two middle rectal (or haemorrhoidal) arteries coming from the internal iliac arteries and two inferior rectal arteries which originate from the internal pudendal arteries in the ischiorectal fossae. The internal pudendal artery is itself a branch of the internal iliac artery. The venous drainage of the large intestine follows its arterial blood supply but of course empties into the portal venous system. The inferior mesenteric vein diverges from the inferior mesenteric artery and passes up behind the pancreas to join the splenic vein. The superior mesenteric vein lies to the right of the superior mesenteric artery and joins the

splenic vein at its junction with the portal vein behind the neck of the pancreas. The lymphatic drainage of the large intestine also follows the blood supply. Small lymph nodes lie close to the marginal artery and also along the arteries leading towards it. The lymph draining through the lymph nodes associated with the branches of the superior mesenteric arteries passes into the intestinal trunk which lies in the root of the small bowel mesentery. The lymph nodes associated with the inferior mesenteric artery drain into the lumbar lymph nodes beside the aorta. Both of these empty into the cisterna chyli which enters the posterior thorax via the diaphragmatic hiatus.

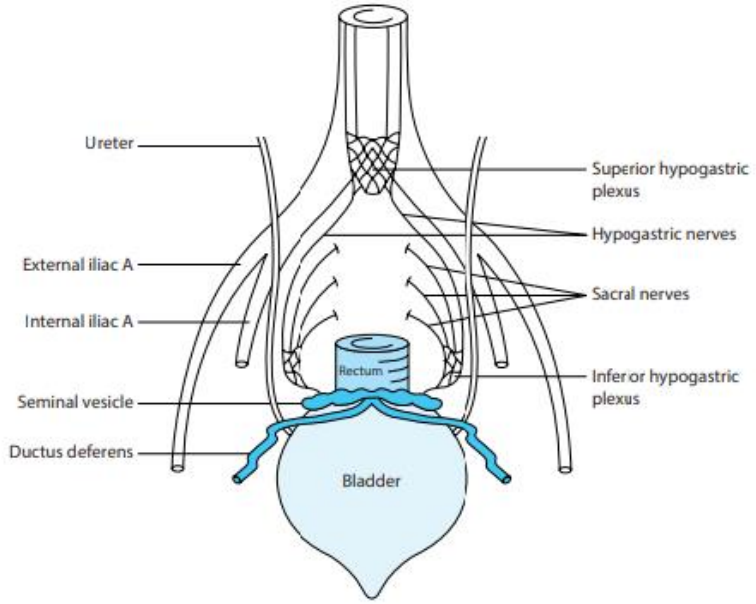
Nerve supply of the large intestine

The nerve supply of the colon and the rectum, like the lymphatics, follows the course of the main vessels. The right colon receives sympathetic nerve fibres from the lower six dorsal ganglia via the superior mesenteric plexus and parasympathetic fibres from the coeliac branch of the posterior vagus nerve. The left colon and rectum are supplied through the upper three lumbar ganglia via the inferior mesenteric, superior hypogastric and inferior hypogastric plexuses. The latter plexus also receives branches from the sacral parasympathetic nerves (nervi erigentes). These nerves remain outside the pelvic fascia and are sometimes injured during mobilization of the rectum.

Physiology of the colon and rectum

Absorption acid excretion

About 1000mL of ileal contents containing 90% water are discharged into the caecum every day in the normal adult. Water absorption takes place during transit through the colon, and only 100–200mL of water is excreted in the faeces. The absorptive capacity of the colon depends on the rate at which ileal contents enter the caecum, and is greater in the right colon than in the left. Normal faeces are composed of 70% water and 30% solids; about 50% of the solids are bacteria and the remainder is composed of food waste and desquamated epithelium. Nutrients such as glucose, amino acids, fatty acids and vitamins can be absorbed slowly through the colonic wall, but only very small amounts of these substances actually reach the caecum under normal circumstances. Sodium absorption is very efficient and is maintained by an active transport mechanism enhanced by mineralocorticoids and glucocorticoids. A normal adult can remain in sodium balance with as little as 5mmol of



The autonomic nerves of the pelvis.

sodium per day in the diet, but following total colectomy and ileostomy the minimum daily requirement increases to about 100mmol to offset losses from the stoma. Chloride and water absorption is passive and follows electrical and osmotic gradients established by the sodium pump. Potassium is actively excreted into the faeces against a concentration gradient and by secretion in mucus. Excessive mucus production (e.g. in colitis or in villous adenomas) may lead to enormous losses of potassium. Only a small amount of bicarbonate is secreted into the colonic lumen in exchange for chloride. A normal bowel habit is hard to define since it is influenced by social and dietary customs. The frequency of bowel movement in Western countries ranges from every 8 hours to once every 2–3 days. Any persistent change in bowel habit is an indication for investigation to exclude organic disease. Diarrhoea may be defined as stools containing more than 300mL of fluid daily, although again this is highly variable. When excessive, it may be debilitating and even fatal if associated with large losses of fluid and electrolytes which are not replaced. Inflammatory disease of the colon or small bowel mucosa may cause excessive exudation of fluid and also lead to diarrhoea, as does anything that decreases the intestinal transit time and decreases absorptive surface area. The symptom of constipation has different meanings for different individuals. It may imply infrequency of bowel movements, hard consistence of the stools or difficulty in evacuation. The pathologies leading to diarrhoea and constipation are dealt with later in the chapter.

Colonic motility

There are three patterns of motor activity in the colon:

•Segmentation. This is the most common type of motor activity seen in the transverse and descending colon and consists of annular contractions that divide the lumen into segments propelling faeces over short distances in both directions.

Segmental contractions form, relax and re-form in different locations in a random fashion, three to eight times per minute.

• Mass movements. These consist of strong contractions moving distally over relatively short distances (30–45 cm) in the transverse and descending colon. These are infrequent and probably occur only a few times each day, often in response to a meal.

• Retrograde peristalsis. This consists of annular contractions moving proximally in the right colon and in the sigmoid and descending colon. This is more frequently seen in experimental animals than in man. The retrograde movement can be shown by observing the spontaneous movement of a radio-opaque marker from the left to right colon.

A complex neurohormonal mechanism is involved in the colonic response to eating which has been inaccurately described as the gastrocolic reflex. This response consists of increased ileal emptying, increased mass movements and an urge to defecate. Other factors influencing colonic motility are physical activity, emotional states and faecal bulk. Thus normal colonic emptying is slow, complex and exceedingly variable. It is difficult to define altered motility in diseased states. There is no orderly laminar flow; some of the material entering the caecum flows past faecal material which has remained from earlier time periods. In general, residue from a meal reaches the caecum after 4 hours and the rectosigmoid by 24 hours. Since there is a large amount of mixing of bowel contents in the colon, residue from a single meal may be passed in the stool for 3–4 days afterwards under normal circumstances. Intraluminal pressure studies of the colon can be performed by the use of small balloons, fine open-ended catheters or telemetry capsules. Such studies indicate that, although pressures of up to 100mmHg can be generated, faecal transport can take place without a rise in intraluminal pressure. However, because specific patterns cannot be correlated with defined diseased states these investigations have little clinical significance. Rhythmic changes in the electrical potential in colonic muscle occur normally at two frequencies, three and nine per minute, respectively. The frequency appears to be approximately 16 per minute in the sigmoid colon with diverticular disease.

Colonic diverticular disease

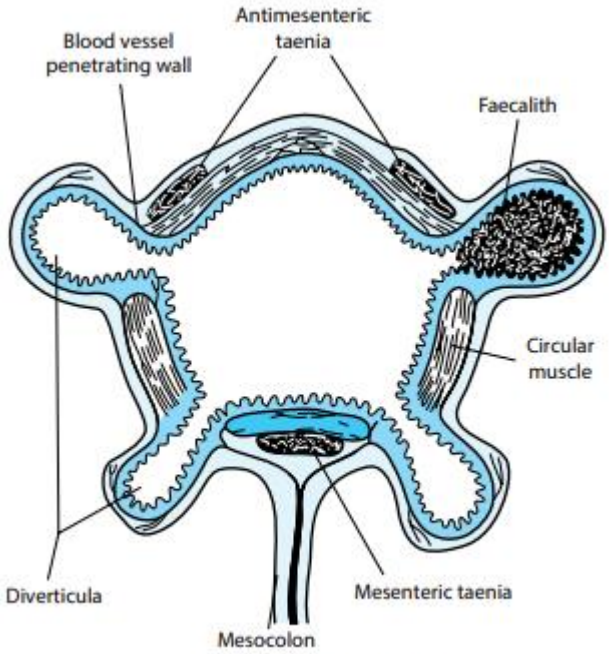
The term ‘diverticular disease’ describes a broad range of clinical pathology relating to the presence of symptomatic colonic diverticula. The disease presents

variably, ranging from mildly symptomatic fluctuation in bowel habit and left lower quadrant abdominal pain, through haemorrhage and localized sepsis to colonic fistulation, perforation and life-threatening peritonitis. In addition, symptoms may present as an isolated occurrence, or develop into a chronic complaint. Considered a disease of Western society and of elderly patients, its incidence is rising together with associated episodes of hospitalization. Despite the significant associated healthcare burden, the aetiology, natural history and optimal management of this disease remains controversial. The terminology applied to describe the different clinical presentations and disease stages associated with diverticular disease can be variable. 'Diverticulosis' refers to the presence of asymptomatic diverticula within the colon in the absence of associated inflammation. The term 'diverticulitis', typically prefixed as 'acute' on symptomatic presentation, can represent a wide spectrum of inflammatory change from localized subclinical inflammation to generalized peritonitis. This term is used interchangeably with 'diverticular disease', although this more commonly describes patients running a more indolent course of disease. Both diverticulitis and diverticular disease can be further described as 'uncomplicated' or 'complicated'. 'Uncomplicated' disease refers to presentations in keeping with localized colonic inflammation, including left-sided abdominal pain, fever and leucocytosis. 'Complicated' diverticular disease describes disease which has progressed beyond local inflammation to one of the known sequelae of abscess formation, colonic stricture, obstruction, haemorrhage, fistula or perforation. The term mychosis is used in the context of diverticular disease to describe muscular shortening and thickening of the colonic wall, commonly seen at surgery. The first noted reference to colonic diverticula as a pathological entity was made by the French surgeon Alexis Littre in the 1700s, although this was not in relation to diverticular disease per se. The current terminology stems from Fleischman, who used the term 'divertikel' in 1815. The pathology we recognize today as diverticular disease was first described by Jean Cruvehiler in 1849. Its absence from early surgical textbooks at this time suggests the disease was not as prevalent, or not as recognized, as today. Mayo reported the first surgical resection for complicated diverticulitis in 1907, advocating primary resection, although the disease remained uncommon. Although this case series promoted primary resection, staged resection consisting of drainage and stoma, interval resection and subsequent stoma reversal remained routine until the development of alternative treatment paradigms in the modern era. Similarly, elective surgery

for recurrent disease has seen a step-change in recent years. Parks' landmark study in 1969 suggested that recurrent disease was more virulent, and more likely to require surgery, hence elective resection was frequently recommended. The original findings of this study have been challenged in recent years, and current research suggests a more benign course of recurrent disease. This, along with the not insignificant morbidity associated with elective resection, means that several of the commonly accepted treatment algorithms for diverticular disease are currently in the process of being redefined.

Diverticular anatomy

Macroscopically, colonic diverticula are acquired saccular protrusions of the colonic wall consisting of colonic mucosa. They do not contain all layers of the bowel wall, are therefore not regarded as true diverticula, and are instead termed false, pulsion or pseudo diverticula. Typically small in size, ranging from 0.5 to 1.0 cm, they occur at points of weakness where the vasa recta blood vessels penetrate the bowel wall (Figure 30.27). They most commonly protrude in four rows situated between the antimesenteric and mesenteric taenia. Colonic diverticula most frequently occur in the sigmoid and left colon and in patients with diverticulosis 90% will have sigmoid involvement. Of note, proximal right-sided colonic diverticula most frequently seen in Asian populations are true diverticula, involving all layers of the bowel wall and therefore may be unrelated phenomena.



Structural abnormalities in diverticular disease.

Aetiology and pathogenesis

The aetiology and pathogenesis of diverticulosis have not been conclusively proven and are potentially multifactorial. Theories relate to structural abnormalities of the colonic wall, motility or neuromuscular abnormality, and the role of dietary fibre. The most commonly cited hypothesis relates to the wide discrepancies in geographical incidence of the disease. Painter and Burkitt suggested in 1971 that the frequency with which the disease was seen in industrialized countries compared with developing nations

related to the diet changes that took place in industrialized countries during the 1800s. Decreased dietary fibre, particularly through the consumption of processed food and roller milling of floor, was shown to reduce stool transit time and reduce stool weight. From this, it was proposed that resulting exaggerated colonic circular muscle contractions caused colonic wall hypertrophy as seen in sigmoid diverticular specimens. These contractions segmented the colon, increasing intraluminal pressure as confirmed on manometry studies (and as predicted by Laplace's law). This in turn could lead to the mucosal herniation through weak points in the bowel wall, as seen in diverticulosis. Subsequent research has not demonstrated hypertrophy or hyperplasia of the taenia coli smooth muscle cells themselves, and it has been proposed that the thickening seen is caused by elastin deposition in a contracted form, leading to bunching of the taenia. Collagen abnormalities have also been implicated, with increased levels of cross-linkage in colons affected by diverticular disease. This would reduce colonic wall compliance, with resulting injury to the submucosa potentially allowing mucosal herniation to occur. Reports of diverticular disease in young patients with Ehlers–Danlos and Marfan syndrome have been cited in support of this. These structural abnormality theories remain an area of research. Other theories relate to abnormalities of the neuromuscular apparatus in the bowel wall. Bassotti and colleagues have demonstrated that interstitial cells of Cajal and glial cells are decreased in colonic diverticular disease, potentially contributing to the disturbances in motility. Studies have investigated changes in serotonin signalling, given its importance in gastrointestinal motility. One indicated an increased presence of serotonin cells in the colonic mucosa of patients with diverticular disease. Others found attenuation of 5-HT transporter expression and function following recent acute diverticulitis. Several studies lend support to the traditional dietary fibre hypothesis, showing a correlation between fibre intake and diverticular disease. The American Health Professionals FollowUp Study showed an inverse correlation between dietary fibre and symptomatic diverticular disease with a relative risk of 0.58. The recent European Prospective Investigation into Cancer and Nutrition (EPIC) concluded that eating a vegetarian diet and a high intake of dietary fibre was associated with a lower risk of admission to hospital or death from diverticular disease. Vegetarians had a 31% lower risk of diverticular disease than meat eaters, confirming a risk seen with meat eaters in other studies. Despite this, there is little evidence behind the common recommendation of increasing dietary fibre intake to prevent

diverticulitis; this has been extrapolated from the established correlations. Most importantly, these hypotheses do not explain the pathogenic mechanism between the development of diverticulosis and subsequent diverticulitis. Similarities with both appendicitis and IBD have been drawn. Stasis or obstruction may lead to bacterial overgrowth and the resulting diverticula and colonic inflammation. Initial theories for this related to diverticular obstruction by a faecalith or food particle, leading to local inflammation and potential perforation. At a histological level, similarities are seen with the appearances of IBD, and several studies are currently exploring the role of aminosalicylate compounds in the treatment of diverticular disease. It has been suggested that some overlap between the diseases may exist. Additional theories include changes in colonic microbiota, ischaemia and visceral hypersensitivity which may all contribute to symptomatology.

Classification of diverticular disease

Hinchey classification and modified Hinchey classification by Sher *et al.*

Hinchey classificatio	Modified Hinchy classification (Sher et al.)
I Pericolic abscess of phlegmon	I Pericolic abscess
II Pelvic, intra-abdominal or retroperitoneal abscess	IIa Distant abscess amenable to percutaneous drainage IIb Complex abscess associated with fistula
III Generalized purulent peritonitis	III Generalized purulent peritonitis
IV Generalized faecal peritonitis	IV Faecal peritonitis

Clinical classification of diverticulitis (adapted from Kohler *et al.*)

Grade	Clinical description	Symptoms
I	Symptomatic uncomplicated disease	Fever, crampy abdominal pain, CT evidence of diverticulitis
II	Recurrent symptomatic disease	Recurrence of above
III	Complicated disease	Haemorrhage Abscess Phlegmon Perforation Purulent and faecal peritonitis Stricture Fistula Obstruction

Comparison between patients and clinical outcomes in diverticular disease is hindered by the differing presentations of uncomplicated vs complicated disease, and the varied way in which complicated disease manifests. Several classifications or scoring systems for the severity of disease have been published. The adoption of different classification systems in different publications has not always helped clarify this area. Most widely used internationally is the Hinchey classification, proposed in 1978 and later modified by Sher and colleagues in 1997. This is used to classify the severity of clinical insult resulting from perforated diverticular disease. Originally based on operative findings, the availability of CT imaging has extended its utility and allowed appropriate categorization of related abscess as recognized in Sher's modification. Further modification has subsequently been proposed by Wasvery and colleagues, adding a stage 0 for uncomplicated disease and introducing Ia and Ib to represent pericolic

inflammation or phlegmon and pericolic abscess, respectively. Köhler and colleagues through the European Association for Endoscopic Surgeons proposed a classification based on clinical severity and presentation, dividing this into symptomatic uncomplicated, recurrent symptomatic and complicated disease by complication. Given the subjective nature of clinical presentation for symptomatic uncomplicated and recurrent symptomatic disease, this is limited in its applicability due to the risk of incorporating an incorrect clinical diagnosis. In the European literature the Hansen–Stock classification has been widely used. This classifies the disease clinically by stage depending on its severity. Siewert and colleagues suggested a similar classification solely for acute complicated diverticulitis based on anatomical location of an abscess. Ambrosetti and colleagues have described a classification system describing diverticulitis as moderate or severe according to CT criteria. Although limiting its application to patients who have therefore undergone CT imaging, it is useful in assisting decision-making by allowing patients to be categorized into optimal pathways for management . The most recent addition to this classification structure has been suggested by Klarenbeek and colleagues in 2011. This represents an attempt to combine the existing classifications into one clinically applicable system, including the most recent developments in imaging modalities and treatments . Other scoring systems may also be utilized in the setting of classifying diverticular disease such as the Peritonitis Severity Score, the Mannheim Peritonitis Score or ColorectalPOSSUM (Physiological and Operative Severity Score for the enUmeration of Mortality and Morbidity). These are not specific to diverticular disease, but are useful in the estimation of patient risk and unit outcome audits.

Clinical presentation

- urological
  - cystitis
  - pyelonephritis
  - neoplasia
- gynaecological
  - pelvic inflammatory disease
  - ectopic pregnancy
  - ruptured ovarian cyst
  - ovarian torsion
- gastrointestinal
  - colorectal neoplasm
  - ischaemic/infectious colitis
  - IBD
  - irritable bowel disease
  - appendicitis (with right-sided sigmoid colon)
- other
  - ruptured iliac artery aneurysm
  - rectus sheath haematoma.

The majority of patients with diverticulosis remain asymptomatic. Symptomatic diverticular disease presents with a broad range of clinical manifestations, resulting from the variable nature of the disease. Symptoms may mimic those of a number of other conditions. This can take the form of acute uncomplicated, chronic uncomplicated or complicated disease.

Thus the spectrum of differential diagnosis in diverticular disease is wide:

Chronic uncomplicated diverticular disease

Chronic uncomplicated diverticular disease may present as episodic abdominal pain localized to the left lower quadrant, altered bowel habit and abdominal distension. The potential differential diagnosis is large, and the overlap with symptoms of irritable bowel disease can be significant making outpatient diagnosis difficult without further investigation. It is often diagnose when other more 'serious' pathology has been excluded. In the elderly patient group a new diagnosis of diverticular disease is frequently reached when the symptoms trigger investigation to exclude an underlying carcinoma.

Acute uncomplicated diverticular disease

Acute uncomplicated diverticular disease classically presents as abdominal pain localized to the left lower quadrant, often associated with a mild fever and leucocytosis. The picture may be variable, and right - sided abdominal pain is not infrequently seen particularly in patients with a long loop of redundant sigmoid colon lying to the right of the midline. Gastrointestinal disturbance is common, with anorexia, nausea and vomiting, constipation and/or diarrhoea frequently reported. Urinary symptoms may also occur secondary to proximity of the bladder to an inflamed sigmoid colon.

Complicated diverticular disease

Complicated disease presents according to the nature of the complication, which is predominantly abscess formation, perforation, fistulation, haemorrhage, stricture or obstruction. Abscess formation is the most common sequela of acute complicated diverticulitis, occurring in approximately 15% of patients. Classic symptoms are in keeping with abscesses elsewhere in the body, namely spiking fever and lassitude potentially accompanied by rigors. Typical locations are pelvic and pericolic, although retroperitoneal sepsis may also develop. The severity of the infection is most commonly classified using the Hinchey classification following CT. A significant localized abscess may progress into a free perforation.

Perforation secondary to diverticular disease occurs in 1–2% of patients and is a potentially life-threatening complication associated with high morbidity and mortality. The term can describe either perforation of a diverticular abscess leading to purulent peritonitis or faeculent peritonitis from contamination of the

peritoneal cavity with stool. Most commonly occurring with a first attack of diverticular disease, patients may not present with a history of diverticulitis. Presentation is similar to a generalized acute abdomen from other causes. The severity of the accompanying septic shock arising from purulent or faeculent peritonitis may help distinguish it from an upper gastrointestinal perforation and chemical peritonitis seen with, for example, a duodenal perforation.

Fistulation occurs in approximately 2% of patients with diverticular disease, and are included in the indications for surgery in 17–27% of patients. They arise from an inflamed colon and associated abscess decompressing into adjacent organs. Most often, this leads to a colovesical, colovaginal fistula, although colouterine, coloenteric and colocutaneous fistulas can occur. Often these patients do not report other associated symptoms and will initially be referred to a urologist or gynaecologist for further investigation. Symptoms are variable depending on the structures involved, although the patient is not usually acutely unwell, as any contained abscess has discharged in order to create the fistula. Colovesical fistulas account for approximately 65% of diverticular disease-related fistulas, and are suggested by recurrent urinary tract infections associated with enteric organisms, pneumaturia or faecaluria. Colovaginal fistulas account for 25% of cases and present with the passage or flatus of faecal material per vagina. Both colovesical and colovaginal fistulas are more common in women who have previously undergone hysterectomy, and it has been hypothesized that the uterus may act as a buffer, preventing the inflamed sigmoid colon reaching the bladder or vagina. Altman and colleagues reported that, compared with women who had neither hysterectomy nor diverticulitis, the risk of fistula surgery increased fourfold in hysterectomized women without diverticulitis, sevenfold in non-hysterectomized women with diverticulitis and 25-fold in hysterectomized women with diverticulitis. In all cases of fistulation, the possibility of an underlying malignancy or IBD giving rise to this should also be excluded. Haemorrhage is not usually associated with acute diverticulitis, but usually in isolation and/or as a result of underlying diverticulosis. It most likely arises from rupture of a blood vessel involved in the herniated mucosa of a diverticulum. As the most common cause of acute lower gastrointestinal bleeding, accounting for up to 40% of cases, it is typically painless, with the only heralding symptom being the urge to defecate. Clots may be passed and the colour of the blood will be variable depending on how proximal the source is located within the colon. Unlike upper gastrointestinal bleeding, although it may initially be profuse, diverticular

bleeding is typically selflimiting and resolves without need for significant intervention.

Approximately 30% of cases are associated with severe blood loss and cardiovascular compromise, although surgical intervention will be required in only 10% of these. In elderly patients, the increasing prevalence of NSAIDs and anticoagulation therapy can complicate management.

Stricture or obstruction at first presentation can be difficult to distinguish from colonic adenocarcinoma, even following radiological imaging, and the diagnosis may not be made until the lesion is resected and histologically examined (Figure 30.29). Stricturing is more common than obstruction, and is thought to develop through repeated episodes of inflammation leading to fibrosis. Patients complain of narrowed stools and constipation, varying according to the degree of structure. Approximately 10% of large bowel obstructions arise secondary to diverticular disease, and small bowel obstruction may also develop through adherence of a loop to an inflamed segment of colon or inflammatory mass. When associated with an episode of acute diverticulitis the obstruction may be functional secondary to colonic oedema and localized sepsis, and will resolve with medical treatment.

Atypical presentations may be seen in the most elderly patients or immunocompromised patients unable to mount a typical systemic inflammatory response. Abdominal pain may not reflect the severity of the clinical problem and overt signs of sepsis may be absent. Evidence suggests higher rates of abscess and free perforation in immunocompromised patients, and they are additionally more likely to fail medical management. These patients should be managed expectantly with close observation, early imaging, and a low threshold for intervention.

Investigation

Colonoscopy

Colonoscopy is controversial in the acute setting because of the potential risk of an iatrogenic perforation of the inflamed colonic wall, or converting a sealed microperforation into a free perforation. It does however play an important role in investigating the cause of acute symptoms in the aftermath of such an attack, and is typically performed 6–8 weeks later when the inflammation has subsided. Westwood and colleagues have recently questioned this tradition of endoscopic

follow-up, given the advent of routine CT on an inpatient for the majority of patients presenting as an acute hospital admission. In their study of 205 patients with acute uncomplicated diverticulitis undergoing colonoscopic investigation following diagnostic CT they showed a yield of 5.4% for advanced colonic neoplasia and 0.5% for colorectal cancer. This equated to the yield for screening asymptomatic individuals and they suggested that in this group of patients CT alone may be considered sufficiently sensitive to exclude the need for further interval endoscopic investigation in all patients. The other common indication for endoscopy is in the investigation of lower gastrointestinal bleeding. In the acute setting this may be hindered by the presence of blood within the lumen compromising the view. Endoscopy is also mandated in the investigation of stricture, when biopsies may be helpful in diagnosing the aetiology, and in the investigation of suspected diverticular fistula.

Ultrasound

Ultrasonography is frequently used in place of CT in European centres. With a reported sensitivity of 84–98% and a specificity of 80–97% in studies, it useful non-invasive, non-ionizing investigation on which acutely inflamed diverticula can be demonstrated. Abdominal ultrasound also has an important role in identifying other potential causes of acute symptoms, particularly relating to gynaecological causes in female patients. It can also be utilized to monitor a known abscess or mass. Uptake of ultrasonography for diagnosis of diverticular disease has not been universal however due to the operator-dependent variability and localized imaging window provided, which may miss other important related findings elsewhere in the abdomen or pelvis.

Computed tomography

CT is considered the primary diagnostic investigation in the acute setting by many, and diagnoses acute diverticulitis with a sensitivity approaching 100% and a specificity of 95%. The main benefit surrounds its ability to identify alternative pathology in the acute setting. In addition, it provides the best modality for characterizing extramural disease and abscess, which have led to a number of diverticular disease classifications based on CT findings as discussed earlier in this chapter. This categorizing of patients can be useful in guiding subsequent clinical management. The most frequent findings consistent with acute diverticular disease are bowel wall thickening, fat stranding and diverticula. Rectal contrast

may also be used to help delineate suspected fistula tracts, or demonstrate perforation. CT is also a useful therapeutic tool for percutaneous drainage of intra-abdominal abscesses. The main concerns relate to exposure to ionizing radiation, particularly in younger patients.

Contrast studies

Contrast studies were historically used routinely prior to the advent of widespread access to CT for initial investigation of suspected diverticular disease. Water-soluble single-contrast enema has now largely been superseded by CT, although it remains an option in resource-poor environments. It may help identify intramural changes and leakage of contrast may aid diagnosis of perforation or fistula; however, it does not provide information relating to extramural disease.

Further investigations

Other investigations may also be required in complicated disease, depending on the specific presentation and concerns. In significant lower gastrointestinal bleeding, mesenteric angiography may be used in order to localize the bleeding point within the colon. This may then be treated radiologically or guide surgical resection. In suspected diverticular fistulation, cystoscopy may be useful in identifying vesical openings and ruling out other urological disease causing symptoms.

Treatment

The optimal treatment of diverticular disease is an area in evolution and the subject of active research. Traditional management recommendations are being questioned, with an increasing move towards conservatism. New medical therapies are being investigated, and new paradigms in surgical management are being proposed.

Medical treatment

For mild, uncomplicated diverticulitis outpatient management with or without antibiotics may be appropriate, depending on the patient's individual situation. Further investigation may be undertaken if warranted after the episode has resolved. If outpatient management fails then hospitalization is required for more active treatment. For patients presenting with more acute systemic symptoms including fever, pain and inability to tolerate oral intake then hospitalization is

appropriate from the outset. Supportive therapy, including antimicrobial therapy, gut rest, intravenous fluids and analgesia may be required together with radiological imaging to confirm diagnosis and stratify treatment. A proportion of patients will fail medical therapy, and approximately 15% of patients develop pericolonic or intramesenteric abscess. In these cases radiological or surgical intervention may ultimately be required. Antibiotics remain the main therapy for symptomatic diverticular disease. The precise regimen will depend on local recommendations; however, typically these will be chosen to cover both aerobes and Gram-negative micro-organisms. In cases of severe sepsis these may be given parenterally until the sepsis is controlled, followed by oral administration. Guidance on the optimum duration of oral antibiotic therapy is lacking, but this will typically be prescribed for 7–10 days. Currently the role of antibiotics in mild uncomplicated diverticular disease is being questioned. In Europe there is a trend towards avoiding antibiotic therapy in these cases, whereas in North America antibiotics are more frequently administered. A Scandinavian study has suggested bowel rest alone in these cases is similar in efficacy to antibiotic therapy. This area is currently being examined further in the DIABOLO trial, a large multicentre randomized control trial in Europe which is investigating a liberal policy, without antibiotics and without the strict requirement for hospital admission. The clinical and economic results are awaited. Risk factor modification is a traditional component of medical therapy based on demonstrated associations, although the evidence that modifying these risk factors imparts a beneficial effect is largely lacking. Increasing dietary fibre, weight loss, smoking cessation and regular physical activity may play a role in reducing the frequency or severity of the disease. Emerging medical therapies include the use of aminosalicylates and probiotics, and active studies continue to investigate their role. Low-grade proinflammatory states are associated with active diverticular disease; therefore, immunomodulatory agents such as 5-aminosalicylic acid (5-ASA) compounds used in the treatment of IBD have garnered interest as potential agents for treating diverticular disease or preventing symptomatic attacks. A meta-analysis of several small trials investigating 5-ASA products in the treatment of diverticulitis has confirmed improved outcomes; however, the results must be interpreted cautiously until larger objective trials have been conducted. Probiotics have been investigated as a similar potential treatment for altering the inflammatory milieu seen in diverticular disease. It has been theorized that the altered bowel motility and transit time may change the colonic microflora. Probiotics are live micro-

organisms, the ingestion of which may restore normal colonic microflora. The beneficial effect of this treatment is yet to be clearly demonstrated.

Radiological intervention

If systemic symptoms and signs of sepsis continue despite optimum antimicrobial therapy, the presence of a diverticular abscess should be suspected. Following confirmatory CT imaging, percutaneous radiologically guided abscess drainage may bring the sepsis under sufficient control for antimicrobial therapy to succeed without further surgical intervention. Despite this, radiological drainage may fail and approximately 20–30% of cases will ultimately require surgical drainage. The options for radiological intervention depend on the size, nature and anatomical location of the abscess(es). A single, small, well-contained abscess.

Surgical treatment

Surgical intervention is reserved for cases in which medical management has failed, or for the complications of diverticular disease. Although the majority of cases of diverticulitis will resolve with medical management, approximately 20% of patients with diverticulitis require surgical treatment at some stage. In recent years there has been an increasing trend towards more conservative management options, and considerable debate still surrounds the need for operative intervention, the timing of this surgery, and the appropriate procedure to perform.

Elective surgery

Recurrent diverticulitis has previously been an accepted indicator for elective sigmoid resection, for both symptom control and the prevention of recurrence and more complicated disease. Widely endorsed guidelines, until recently, recommended surgery for any patient who had experienced two attacks of acute diverticulitis requiring hospitalization. Such elective resection in diverticular disease carries not insignificant risks, with morbidity estimated at 25–50% and a mortality of 1–15%, increasing with age. This was justified by historical studies suggesting high rates of recurrence with hospitalization and complicated disease after repeated attacks of diverticulitis. Contemporary studies have challenged this justification, suggesting that the disease in fact follows a more indolent path. The current trend is therefore moving towards more conservative management, and this policy has not so far led to an increase in patients presenting with

complicated disease. In addition, there has been a growing recognition that such elective surgery does not always improve the patients' symptomatology and disease recurrence may still occur in 3–13%. Elective surgery should now only be offered following a full consideration of factors including the patient's age, comorbidities, severity of disease, complications and risk of further episodes. Special cases include patients in remote locations who may have difficulty in accessing medical care for the complications of diverticular disease when they occur, and those in whom repeated hospitalization is unacceptable for work or lifestyle reasons.

Diverticular fistulas may occasionally close spontaneously when the acute inflammatory process settles. More often those not resolving with conservative management require surgical treatment unless patient choice or operative risk prevents this. Patients and clinicians must be aware of the small risk of underlying neoplasia driving the fistulating process rather than a pure diverticular phenomenon. Often this can only be established on histological examination of the excised specimen. The operation should be timed to ensure the patient is nutritionally optimized and inflammation is controlled or quiescent. An attempt at establishing the location of the fistula should be undertaken prior to surgery through appropriate contrast imaging or endoscopic examination. Despite this, at surgery the tract may not be clearly identified and resection of the involved portion of colon may be sufficient to resolve the problem. Primary anastomosis will usually be performed, and the omentum may be mobilized and placed between the bowel and other pelvic organs. The surgery may be technically difficult due to the significant inflammatory mass often associated with such cases, and anatomy may be distorted. Placement of ureteric stents may be considered prior to surgery. Although case series report success undertaking such procedures laparoscopically, their complexity makes this a difficult undertaking.
Emergency surgery

Haemorrhage secondary to diverticulosis is an infrequent cause for emergency surgery, and rarely with symptomatic diverticulitis. As discussed earlier in this chapter, although approximately onethird of patients with bleeding experience severe blood loss this will largely spontaneously settle with only supportive therapy. Only 10–20% of patients will require further intervention. This most commonly takes the form of interventional radiological treatment with mesenteric angiography and embolization of the vessel feeding the bleeding point

as discussed earlier in this chapter. Rarely, patients will nonetheless require surgery due to massive recurrent or ongoing bleeding, or failed mesenteric angiography.

The need for surgery in this patient group is related to the ongoing transfusion requirement, with one study indicating that in those receiving ≥4 units of blood 60% will ultimately require surgery. With ≤4 units only 1.5% required surgery. Patients coming to surgery are typically haemodynamically unstable, high-risk cases.

Once an upper gastrointestinal cause is clearly excluded with an oesophagogastroduodenoscopy, laparotomy and on - table colonoscopy may be attempted. The colon is lavaged through the appendix stump in order to clear retained blood and improve the endoscopic view. This may be unrewarding; however, if a bleeding point is clearly identified then a directed segmental colonic resection can be performed with anastomosis or exteriorization of the bowel. If no bleeding point is seen then blind subtotal colectomy can be performed with end ileostomy and mucus fistula. In this situation the patient is often in extemis and the surgery has substantial morbidity and mortality.

Stricture or bowel obstruction may result from fibrosis and scarring caused by recurrent episodes of inflammation. Stricturing is seen more frequently than obstruction, which is usually incomplete. Management of these cases depends on the degree of obstruction and urgency with which it presents. With strictures and incomplete obstruction, bowel rest and treatment of the underlying inflammation may be all that is required to get a patient over an acute episode. Full outpatient radiological and endoscopic investigation may then be performed. In cases of complete obstruction, emergency management will be required. In both scenarios, colonic stenting can be considered, either as a temporizing measure and bridge to surgery or as a non-operative option. The procedure is technically more difficult than that for colorectal cancer, because of the longer portion of bowel typically involved. The risk of stent migration is high, and series published so far indicate that complications and need for reintervention are both common. Surgical options include on - table colonic lavage, resection and primary anastomosis, with or without covering ileostomy or alternatively the traditional Hartmann's procedure. Occasionally, small bowel obstruction may develop through its adherence to or involvement in an inflammatory mass. Although CT scanning may help differentiate between primary small bowel obstruction and a

secondary cause relating to diverticular disease, exploratory surgery may still be required.

Perforated diverticular disease has traditionally mandated surgery unless clinically futile. Classic dogma taught that Hinchey stage I and potentially stage II diverticular perforations are treated surgically with resection and immediate anastomosis. Resection and diversion via a Hartmann's procedure were reserved for perforated stage III and IV disease. This was based on the theory that an anastomosis in the setting of local infection and/or systemic inflammation is associated with a high risk of anastomotic leak. Evidence is now growing that this may be overtreatment and that such a radical approach may be avoided in many cases. The theory for this comes from the concept of microperforation of the bowel, which subsequently seals. Purulent contamination may therefore not be in direct continuity with the lumen, and may arise from a ruptured interloop abscess.

For Hinchey II and III patients, studies suggest that laparoscopy with copious peritoneal washout with continued antimicrobial therapy may be better treatment provided faecal contamination or free perforation is not seen. If these are seen, conversion to open surgery is still recommended. One prospective multi-institutional study has shown laparoscopic washout is associated with significantly improved outcomes compared with standard laparotomy, with morbidity of 4% and mortality of 3%. Such treatment remains controversial in some quarters, and randomized controlled trials are under way. The surgical options in perforated diverticular disease can be summarized as follows:

- conservative
  - laparoscopic lavage and drainage
  - laparotomy ± suture, drainage and stoma formation
  - exteriorization of sigmoid loop
- radical
  - resection and diversion without anastomosis (Hartmann's procedure)
  - resection and primary anastomosis
  - resection and primary anastomosis with proximal diversionary stoma.

When a laparotomy is required, the sigmoid colon should be resected in order to remove the inflammatory focus, rather than simple drainage and washout. A one-stage procedure is preferable in order to avoid the mortality and morbidity associated with further surgery; however, careful patient selection is required for this. A primary anastomosis should only be performed in an adequately resuscitated, cardiovascularly stable patient and where appropriate high-level perioperative care is available. Splenic mobilization should be

considered in order to reduce tension on the join. When concerns arise, defaulting to a stoma may be the safest option.

The preference for one-stage surgery has developed partly through increasing experience in performing anastomoses in what would previously have been considered adverse conditions, partly through better patient selection and partly through improved anaesthesia and perioperative care. In parallel with this, studies have shown the high levels of morbidity and mortality associated with a Hartmann's procedure. Stoma complications of over 10% are reported in this setting, and given the elderly population typically affected up to 35% of patients may never have their colostomy reversed due to the risks of further surgery or patient preference. A recent systematic review reported mortality of 9.6% vs 15.1% for primary anastomosis versus Hartmann's resection for perforated diverticular disease.

In this population it is also important to ensure sigmoidoscopy is performed prior to resectional surgery in order to exclude coexistent distal adenocarcinoma, as this may influence the surgery. Sigmoidoscopy can be quickly performed on the operating table so as to avoid any delay to the emergency surgery.

In summary, our understanding of diverticular disease and the optimum treatments for this are in a state of transition. Although diverticular disease is becoming more common, its natural history is still being defined. Several theories regarding the aetiology of the condition exist, and numerous epidemiological risk factors have been identified, however the underlying pathogenesis has not yet been proved. New medical treatments are being investigated, and classically held views regarding the optimum surgical management are being challenged by new paradigms.

Ulcerative colitis

Ulcerative colitis is a distinctive form of inflammatory disease affecting the large intestine. Inflammation of the colon (colitis) has varied aetiology but the various disorders can be grouped as infective or non-infective:

- Infective
  - Virus
    - Cytomegalovirus (CMV)
  - Bacteria
    - *Campylobacter*
    - *Escherichia coli*
    - *Shigella*
    - *Clostridium difficile*
    - *Chlamydia*
    - *Gonococcus*
  - Protozoa
  - Amoebiasis
  - *Cryptosporidium**
  - *Giardia**
- Non-infective
  - Ulcerative colitis
  - Crohn's disease
  - Radiation enteritis
  - Drug-induced colitis

Ulcerative colitis is a disease confined to the large bowel mucosa. The disease chiefly affects the young and has an equal sex distribution. The annual incidence per 100 000 population is similar for males and females up to the fourth decade of life. As a rule of thumb, it is useful to remember incidences of 10 and 5 per 100 000 for ulcerative colitis and Crohn's disease respectively. The incidence of ulcerative colitis has changed little over the last 30 years, whereas Crohn's disease has increased about fivefold, although this may now be stabilizing and is possibly in decline. The prevalence of ulcerative colitis is about 160 per 100 000 population (compared with about 50 per 100 000 for Crohn's disease). This means that there are around 100 000 people affected in the UK.

Epidemiology and aetiology

Genetics Between 10% and 20% of affected individuals have a firstdegree relative with IBD. There is a concordance within Jewish families, a low incidence in spouses and absence of IBD in families of adopted probands. Crohn's disease and ulcerative colitis can occur in the same family, and the overlap of features of the two diseases (indeterminate colitis) of 10–15% and the change of diagnosis from one to the other in a further 10% may be a feature of genetic heterogeneity. While it has been suggested that both diseases share some similar gene loci with other genes defining each condition, it is noteworthy that there are no reports of mixed Crohn's disease and ulcerative colitis among monozygotic twins. Extra-alimentary manifestations, including ankylosing spondylitis and primary sclerosing cholangitis, are more common in first-degree relatives of affected propositi; both have HLA associations, including HLA-B27 and HLA-B8 respectively. Ulcerative colitis is more common in white people than in black people or Arabs. Whether this is genetic or environmental is unknown. IBD has a low incidence in developing countries.

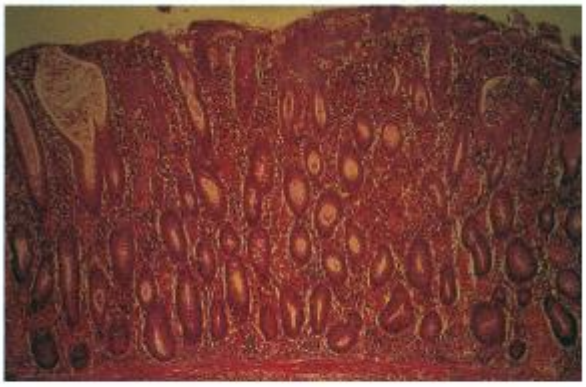
Environmental factors

The incidence of ulcerative colitis rises about 10 years ahead of Crohn's disease, suggesting environmental influences. There is evidence that NSAIDs can be

associated with IBD in humans. It has long been recognized that some patients with ulcerative colitis have a history of infective proctocolitis. Smoking is protective in ulcerative colitis but not in Crohn's disease. It also appears to be true for pouchitis. Data are conflicting on the influence of oral contraceptives, and most of the information relates to Crohn's disease. No causative dietary factor has been identified in humans. However, lactose intolerance can accompany ulcerative colitis, although this is rare.

Pathogenesis

Inflammation is confined to the large intestine, which includes the colon, rectum and upper anal canal. The mucosal columnar glandular epithelium extends into the anal canal to the anal transitional zone, which varies in longitudinal length from a few millimetres to over a centimetre. The anatomical extent of ulcerative colitis varies from involvement of the upper anal canal and rectum alone (proctitis) to the colon more proximally (proctocolitis). The rectum is always involved for all practical purposes, although relative rectal sparing can occur in patients receiving local anti-inflammatory treatment. A spared rectum not associated with local treatment should raise the suspicion of Crohn's disease. Backwash ileitis occurs only in cases with colonic extension to the ileocaecal junction. Anal disease occurs in about 10% of cases coming to proctocolectomy. The lesion is usually minor, e.g. a low fistula or fissure. Rectovaginal fistula can occasionally occur in ulcerative colitis. The inflammation in the colon and rectum is diffuse without intervening normal mucosa. Ulceration causes bleeding and in patients with severe disease the inflammatory exudate results in loss of water, electrolyte and protein which may be as great as 200 g per 24 hours.



Histological appearances of ulcerative colitis. Note the crypt abscesses.

Clinical presentation

At presentation, approximately 50% of patients have disease confined to the rectum (proctitis). In 30% this extends to the left colon (proctosigmoiditis) and

in a further 20% disease extends beyond the splenic flexure (extensive colitis). Symptoms are local and general. The severity of the former and the presence of the latter depend largely on the anatomical extent of the disease. Ulcerative colitis is characterized by exacerbations and remissions. Bloody diarrhoea with urgency is the hallmark of colitis.

Proctitis

Symptoms include bleeding and mucus secretion. Sometimes constipation occurs but more often there is increased frequency of defecation. Rectal irritability may result in urgency of defecation. Systemic symptoms are very uncommon. Patients do not suffer from disturbances of growth, and only rarely from extra-alimentary manifestations or subsequent cancer. There is a tendency for proctitis to extend proximally with time. Proctosigmoiditis and extensive colitis Proximal extension to the left colon and more proximally leads to worsening local symptoms and systemic disturbances in some cases. Urgency is the most incapacitating local symptom. When severe, patients may have warning of impending defecation of a few seconds only. In such cases, urge incontinence can occur. Severe symptoms often dominate the patient's life and seriously affect work and family life. The protein-losing enteropathy may lead to malnutrition with loss of lean body mass and anaemia. Retardation of growth in children may be a feature of extensive colitis. In acutely ill patients, water and electrolyte loss may cause hypovolaemia and breakdown of the mucosal barrier may lead to toxicity. Exacerbations may be precipitated by anxiety or stress but usually there is no recognizable causative factor. The disease may be of the acute relapsing type, with acute episodes interspersed by periods of complete resolution. Alternatively it may take the form of persisting chronic disease. Such patients may develop acute exacerbations that settle only partially on treatment. Patients with extensive disease are more likely to have associated extra-alimentary manifestations and are at greater risk of developing malignancy. These complications can occur in patients with disease confined to the left side of the colon but are much more frequent in extensive colitis.

Acute presentation

About 5% of patients present with acute severe colitis as the first manifestation of the disease. The patient will be ill with severe local symptoms, weight loss, anorexia, and water and sodium depletion. Intensive medical

treatment has a high chance (70%) of inducing remission but when unsuccessful urgent or semi-urgent surgery will be necessary. Acute severe colitis may progress to toxic dilatation recognized by distension of the colon to a diameter greater than 6cm on a plain radiograph. Perforation is a rare but serious occurrence with a mortality still approaching 40%. Rarely deep ulceration (usually in the rectum) may cause severe bleeding.

Extra-alimentary manifestations of ulcerative colitis

Up to one-third of patients with ulcerative colitis will develop at least one extra-alimentary manifestation during the course of the illness. These can be divided into those related or not to disease activity. Amyloid or hypertrophic osteoarthropathy are rare and are the result long-standing chronic illness.

Arthropathy

Arthropathy is the commonest extra-alimentary manifestation. It can be divided into three broad groups. Activity - related polyarthropathy occurs in up to 20% of patients and is more likely in those with extensive disease. It affects predominantly the large joints of the limbs, knees being the most common. The arthropathy is fleeting and asymmetrical and is rheumatoid factor negative. It disappears when medical treatment induces a remission or after proctocolectomy. It can develop in patients with pouchitis after restorative proctocolectomy. Ankylosisng spondylitis is an axial arthropathy involving the sacroiliac joints and one or more vertebrae and occurs in up to 5% of patients. The majority of such cases are HLA-B27 positive. The disease is unrelated to the activity of colitis and does not respond to proctocolectomy. There may be a genetic basis. Asymptomatic sacroileitis is an arthropathy limited to the sacroiliac joint and is HLA-B27 negative. It occurs more frequently than ankylosing spondylitis and is also unaffected by treatment for the colitis.

Liver

Ulcerative colitis - associated hepatic and extrahepatic disorders occur in up to 5% of cases, predominantly in those with extensive colonic involvement. Fatty degeneration is common, but has no obvious clinical importance. Parenchymal liver disease of the chronic active hepatitis type and cirrhosis can occur. The latter may lead to portal hypertension. Primary sclerosing cholangitis is more often seen

in ulcerative colitis than in Crohn's colitis. The disease is characterized by a fibrous inflammatory reaction within the biliary tree leading to multiple intra- and extrahepatic stenoses. The diagnosis is made on endoscopic retrograde cholangiopancreatography or MRI. There is no apparent relationship between duration of disease and disease activity, although patients with primary sclerosing cholangitis undergoing restorative proctocolectomy have a higher subsequent incidence of pouchitis and dysplasia in the ileal pouch mucosa. Treatment by steroids, colectomy or antibiotics is ineffectual and ultimately the disease progresses to liver failure. Such patients may be considered for liver transplantation. Cholangiocarcinoma is a rare association with ulcerative colitis. There may be an induction period of many years and the risk appears to continue even after proctocolectomy.

Skin

Erythema nodosum is the commonest cutaneous manifestation of IBD. It occurs more often in Crohn's disease. The condition is activity related. Pyoderma gangrenosum is more often associated with ulcerative colitis. It usually occurs in the lower limb as a circumscribed area of erythema with a punched-out ulcerated centre. Lesions may be multiple and occasionally are very extensive. Proctocolectomy is associated with healing in about 50% although this may take weeks to months.

Eyes

Uveitis is rare and is not related to disease activity. The condition can lead to scarring with visual impairment and ophthalmological management is essential. Episcleritis is activity related and occurs more often in Crohn's disease. It does not lead to chronic changes.

Investigation

In the tropics, infective causes constitute the vast majority of causes of colitis. In temperate regions infective causes may occur in hospitals and long-stay institutions. The diagnosis is made by histopathological examination of biopsy material taken during endoscopy having excluded microbiological causes. Endoscopy will determine the extent of inflammation in the assessment of severity.

Microbiology

A specimen of stool must be sent for microbiological examination. If amoebiasis is suspected, the specimen should be examined in the laboratory within a few hours. In addition, diagnosis of amoebiasis requires a biopsy to demonstrate cysts. Shigella, C. difficile and Campylobacter infection should be excluded. These may occur in epidemics in institutions with a significant mortality in frail elderly patients. The microbiologist should be warned on the request form that these could be present.

Proctitis can be caused by gonorrhoeal and chlamydial infection. The inflammation is catarrhal and consists of an erythematous flare associated with a purulent exudate. It rarely extends proximally beyond a few centimetres from the anal verge. When suspected, rectal, urethral and vaginal swabs should be taken. Again the microbiologist should be forewarned. Opportunistic infection may cause proctocolitis in immunocompromised patients (e.g. those with human immunodeficiency virus infection) or those on immunosuppressive drugs. Examples include cytomegalovirus, Mycobacterium avium-intracellulare and cryptosporidia.

Endoscopy

Loss of the vascular pattern (the submucosal vessels seen through the transparent mucosa) is the most sensitive sign of inflammation. This is due to oedema of the mucosa which makes it opaque. Oedema also causes fine granularity in which there is a delicate regular stippled appearance of the mucosal surface. More severe changes include erythema, contact bleeding and frank ulceration. Where previous acute attacks have been followed by repair, mucosal regeneration nodules (coarse granularity) or pseudopolyps may be seen. Pseudopolyps represent tags of mucosa that have been partially detached during the active episode and remain as projections after healing of ulceration. Rigid sigmoidoscopy will only visualize the rectum. Colonoscopy allows assessment of the proximal extent of the disease.

Histopathology

Histopathological examination of a mucosal biopsy is the basis of diagnosis.

Biopsy technique

A biopsy is obligatory and is most easily obtained during rigid sigmoidoscopy. Colonoscopy used for surveillance allows multiple biopsies to be

taken. Perforation and bleeding are potential complications. The patient must be asked whether anticoagulants or immunosuppressive drugs are being taken before a biopsy is performed. The biopsy taken during rigid rectoscopy itself should be obtained with forceps with a circular cusp that minimizes the depth of penetration. The optimal site is about 7 cm from the anal verge in the posterior quadrant of the rectum. Adequate vision during rectoscopy must be assured and the jaws of the forceps are firmly closed, taking a bite of mucosa and submucosa. After the biopsy has been taken, the site must be inspected for bleeding. If this persists, a topical solution of epinephrine (adrenaline) 1 in 1000 soaked in a small swab should be applied to the biopsy site. The biopsy should be oriented onto a piece of absorbent paper and placed in formalin (10%).

Histopathological features

Active disease

In active disease, there is mucosal thickening with infiltration of the lamina propria by neutrophils, plasma cells, lymphocytes, eosinophils and mast cells. Mucin within goblet cells is discharged so that these are less evident or absent (goblet cell depletion). The degree of neutrophil infiltration is the best histopathological marker of severity. In mild disease, neutrophils are confined to the lamina propria. Extrusion of neutrophils into the crypt lumen forms a crypt abscess, the number of which correlates with the severity of disease. Mucosal ulceration is partly the result of rupture of crypt abscesses leading to mucosal destruction. Damage to the crypt basal epithelium leads to loss of crypts. Attempts at regeneration may be mistaken for dysplasia but the presence of more normal cells towards the luminal surface allows these to be distinguished. There may be branching of crypts owing to regeneration following crypt epithelial damage.

Acute severe colitis

Progression of these acute changes occurs in cases with acute severe colitis. Ulceration can be very extensive, leaving large areas of exposed muscularis propria covered with granulation tissue. This may be associated with thinning of the musculature and colonic dilatation. Inflammation may be transmural and fissure formation may be seen. Colitis in remission may leave a distorted architectural pattern with crypt depletion. Mucosal cells that remain often regain normal function and show retained mucin as identifiable goblet cells. A chronic

inflammatory cell exudate in the lamina propria is likely to be present, although this may be very mild in patients in remission for long periods. Paneth cell metaplasia indicates episodes of previous colitis.

Ulcerative colitis/Crohn's disease

Diagnostic difficulties in differentiating ulcerative colitis and Crohn's disease have been recognized for many years. The pathological criteria distinguishing them are shown in Table 30.7.

Indeterminate colitis

In some patients insufficient numbers of these diagnostic attributes are present or there is considerable overlap and atypical features are seen. Thus it may be impossible for the pathologist to separate the two diseases, which may be reported as unclassified colitis or as unclassified colitis with additional indication of the possible or probable presence of Crohn's disease or ulcerative colitis. In about 10% of cases, however, the pathologist will be able to state only that the colitis is indeterminate.

Indeterminate colitis is not a disease entity. It is a term which indicates that the histopathologist is unable to come to a firm diagnosis owing to the presence of features of both conditions. Usually the dilemma arises in emergency colectomy specimens where severe inflammation may be combined with features of ulcerative colitis and Crohn's disease. In trying to resolve the diagnostic dilemma more biopsies should be taken. If the patient has had a colectomy for acute disease, then these will come from the rectal stump which may have already developed diversion proctitis making the histopathologist's task more difficult. The small bowel should be examined by endoscopy or radiology which if abnormal is suggestive of Crohn's disease as would be the presence of an anal lesion.

When histopathological, radiological and clinical features are considered together, patients with indeterminate colitis can usually be judged to incline more to Crohn's disease or ulcerative colitis. Where they cannot, the natural history tends to incline to that of ulcerative colitis.

Radiology

A plain abdominal radiograph is the most useful means of identifying colonic dilatation. There has been a movement away from tubular contrast radiology to CT. The instant barium enema is now rarely used although it gives an excellent record of the extent of the disease in most cases.

Treatment

Best care is likely to be achieved by a multidisciplinary team including medical staff, specialist nurses, nutritionists and stomatherapists with social and psychological support. Collaboration between gastroenterologist and surgeon is essential and should include patient sharing where appropriate, joint outpatient consultations for difficult cases, and early involvement of the surgeon in acute disease.

Medical treatment

Medical treatment involves bed rest and the correction of water and electrolyte depletion by intravenous infusion. Severely anaemic patients should be given blood. The patient should be encouraged to eat a high protein and calorie diet. Intravenous nutrition may be indicated in severely malnourished patients, as judged by a decrease in lean body mass and serum albumin.

Intravenous prednisolone (60mg daily) and an H2 -receptor antagonist or a proton pump inhibitor to protect against upper gastrointestinal ulceration are given. Ciclosporin has been reported to induce remission in over 50% of patients unresponsive to steroids but early relapse may occur, resulting in the same clinical situation within a short period.

More recently, a number of studies have reported the use of biological agents for the treatment of acute ulcerative colitis. Infliximab (Remicade Centocor, Malvern, PA), a chimeric (75% mouse, 25% human) anti-tumour necrosis factor (TNF)-α monoclonal antibody, and Adalimumab (Humira, Abott), a humanized anti-TNF antibody, are currently used to modulate the proinflammatory processes central to the pathogenesis of IBD. In a study of 30 patients with active ulcerative colitis treated with infliximab between 2000 and 2006 at Oxford, 53% of patients came to colectomy at a median time of 140 days after their first infusion (range 4–607). Of those avoiding colectomy, only 17% sustained a steroid-free remission. The role of immunotherapy in the acute setting remains to be established in a number of randomized controlled trials.

Patients with severe acute colitis require admission to hospital. Initial treatment is medical but about 30% of patients will come to surgery. Surgery is absolutely indicated in cases with acute toxic dilatation or perforation.

Monitoring is essential to assess improvement or deterioration. The pulse rate, temperature and blood pressure are regularly recorded. The patient should be weighed on admission and twice weekly thereafter. Blood should be sent for haemoglobin, albumin and electrolyte estimations. A stool chart is essential. This should record every defecation with an assessment of volume and consistency of stool and the presence or absence of blood on each occasion. The abdomen should be examined regularly. Distension suggests the possible development of toxic megacolon. A plain radiograph will allow assessment of the colonic diameter. Abdominal tenderness and rigidity suggests local or general peritonitis. The presence of intramural gas on the plain abdominal radiograph is a sign of imminent perforation and is therefore an indication for immediate surgery.

Unresponsiveness to medical treatment in acute colitis

Failure to respond to medical treatment should be recognized early. The gastroenterologist and surgeon should confer at least daily to decide whether there has been improvement, stagnation or deterioration. Deterioration despite adequate medical treatment should be an indication for surgery. Stagnation over several days with no sign of improvement should also be an indication for operation. Clinical indicators that surgery is likely to be necessary at the time of admission include a frequency of defecation of over 10 times per 24 hours with the passage of blood at every defecation attempt, low albumin, low haemoglobin and a fall in lean body mass of more than 10%. Previous acute attacks, poor general health and social circumstances affected by the disease should be taken into account. A significant history of chronic illness and social incapacity should sway the decision in favour of surgery.

Medical treatment of proctitis

Most patients are satisfactorily treated medically by a combination of steroids and 5-ASA preparations. The former is intended to induce a remission, the latter to maintain a remission once achieved. Both can be given as suppositories or as an enema, the choice depending on the proximal extent of disease. Steroid preparations such as budesonide have a lesser tendency for absorption. An oral 5-ASA preparation should also be prescribed from the

beginning. Modern 5-ASA drugs (Asacol, Pentasa and Balsalazide) no longer contain sulphonamide, which was responsible for some of the side effects of salazopyrine. They are formulated to protect the aspirin from degradation before it arrives in the colon. Proctitis refractory to this treatment may respond to other preparations, including bismuth, nicotine and witchhazel. Rarely, patients with persisting severe symptoms may require surgery.

Surgical treatment

Acute colitis (emergency surgery) Surgery has a major role in the management of acute colitis. The need for surgery is greatest during the first year after onset of the disease. The indications and their relative frequency are shown in Table 30.8 and Figure 30.34, respectively.

Colectomy with ileostomy and preservation of the rectum

This is the operation of choice for acute severe colitis. For all surgery for ulcerative colitis the reversed Trelendenberg position with the legs raised (Lloyd-Davies) should be used, thereby allowing access to the rectum. The bladder is routinely catheterised. It is helpful to insert a proctoscope before starting to drain the rectum and deflate the bowel.

Ileostomy trephine and incision

When an ileostomy forms part of the procedure, the trephine should be made before opening the abdomen. For open colectomy this should be midline. This can be limited to 7cm or less in a thin patient. A paramedian incision is no longer appropriate. For laparoscopic colectomy a small incision is made to remove the specimen. It also allows a manual port if handassisted laparoscopy is used. On opening the abdomen, care must be taken to avoid perforation. Where adhesions have formed between the colon and the parietes, dissection should be made within the latter.

Operative steps

These are discussed below.

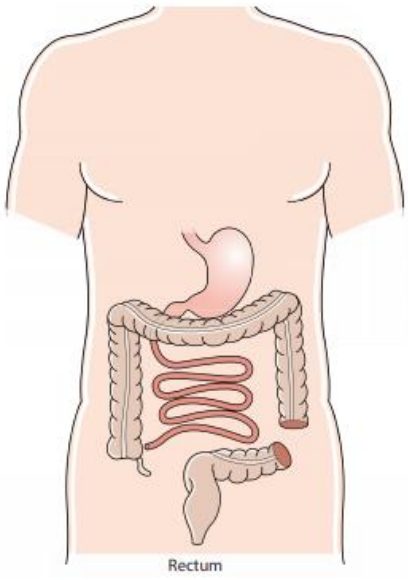
• Mobilization of the right colon. The surgeon stands on the patient's left side.

• Division of the bowel. The bowel is divided at the ileocaecal junction before proceeding further. This allows control of the right colon manually to allow safe division of the ileocolic and right colic vessels, by avoiding tension.

• Division of vessels of the transverse colon. • Mobilization of the left colon. The surgeon moves to the other side of the table to do this.

• Mobilization of the splenic flexure. The splenic flexure is often drawn down owing to shortening of the bowel due to the disease process and may be very easy to mobilize.

• Division of the sigmoid. The level of division should allow sufficient length of distal bowel to be able to exteriorize it through the anterior abdominal wall whether a mucous fistula is formed or not. Division at the level of the peritoneal reflection leaves a distal stump that is too short to be exteriorized in the uncommon event of breakdown of the distal suture line and also makes identification of the rectum at a subsequent operation difficult. A long rectosigmoid stump should therefore be aimed for unless it is necessary to remove the rectum in the case of severe bleeding.



Emergency colectomy: level of division of the sigmoid allowing adequate mobility for exteriorization.

Colectomy with ileorectal anastomosis

Indications Colectomy with ileorectal anastomosis is a well-tolerated one-stage procedure. Before the introduction of restorative proctocolectomy it was the only procedure which avoided an ileostomy being used in 10% to over 80% of

patients according to the preference of the surgeon. Since then, its incidence has fallen to below 10%.

The indications are as follows:

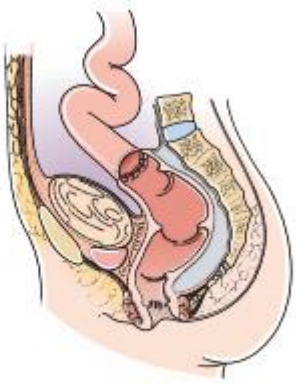
• a non- or mildly inflamed rectum with good compliance

• absence of dysplasia anywhere in the large bowel

• adequate anal sphincter

• availability of the patient for follow-up

• presence of disseminated colonic carcinoma with relative rectal sparing. The patient must also be prepared to be followed by annual rectoscopy with biopsy owing to the risk of malignancy in the rectal stump which is about 5% at 20 years. If this is not possible then the operation should not be advised.



Colectomy with ileorectal anastomosis.

Operative steps

The technique is identical to colectomy with ileostomy up to the point of removal of the specimen. The upper rectum is very accessible for manual or stapled anastomosis.

Large bowel polyps

Polyps

The term polyp is rather imprecise and in its broadest sense can be taken to mean a protuberant growth which can be either benign or malignant. As far as the colon and rectum are

concerned, however, the term is usually taken to mean a benign swelling arising from the colonic or rectal mucosa, although, as it will be seen later on, certain types of polyp may contain a malignant or invasive focus and may indeed be an essential precursor in the development of colorectal cancer. Colorectal polyps may be inflammatory, hamartomatous, metaplastic or adenomatous. These will

be dealt with in turn, but particular attention will be paid to the adenomatous variety in view of their close association with colorectal cancer.

Inflammatory polyps

These occur in ulcerative colitis, Crohn's colitis, diverticulitis, chronic dysentery and in benign lymphomatous lesions of the colon. They are sometimes referred to as pseudopolyps as they are commonly formed from an island of hypertrophied mucosa in an area of inflammation and ulceration. These polyps tend to be small, rarely exceeding 0.5mm in diameter and consist of inflamed congested mucosa with oedematous changes in the submucosa.

Hamartomatous polyps

Hamartomatous polyps may be found in two forms, as juvenile polyps and as the familial Peutz–Jeghers syndrome. Juvenile polyps are found in infants or children and are often multiple, being round or oval with a smooth surface. At the time of diagnosis most lesions are pedunculated with a transition from normal colonic mucosa to a type of glandular tissue at the junction of the stalk and the polyp. The polypoidal substance consists of vascular tissue infiltrated by inflammatory cells and contains cystic spaces maintained by mucus-secreting columnar cells. There is a familial tendency in juvenile polyposis with the majority of patients presenting before the age of 10 years. Male children predominate over female. Fortunately, they are single in 70%, and 70% occur in the rectum and distal sigmoid colon. The polyps occurring in the Peutz–Jeghers syndrome are associated with pigmented lesions (a bluish brown discoloration) on the face and on the lingual and buccal mucosa. Here the familial tendency is very strong.

The polyps are almost always multiple and are found more commonly in the small bowel than in the colon or rectum. On histological examination the basic malformation is found in the muscularis mucosae. Unlike juvenile polyposis there is a significant malignant potential and there are reports of carcinoma arising in young patients with this syndrome.

Metaplastic polyps

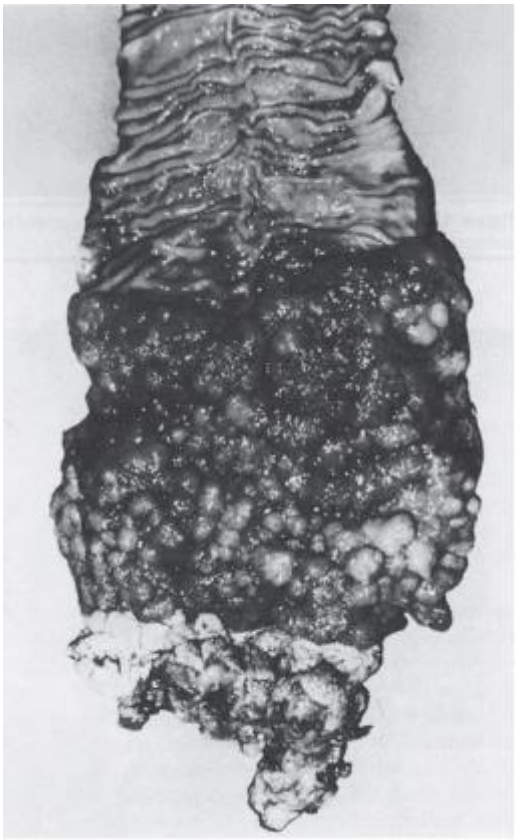
These are generally plaque-like growths which vary in size from 1 to 2mm but rarely exceed 5mm in maximum diameter. Although they are most commonly

found in the rectum, the whole of the large bowel is susceptible. There is no specific age distribution or predisposing factor. On histological examination there is lengthening of the mucosal glands with dilatation of the goblet cells and evidence of inflammatory infiltration of the lamina propria. It is not understood why these lesions arise but they are very rarely symptomatic and do not appear to be premalignant. There is however some evidence that the presence of metaplastic polyps in the rectum or distal colon may be associated with an increased risk of adenomatous polyps or even carcinoma in the more proximal colon.

Adenomatous polyps

Pathology

Adenomatous polyps are benign neoplastic growths arising from the mucosa of the intestine and although they may occur anywhere between the stomach and the rectum, they are most common in the large intestine. In the Western world they are extremely common and postmortem studies indicate that they are found in more than 30% of people over the age of 60 years. The distribution of polyps is similar to that of adenocarcinoma, i.e. commonest in the rectum and left side of the colon, rare in the transverse colon and with a slight increase in the incidence in the right side of the colon and caecum. Adenomas are highly variable in size and macroscopically may be pedunculated or sessile. Recently the concept of the flat adenoma, which can be defined as an area of adenomatous change barely discernible macroscopically has emerged, but the significance of these lesions has yet to be established. In the colon, adenomas are normally pedunculated whereas in the rectum they are commonly sessile. The villous papilloma is a sessile adenoma made up of frond-like strands which grows as a carpet on the rectal mucosa.



Extensive villous adenoma of the rectum.

Histologically, the epithelium in an adenoma can be arranged in tubular pattern consisting of closely packed glands or a villous pattern where the epithelial cells are arranged on frondlike extensions from the surface of the tumour. In practice the majority of adenomatous polyps display a mixture of tubular and villous patterns and can be described as tubulovillous. When the pathologist examines an adenoma it is important to establish whether or not there are any areas of invasion where dysplastic cells have transgressed the basement membrane into the fibrous stalk of the polyp. This will be found in about 50% of all adenomas that are over 2 cm in maximum diameter. In general, villous adenomas are more likely to undergo malignant change than tubular adenomas but this is by no means an absolute rule.

Aetiology

The aetiology of adenomatous polyps is essentially the same as that of colorectal cancer, indeed it is believed that the majority of colorectal cancers arise from pre-existing adenomatous polyps (see next section). In summary, although environmental factors (probably mainly dietary) have important implications for the formation of polyps, the genetic background is crucial. Not only are there dominantly inherited mutations which predispose to the development of polyps and cancer (see sections Familial adenomatous polyposis and Hereditary non-polyposis colorectal cancer) but there are also more subtle genetic variations which have an important impact on the predisposition to develop adenomatous polyps. This will be dealt with in detail in the section Colorectal cancer.

Clinical features

Most adenomatous polyps are asymptomatic and the diagnosis is made on routine examination. Nevertheless, both occult and frank bleeding can occur and patients may present with either rectal bleeding or anaemia. Occasionally, polyps may be extruded from the anal canal and may be misdiagnosed as prolapsing haemorrhoids. The retrograde propulsion of larger pedunculated polyps may produce abdominal pain and in extreme cases lead to the development of colocolic intussusception.

Rectal polyps may be accompanied by tenesmus and a change in bowel habit to diarrhoea. This may be the result of mucoid discharge from the surface of the polyps. This feature is particularly common with villous papillomas where spurious diarrhoea from the abundant mucus discharge leads to a failure in

health, dehydration and electrolyte disturbance. In the mucus, sodium and chloride concentrations are similar to plasma but the potassium concentrations are between three and 20 times greater. Thus in larger papillomas, hypokalaemia and metabolic acidosis may result in lethargy, muscle weakness, mental confusion and in some extreme cases renal failure. These metabolic disturbances require attention prior to any attempt at surgical treatment.

Diagnosis

The diagnosis of adenomatous polyps is made on either large bowel endoscopy or barium enema. Large bowel endoscopy (colonoscopy in particular) is more sensitive at identifying polyps but large polyps can easily be seen on a high-quality, double-contrast barium enema. In some instances, it may be difficult to distinguish among a polyp, a diverticulum and faecal material, and this gives rise to a significant false-positive rate of polyp detection using barium enema. It must also be stressed, however, that colonoscopy is not 100% sensitive either and it has been demonstrated that repeat colonoscopy can often demonstrate lesions which were missed on the previous colonoscopy and miss lesions which had already been seen. One advantage of endoscopic diagnosis of polyps is that a biopsy can be taken of the larger polyps. This is important as it is often difficult to distinguish between a large benign adenomatous polyp and a polypoid carcinoma. Colonoscopy also offers the opportunity to remove the polyp using snare diathermy .

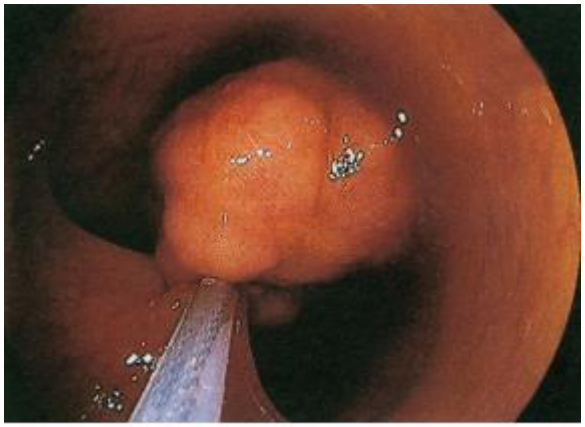
Treatment

The mainstay of treatment of adenomatous polyps is endoscopic polypectomy. Using a colonoscope, a wire loop is placed around the stalk of a pedunculated polyp; a blended current is then passed along the snare which is gradually tightened until it cuts through the stalk (Figure 30.53). The polyp should then be retrieved for histological examination. Occasionally, polypectomy may be accompanied by brisk arterial bleeding. If this occurs, the endoscopist should grasp the bleeding stalk with the snare, tighten the snare and hold in place for about 5 minutes without using diathermy current. This will usually stop the bleeding.

Another important complication of polypectomy is perforation of the colon. All patients undergoing colonoscopy with polypectomy should be warned of the risks of bleeding and perforation, as both may require emergency surgery. When a

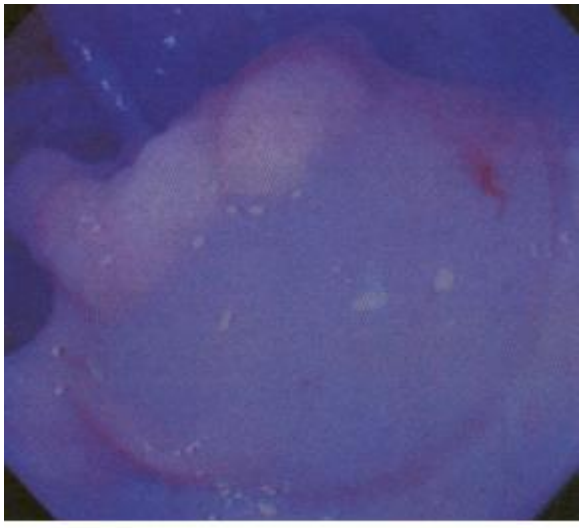
colonic polyp is sessile or has a very broad stalk, then it may be hazardous to carry out a standard polypectomy owing to the risk of perforation. In this case it is possible to elevate the polyp by injecting saline into the submucosa. This will allow much safer polypectomy. In very large polyps, it may be possible to carry out piecemeal snare excision. This is a difficult and hazardous procedure and should only be carried out by experienced endoscopists.

When a polyp has been completely excised and sent for histology, a focus of invasion will occasionally be found within the polyp making it a polyp cancer. It is generally agreed that if the polyp has been removed in one piece and the invasion does not extend to the resection margin then further surgery is not required. However, if the resection margin is involved, the patient should then be offered a colectomy. For this reason, if an endoscopist is concerned that a polyp may in fact be a cancer, it is useful to mark the site of excision using an intramural injection of India ink via the colonoscope. Having said this, many surgeons will take the view that a large polyp in the colon is highly likely to be malignant, even in the absence of confirmatory biopsies and will advise the patient to go straight to colectomy.



Snare excision of a polyp.

Although these endoscopic approaches are suitable for the majority of colonic polyps, rectal polyps pose a different problem. Occasionally, a large pedunculated prolapsing rectal polyp can be pulled out through the anal canal and the stalk simply ligated and divided. However, the majority of rectal polyps are sessile and require a different approach. Traditionally a low sessile rectal polyp was treated by submucosal saline injection to lift it away from the muscle wall and then transanal excision using Park's anal retractors to gain access.



Elevation of polyp after submucosal saline injection.

For polyps which are higher in the rectum, however, this is often not feasible, and for this reason TEMS was developed. This employs a sophisticated operating sigmoidoscope with a binocular optical system, which, by means of continuous

insufflation and suction, offers an excellent operating environment to remove such polyps. When these polyps are situated posteriorly, it is safe to remove a fullthickness disc of rectal wall and this is a sensible precaution as a proportion of such polyps will harbour invasive malignancy. However, anteriorly above about 10 cm this is hazardous as it may lead to perforation into the peritoneal cavity and a submucosal technique must be employed. An alternative procedure is the trans-sphincteric approach described by Yorke Mason, which involves formal division of the anal sphincter mechanism in order to gain access to the rectum posteriorly. Owing to the currently available sophisticated techniques for transanal excision, however, this approach is seldom used nowadays. Occasionally, a patient will have such an extensive carpet of adenoma throughout the rectum that a transanal approach is not feasible. In this case a total proctectomy (i.e. an anterior resection with a mucosectomy down to the dentate line) has to be carried out. Continuity can then be restored by means of a sutured coloanal anastomosis.

Haemorrhoids

The term haemorrhoids or 'piles' means different things to different people and many patients will use these words to describe a wide variety of anorectal conditions. To the surgeon, however, it refers to abnormalities of the vascular cushions of the anus.

Pathology and aetiology

The anal cushions consist of three spaces filled by arteriovenous communications supported by a fibrous matrix and smooth muscle lying within the anal canal. This allows the anal lining to expand during defaecation but yet to form a complete seal when the anal canal is closed. The arterial supply for these cushions comes from the superior, middle and inferior rectal arteries. Haemorrhoids are thought to result from degeneration of the smooth muscle and fibroelastic tissue that supports the cushions, allowing them to prolapse into the anal canal. However, the underlying reasons for this degeneration are not clear and although constipation and straining at stool have been implicated, the evidence for this is patchy. There is a family history in about 50% of cases and it is therefore possible that a genetic predisposition exists.

Clinical features

The most common symptom is bleeding at defaecation. Commonly this is bright red and follows immediately after defaecation. Typically this is painless but may be quite profuse and frightening for the patient. Other symptoms include perianal swelling, pruritus and minor soiling. Pain from haemorrhoids is associated with complications. Clinically, haemorrhoids can be classified into four groups:

1 internal haemorrhoids presenting with bleeding alone (first degree)

2 haemorrhoids which prolapse on defaecation but reduce spontaneously (second degree)

3 haemorrhoids which prolapse and require manual reduction (third degree)

4 irreducibly prolapsed haemorrhoids (fourth degree).

Thus, on examination the external appearances will depend on the degree of prolapse and the anal canal may in fact look normal. Skin tags around the anal orifice are common and mucosa may be seen to prolapse . Digital rectal examination is generally normal. The main diagnostic test is proctoscopy, which gives a good view of the internal anal cushions. It is also essential to examine the rectum with a rigid or flexible sigmoidoscope at least to exclude other lesions.

Investigations

As far as making the diagnosis of haemorrhoids is concerned, investigations other than those mentioned above are unnecessary. However, if there is any doubt about the source of bleeding then a full colonic examination in the form of a flexible sigmoidoscopy and barium enema or a total colonoscopy should be carried out. This would be indicated when there are other symptoms such as change of bowel habit or lower abdominal pain or if the patient is in the high-risk age range for colorectal cancer (i.e. over 50 years of age).

Complications

The complications of haemorrhoids include thrombosis, massive haemorrhage and faecal incontinence.

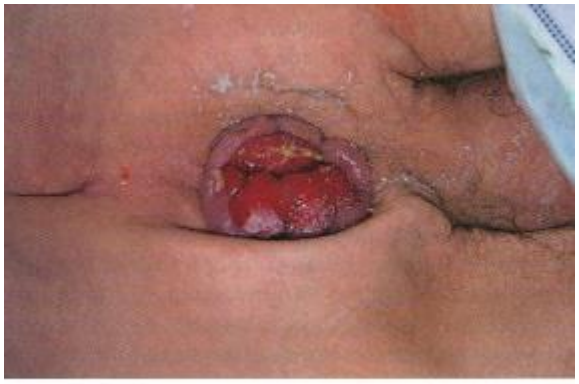
Thrombosis

When haemorrhoids become irreducible, intravascular thrombosis and oedema may ensue owing to strangulation of the blood supply. This gives rise to severe

pain and on examination swollen bluish external haemorrhoids will be seen. Occasionally these may become gangrenous.

Massive bleeding

Very occasionally patients may bleed so profusely from haemorrhoids that they become shocked and require resuscitation with blood transfusion. More commonly, although still relatively unusually, a patient may develop iron-deficiency anaemia from regular bleeding episodes. Attributing iron-deficiency anaemia to haemorrhoids should generally only take place after full investigation to exclude other sources.



Prolapsing haemorrhoids.

Incontinence

Pruritus and minor soiling are relatively common owing to leakage of mucus and liquid faeces from the rectum. This is thought to be due to a poor sealing mechanism owing to displacement of the anal cushions. This may be compounded by a certain amount of sensory impairment in the anal canal. Under most circumstances these symptoms are not particularly disabling but occasionally patients may find them extremely troublesome. If this is the case, the patient should be fully investigated for incontinence as there may be some other underlying cause.

Treatment

The majority of patients with symptomatic haemorrhoids do not need active intervention. Often reassurance after exclusion of serious disease is sufficient. If the patient is finding that constipation or straining is an important feature then bulk laxatives may be of value. Topical ointments may help by providing lubrication but their value is unclear. Diltiazem cream 2% has benefits for reduction of haemarrhoids. It has to be applied twice a day for 6 weeks. If patients have a benefit, they continue for another 6 weeks. Active intervention for haemorrhoids can be divided into two broad areas: (1) outpatient procedures and (2) surgery.

Outpatient procedures

Injection sclerotherapy

For many years injection of sclerosant (most commonly 5% phenol in almond or arachis oil) has been used for the treatment of haemorrhoids. This is injected using a long needle via a proctoscope and 3–5mL of sclerosant should be injected into the submucosa well above the dentate line at each haemorrhoidal site. The underlying aim is to produce a fibrous reaction within the anal cushion to reduce the degree of prolapse. Care must be taken not to inject too superficially, as this will lead to ulceration, or too deeply, as this will be ineffective. If the injection is too deep it is also possible to damage the prostate or the seminal vesicles and perirectal sepsis has been reported.

Rubber band ligation

An alternative to injection sclerotherapy is rubber band ligation and indeed in randomized trials it has been shown to be more effective. This involves placing tight rubber bands around the prolapsing cushion at least 1.5 cm above the dentate line. There are various devices for achieving this but most are used via a proctoscope. Perhaps the simplest device is a suction tube to which the band is mounted . The mucosa is then sucked into the tube and a special triggering device is used to push the band off the end of the tube. More than one band can be inserted at one time although it may be necessary to repeat the procedure. The surgeon should be very careful to apply the band above the dentate line as failure to do this will lead to immediate severe pain. If this happens it is necessary to remove the band by cutting onto it with the tip of a scalpel blade, and this may sometimes necessitate a general anaesthetic. After the procedure patients should be warned to expect some bleeding at between 5 and 10 days when the necrotic cushion separates. They may also expect to have some aching, which may be relieved by warm baths and non-steroidal anti-inflammatory drugs.

Other outpatient techniques

Bipolar coagulation, infrared photocoagulation, laser photocoagulation and cryotherapy have all been used but none has gained popularity. The true efficacy of outpatient procedures is not clear. There have been a number of comparative randomized studies that tend to favour rubber band ligation, but unfortunately stratification for severity of disease and the use of no-treatment controls has been lacking. There is little doubt that these procedures have a strong placebo

effect and further research is required to establish their precise role in the management of haemorrhoids.

Surgery

In patients who have had failed to benefit from outpatient treatment, the surgical approach becomes necessary. In addition there is a feeling among many colorectal surgeons that patients with severely prolapsing haemorrhoids or in whom bleeding is a major concern should have primary surgical intervention. Currently, there are two widely used surgical approaches: haemorrhoidectomy and stapled anopexy. Haemorrhoidal artery ligation or dearterialization is a more recent approach that is gaining popularity.

Haemorrhoidectomy

There are many different varieties of haemorrhoidectomy but the basic principle is to excise the prolapsing anal cushions while maintaining mucocutaneous continuity between the areas of excision. Each haemorrhoid is grasped close to the mucocutaneous junction in turn and excised in the plane immediately outside the internal sphincter using diathermy. Associated skin tags are included in the excision. If this is done carefully the pedicle will merely consist of a thin strip of mucosa and can be transected directly with diathermy, although some surgeons still prefer to ligate the pedicle. Variations include operating position and whether the mucosa is closed by suture or left open. Lithotomy position is most common in UK practice with prone or left lateral most common in the USA.

Traditionally, after haemorrhoidectomy patients were kept in hospital until their bowels had moved but with careful preparation and community support haemorrhoidectomy can be carried out as a day case. It is even possible to perform the procedure under regional anaesthesia. Steps to diminish pain postoperatively and particularly at the time of defecation include the use of metronidazole, laxative regimens and the use of topical glyceryl trinitrate (GTN) or diltiazem. There is some support for mucosal closure, as in the Ferguson haemorrhoidectomy, compared with the open approach, as in the Milligan–Morgan haemorrhoidectomy, but evidence is not strong in any of these areas.

Stapled anopexy

The term stapled haemorrhoidectomy is inaccurate since the intention is not to excise the prolapsed haemorrhoidal cushions but relocate and fix them; the term

stapled anopexy is more appropriate, although less popularly applied. The procedure has attracted a great deal of interest in the last few years. The principle of this operation is to carry out excision of a circumferential strip of mucosa above the dentate line and to simultaneously close the defect. This pulls the mucosa and therefore the anal cushions back up into their normal position, thus restoring the anatomy of the anal canal. This is done using a specially designed proctoscope and circular end-to-end anastomosing stapler.

A purse string is inserted in the rectal mucosa 3cm above the dentate line, the stapler is inserted and the purse string tightened around the centre ro . The stapler is then fired, simultaneously excising the mucosa and stapling the two cut ends together. After excising a cam 'doughnut' a circumferential staple line should be left 1cm above the dentate line. If the stapling is performed too proximally it will be ineffective in elevating the anal cushions, and if performed too distally will risk interference with the sphincter complex. Immediately after the procedure the staple line must be inspected for bleeding points, which can be oversewn. In theory, confining the surgery to the less sensate area above the mucocutaneous junction should be less painful and randomized controlled trials have consistently shown an advantage to the stapled technique in terms of pain. Haemorrhoidectomy has an unfortunate reputation for pain among the general public with many postponing or avoiding intervention. Although certainly not painless, stapled haemorrhoidectomy does appear to increase patient acceptability and may help facilitate day-case surgery. In expert hands the technique appears at least as effective as excisional haemorrhoidectomy but there may be an increased risk of recurrent prolapse over time. It has not been possible to demonstrate a functional advantage despite a more restorative approach. Although large numbers of cases have now been performed uneventfully, care is required as perforation and complete closure of the rectum have occurred (see also Haemorrhoids and incontinence).

Haemorrhoid artery ligation/dearterialization

The latest approach in the quest for an effective but painless treatment for haemorrhoids involves identifying and ligating the arterial inflow to the haemorrhoidal pedicles. This is achieved by using specially designed proctoscopes

incorporating a Doppler device and facilitating guided haemorrhoidal artery suture and ligation. The latest variation allows expansion of the technique to treat haemorrhoidal prolapse by suture fixation (rectoanal repair).

The technique is disseminating rapidly with several supportive publications. It appears to be well tolerated and have minimal adverse consequences so far.

Treating complications of haemorrhoids

The patient with strangulated thrombosed haemorrhoids usually requires hospitalization for adequate analgesia and bed rest. Cold compresses applied directly to the haemorrhoids are also beneficial. Surgeons are divided as to whether or not early haemorrhoidectomy should be carried out in these patients and often an individual decision has to be made on the basis of the severity and duration of the symptoms. Those against intervention argue that following an episode of strangulation haemorrhoid symptoms often resolve spontaneously. Occasional cases of portal pyaemia have been reported.

A case of prolapsed thrombosed haemorrhoids need to be distinguished from that of a thrombosed external haemorrhoid. This is another misnomer as the latter condition is simply a haematoma forming in relation to the external haemorrhoidal plexus and quite separate from the anal cushions. Incision and evacuation of the clot will rapidly relieve symptoms.

Massive bleeding usually requires haemorrhoidectomy and should be distinguished from the occasional case of variceal bleeding secondary to portosystemic shunting at the anorectal junction. Minor degrees of incontinence usually respond well to carefully performed haemorrhoid surgery.

Anal fissure

An anal fissure is a linear ulcer that occurs in the anal canal just distal to the dentate line. This may be caused by Crohn's disease or trauma but most commonly it is a primary condition. It affects both men and women and the highest incidence is in the third and fourth decades of life. It may occur soon after pregnancy and vaginal delivery.

Aetiology and pathology

The initiating factors in anal fissure are unclear, although minor anal trauma caused by passage of a constipated stool has been suggested. The main

underlying pathology, however, appears to be a high resting anal pressure caused by increased internal sphincter tone. The blood supply to the anal canal has to pass through the internal sphincter and therefore spasm of this muscle reduces the blood flow and the oxygen tension in the skin of the anal canal. Interestingly, the fissures tend to occur at the watershed of the blood supply, i.e. the anterior and posterior midline in women and the posterior midline in men.

Clinical features

The typical clinical features are of pain on defecation associated with bright red bleeding. This may be associated with pruritus ani and discharge of mucus. On examination there is usually a skin tag overlying the fissure and the fissure itself can be seen by everting the anal canal using lateral traction (Figure 31.32). This will reveal a sharply defined ulcer and it may be possible to see the lower fibres of the internal sphincter at its base. Digital rectal examination or proctoscopy should not be attempted in the conscious patient as this will cause considerable discomfort.

Diagnosis

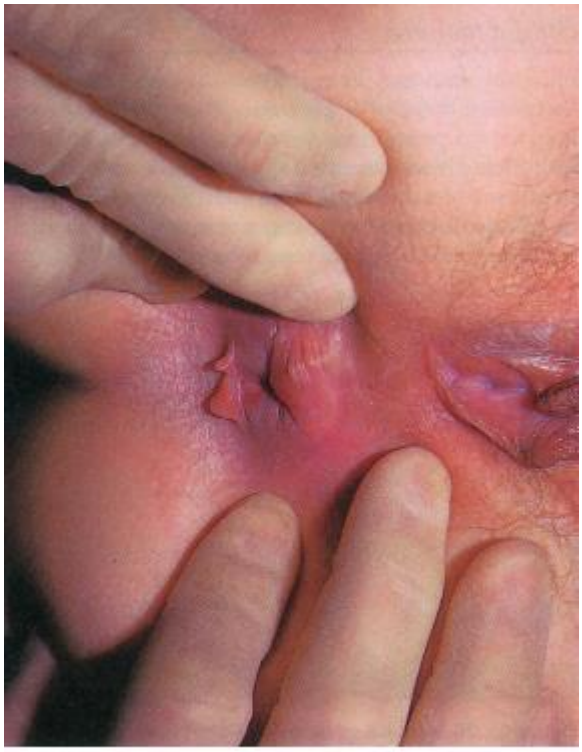
In terms of making a diagnosis this is done clinically, but it is important to exclude other conditions such as Crohn's disease or malignancy. This can be established by examination under anaesthesia and biopsy when appropriate.

Treatment

The underlying principle of treating anal fissures is to reduce the internal anal sphincter tone. In patients with minimal symptoms this may be achieved by topical application of a local anaesthetic and bulk laxatives. In patients with more severe symptoms, however, the use of 0.2% GT) cream applied two or three times a day can produce healing of fissures in about 50% of cases. Topical diltiazem has a similar effect but may be better tolerated. These agents probably act by both relaxing internal anal sphincter and improving the blood flow owing to their vasoactive properties. The healing takes about 6–12 weeks. For the patient unable or unwilling to use topical application oral nifedipine can have a similar effect.

If these approaches fail then a trial of temporary paralysis of the internal anal sphincter by botulinum toxin type A injection is worth trying and has the advantage that the effects will wear off without leaving any permanent sphincter

compromise. The dosage and site of injection (intersphincteric versus intramuscular) is debated as is the exact mode of action of botulinum toxin in this situation. Few adverse effects have been reported and this approach may reasonably be positioned between topical treatments and surgical intervention. If this does not work the first time, it can be repeated after 6 weeks.



Anal fissure.

If medical treatment and botulinum toxin injection fail then a surgical approach becomes necessary. Historically, forced anal dilatation (Lord procedure) was performed and, although effective, this was associated with an unacceptable level of incontinence. The surgical treatment of choice is now a lateral sphincterotomy, which involves dividing the internal sphincter at one point on the lateral wall of the anal canal up to the level of the dentate line. This is achieved by inserting an anal retractor so that the internal sphincter is gently stretched and easily palpable. A small incision is then made on the lateral aspect of the anal canal just below the internal sphincter. Using scissors the intersphincteric plane is developed, as is the plane between the anal skin and the internal sphincter. The scissors are then used to divide the sphincter and bleeding is controlled by firm finger pressure. Lateral sphincterotomy is said to be successful in about 95% of cases, but patients should be warned that it can be associated with minor degrees of incontinence to flatus or mucus. Adjustments to the surgical technique to diminish the incidence of incontinence include never extending the sphincter incision above the top of the fissure or beyond the dentate line.

An alternative approach is required in refractory cases and when there is pre-existing sphincter compromise. These are frequently cases in which sphincter pressure is low as opposed to the ultra-high pressures usually noted in anal fissure. These cases merit full investigation by anorectal physiology and anal ultrasound. Ultrasound may reveal a fully intact internal anal sphincter in cases of failed lateral sphincterotomy and suggest that the procedure was inadequate and

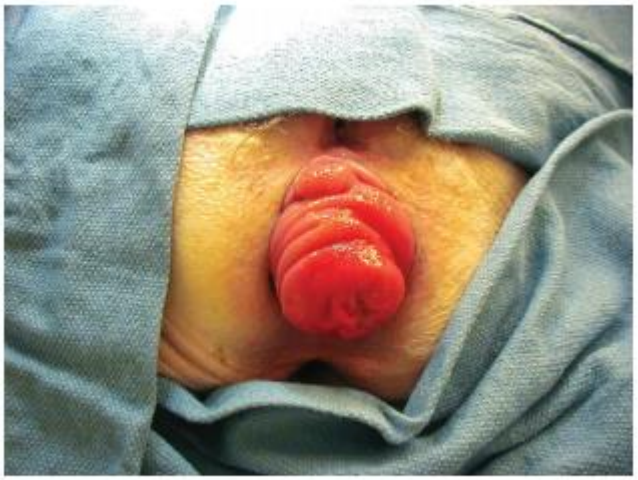
may be repeated. Otherwise, a mucosal advancement flap can be used with a fissurectomy or the 'house' advancement flap, which aims to cover the exposed internal anal sphincter fibres by advancing skin from the anal verge upwards to cover the excised fissure bed.

Rectal prolapsed

The definition of rectal prolapse is the protrusion of the full thickness of the rectal wall through the anus. This protrusion differs from mucosal prolapse and internal intussusception. In cases of mucosal prolapse, only the inner mucosal layer protrudes through the anus, leaving the muscular layers behind. In rectoanal intussusception the prolapsed tissue may be partial or full thickness, but remains confined to the rectal lumen. The type of prolapse will help direct the appropriate therapeutic option.

The true incidence of rectal prolapse is unclear, although it is more common towards the seventh decade of life. The majority of these patients are elderly females. Patients with rectal prolapse often complain of a mass protruding from the anus, initially with straining then progressing to exteriorization with any increase in abdominal pressure and finally even at rest or simple movements. Chronic prolapse can result in an inflamed and ulcerated mucosa and bleeding. Faecal incontinence is a significant feature of full - thickness prolapse, which may be a consequence of sphincteric disruption or pudendal stretching.

The clinical assessment of rectal prolapse often requires reproduction of the prolapse. The patient may need to sit on a toilet and strain or use enemas to reproduce the event in order to fully evaluate the extent of prolapse. Full-thickness prolapse presents with concentric folds and double rectal walls on palpation, while mucosal prolapse is visualized as radial folds that do not protrude more than 3–5cm.



Full-thickness rectal prolapse. Note the concentric folds and palpable double wall of rectum.

Surgical management of rectal prolapse

Rectal prolapse surgery can be performed on patients in whom conservative measures have failed, and prolapse is causing significant reduction in quality of life. Surgery can

be performed via the perineal route, which is less metabolically challenging, and suitable for patients who are more frail, or the abdominal route, which may be laparoscopic or open, and suitable for patients who are more physically fit. Transperineal operations include perineal rectosigmoidectomy (Altemeier's procedure), which is a full-thickness excision and reanastomosis of the rectosigmoid, and Delorme's procedure, which is a mucosal excision and subsequent muscular plication. Neither procedure involves fixation of the rectum to a bony prominence. Both procedures are not technically demanding, and can be repeated as necessary. However, perineal procedures generally carry a recurrence rate in the region of up to 16% for perineal rectosigmoidectomy and 40% for Delorme's procedure at 5 years. Perineal procedures carry the advantage of having lower morbidity than abdominal procedures, with the absence of an abdominal incision, less adhesion formation, no intraabdominal anastomosis and a relatively short hospitalization.

Abdominal operations include anterior resection of rectum and dorsal and ventral mesh rectopexy, plus or minus anterior resection. The fixation of rectum to sacrum may provide better results in treating rectal prolapse, and presents the lowest recurrence rate when combined with sigmoid resection (2–10%). Dorsal rectopexy frequently results in chronic constipation because of the necessary mesorectal dissection and damage to the hypogastric plexi of nerves. Ventral rectopexy has recently become popular, as it is associated with a significantly lower incidence of constipation. There have been reports of patients with ventral rectopexies requiring a posterior STARR procedure to address the angular change in the resultant rectum. The longevity of the rectopexy procedure is significant, with fewer than 5% recurrences over 5 years. The management of recurrent rectal prolapse depends on the previous procedure. Abdominal approaches should be avoided if the patient has undergone previous perineal rectosigmoidectomy, especially if sigmoid resection is contemplated, because of the potential ischaemia and necrosis of the intervening segment between anastomoses. A repeat rectosigmoidectomy can be safely performed in this patient group.

Perianal sepsis and fistula in ano

Perianal sepsis and fistula formation may be associated with a number of disease processes including Crohn's disease, malignancy, tuberculosis, pilonidal sinus and trauma. In the majority of patients, however, the condition is idiopathic.

Pathology and aetiology

Central to current thinking regarding perianal sepsis and fistula formation are the anal glands. These glands are situated in the intersphincteric space and open into the anal canal at the dentate line via a duct that transverses the internal sphincter. The function of these glands is not clear but as they secrete mucin they may have a lubricant function. It is thought these glands may become infected if the duct becomes blocked and, when this occurs, pus accumulates within the gland. The pus may then track superiorly, inferiorly, laterally or circumferentially.

Most commonly, the pus will pass downwards in the intersphincteric plane to form a perianal abscess. It may also find its way through the external sphincter into the ischiorectal fossa and thus form an ischiorectal abscess. More rarely the pus may tract up in the intersphincteric plane and form an intersphincteric abscess or discharge down into the ischiorectal fossa through the levator ani muscles . When a perianal or ischiorectal abscess discharges through the skin, either spontaneously or as a result of surgical intervention, it may resolve completely. However, if the duct between the gland and the dentate line remains patent and becomes infected, the patient may then be left with a fistulous communication between the dentate line and the skin. According to the mode of spread the fistulous tracts can be classified in the following way:

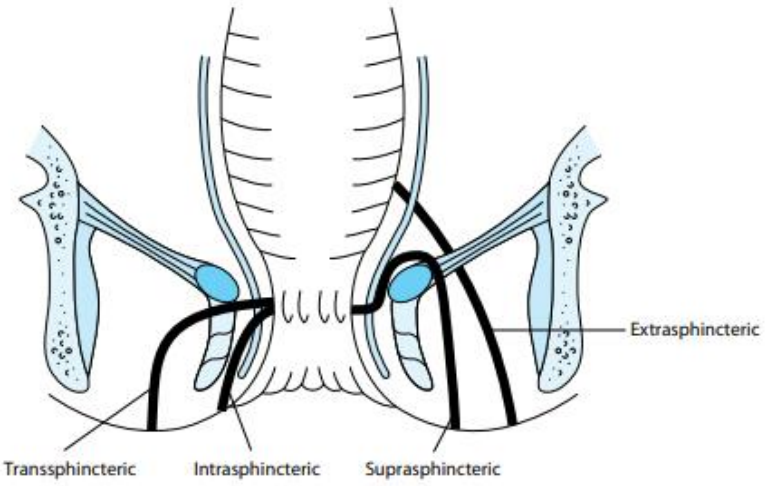
• intersphincteric

• trans-sphincteric

• suprasphincteric

• extrasphincteric.

Intersphincteric fistulas make up about 50% of all fistulas and usually consist of a straightforward tract between the dentate line and the skin incorporating part of the internal sphincter. However, some of these can have a high intersphincteric extension and even a high



Anorectal fistulas.

opening into the rectum.

Trans-sphincteric fistulas account for about 30% and consist of a tract passing through the external sphincter. This may be low or high and occasionally may be associated with a blind high tract in the ischiorectal fossa which may even penetrate the levator ani muscles.

Suprasphincteric fistulas run above the puborectalis muscle and then descend down through the levator ani muscles into the ischiorectal fossa.

Extrasphincteric fistulas bypass the sphincter complex completely and extend from the lower rectum through the levator ani muscles and into the ischiorectal fossa.

This classification is important as it has implications for treatment.

Clinical features

The patient with a perianal sepsis usually presents with severe perianal pain, fever and malaise. The abscess may discharge spontaneously, but because of the severity of symptoms patients often present at hospital before this has occurred. On examination a perianal abscess is usually very obvious as a tender red swelling at the anal margin. The ischiorectal abscess, however, may be quite deep seated and more difficult to detect on clinical examination, although pressure on the ischiorectal fossa will usually give rise to severe pain. In an advanced ischiorectal abscess a large area of tender induration will be seen. Occasionally, a patient may present with an intersphincteric abscess that has not pointed at the anal canal and this can be very difficult to detect clinically. Thus, in the patient with a severe acute anal or perianal pain an examination under anaesthetic should be carried out. If there is an intersphincteric abscess this will be felt as induration through the rectal wall.

A fistula may present after discharge or incision of an abscess, usually after all the inflammation and induration has settled down in about 50% of all abscesses. Alternatively, there may be no history of abscess formation and the fistula may appear to arise de novo. The main symptoms are of discharge and pruritus, although occasionally a patient may notice the passage of flatus through a fistula track. There may also be a history of recurring episodes of pain relieved by discharge from the fistula.

On examination a punctate opening (or openings) can be seen, usually close to the anal verge, although sometimes a few centimetres away. Pus or serosanguinous discharge may been seen exuding from the opening particularly if digital pressure is applied over the fistula. Careful firm palpation between the opening and the anal canal will often reveal the fistulous track as a subcutaneous 'cord'. Goodsall's rule (Figure 31.26) is useful when estimating the course of a fistula; this states that, if a fistulous opening is posterior to an imaginary line drawn transversely through the middle of the anus, the track will curve round so that it opens into the dentate line in the posterior midline, whereas, if the opening is anterior, the track will be radial (i.e. it follows a straight line from the opening to the dentate line). The main exception to this rule is the anterior opening, which is ≥3 cm from the anal verge, as this may be 'horseshoeing' round from the posterior midline.

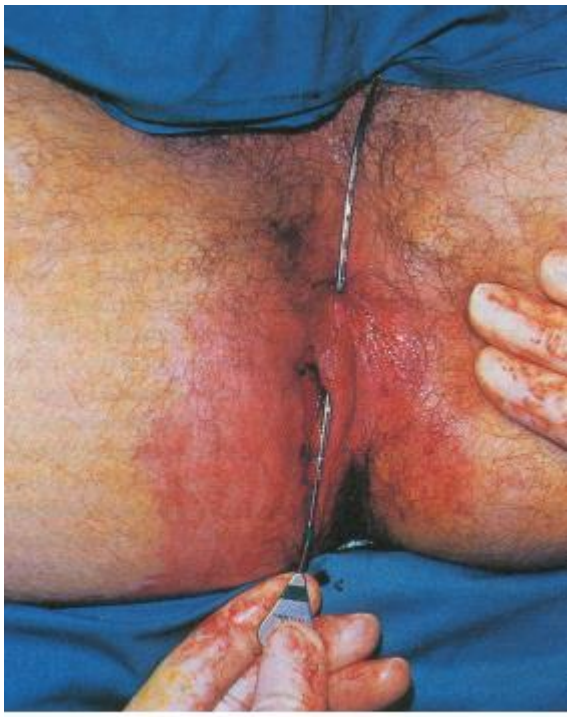
Diagnosis

The diagnosis of an abscess is usually made clinically, and its exact position relative to the sphincter complex is made at operation. Likewise, the course of a fistula is usually established by examining the patient under general anaesthesia using specially designed fistula probes. The most widely used probes are those designed by Lockhart-Mummery; these are available in a variety of configurations, and the most useful are slightly curved . The flat handle allows for precise manoeuvring of the probe, and the groove is useful for laying the fistula open (see below). Under normal circumstances it is relatively easy to pass the probe from the external opening along the tract to the internal opening at the dentate line . In doing this it is important not to use too much force, as it is possible to create a false tract in so doing. If it proves to be impossible to find the internal opening using this technique a valuable manoeuvre is to instil a very small amount of hydrogen peroxide into the external opening by means of a fine cannula. With an Eisenhammer retractor in place within the anal canal it is then usually possible to see bubbles appearing at the site of the internal opening. This will guide further probing. In the process of probing a fistula it is important to look for extensions to the main tract. Indeed, if the probe cannot easily pass along the tract it is probably falling into a blind extension bypassing the main tract. Although examination under anaesthetic with probing is the most useful diagnostic approach, some forms of imaging may also be of value. Fistulography has been used in the past. However, its accuracy is sparse. Currently, fistulography has

been superseded by endosonography and MRI . It appears to be particularly useful in complex fistulas with secondary extensions and abscesses and, although it is perhaps no more accurate than an examination under anaesthetic by an experienced coloproctologist, a high-quality MRI of the sphincter complex may be useful as a 'road map' to guide the surgeon. Transanal ultrasound is undertaken using a 10mHz mechanically rotated transducer to give a 360o axial image.

Treatment

The initial treatment of a perianal or ischiorectal abscess which is pointing onto the skin is fairly straightforward. The abscess cavity should be incised and drained and an opening in the skin made that is large enough to allow continued drainage of the abscess. The cavity should not be packed tightly as this makes it uncomfortable for the patient and actually impedes drainage of the abscess cavity. A large cavity may require dressing changes in hospital until it can be managed by a community nurse. Alternatively, it is possible to put a counterincision in a large abscess and leave a drain. The patient now has to irrigate the wound in the shower twice a day but can be managed as an outpatient. The patient should always be followed up in the outpatient department to look for the development of a fistula when the cavity itself has healed. One of the complications of incision and drainage of a perianal or ischiorectal abscess is the iatrogenic production of a high fistula by overenthusiastic curetting of the abscess cavity or injudicious probing for a fistula. This must be avoided at all costs.



Probe in a fistula *in ano*.

The best treatment for fistula in ano is undoubtedly a fistulotomy, laying open of the entire tract, curetting out of the granulation tissue and leaving the wound to granulate. This is done most easily by cutting down onto the groove on the concave aspect of the fistula probe, and then using a small Volkmann's spoon to curette. Some surgeons will marsupialize the tract by suturing the divided wound edge to the edges of the fibrous tract and this is said to result in faster healing. A

fistulotomy is generally safe with a simple intersphincteric fistula as the only muscle that will be divided will be the lower part of the internal sphincter. If, however, the fistula is transsphincteric then a fistulotomy creates a risk of incontinence. A fistulotomy of a suprasphincteric or extrasphincteric fistula will inevitably result in complete incontinence.

In the case of a low trans-sphincteric fistula that only involves a small part of the lower external sphincter, a fistulotomy is relatively safe. However, at the time of the initial examination it is important to assess exactly how much of the external sphincter is involved. It is therefore safer to establish drainage of the fistula by inserting a seton through the fistula tract. This can simply be a length of suture material or a vascular sling loosely tied. The seton controls the situation by ensuring ongoing drainage through the tract and preventing recurrent abscess formation. When the patient is awake after the procedure it is then easier to assess the anal sphincter and, if the surgeon is confident that only a small part of the external sphincter is involved, the patient can go back for laying open of the fistula.

If, however, a significant part of the external sphincter muscle is involved or if the fistula proves to be suprasphincteric, then a more conservative approach must be taken. The most widely used approach is to ensure complete drainage of all the sepsis and to leave the seton in place for several weeks.

When all the sepsis and inflammation has resolved the seton can then be removed and this will result in healing in about 50% of cases. If this fails, the seton must be reinserted and a careful discussion with the patient must take place. If fistulotomy will leave some external sphincter muscle intact then this is a feasible option as long as the patient is aware that a certain degree of incontinence may result. The situation is always more difficult when some pre-existing sphincter compromise is at play and for women in general, in whom a shorter sphincter and the potential adverse effect of childbirth on the continent mechanism require to be considered. When a seton is left in place for a longer time, it may work its way towards the surface. In this way the high fistula becomes a low fistula. The seton gradually cuts its way through the fistula tract leaving fibrosis behind it. Although the sphincter is divided more slowly than in fistulotomy the effect on continence is not very much different with some degree of incontinence in about 60% of cases.

An alternative procedure for the trans-sphincteric fistula is fistulectomy and advancement flap repair. Here the fistula tract is excised or cored out by following it up from the external orifice and dissecting through the external sphincter muscle. A flap of mucosa and internal sphincter is then raised above the internal opening and sutured down over it. This is successful in some cases, but the overall rate of success is difficult to estimate. Repeating the procedure can increase the success rate, but repeated interventions with scarring and fibrosis will in themselves impair anal canal function. A degree of laxity in the mucosa is helpful in raising a flap and may account for the lower success rates in males and in posterior versus anterior fistulas. With a high trans-sphincteric fistula or a suprasphincteric fistula incontinence is inevitable with fistulotomy, and one approach is to give the patient a temporary colostomy, lay open the fistula and then carry out a sphincter repair after healing has taken place. Alternatively the patient may opt to live with a longterm loose seton in place. This approach is the safest in a variety of situations, including multiple fistulas and Crohn's disease.

A variety of approaches have been taken to try and induce healing of anal fistulas without resorting to fistulotomy or fistulectomy. In general, the aim has been to fill the tract with biological material in the hope that it will be slowly replaced by the body's own tissue, leaving a healed fistula tract. Injection of fibrin glue was used quite extensively. Initial results seemed promising but long-term results have been poor with high recurrence rates. The liquid consistency of fibrin glue is possibly not ideal for the purpose of closing anorectal fistulas, because the glue is easily extruded from the fistula tract by increased intraluminal pressure. A plug fabricated from porcine collagen, which is claimed to stimulate tissue remodelling leading to closure of the fistulous tract, is currently being used. The tract is cleaned out, the plug is pulled into position and then sutured in place with the mucosa closed over the top (Figure 31.30). A multicentre randomized controlled trial is now taking place in the UK comparing this plug with a cutting seton approach (Fistula In Ano Trial – FIAT).

The extrasphincteric complex fistula, which is usually secondary to Crohn's disease or trauma, is particularly difficult to deal with and, particularly in Crohn's disease, it may be better to avoid surgical interference. In such patients, a longterm seton may be the answer and some patients may require a defunctioning colostomy. After resolution of the sepsis, laying open of the fistula

and subsequent sphincter repair may be possible, but the ultimate results can be less than ideal.

Current interest surrounds the LIFT (ligation of intersphincteric fistula tract) procedure. This involves dissection upwards in the intersphincteric space to the level of the fistula which is then ligated and divided. The external fistula is curetted. The technique is attractive as it has minimal impact on the sphincter muscle. A variation is to insert a small sheet of biomaterial between the divided ends of sphincter (BioLIFT). Early results are encouraging but wider experience and longterm follow-up are required to fully assess its role.

Colorectal cancer

Aetiology and associated risk factors The colorectal cancer risk is influenced by both environmental and genetic factors, with the lifetime risk up to 5%. There are a number of studies investigating the aetiology of colorectal cancer, but the exact cause is still not fully clear. There are a number of environmental and genetic factors that have been strongly associated with the disease and therefore are considered to play a role in the carcinogenesis. The exact mechanism is not fully understood yet.

Environmental factors

Diet is one of the major environmental risk factors that have been associated with colorectal cancer. It has been suggested that the Western diet is a strong risk factor (WHO Cancer report). The consumption of red meat and poultry was shown to be associated with colorectal cancer and might be a result of the exposure to carcinogens that can be created when cooking. The role of red meat was also assessed by three meta-analyses that confirmed the above statement. One study demonstrated a link to colorectal cancer when the meat was well cooked. This study suggested that carcinogens formed on the surface of wellcooked red meat might be responsible for this link, particularly for rectal cancer. Myoglobin has also been suggested to play a part in the carcinogenesis. There are a vast number of publications debating the value and role of dietary fibre in colorectal cancer. There was initially a negative association of dietary fibre intake to colorectal cancer. The majority of the published studies showed that lower intake of vegetables and therefore fibre was associated with a lower risk of colon cancer. However, this claim was soon challenged by two meta-analyses that confirmed the benefits of a high intake of vegetables in terms of reducing the risk

of colorectal cancer. Trock et al. demonstrated a reduction of risk by 40%, and Howe et al. by 50%. More recent prospective studies have been performed but did not demonstrate significant association of high-fibre intake as protective factors for cancer.

Micronutrients and vitamins have also been investigated. A study investigating the level of folate in serum showed that low serum levels were associated with higher rates of colorectal cancer. One meta-analysis showed that the risk was higher for colorectal cancer when the serum levels of vitamin D were reduced. Calcium and aspirin have been shown to reduce the risk for colorectal adenoma formation, thus reducing the risk of colorectal cancer.

Obesity is associated with a higher risk of colorectal cancer. Obesity is shown to increase the risk by around 25% in overweight males and 50% in obese males. The same study showed a non-statistically significant risk of 9% for obese females. Larger waist size was also linked to higher risk for both males and females. Physical activity has been shown to play an important role. High levels of physical activity have been shown to substantially reduce the risk, especially in males by almost 25%. Physical inactivity has been suggested to be responsible for up to 14% of the new colon cancer cases in the Western countries.

Familial and genetic factors

Family history of colorectal cancer is known to increase the risk of colorectal cancer. The risk doubles when there is a history of a first-degree family member diagnosed with colorectal cancer. The risk is even higher when the relatives were diagnosed with the disease at a younger age (younger than 50) and for patients with more than one relative with the disease.

FAP and the hereditary non-polyposis colorectal cancer (HNPCC) have been linked with about 5% of the diagnosed colorectal cancers. The presence of early multiple bowel adenomas is the distinctive feature of FAP, which is responsible for about 1% of colorectal cancers. The presence of FAP increases the risk of developing colorectal cancer to 100% by the age of 40. It is an autosomal dominant disorder, caused by a germline mutation in the adenomatous polyposis coli (APC) gene, which is a tumour suppressor gene and found on chromosome 5q21. Early onset of colorectal cancer is typical for HNPCC, which is estimated to be responsible for 1–4% of colon cancers. HNPCC is secondary to germline DNA mismatch repair gene mutations in the majority of the cases. The overall lifetime cancer risk of

developing colorectal cancer in patients with these mutations by the age of 70 is 91% for men and 61% for women. The same study demonstrated that the risk was significantly higher for males (74%) than for females (30%; p=0.006).

Other, rarer syndromes have also been shown to increase the risk of developing colorectal cancer. Peutz – Jeghers syndrome is an autosomal dominant condition characterized by the presence of hamartomatous polyps in the small bowel and mucocutaneous melanin pigmentation of the perioral region, hands and feet. Patients with the syndrome have 39% higher risk of developing colorectal cancer by the age of 70. MYH (mutY homolog gene mutation)-associated polyposis and juvenile polyposis, both autosomal dominant conditions, have been associated with 35– 53% and 17–68% increased risk of colorectal cancer respectively.

Inflammatory bowel disease

Patients diagnosed with IBD carry higher risk of colorectal cancer than the general population. This risk has been extensively investigated and is well established. The risk of colorectal cancer in ulcerative colitis patients is more pronounced. Colorectal cancer on the background of chronic ulcerative colitis has been described as being more aggressive and with poorer prognosis than the general population. The prevalence of colorectal cancer in patients with ulcerative colitis has been reported up to 3.7%. The cumulative risk of developing colorectal cancer has been reported to increase by 1% per year after 10 years from the date of onset of active ulcerative colitis. The relative risk of developing colorectal cancer on the background of ulcerative colitis at an early age has been reported up to 38-fold, with reports of 14.8 and 2.8 for patients with pancolitis and left-sided colitis respectively. The risk of developing colorectal cancer in ulcerative colitis has also been found to be significantly increased in patients with total colitis.

Earlier studies showed that the incidence of colorectal cancer in patients with CD was higher cancer, with other studies showing that CD-associated colorectal cancer had a poorer prognosis than sporadic colorectal cancer. The risk ratio for developing colorectal cancer on the background of CD is 4.5 in patients with colonic disease, compared with the general population, with a cumulative risk of 2.9% at 10 years.

Clinical presentation

Initially patients with colorectal cancer may be asymptomatic. As the disease progresses, development of systemic symptoms and symptoms specific to the location of the cancer may be observed. Rectal bleeding is one of the commonest presentations of colorectal cancer. However, it may be the result of a benign pathology and therefore has low positive predictive value for colorectal cancer. The prevalence, in populations of over 20 years old, ranges between 15.5% and 24%. A recent meta-analysis of 13 studies with 18 634 patients showed a positive predictive value (PPV) of 5.3% that increased to 8.1% in a subgroup analysis of studies that included patients aged 50 or more. Heterogeneity was moderate. A study by Fine et al. suggested that the colour of bleeding reported by patients was inaccurate because of misinterpretation and the use of a colour card was proposed in order to help patients to be more accurate. The same study reported that the majority of bright red bleeding (about 83%) was linked to distal (60cm) pathology. However, more proximal pathology was identified in 20 of 217 (9.22%) patients. A community-based trial showed that colorectal cancer was more likely when the blood was mixed with faeces and when it was reported as being dark (likelihood ratio 3.0). The picture is slightly different in secondary care, with the positive predictive value for colorectal cancer being higher for patients with a history of dark and bright blood (13.2%) than dark blood (10.6%) or bright blood alone (4.3%), and for when the blood was mixed with faeces (11.0%) than when separated from stools (3.4%). One study of 226 patients evaluated the risk of rectal bleeding in the presence of perianal symptoms and showed a negative likelihood ratio of 2.90, with poor sensitivity and specificity. This suggests that rectal bleeding in the presence of perianal symptoms carries low risk of colorectal cancer.

Patients with colorectal cancer often present with a ‘change in bowel habit’. The definition in the UK refers to the passage of loose stools and/or increased frequency of defecation that persists for more than 6 weeks. It has been reported by a number of studies that change in bowel habit may occur in up to 91% of patients with distal and 61% of patients with proximal cancer. Constipation is not currently considered as a risk factor; recent studies suggest that there may be a link and therefore should be used to refer patients for further investigation. A meta-analysis showed a positive likelihood ratio of 1.8 and negative likelihood ratio of 0.7 when it is associated with rectal bleeding.

Unintentional weight loss has been regarded as a predicting factor of colorectal cancer. However there has been no conclusion about the percentage of weight loss that should be considered clinically relevant. Loss of 5% of weight in a period between 6 and 12 months is generally considered significant. In colorectal cancer, weight loss is usually a late presentation with a median of 27 (9–42) weeks. The same study demonstrated a higher incidence of weight loss for proximal (46%) than distal (34%) tumours, but did not achieve statistical significance. The positive predictive value for colorectal cancer in patients with weight loss increases to 4.7% when it is associated with rectal bleeding, to 3.1% with diarrhoea and 3% with constipation. A recent meta-analysis of six studies that included 1468 patients demonstrated that the combination of weight loss and rectal bleeding increased the positive likelihood ratio to 1.88 and negative likelihood ratio to 0.93. There was low heterogeneity among the included studies.

Abdominal pain may be the first presentation. Up to 30% of the general population experience abdominal pain every year. This is usually a result of a benign condition. It has low sensitivity and specificity when it is the sole symptom. A recent metaanalysis demonstrated a positive likelihood ratio of 2.47 and negative likelihood ratio of 0.75. The same study showed low sensitivity (31%) but high specificity (91%). The heterogeneity between the studies was high though. The positive predictive value increased to 7.6% when abdominal pain was associated with rectal bleeding but the between-study heterogeneity remained high. Hamilton et al. demonstrated that there was an increased PPV of 3.4% when it was associated with weight loss, 3.1% with rectal bleeding and 1.9% with diarrhoea.

Investigation

Clinical examination A full history and examination is essential before any management plan is made. History should also include questions assessing the patient's performance status and comorbidities, as this information may be pivotal to the management plan. During clinical examination the patient should be specifically inspected for signs of anaemia, jaundice (suggestive of liver metastases) and malaise, and examined for abdominal distension, ascites, masses and abdominal tenderness. Digital rectal examination is essential, as it can provide essential information at the initial stage if the tumour is within the lower rectum.

Blood tests

Routine blood tests are performed including a full blood count, urea and electrolytes, liver function tests and coagulation. More specifically, the haemoglobin is measured to confirm or rule out anaemia, the liver function tests and coagulation to assess the liver function and raise any question of liver metastases.

Tumour markers, such as the carcinoembryonic antigen (CEA) and cancer antigen (CA) 19-9 are measured. CEA is an oncofetal antigen discovered in 1968. It is a product of the normal fetal gut tissue and epithelial tumours, especially those of the large bowel. It can, however, increase in smokers, IBD, pancreatitis, liver disease and in patients with epithelial tumours at other sites. CA 19-9 was discovered in patients with colon and pancreatic cancer. Negative CEA and CA 19-19, with a cut-off of 5ng/mL and 37U/mL, respectively, have been shown to significantly improve the overall survival following potentially curative operation. Care should be taken with mucin-producing and poorly differentiated adenocarcinomas of the colon and rectum which do not often present with a raised CEA and are classed as high-risk tumours for local failure and poor longterm prognosis.

Proctoscopy and rigid sigmoidoscopy

Proctoscopy and/or rigid sigmoidoscopy can be performed in an outpatient setting, confirming a lower rectal tumour and providing at the same time the opportunity to get biopsies from the tumour and therefore plan the patient's further management more efficiently. In addition, it may reveal a different pathology (i.e. IBD) or the presence of streaks of blood in the lumen of the rectum, which is strongly indicative of pathology in the sigmoid above the reach of the rigid rectoscope.

Flexible sigmoidoscopy

Flexible sigmoidoscopy can be performed relatively quickly and is virtually risk free. Therefore, the National Institute for Health and Clinical Excellence (NICE) in the UK suggests it to be the most appropriate initial investigation for the majority of symptomatic patients, especially the patients with symptoms suggesting possible left-sided lesions. Flexible sigmoidoscope can only reach up to 60 cm.

When there are no positive findings in patients without any right-sided symptoms, the likelihood of right-sided cancer is low.

Colonoscopy

NICE advises the use of colonoscopy in patients who are considered at high risk of developing colon cancer because of older age, a clinically palpable abdominal mass, iron-deficiency anaemia or symptoms such as abdominal pain with loss of appetite and weight. Colonoscopy is also necessary when there is significant clinical doubt following a flexible sigmoidoscopy. Diagnostic colonoscopy is usually appropriate for patients with right-sided symptoms, except for those with palpable masses, for whom imaging may be more appropriate (barium enema or CT). Colonoscopy should also be considered when a rectal tumour is diagnosed in order to exclude any synchronous tumours. About 4% of patients diagnosed with primary colorectal cancer will have synchronous colon cancers. Colonoscopy enables direct visualization of the tumour and its extent with assessment of its fixity to the surrounding tissues; and its potential for complete obstruction. In addition, biopsies can be taken and any significant active bleeding can be controlled.

Imaging

Accurate identification of metastatic disease is key for the decision to operate on a patient. CT colonography and barium enema can be used instead of colonoscopy for the initial diagnosis of the tumour. For colon cancer, a CT scan is used to stage local disease and assess for potential distant metastases (i.e. lung, liver). CT of the chest, abdomen and pelvis is usually requested. For rectal cancers MRI is used to stage local disease and CT to assess potential distant metastases. There is not enough evidence yet to support the regular use of positron emission tomography (PET) or PET/CT scans in this group of patients. It is therefore employed when clinical and radiological findings from other scans are equivocal. For low rectal cancers endorectal ultrasound has been used with high accuracy for the early stage cancers.

CT colonography

CT colonography is a modern application of the conventional CT scan that can provide information on the entire colon. It is minimally invasive and has been reported to be easier to tolerate by the patients. It is required that the patients

undergo bowel preparation before the procedure, although more recent studies have been investigating the potential to avoid this. The side effect of this modality is the increased radiation that patients receive compared with conventional colonoscopy. Low radiation protocols are currently being investigated, with early results showing that it is feasible to provide good images. There have been three large multicentre studies published with significant differences reported in terms of the accuracy of CT colonography. Pickhardt et al. found comparable sensitivities between CT colonography (86%) and colonoscopy (90%) in the detection of polyps of ≥6mm.

However, subsequent publications showed that colonoscopy was significantly superior to CT colonography by detecting up to 99% of lesions/polyps. A more recent study showed that CT colonography has high sensitivity (97%) and specificity (about 91%) and that it can be more accurate than barium enema studies, but less accurate than colonoscopy. A meta-analysis of 49 studies of 11 151 patients was performed to assess the sensitivity of CT colonography and colonoscopy and demonstrated high sensitivity for both tests with moderate between-study heterogeneity when both cathartic and tagging factors were combined for bowel preparation.

Barium enema

Barium enema is well established in the NHS in the UK. It is safe and readily available. Bowel preparation is necessary but there is no need to sedate the patient, and therefore the patient can be discharged home the same day. Barium enema on its own has lower sensitivity than colonoscopy, and any negative results should be interpreted carefully in association with the patient's symptoms. Another disadvantage of barium enema is the inability to take tissue biopsies or remove any polyps. For these reasons its use has been declining in NHS centres where colonoscopy or CT colonography is available.

Locoregional staging

Computed tomography CT is extensively used in the preoperative assessment of patients with colorectal cancer. It is used to assess local and distant disease. Studies have shown variability in the assessment of the extent of local disease. Modern CT scanners can also provide detailed reconstructed three-dimensional images that can help to improve the interpretation of two-dimensional images. A recent study has demonstrated that modern CT scanners are highly accurate in

predicting the pathological T stage (pT). The diagnostic accuracy for T2 and T3 was 94.1%, and 100% for T4 tumours. The same study demonstrated high diagnostic accuracy (80.5%) for detecting lymph node metastases. In older studies, CT was less accurate in detecting and staging tumour and lymph node metastases.

CT criteria for colonic T staging

Stage	Description
T1	Intraluminal lesion without evidence of bowel wall invasion (bowel wall thickening)
T2	Thickened colon wall without disruption of muscularis propria
T3	Thickened colon wall with discrete mass extending into pericolic fat
T4a	Irregular advancing edge of tumour penetrating adjacent organs
T4b	Breach of the peritoneum that covers the colon
T4c	Perforated tumours with evidence of pericolonic gas and free fluid in the abdomen

A large study demonstrated that lymph node metastases were often missed by CT. This may be because of the weakness of CT to detect micrometastases and microalterations of the lymph node shape and size. In order to address this, Kanamato et al. analysed every lymph node that was detectable by CT and measured its longest and shortest diameter. They demonstrated that a short–long-axis diameter ratio of 0.8 or greater as an index for the diagnosis of metastatic lymph nodes achieved an accuracy index of 80% per node. As the technology evolves, it is likely that CT scanners will become even more accurate. There are radiological criteria for CT that have been established in order to facilitate radiological staging. The criteria are described in.

Magnetic resonance imaging

MRI is the investigation of choice for pretreatment local (T) staging for rectal tumours. High-resolution MRI can clearly depict the bowel layers and accurately detect the depth of tumour invasion, extramural invasion and the relationship of the tumour to the surrounding anatomical structures. MRI has high diagnostic accuracy in predicting the T stage and the distance of the tumour from the circumferential margins. It correctly anticipates the subsequent histopathological examination in 85% of T3 and T4 tumours. MRI has a sensitivity of 94% and a specificity of 85% in determining the relationship between the advancing border of the tumour and the fascia propria of the rectum. Involvement of the fascia propria predicts a positive surgical circumferential margin. MRI also allows the subcategorization of T3 tumours into T3a and T3b. Nodal staging has been always challenging as micrometastases may not be diagnosed.

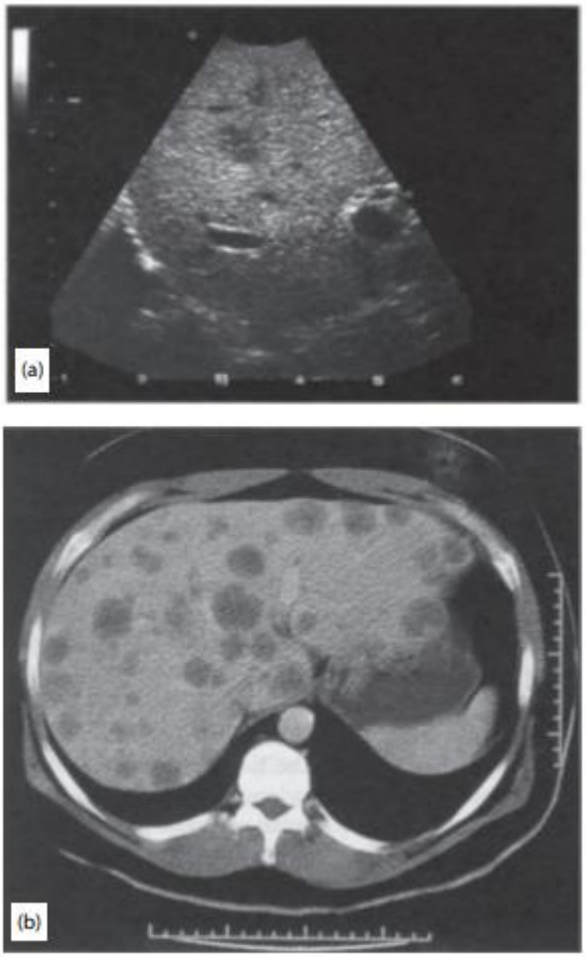
Nodal staging, whether assessed by ultrasound or MRI, is still unreliable, but analysis of the consistency of the node and the regularity of its capsule as demonstrated on MRI may improve its accuracy. Brown et al. showed that the

heterogeneity of the lymph node's MRI signal was a highly specific discriminator, raising the diagnostic sensitivity to 85% and specificity of 97%. A recent study by Kim et al. showed the diagnostic accuracy of MRI to be 83% in detecting lymph node metastases compared with the 70% accuracy of PET. Combining both modalities increased the diagnostic accuracy to 90%. More recently, the use of nanoparticle-enhanced MRI (using ultrasmall superparamagnetic iron oxide as a contrast agent) is reported to have sensitivity in the order of 92% and specificity of around 96% for detecting lymph node metastases.

Imaging for distant metastases

Identification of distant metastases is crucial in the management of patients with colorectal cancer as it drastically changes the management of patients. CT and MRI have demonstrated high sensitivity in detecting distant metastases, providing detailed anatomical information of the affected organ and tumour extension into surrounding tissues.

A meta-analysis investigating the value of ultrasound scan (USS), CT, MRI and PET in detecting liver colorectal of distant metastases. However, the authors admitted that only eight of 27 (29.6%) studies were of high quality, fulfilling their quality criteria at least by 80%.



(a) Ultrasound appearance of hepatic metastases – image obtained at intraoperative contact ultrasound. (b) CT scan showing multiple hepatic metastases.

Medical treatment

Each individual case must be discussed in a multidisciplinary setting before any decision for medical or surgical therapy is made. Medical therapy can be in the

form of neoadjuvant (preoperative), adjuvant (postoperative) or palliative, chemotherapy or chemoradiotherapy.

Neoadjuvant therapy

Neoadjuvant radiotherapy

Neoadjuvant radiotherapy or chemoradiotherapy is the most common application of preoperative medical therapy in colorectal cancer. It is mainly used for high-risk rectal cancer, when the margins are threatened or when a tumour response may change the type of surgery required to achieve complete clearance (perform an anterior resection instead of abdominoperineal resection) and preserve the anus and bowel function. Neoadjuvant chemoradiotherapy is used for T3, T4, lymph node-positive disease, and when the resection margins are threatened. For chemoradiotherapy, capecitabine and bevacizumab are the chemotherapeutic agents that are commonly used to increase the tumour's sensitivity to radiation.

Studies have shown that neoadjuvant chemoradiotherapy can reduce the local recurrence rate without changing the overall survival rates. A meta-analysis of randomized controlled studies with patients who underwent neoadjuvant therapy prior to surgery for resectable rectal cancer showed a marginal benefit for neoadjuvant radiotherapy that did not reach statistical significance. The same study showed that the cancer-specific survival and local recurrence rate were reduced in the group of neoadjuvant radiotherapy. In a more recent study the use of preoperative short-term radiotherapy reduced the 10year local recurrence by more than 50% without an overall survival benefit.

Neoadjuvant chemotherapy

Chemotherapy prior to surgery is used to reduce the tumour's size so that it can be resected with higher likelihood of complete resection and fewer operative complications. 5-Fluorouracil (5FU) is the first-choice chemotherapy drug for colorectal cancer and is used intravenously. It can be used in combination with leucovorin (folinic acid) that has been shown to increase its action. More recently the same medication has been developed in a form that can be taken orally (capecitabine).

There are new chemotherapeutic agents that can be combined with 5FU or used alone. These include irinotecan, oxaliplating, cetuximab and penitumumab. These, apart from cetuximab, are usually used in combination with 5FU.

Preoperative chemotherapy has not been extensively used for patients in whom surgery is likely to completely remove the primary cancer. There is currently an ongoing randomized controlled study in the UK that is investigating the value of neoadjuvant chemotherapy in patients who would otherwise undergo colonic surgery and adjuvant chemotherapy (FOxTROT). This study is based on the observation that in other cancers preoperative chemotherapy has proved to be more effective than postoperative chemotherapy. This has generated the following hypotheses:

• Early optimal systemic therapy may be more effective at eradicating distant metastases than the same treatment given after the delay and immunological stress of colonic surgery.

• Shrinking the primary colon tumour before surgery may reduce the risk of incomplete surgical excision, and the risk of tumour cell shedding during surgery.

Adjuvant medical therapy

The aim of adjuvant chemotherapy is to prevent the dissemination of the disease in high-risk patients. This is discussed at the local multidisciplinary meetings with the results of the histopathology. Adjuvant chemotherapy has been shown to increase the overall survival and disease-free survival.

These are the factors that are considered to increase the risk of dissemination and used as an indication for adjuvant chemotherapy:

• positive lymph nodes

• poorly differentiated cancer cells

• vascular and perineural invasion

• T4 cancer

• evidence of localized distant metastasis during the operation.

Chemotherapy regimes that are currently in use are the following:

• FOLFOX – leucovorin, 5 Fu and oxaliplatin

• XELOX – a combination of oxaliplatin and capecitabine

• capecitabine/5 Fu

• tegafur and uracil capsules.

Palliative therapy

Radiation and chemotherapy have been used in patients who have irresectable (tumour that cannot be resected) or inoperable (tumour that is not indicated to be operated due to dissemination) disease. Radiotherapy is usually used to alleviate symptoms related to tumour expansion such as pain, lower leg oedema and lymphoedema. The aim is to shrink the tumour and reduce the effect to the local tissues. Systemic chemotherapy is used to control the dissemination of the disease and prolong the overall survival. A meta-analysis of seven trials with 866 patients showed a 35% reduction in the risk of death and 16% in the improvement of survival within a year when palliative chemotherapy was used. There was also an improvement of survival by 3.7 months (median).

Surgical treatment

Colon cancer surgery

Historical perspective

The intestinal suture was considered a 'mighty procedure in a highly vulnerable organ', and therefore a dangerous option for surgery. The formation of a stoma used to be the surgical treatment of choice for any intestinal disease and injury. Colostomy was introduced as a palliative procedure for patients with obstructive bowel cancer in 1839. Until then, patients diagnosed with obstructive cancer would have either died or been relieved by a spontaneous fistula.

Reybard of Lyons in 1833 was the first to perform a successful resection of the sigmoid colon, but he found great opposition from the Paris Academy of Medicine.The mortality following a colonic resection ranged from 60% before 1889 to 37% by 1900. Owing to the high mortality rates the staged extraperitoneal resection technique (exteriorization–resection) was usually employed.

Bowel resection with anastomosis was not widely performed until the introduction of antibiotics. The sigmoid colectomy was always performed with the formation of a defunctioning colostomy until the 1950s. At that time, the interest of the surgical community was focused on ways to reduce faecal contamination during the procedure. A number of published articles discussed the application of

non-crushing clamps and the value of limited inversion of the anastomosis. Stapling techniques were reported as early as 1908, but they have found widespread application more recently.

Procedures

The aim of surgery is to achieve complete tumour resection with an adequate resection margin, along with draining the lymphatics and the creation of a tension-free anastomosis. The following operations are performed for colon cancer:

• right colectomy

• transverse colectomy

• left colectomy

• sigmoid colectomy

• sutotal/total colectomy

• palliative resection (limited).

The majority of the new surgeons prefer to perform colon operations through a midline incision, the extent of which (upper, lower or full extent) is dictated by the type of colectomy, position of the tumour and body habitus. The advantages of a midline incision are the following:

• it facilitates quick entrance into the peritoneal cavity

• it provides adequate access to both sides of the abdomen

• it allows the formation of either a colostomy or ileostomy to either side

• the wound heals strongly and is easily closed in a single layer.

A right-sided oblique incision may be employed for right colon resections. The wound from this type of incision heals well and is possibly associated with less discomfort for the patient. However, the exposure may be compromised.

This type of incision is mainly used by older surgeons who had been trained to do this technique, as previously it was more widely used. The introduction of minimally invasive surgery resulted in this technique being abandoned. Minimally invasive surgery enabled faster bowel function recovery and feeding of patients.

This was associated with less discomfort and shortened hospital stay. These results led to the introduction of minilaparotomy. Minilaparotomy has been defined as complete resection performed through a skin incision less than 7 cm long. Minilaparotomy for colon cancers was shown to be feasible, with comparable oncological outcomes, and was found to reduce hospital stay and analgesia requirements. Body habitus was found to be a crucial factor in the completion of the procedure without extending the incision.

When the peritoneal cavity is entered, it is important to check for the presence of metastatic liver and/or lymph node disease and peritoneal dissemination. The tumour is examined at the end after excluding dissemination of disease. Resectability and fixation of the tumour to the surrounding tissues are evaluated with minimum handling. Large lymph nodes can be inflammatory and, when in doubt, a frozen section can be sent for histopathological examination.

Right hemicolectomy

Right hemicolectomy is performed to resect lesions located at the caecum, ascending colon and hepatic flexure. The blood supply in this area originates from the ileocolic and right colic arteries, and therefore dictates the use of right hemicolectomy. The positioning of the surgeon is a matter of personal preference. The small bowel is retracted into the left upper quadrant of the abdominal cavity, exposing the root of the mesentery and the base of the transverse mesocolon. The right colic and right branch of the middle colic vessels may be ligated at the beginning of the dissection. This can be more challenging in the obese patient and may not be safe to perform. The small incision in the root of the mesentery is extended to the point that the transverse colon will be divided. The mesenteric and mesocolic vessels are ligated, limiting the entire blood supply to the tumour. In more recent years, the implementation of vessel-sealing devices has changed the way that this part of the procedure is performed. These devices can securely seal small vessels (up to 7mm diameter) within a few seconds. This technique can replace the use of clips and sutures.

The congenital peritoneal adhesions along the lateral gutter are divided using coagulation diathermy, elevating the terminal ileum and right colon from the retroperitoneal structures. This will include any lateral peritoneum involved by serosal tumour with the specimen. Care must be taken to avoid injury to the ureter, spermatic or ovarian vessels, and inferior vena cava. The ileocaecal fold of

Treves is incised to prepare the terminal ileum. As the colon is elevated from the retroperitoneum, the second part of the duodenum will appear and should be displaced with care to avoid any injury. The hepatic flexure is freed from the developmental gallbladder and liver adhesions using diathermy or vessel-sealing devices. When the colon is sufficiently lifted the head of the pancreas will be visible indicating that the duodenum is adequately cleared from the field. Clamping of the blood supply can be safely performed now following development of the avascular plane around the vessels.

The lesser sac is entered by dividing the greater omentum, enabling at the same time the retraction of the posterior wall of the stomach away from the dissecting field and therefore reducing the risk of injury. The dissection of the greater omentum continues until the hepatic flexure is fully mobile. The omentum is incised at the point of the anastomosis. The bowel is therefore ready for resection. On rare occasions that the tumour invades the duodenum and the pancreas the tumour can be resected en bloc with the duodenum and pancreas (pancreaticoduodenectomy). This will require the employment of an expert hepatobiliary surgeon.

Surgery for transverse colon cancer

Cancer of the transverse colon can often be a challenge for the colorectal surgeon. The blood supply to this area is derived from the middle, right and left colic vessels and an anastomosis at the splenic flexure may beat risk of ischaemia, as the blood supply solely from the inferior mesenteric artery may not be sufficient. This is not usually an issue when dealing with tumours at the hepatic flexure, as there is sufficient blood supply from the ileocolic and right colic vessels.

The lymphatic drainage is also an issue when dealing with tumours of the transverse colon. Cancer in the transverse colon can metastasize to regional lymphatics through the middle colic, right colic and left colic branches. This is why some surgeons suggest subtotal colectomy as an option for this type of cancer. Other surgeons decide the type of surgery based on the location in the transverse colon, suggesting right hemicolectomy for proximal lesions and left partial or left hemicolectomy for distal lesions. For the latter the anastomosis can be performed between the transverse and the sigmoid colon. This will require mobilization of the right colon as well. Limited transverse colectomy may be considered for

palliation. As mentioned, however, as a cancer operation it may be inadequate. Mobilization of the splenic flexure and left colon are explained in the section Rectal cancer surgery. The same principles are applicable here.

When the tumour invades the spleen or when the spleen is at risk, en bloc splenectomy may be indicated. In these circumstances the spleen and tail of the pancreas may need to be removed en bloc with the colon. Splenectomy in association with colonic resection for cancer is associated with a high morbidity and mortality rate. The incidence of unintentional splenic injury during mobilization of the splenic flexure is around 1%. Splenectomy increases the rate of postoperative sepsis in this group of patients.

Left hemicolectomy (partial colectomy)

Left hemicolectomy is performed for tumours involving the distal transverse colon, splenic flexure and descending colon. In this procedure, the proximal right branch of the middle colic and the inferior mesenteric artery should be preserved. The left colic artery is ligated. The anastomosis is performed between the midtransverse and the upper sigmoid colon. The technique of the mobilization is described in the section Rectal cancer surgery.

Subtotal/total colectomy

Subtotal colectomy involves the removal of most of the colon with an anastomosis between the ileum and the sigmoid or descending colon. Total colectomy involves the removal of the entire colon with an ileorectal anastomosis at the rectosigmoid junction. It is a more extensive operation with the advantage of an easier to perform anastomosis and maximum harvesting of lymph nodes. Subtotal or total colectomy may be indicated for the following:

- synchronous left- and right-sided tumours
- multiple tumours (benign or malignant or both) are present
- FAP and IBD
- HNPCC • when a resection has been previously performed
- technical limitations.

Complete mesocolic excision

Surgery for rectal cancer has become standardized with the implementation of TME. Until recently, there had not been any attempts to standardize surgery for colon cancer. Hohenberger et al. suggested the equivalent to the TME procedure, the complete mesocolic excision (CME) of the colon. The concept of CME is based on the same embryological principles of TME. It involves sharp dissection of the visceral plane from the retroperitoneal one with high ligation of the draining/ supplying vessels, aiming to prevent any injury of the visceral fascia layer that may potentially result in spillage of tumour cells and dissemination of the disease.

For right colon cancers the mobilization of the duodenum with the pancreatic head (Kocher manoeuvre) and the mesenteric root up to the origin of the superior mesenteric artery is essential to maximize the exposure of the supplying vessels. The attachments of the mesenteric plane to the duodenum and the uncinate process are dissected in order to gain full access to the superior mesenteric vessels. The right colon is fully mobilized and therefore can be twisted (clockwise) to provide easier access to the central part of the superior mesenteric vessels. The ileocolic and, when present, the right colic vessels should be divided at their origin from the superior mesenteric vessels. For caecal and ascending colon cancer, ligation of the right branches of the middle colic vessels is adequate. The colon is divided at the level of the middle colic vessels. For hepatic flexure cancer, the transverse colon is resected at the splenic flexure.

For transverse colon cancers, the proximal part of the colon can be preserved and anastomosed to the sigmoid colon. The right colic vein drains into the superior gastroepiploic vein. It is usually ligated first to prevent severe haemorrhage from accidental injury. The superior mesenteric vessels are subsequently exposed, as the veins supplying the arteries are divided centrally. Attempt should be made to preserve the surrounding autonomous nervous plexus to avoid the risk of bowel dysfunction. If there are suspicious pancreatic lymph nodes they can be dissected off the pancreatic head with central ligation of the right gastroepiploic artery. The superior pancreaticoduodenal artery is usually preserved.

For left colon tumours, mobilization of the splenic flexure with the left colon is performed. This is described in more detail in the section Rectal cancer surgery. Once the left colon is completely dissected off the retroperitoneum, the greater omentum is divided from the transverse colon to facilitate full exposure of the lesser sac and the subsequent division of the final two layers of the transverse mesocolon at the lower edge of the pancreas. It is important that during this

process the integrity of the mesocolon is maintained in a similar way to the mesorectal excision for rectal cancer.

The root of the inferior mesenteric artery is usually preserved. The left colon artery is centrally ligated including the lymph nodes at the origin of the inferior mesenteric artery. Preservation of the superior hypogastric plexus is also important. For cancers of the mid-descending colon down to the sigmoid colon, the root of the inferior mesenteric artery and the inferior mesenteric vein below the pancreas are ligated. The proximal colonic division is performed at the point between the left transverse colon and the proximal descending colon and depends on the tumour site. The resection is performed distally in the upper third of the rectum.

Screening for colorectal cancer

It is well established that 'early' colorectal tumours (i.e. stage A) have a much better prognosis than more advanced tumours, and it therefore makes sense to try to diagnose colorectal cancer at an early stage. Unfortunately, the common symptoms of colorectal cancer (i.e. change of bowel habit, abdominal pain and rectal bleeding) only occur when the tumour is relatively locally advanced. The only way in which to reliably identify cancers at an early stage is to employ population screening, and a great deal of research effort has gone into this approach over the past 20 years.

There are now three population-based randomized controlled trials which have demonstrated that screening asymptomatic populations with faecal occult blood testing can reduce disease-specific mortality by 15–30%. Unfortunately, standard faecal occult blood testing, which is positive in about 2% of an unselected population between the ages of 50 and 75 years, is only about 50% sensitive. Thus a negative faecal occult blood test is not a guarantee that colorectal cancer is not present, although a positive test is associated with a 50% chance of colorectal neoplasia and a 12% chance of having invasive malignancy. It is possible to increase the sensitivity of faecal occult blood testing, but this reduces the specificity to such an extent that a large number of negative investigations would have to be carried out.

Another approach to screening is to use flexible endoscopy as the primary screening test. To use colonoscopy in this way would be impractical, but flexible sigmoidoscopy is a more realistic proposition. As 75% of colorectal cancers are

within the reach of this instrument and as the presence of an adenomatous polyp in the left colon is an indicator of possible neoplasia on the right of the colon, then a 'once-only' flexible sigmoidoscopy may be a useful screening test. This is the subject of a randomized trial carried out in the UK, but mortality data from this trial are not yet available. One of the problems with all forms of colorectal cancer screening is the low compliance rate, which is presumably related to a mixture of ignorance, fear and distaste.

Tests and tasks

1. The main reason of acute anal abscess is:

1. Hemorrhoids

2. Injury of rectal mucosa after medical procedures

3. *Microtraumas of rectal mucosa*

4. Bullet wound of the rectum

5. Inflammatory diseases of organs neighbor to the rectum

2. What therapeutic methods should be used in acute anal abscess?

1. Massive antibacterial therapy

2. Physiotherapy

3. Emergency surgery

4. Elective operation

Choose the correct answer combination: a) 1, 2 b) 1, 4 c) 1, 2, 4 d) 2, 3 *e) 1, 3*

3. From what diseases acute anal abscess should be differentiated?

1. Buttock carbuncle

2. Buttock abscess

3. Prostate abscess

4. Suppuration of coccygeal cysts

5. Bartholinitis

Choose the correct answer combination: a) 1, 2 b) 3, 5 c) 4 d) All variants are correct e) All variants are incorrect

4. These principles should be followed in therapy of acute anal abscess:

1. Early surgery

2. Adequate opening and sanitization of a suppurative focus

3. Excision of the internal aperture

4. Adequate draining

Choose the correct answer combination: a) 1, 2 b) 1, 2, 4 c) 1, 3 d) 2, 4 e) All variants are correct

5. Which of the following measures are important for acute anal abscess prevention?

1. Cleansing enemas

2. Medicinal enemas

3. Saline laxatives

4. Treatment of the accompanying proctological and gastro-intestinal diseases

5. Washing of the perineum after defecation instead of toilet paper use

Choose the correct answer combination: a) 1, 2 b) 1, 3, 4 c) 2, 3, 5 d) 2, 4, 5 e) All variants are correct

6. The following symptom complex is characteristic of rectal fissure (the choice depends on the disease stage):

1. Moderate pain in the anal region, increasing during defecation, anal itch, voluminous bleeding after defecation.

2. Feeling of incomplete emptying after defecation, blood-coloured ribbon stool, tenesmus, unstable stool, defluvium, sometimes single portions of dark blood

3. Unstable stool, feeling of heaviness in the pelvic area, feces of normal configuration with dark or crimson blood, scybalous stool, the abdomen is bloated and unrelieved with poor stool

4. Frequent liquid stool, tenesmus, mucous and bloody discharge, sometimes profuse diarrhea, possible temperature reaction

5. Severe pain after defecation, 2-3 drops of blood after defecation, fear of stool, chronic constipation

7. The most often form of paraproctitis is:

1. Subcutaneous paraproctitis

2. Submucous paraproctitis

3. Ischiorectal paraproctitis

4. Pelviorectal abscess

5. Intercondyloid paraproctitis

8. Surgery on acute anal abscess should be performed under:

1. Intravenous anasthesia

2. Local anesthesia

3. Sacral anesthesia

4. Peridural anesthesia

5. Any kind of anesthesia, except local anesthesia

9. In case of hemorrhoidal boluses acute thrombosis ambulotary therapy it is most rationally to:

1. Indicate laxatives (magnesium sulfate), lead water, intake of aescusan or aspirin, suppositories with belladonna

2. Novocaine block, reduction of a hemorrhoid

3. Indicate analgetics, fomentations during first 2-3 days, rest cure, heparin ointment dressing and a diet

4. Remove thrombosed boluses

5. Apply sclerosing therapy

10. Coccygeal epithelial course:

1. Is connected to the sacrum

2. Is connected to the tip

3. Ends blindly in the subcutaneous tissue of the inter-buttock area

4. Is situated between the posterior rectal surface and the anterior sacral surface

5. Is communicated with the rectal lumen

I. Patient M., 58 years old, went to the doctor in connection with the appearance of pain in the lower abdomen and with diarrhea (more than a month marks the alternation of constipation and diarrhea). The general condition of the patient is satisfactory. Appetite saved. The tongue is wet, clean. The abdomen is not swollen, soft, painful with deep palpation in the left iliac region. Body temperature 37.3 ° C. A digital examination of the rectum revealed no pathology. In connection with a suspected colon disease, irrigoscopy was performed and sigmoid colon diverticula were detected.

1) What complication of diverticulosis can be suspected?

2) What diseases do you need to have a differential diagnosis with?

3) What types of surgical intervention can be used in the absence of the effect of conservative treatment?

II. Patient I., 30 years old, complains of the presence of a fistula with a purulent compartment in the perineum, which has existed for about a year. On examination, a fistula with a small purulent discharge and maceration of the skin around it was found on the perineal skin. On palpation, a dense cord is detected in the subcutaneous tissue.

1) Make a preliminary diagnosis.

2) Prescribe a conservative treatment.

3) What types of operations can a patient perform?

III. Patient N., 62 years old, complains of pulling pain in the perineum, mucus from the rectum. A digital examination of the rectum in its ampullar section revealed several formations with a diameter of 0.5 to 3 cm with a clearly defined leg. These formations are displaced along with the intestinal mucosa.

1) Make a preliminary diagnosis.

2) Define surgical tactics in this case.

3) What are the methods of surgical treatment of this pathology?

Answers to Tests

1. – 3
2. – e
3. – e
4. – e
5. – d
6. – 5
7. – 1
8. – 5
9. – 3
10. – 3

Answers to Tasks

I.

1)Chronic diverticulitis;

2)Colon cancer,Crohn's disease,nonspecific ulcerative colitis,polyposis of the large intestine.

3)With single diverticulums - their excision, and with multiple - resection of the affected part of the colon.

II.

1) Chronic paraproctitis. Fistulous form.

2) Diet therapy (regulation of stool); sedentary warm baths; rising shower; washing fistulous passages with antibiotics; hemorrhoidal suppositories; microclysters with sea buckthorn oil.

3) With intrasphincteric fistula: excision of the fistula into the lumen of the rectum.

Transsphincteric fistulas are eliminated by excision of the fistula into the intestinal lumen with closure of the deep layers of the wound or ligature method

With extrasphincteric fistulas: complete excision of the fistula and suturing of the inner hole; application of the ligature method.

III.

1) Polyps of the rectum.

2) With morphological confirmation, surgical treatment is indicated (polyp removal).

3) Endoscopic papilectomy with electrocoagulation of the leg;

Transanal excision of polyps with suturing on the wound of the mucosa.

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