Colorectum

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6.1 Anatomy

The colon and rectum together measure between 125 and 140 cm in the adult. The colon is divided into the cecum (10 cm) and ascending (15 cm), transverse (40 cm), descending (25 cm), and sigmoid (25-40 cm) colons. The rectum measures approximately 13-15 cm (Fig. 6.1). The main function of the colon is absorption of water and electrolytes and the storage of fecal material until it can be excreted. The cecum is that part that lies below the ileocecal valve and receives the opening of the appendix. It is mostly surrounded by peritoneum, allowing it to be mobile in the right iliac fossa. The base of the appendix is attached to the posteromedial surface of the cecum. The

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ascending colon extends upward from the cecum to the inferior surface of the right lobe of liver. Here it becomes continuous with the transverse colon by turning sharply to the left, forming the right colic or hepatic flexure. The ascending colon is bound to the posterior abdominal wall by peritoneum covering its front and sides. The transverse colon extends from the hepatic flexure to the left, hanging downward and then ascending to the inferior surface of the spleen, where it turns sharply downward to form the left colic or splenic flexure. The transverse colon is completely surrounded by peritoneum with the transverse mesocolon being attached to its superior border (the length of the transverse mesocolon accounts for the variability in the position of the transverse colon) and the greater omentum to its lower border. The descending colon extends downward from the splenic flexure to the left side of the pelvic brim. It is bound to the posterior abdominal wall by peritoneum covering its sides and front. The sigmoid colon is continuous with the descending colon and hangs as a loop into the pelvic cavity. It is completely surrounded by peritoneum and a fan-shaped piece of mesentery attaches it to the posterior abdominal wall, thus allowing mobility. The rectum begins as a continuation of the sigmoid colon in front of the third sacral vertebra and follows the curvature of the sacrum and coccyx to where it pierces the pelvic floor to become continuous with the anal canal. Peritoneum covers the anterior and lateral surfaces of the upper third and the anterior surface of the middle third, the lower third being devoid of

Fig. 6.1 Colorectum (Used with the permission of the Union for International Cancer Control (UICC), Geneva, Switzerland. The original source for this material is from Wittekind et al. (2005))

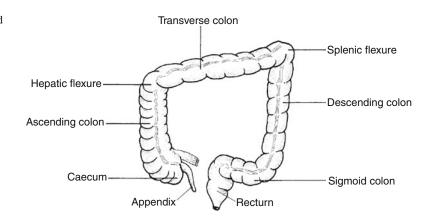
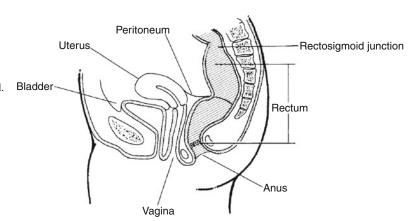


Fig. 6.2 Rectosigmoid and peritoneal reflection (lateral view) (Used with the permission of the Union for International Cancer Control (UICC), Geneva, Switzerland. The original source for this material is from Wittekind et al. (2005))



a peritoneal covering. At the junction of the middle and lower third, the peritoneum is reflected onto the posterior surface of the upper vagina in the female to form the rectovaginal pouch (pouch of Douglas) and onto the upper part of the posterior bladder in the male, forming the rectovesical pouch (Fig. 6.2). The extent of serosal covering in the colorectum is illustrated in Fig. 6.3. The rectum is surrounded by a bilobed encapsulated fatty structure which is bulkier posterolaterally than anteriorly – the mesorectum.

The small and large intestines differ in their appearance in a number of ways:

- The longitudinal muscle in the small intestine forms a continuous layer, whereas in the colon it comprises three bands called *taeniae coli*. However, in the rectum, the taeniae coli come together to form a broad band on the anterior and posterior surfaces.
- The wall of the colon is sacculated, whereas the small intestine is smooth.

- The colon has "fatty tags" called appendices epiploicae.
- The permanent mucous membrane folds (*plicae circulares*) in the small intestine are not present in the colon.

Microscopically the colonic mucosa is made up of tubular crypts lined by columnar epithelium with mucin-secreting goblet cells and endocrine cells also being present.

Lymphovascular drainage:

Embryologically the gastrointestinal tract is divided into three segments (fore, mid, and hindgut) with each region being supplied by its own artery:

- Celiac artery supplies the foregut (distal esophagus to the mid-portion of the second part of the duodenum).
- Superior mesenteric artery supplies the midgut (mid-portion of the second part of the duodenum to the junction of the proximal two-thirds and distal third of the transverse colon).

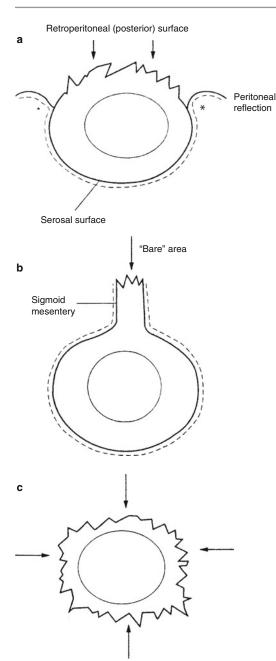


Fig. 6.3 Extent of serosal covering of the large intestine. *Arrows* indicate the "bare" non-peritonealized areas of different levels. (a) The ascending and descending colon are devoid of peritoneum on their posterior surface. (b) The sigmoid colon is completely covered with peritoneum, which extends over the mesentery. (c) The lower rectum lies beneath the pelvic peritoneal reflection. The *asterisks* in (a) indicate the sites where serosal involvement by tumor is likely to occur (Reprinted, with permission, from Burroughs and Williams (2000))

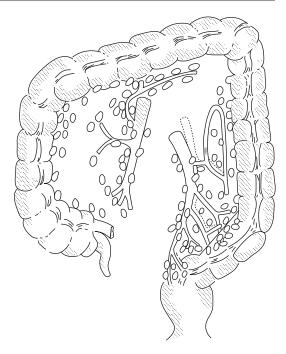


Fig. 6.4 Colorectum: regional lymph nodes are the pericolic, perirectal and those along the ileocolic, right colic, middle colic, left colic, inferior mesenteric, superior rectal (hemorrhoidal) and internal iliac arteries. (Used with permission of the Union for International Cancer Control (UICC), Geneva, Switzerland. The original source for this material is from Wittekind et al. (2005))

 Inferior mesenteric artery supplies the hindgut (distal third of the transverse colon to the junction of the superior and inferior half of the anal canal).

The rectum is also supplied by branches of the internal iliac artery. The anastomosis of the colic arteries around the concavity of the colon forms the marginal artery. The venous drainage of the colon is to the portal venous system and the rectum to the inferior mesenteric and internal iliac veins.

The lymphatics accompany the colic vessels draining to the superior and inferior mesenteric nodes. Those from the rectum drain into nodes (pararectal nodes) situated in the perirectal connective tissue (mesorectum) and thence to the superior mesenteric and internal iliac nodes (Fig. 6.4).

6.2 Clinical Presentation

There is considerable variability in the clinical presentation of colorectal disease.

Angiodysplasia usually presents with persistent occult bleeding or repeated small bleeds. In colonic ischemia/infarction there may be a history of arrhythmia or cardiac failure, and it may present acutely with abdominal pain and bloody diarrhea or less acutely with stricturing and symptoms of obstruction. Infective conditions usually lead to diarrhea, crampy abdominal pain, and fever. A careful antibiotic drug history should be obtained if pseudomembranous colitis is suspected. Inflammatory bowel disease may have an indolent presentation with lethargy, anorexia, and weight loss. However, more characteristic symptoms of ulcerative colitis include bloody diarrhea (>10 stools/day), urgency, and abdominal pain. Peritonitis and systemic sepsis may occur with toxic megacolon and perforation. Colorectal Crohn's disease characteristically presents with diarrhea. Obstruction due to stricturing may occur and fistulae leading to specific symptoms (e.g., colovesical - pneumaturia and recurrent urinary infection; rectovaginal - fecal discharge per vagina). Perianal fissures/fistulae and anorectal sepsis are relatively common in Crohn's disease. Extragastrointestinal manifestations of inflammatory bowel disease include fingerclubbing and erythema nodosum. Diverticular disease may present insidiously with lower abdominal pain or fistula formation (e.g., colovesical), or acutely as acute diverticulitis (abdominal pain, diarrhea, and localized peritonitis), pericolic abscess, obstruction (due to stricturing), perforation (generalized peritonitis), or hemorrhage (relatively rare).

Adenomatous polyps are usually asymptomatic, but large villous adenomas in the rectum may elicit an alteration in bowel habit, mucus per rectum (may cause pruritis ani), tenesmus (a sensation of incomplete evacuation), and electrolyte loss (particularly potassium). Colorectal carcinoma is usually asymptomatic early in its existence and later may present with nonspecific symptomatology such as an alteration in bowel habit, mucus PR, abdominal mass or discomfort, and PR bleeding (may be occult and can lead to iron-deficiency anemia). As a rule, the more proximal the tumor, the darker the blood. Tumors in the right colon are more likely to be ulcerated and so tend to present with PR bleeding, whereas

tumors of the left colon are often constrictive and present with obstruction – this is compounded by the fact that the fecal material is more solid in the distal colon. Perforation may occur either through the tumor itself or distant and proximal to it due to obstruction and back pressure, e.g., in the cecal pouch. Rectal tumors can lead to tenesmus and local invasion may produce back pain and sciatica (involvement of the sacral plexus), rectovaginal fistula, etc. Liver metastases may cause clinical jaundice.

6.3 Clinical Investigations

- FBP iron-deficiency anemia as a result of PR bleeding.
- U&E electrolyte disturbance in diarrhea/ mucus PR.
- LFTs deranged in liver metastases or in the hepatobiliary manifestations of Crohn's disease.
- C-reactive protein/ESR allows the activity of inflammatory bowel disease to be monitored.
- Stool culture rule out infective colitis.
- Fecal occult blood will detect occult bleeding.
- CXR will detect pulmonary metastases.
- AXR will show signs of colonic obstruction. Any dilatation of the colon >6 cm in diameter heralds the onset of toxic megacolon. In ischemic colitis, there will be dilated colon with characteristic "thumbprinting." In colovesical fistula, gas is present in the bladder. Free intraperitoneal gas will be seen in colonic perforation. In patients being investigated for chronic constipation, radio-opaque markers are ingested and an AXR is taken 5 days later with passage of <80% of the markers considered abnormal.
- Barium enema widely used investigation in colorectal disease. There will be characteristic "thumbprint" filling defects caused by edematous mucosa in ischemia/infarction. The extent of ulcerative colitis can be assessed and in Crohn's disease it will show skip lesions, areas of stricturing, and any fistulae. It will reveal the presence of diverticula. Barium enema is useful in the detection of large polyps and carcinomas

with constricting tumors producing a characteristic "apple core" lesion. However, it will not reliably define rectal lesions.

- CT scan will detect a pericolic abscess (can be drained under CT guidance) and is useful in showing the site of a tumor and any metastatic spread. CT colonogram and barium enema can be of use in a medically unfit patient or where there is a distal stricture not passable by the colonoscope.
- MRI scan and ELUS allow assessment of local pelvic tumor spread in rectal carcinoma for staging purposes and selection for neoadjuvant therapy.
- PET CT scan helps to distinguish recurrent carcinoma from post-radiotherapy fibrosis in the pelvis and to detect occult distant metastases.
- Angiography will demonstrate a bleeding point, e.g., in angiodysplasia if there is active bleeding >2 ml/min.
- Cytology examination of ascitic fluid or peritoneal washings.
- Endoscopy and biopsy inspection and biopsy of the mucosa, determination of disease distribution, solitary or multiple lesions.
- Laparoscopy staging laparoscopy may be undertaken and any peritoneal deposits biopsied.
- CEA serum levels elevated in colorectal neoplasia particularly in metastatic or recurrent disease.

6.4 Pathological Conditions

6.4.1 Non-neoplastic Conditions

These comprise inflammatory (acute or chronic), mechanical, ischemic, and iatrogenic disorders.

6.4.1.1 Inflammatory Disorders

Acute proctocolitis: Infective or drug-induced, e.g., antibiotics, there is preservation of the mucosal architecture and acute inflammation with biopsies only being submitted if symptoms persist beyond several weeks. Infective cases (campylobacter, shigella, salmonella) are usually self-limited and culture positive in only 40% of

cases. Drug-induced inflammation often responds to its withdrawal.

Chronic proctocolitis: Characterized by disturbance of the mucosal architecture and a chronic inflammatory cell infiltrate ± foci of active inflammation. Commonly due to idiopathic chronic inflammatory bowel disease (CIBD) but also seen overlying diverticulosis and pneumatosis coli, in infection (shigella, amoebiasis, schistosomiasis), obstructive enterocolitis, and with drugs. Microbiological culture and travel and drug history should always be ascertained in patients with chronic diarrhea.

CIBD – ulcerative colitis and Crohn's disease: The latter has been discussed previously (see Chap. 5) but can present either as isolated colonic disease or associated with ileitis. It is a segmental, transmural chronic inflammatory condition and there is often rectal sparing but anal disease (fissure, fistula, abscess) present. The segmental distribution, focality of inflammation, presence of granulomas, and ileal component are all useful diagnostic pointers in colonoscopic biopsy or resection specimens. Recurrence elsewhere in the gut is not uncommon despite surgical resection, and, because of this, Crohn's disease is a contraindication to pouch formation in restorative proctocolectomy. Occasionally it presents isolated to the appendix or sigmoid colon coexisting with diverticulitis.

In contrast to this ulcerative colitis is a diffuse, chronic active mucosal inflammatory condition involving the rectum and a contiguous length of large intestine, e.g., left-sided proctocolitis or pancolitis. It is of variable severity with episodic exacerbations and remissions - acute fulminant colitis may be complicated by severe hemorrhage, toxic dilatation or megacolon, perforation, and peritonitis. Other complications include mucosal dysplasia and malignancy (usually adenocarcinoma) in extensive disease of long-standing duration (pancolitis >10 years). Villiform or polypoid DALMs (dysplasia-associated lesions or masses) can be difficult to distinguish from the much more common inflammatory mucosal polyps and may harbor underlying adenocarcinoma. Alternatively dysplasia may occur in flat mucosa, and colonoscopic surveillance of chronic colitis involves sequential mucosal sampling as well as target biopsy of any macroscopic abnormality.

Biopsy orientation onto a polycarbonate strip aids subsequent localization of any histological abnormalities. Macroscopically ulcerative colitis shows mucosal granularity, linear or confluent ulceration, and polyps of varying size. The terminal ileum is only involved in severe pancolitis over a length of 1–2 cm (backwash ileitis) and although there is usually proctitis, the rectum may be spared due to treatment effects, e.g., predsol enemas. Extraintestinal effects include arthritis, iritis, and, in the liver, primary sclerosing cholangitis which can lead to cirrhosis and cholangiocarcinoma.

In a minority of cases, clear distinction cannot be made between ulcerative colitis and Crohn's disease on macroscopic/colonoscopic and microscopic examination – so-called indeterminate colitis (in a resection specimen) or CIBD, unclassified (in biopsy material).

Diversion proctocolitis: Follows fecal stream diversion, e.g., after ileostomy or colostomy for tumor, trauma, or CIBD. The defunctioned segment develops florid reactive lymphoid hyperplasia which can be mucosal or transmural, mimicking or superimposed on an underlying inflammatory disorder such as CIBD. Persistent severe symptoms may necessitate surgical excision of the segment, e.g., the rectal stump following colectomy for ulcerative colitis.

Microscopic colitis: Minimal inflammation may be apparent grossly or histologically for various reasons, e.g., treated CIBD, postinfection, drug ingestion, uremia, stercoral trauma, etc. However, microscopic colitis which causes chronic, voluminous watery diarrhea is radiologically and colonoscopically normal. It occurs in middle-aged to elderly women and has variable associations with HLA type, autoimmune diseases, and NSAID ingestion. Diagnosis is by histology with a normal architecture and transmucosal infiltrate of chronic inflammatory cells. Its main variants, collagenous and lymphocytic colitis, show a thickened subepithelial collagen band and excess surface intraepithelial lymphocytes, respectively. Not infrequently there is spontaneous resolution or response to antiinflammatory therapy.

Infective proctocolitis: Investigation includes microbiological culture with microscopy for

cysts (amoebiasis) and ova (schistosomiasis). Infection should be considered particularly where there is a history of travel or immunosuppression, e.g., AIDS, chemotherapy or post-transplant. In immunosuppression, infection with unusual opportunistic organisms can occur, e.g., cryptosporidiosis, atypical mycobacteria.

6.4.1.2 Mechanical Disorders

Melanosis coli: Characterized by pigmented macrophages in the lamina propria that impart a dusky mucosal appearance mimicking ischemia. The pigment is lipofuscin and degenerative in nature thought to relate to cellular apoptosis. There is an association with use of laxatives and bowel dysmotility.

Volvulus: Usually comprises a markedly dilated atonic sigmoid colon in either Africans (due to a high-fiber diet with bulky stools) or constipation-related-acquired megacolon in the elderly. The sigmoid loop twists on its mesentery, obstructs, and may become secondarily ischemic. Resection specimens are often dilated, thinned, and featureless. Melanosis coli may be present. Congenital megacolon and Hirschsprung's disease are discussed elsewhere (see Chap. 21).

Pneumatosis coli: Submucosal gas cysts lined by macrophages and giant cells with overlying mucosal chronic inflammation or pseudolipomatosis. There is an association with volvulus, constipation, diverticulosis, and chronic obstructive airways disease. Pathogenesis relates to retroperitoneal tracking of air into the bowel mesentery, abnormal luminal gas production linked to the increased intraluminal pressure seen in the above disorders, and introduction of gas during endoscopy. About 50% of cases resolve, but recurrent or severe lesions may require colectomy of the involved segment.

Obstructive enterocolitis: Continuous or segmental areas of inflammation or ulceration adjacent to or distant from an obstructing distal lesion, e.g., annular carcinoma or diverticulosis. Small bowel may also be involved with mimicry of Crohn's disease. A dilated, thinned cecal pouch can become ischemic and perforate.

Diverticulosis: Very common in Western society due to a low-fiber diet, high intraluminal pres-

and subsequent transmural mucosal herniation in the sigmoid colon through points of vessel entry from the mesentery. Presentation is with altered bowel habit, per rectum bleeding, left iliac fossa pain or a mass. The latter implies diverticulitis with possible perforation and pericolonic reaction/abscess formation. pyemia, liver abscesses, and peritonitis can ensue. The diverticular segment is thickened and contracted with muscle coat hypertrophy and visible diverticular pouches in the muscularis and mesenteric fat. They may be filled and obstructed with fecal or vegetable debris, and ulcerated with a coating of pericolonic exudate and abscess. The concertina-like redundant mucosal folds can show crescentic colitis due to abrasion of their tips by the passing fecal stream. Occasionally the chronic inflammation may be transmural and granulomatous mimicking or coexisting with Crohn's disease. Treatment is often conservative, e.g., by diet alteration, but severe or complicated cases require colectomy. Copresentation with an occult carcinoma within the strictured segment must be excluded by careful pathological examination.

Mucosal prolapse: A mechanism producing reactive mucosal changes of crypt hyperplasia, smooth muscle thickening of the lamina propria, and variable surface erosion. It is common to a number of situations including solitary rectal ulcer syndrome (SRUS), inflammatory cloacogenic polyp, diverticular-related crescentic colitis, mucosa adjacent to a polyp, stricture or tumor, stercoral trauma, and the mucocutaneous junction of stomas. In SRUS, there is a history of abnormal anterior rectal wall descent due to straining at defecation. This results in induration of the wall that can mimic a plaque of tumor on palpation and rectoscopy. Biopsy is diagnostic and treatment is usually conservative, related to better stool habit - occasional cases require resection of the involved sleeve of mucous membrane with apposition and plication of the intervening muscle (Delorme's procedure).

6.4.1.3 Ischemic Disorders

The pathogenesis of intestinal ischemia has been previously discussed and in the large intestine is often due to mesenteric vascular insufficiency because of systemic hypotension (myocardial infarction, cardiac arrhythmia, blood loss) or mesenteric atheroma/thrombosis/embolism. Acute lesions may resolve if mucosa-confined but are potentially fatal if transmural. Late or chronic ischemia has a predilection for the splenic flexure and rectosigmoid watershed areas of vascular supply. This can result in nonspecific ulceroinflammatory and stricturing lesions - end-stage changes that can be produced by various other conditions, e.g., CIBD, infection (E. coli 0157:H7 bacterium), pseudomembranous colitis due to Clostridium difficle overgrowth, obstructive enterocolitis, and stercoral trauma. Occasional cases are due to vasculitis or amyloid infiltration. Assessment of resection limit viability and mural/ mesenteric vessels is necessary in ischemia.

A vascular abnormality that can present with iron-deficiency anemia in elderly patients is colonic angiodysplasia. Thought to be degenerative in nature due to increased intraluminal pressure compressing mural vessels, the commonest site is the cecum. Operative injection of radio-opaque contrast may be needed to demonstrate areas of vascular ectasia so that targeted blocks can be sampled. The ectatic vessels involve the submucosa and lamina propria.

6.4.1.4 latrogenic Disorders

These include drugs, radiation therapy, and graft versus host disease.

Drugs: NSAIDs should always be considered in the presence of any unusual colitis, localized ulceration, stricture, perforation, or mucosal diaphragm formation. Antibiotics can commonly cause dysfunctional diarrhea, an acute proctocolitis, or, particularly in the elderly, pseudomembranous colitis. The latter is due to the production of Clostridium difficile toxin leading to ischemictype lesions with yellow surface plaques of acute inflammatory and fibrinous pseudomembrane. Severe cases result in end-stage ulceration and colectomy may be indicated although initial treatment is with appropriate antibiotics.

Radiation therapy: Acute and chronic phases with the potential for mucosal healing and usually produced by radiotherapy for pelvic (uterine

cervix, rectum, prostate) or retroperitoneal cancer. Acute radiation proctocolitis is normally self-limited and seldom biopsied. Chronic changes result in mucosal atrophy, hyaline fibrosis, vascular thickening, and strictures.

Graft versus host disease: Immunosuppressed bone marrow transplant patients risk developing a range of acute and chronic changes similar to those seen in radiation damage.

6.4.2 Neoplastic Conditions

Serrated polyps: Simple hyperplastic or metaplastic polyps are benign and more prevalent in the left colon/rectum with increasing age. Sessile serrated polyps/adenomas are recently recognized lesions thought to be precursors of rightsided serrated pathway carcinomas, typically in elderly females. Traditional serrated adenomas are more commonly distal and share some morphological features and cancer risk of conventional adenomas.

Adenoma (conventional): Designated as tubular, tubulovillous, or villous, depending on the relative proportions of glands and fronds present and composed of low- or high-grade dysplastic epithelium. Increasing in frequency with age, and in the left colon, the risk of malignancy relates to the size (>2 cm=40-50% risk), degree of villous morphology, and grade of dysplasia. Tubular adenomas are nodular and tend to develop a distinct stalk, whereas villous lesions are sessile. Stalked adenomas can twist and prolapse (typically in the sigmoid colon) resulting in glandular herniation into the submucosa that mimics invasive carcinoma - the low power lobular configuration, the presence of lamina propria hemosiderin, and lack of stromal fibrous desmoplasia are useful histological clues to benignity. Invasive carcinoma is defined by the presence of neoplastic epithelium infiltrating submucosa, and in stalked adenomas, polypectomy may be considered therapeutic if the tumor is well or moderately differentiated and does not show lymphovascular invasion or involvement (<1 mm) of the diathermied polyp base. Otherwise colonic resection is required and, therefore, good orientation of the adenoma to its stalk and assessment of the base are crucial. In contrast, invasion in a sessile adenoma accesses true mural submucosa, and colonic resection is usually considered more appropriate based on greater risk of lymph node metastases, unless the patient is very elderly or medically unfit. Local mucosal resection is an option, but in such cases further radical surgery is required if the cancer involves muscle coat, the base of the specimen, lymphovascular channels, or is poorly differentiated.

It is not unusual for patients to have several sporadic adenomas, but in FAP, there are hundreds or thousands with progression to colorectal cancer 20–30 years earlier than average, indicating a need for prophylactic colectomy. There is also a strong association of FAP with duodenal adenomas and periampullary carcinoma.

Flat adenomas are less common and difficult to identify macroscopically without the use of magnification or dye spray technique. They have proportionately higher grades of dysplasia and frequency of carcinoma and may account for a proportion of the 30% of carcinomas without an identifiable adenoma at their edge.

In the UK, the National Bowel Cancer Screening Programme targets the detection of adenomas in asymptomatic patients in an attempt to prevent cancer formation. It invites the population aged 60–69 years to participate in 2-yearly fecal occult blood testing. About 2% have a positive result and are referred for colonoscopy (or CT colonogram, depending on fitness and availability of local resources) and 50% of these will have a detectable abnormality (80% adenoma or tumor: 20% others, e.g., CIBD). Initial results have shown a significant yield of precancerous adenomas and a shift toward a higher frequency of early stage (Dukes' A) cancers. Future plans for the screening program include age extension to 75 years and one-off flexible sigmoidoscopy at age 55 years.

Adenocarcinoma: Comprising the vast majority of colorectal malignancies, 80–85% are moderately differentiated adenocarcinoma of no special type. A minority are mucinous, signet ring cell, or poorly differentiated. Distribution is throughout the colorectum although rectosigmoid is the commonest site (50% of cases), 10–15% of sporadic cases are multiple, occurring either synchronously

or subsequently/metachronously. Predisposing conditions are chronic ulcerative colitis, FAP, and hereditary non-polyposis colorectal cancer (HNPCC) or Lynch syndrome. In HNPCC, there is a tendency for right-sided cancers, which may be multiple, mucinous, or poorly differentiated and with a family history of cancer at a younger age (<50 years), also involving other sites, e.g., uterus, stomach, ovary, ureter, and small intestine. Its genetic basis is different from that of sporadic colorectal cancer, due to a deficiency in one of the DNA mismatch repair proteins caused by a heritable germline mutation.

As previously noted, the cancer site and its macroscopic growth pattern influence clinical presentation. Important prognostic indicators are the extent of local tumor spread, a circumscribed or infiltrative margin, involvement of the serosa, longitudinal or mesocolic/mesorectal resection margins, and tumor perforation. Tumor present within ≤1 mm defines involvement of the mesenteric margin irrespective of whether it is nodal, lymphovascular or direct spread. Generally a macroscopic clearance of 2-3 cm from a longitudinal margin is satisfactory unless histology shows the cancer to be unusually infiltrative or poorly differentiated. All mesenteric lymph nodes should be identified, counted, and sampled (aiming for a departmental median count of at least 12 nodes per specimen) and a suture tie limit node identified – in some colectomy specimens this may mean more than one. Involvement of adjacent organs or structures (e.g., abdominal wall) is documented and predisposing lesions such as adenoma(s) or colitis represented. Multiple tumors are dissected and staged individually with respect to mural and nodal spread.

Other cancers: Carcinoid tumors are usually small incidental mucosal rectal polyps, GISTs are rare, and malignant lymphoma can complicate ulcerative colitis or AIDS.

Prognosis: Relates mainly to the depth of tumor spread, lymph node involvement, and adequacy of local excision with overall 5-year survival 35–40%. Cancers confined to the mucous membrane or wall do much better than those that invade beyond this or show nodal disease. Adverse prognostic indicators also include a mucinous character, poor differentiation, tumor

perforation, obstruction, and resection margin involvement. It is estimated that about 50% of patients are cured, 10% die from local recurrence and 40% from lymphatic and vascular spread. Treatment is typically surgical excision with adjuvant chemotherapy considered for cancers showing poor differentiation, nodal, peritoneal, and/or extramural vascular spread, tumor perforation, or resection margin involvement. Rectal cancers often receive 5-day short-course preoperative radiotherapy in an attempt to downstage the lesion or facilitate resection. This usually does not produce the marked macroscopic and histological features of regression that can be seen with the alternative 6-week-long course of neoadjuvant chemoradiotherapy. The latter is given to patients with clinically fixed tumors that show significant spread on MRI scan into the mesorectum, its nodes, or near its investing fascia (circumferential radial margin: CRM).

6.5 Surgical Pathology Specimens: Clinical Aspects

6.5.1 Biopsy Specimens

A number of procedures can be undertaken to obtain biopsy specimens from the colorectal mucosa:

Proctoscopy is used to inspect the distal rectum and anal canal.

Sigmoidoscopy can be carried out by using either a rigid or flexible sigmoidoscope. Rigid sigmoidoscopy is usually done without bowel preparation at the bedside or in the outpatient clinic. A hollow rigid plastic tube measuring 25 cm in length with an attached light and air supply is inserted into the rectum up to the distal sigmoid colon. Forceps can be passed through the tube to biopsy any lesion visualized. The scope is also used to assess tumor fixation and its distance from the anus. Flexible sigmoidoscopy (and colonoscopy) involves formal bowel preparation. A flexible fiber-optic endoscope is inserted and works in the same way as an upper GI endoscope, with a controllable tip and ports for inserting instruments, e.g., forceps, snare, etc. This should visualize up to the proximal sigmoid colon.

Colonoscopy is carried out using a colonoscope which is essentially a longer sigmoidoscope, with scopes of different lengths available (ranging from 140 to 185 cm). An experienced endoscopist should be able to pass the endoscope through the ileocecal valve to visualize the terminal ileum. Intraoperative endoscopy can be used during a laparotomy to, for instance, locate lesions, e.g., polyps found by barium enema that require localized resection and which cannot be palpated by the surgeon.

Biopsy specimens can be taken from the colonic mucosa by forceps passed through the endoscope in much the same way as that used in upper GI endoscopy. The colonoscopic management of polyps is important and depends on the size and type of polyp:

- Large pedunculated polyps can be removed by "snaring." A circular wire is passed over the polyp onto its stalk. An electrical current is passed along the wire to coagulate the vessels in the base of the stalk, which is then transected by closing the wire. If the stalk is large, adrenaline can be injected into the base to minimize bleeding. The polyp is retrieved by using the snare, a Dormia basket or suction.
- Smaller polyps (5–7 mm) can also be snared and removed by suction.
- Polyps <5 mm can be removed by "hot biopsy."
 Biopsy forceps grasp the polyp and a current
 is applied to electrocoagulate the base, and
 then the head of the polyp is pulled off by the
 forceps.
- Broad-based sessile polyps can either be removed piecemeal using the snare or by injection polypectomy. This involves injecting adrenaline solution into the submucosa around the polyp, raising it, and allowing it to be snared completely. This method can be used for polyps up to 5 cm in diameter.
- In patients with multiple small polyps, these can be highlighted by spraying dye onto the mucosa. This will reveal polyps 0.5 mm and larger as pale areas on a blue background.
- If the endoscopist is concerned that a polyp may be malignant, the site of polypectomy can be marked by tattooing the bowel mucosa with India ink. This allows the site to be revisited at a later date.

Submucosal lesions can be sampled by endoscopic FNA. Colonoscopy may also be used as a therapeutic tool, e.g., foci of angiodysplasia may be coagulated using hot biopsy forceps.

6.5.2 Resection Specimens

Resection of the colon and rectum is performed for a wide variety of both non-neoplastic and neoplastic conditions (Table 6.1), the type of procedure depending on the site and nature of the lesion, e.g., a malignant tumor, will require a more extensive resection than that for a large adenomatous polyp. Likewise the extent of mesenteric resection will depend on the type of lesion, i.e., wide mesenteric resection for neoplastic lesions and limited resection for nonneoplastic conditions. It also depends on the "intention" of the surgery for a malignant condition, i.e., a wide mesenteric resection with proximal ligation of vessels and, hence, removal of lymph node groups if the intention is curative, or limited, if the disease is advanced and the intention is palliative. The variety of terms used to describe the different types of colonic resection (colectomy) is depicted in Fig. 6.5. Choice is also determined by the distribution or multiplicity of lesions detected at preoperative colonoscopy. Planned elective laparoscopic sur-

Table 6.1 Colorectal resections

Specific	Diverticular disease
	Volvulus
	Pneumatosis coli
	Colonic angiodysplasia
	Rectal stump (CIBD, diversion proctitis)
	Rectal mucosa (prolapse)
Ulceroinflammatory	Ulcerative colitis
	Crohn's disease
	Pseudomembranous colitis
	Ischemia
Neoplasia	Large or multiple
	adenomas
	Carcinoma
	Malignant lymphoma

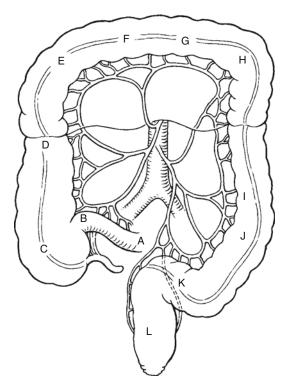


Fig. 6.5 Types of colonic resection. A → C Ileocecectomy; $\pm A + B \rightarrow D$ Ascending colectomy; $\pm A + B \rightarrow F$ Right hemicolectomy; $\pm A + B \rightarrow G$ Extended right hemicolectomy; $\pm E + F \rightarrow G \pm H$ Transverse colectomy; $G \rightarrow I$ Left hemicolectomy; $F \rightarrow I$ Extended left hemicolectomy; J + K Sigmoid colectomy; $\pm A + B \rightarrow J$ Subtotal colectomy; $\pm A + B \rightarrow L$ Total colectomy; $\pm A + B \rightarrow L$ Total proctocolectomy; L Proctectomy (Reprinted, with permission, from Fielding and Goldberg (2002))

gery is the preferred option – open abdominal surgery (laparotomy) may be necessary for extensive disease, or if the patient presents as an acute emergency.

6.5.2.1 Resection in Neoplastic Conditions

Adenomatous polyps – As discussed above, the majority of adenomatous lesions can be removed by endoscopic techniques. However, large sessile polyps >5 cm in diameter and occupying more than one-third of the colon circumference should be removed by a localized resection. Sessile adenomas in the rectum can be removed by *transanal submucosal resection*. In this procedure, adrenaline solution is infiltrated into the submucosa around the lesion and the mucosa is incised by scissors 1 cm from the lesion. This can then be

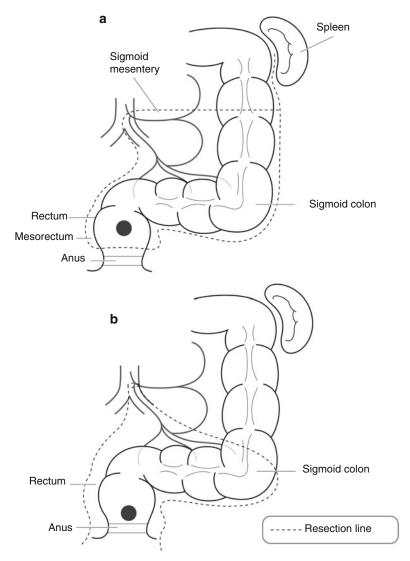
easily lifted off the circular muscle in a single piece and the mucosal defect is closed by sutures. This "advanced" polypectomy with submucosal infiltration is termed endoscopic mucosal resection (EMR). Extensions of this are endoscopic submucosal dissection (ESD) and transanal endoscopic microsurgery (TEMS) providing complete mucosectomy to full-wall-thickness specimens. These "big biopsy" specimens are both diagnostic (benign vs. malignant) and potentially therapeutic, allowing assessment of risk factors that might necessitate subsequent radical surgery, e.g., deep margin status, substaging of submucosal invasion, and lymphovascular involvement in carcinoma. Occasionally large rectal polyps may require formal proctectomy or anterior resection.

Malignant lesions – The type of resection for colonic tumors will depend on the site of the lesion and the intent of the surgery. As previously stated, the colonic lymphatics accompany the main blood vessels and the extent of resection depends on the lymphatic clearance required. In cancer operations of curative intent, the affected colon with its lymphovascular mesenteric pedicle is resected. Continuity is restored by either an ileocolic or colocolic end-to-end anastomosis. However, on occasion, an end ileostomy/colostomy may be required if the surgeon thinks that primary anastomosis would be compromised (e.g., if there is extensive intraperitoneal contamination).

The curative resection of rectal tumors may be carried out by one of two methods:

Anterior resection of rectum - In this procedure, the rectum is mobilized by entering the fascial plane around the mesorectum. This allows the rectum to be removed en bloc with the mesorectum which contains the initial draining lymphovascular channels and nodes (low anterior resection and total mesorectal excision – TME) (Fig. 6.6a). Continuity is reestablished by a stapling device forming an endto-end colorectal anastomosis. Occasionally, in low anastomoses, a protective loop colostomy/ ileostomy may be fashioned to divert the fecal stream. This can be closed at a later date. To obtain an adequate length of colon to form a safe anastomosis, the splenic flexure will usually need to be mobilized. On occasion, the

Fig. 6.6 (a) Resection in low anterior resection and total mesorectal excision; (b) resection in abdominoperineal excision (Reproduced, with permission, from Allen and Cameron (2004))



spleen may be damaged during this mobilization and a splenectomy would then have to be performed. In cases where the tumor is in the proximal rectum, a high anterior resection and mesorectal division can be employed. This entails division of the rectum and mesorectum 5 cm distal to the tumor and allows a larger rectal stump for anastomosis.

 Abdominoperineal (AP) excision of rectum (APER) – In this procedure, the rectum is mobilized as above and the colon is divided at the apex of the sigmoid. The anal canal and distal rectum are then resected from below via the perineal route (Fig. 6.6b). The entire rectum (and mesorectum) and anus are then removed en bloc. The perineal wound is closed and a permanent end colostomy is fashioned in the left iliac fossa using the transected end of the sigmoid colon.

Until the early 1980s, anterior resection was used in less than 50% of patients with rectal tumors, i.e., those in the proximal rectum. However, it is now used for approximately 90% of tumors in the rectum. Initially it was feared that because less tissue is excised and the clearance of the distal margin is not as great during

anterior resection, there would be increased local recurrence rates if anterior resection was used for low rectal tumors. However, it appears that the degree of lateral clearance is similar in the two procedures and that a distal clearance of 2 cm is adequate to prevent local recurrence. Given the physical and psychological problems associated with a permanent colostomy, and the higher incidence of bladder and sexual problems in patients undergoing AP resection, it is felt that a sphincter-saving procedure (i.e., anterior resection) should be employed whenever possible. However, tumors extending to less than 2 cm from the anorectal junction (i.e., less than 6 cm from the anal verge) should be treated by AP resection.

Occasionally, in a medically unfit patient, localized resection is used for a well-differentiated, pT1 rectal cancer that is <3 cm in diameter. Accurate preoperative staging is crucial in selection of these patients and some may then need to proceed to salvage resection if adverse pathological features are identified in the pathological specimen, e.g., poor differentiation, lymphovascular involvement, or invasion of the deep margin or muscle coat. Sometimes patients with obstructing cancers undergo piecemeal resection (essentially palliative and non-curative), partial laser ablation, or stenting to restore intestinal continuity and avoid the risk of perforation. This may even allow resection to be carried out more safely at a later date.

6.5.2.2 Resection in Non-neoplastic Conditions

Hartmann's procedure – This is one of the most commonly used emergency operations for colorectal disease. Although this was initially devised for the elective treatment of proximal rectal tumors, it is now usually used in the emergency setting to treat conditions such as perforated diverticular disease (most commonly), perforated tumor, etc. The procedure itself is defined as resection of the sigmoid colon (and a variable length of proximal rectum if required) with the fashioning of a terminal-end colostomy and closure of the rectal stump. The colostomy may be reversed at a later date by forming an end-to-end colorectal anastomosis.

Nonacute-presenting diverticular disease is usually treated surgically by either sigmoid colectomy or left hemicolectomy depending on the extent of the disease.

Surgery in colorectal inflammatory bowel disease – The surgical management of colorectal Crohn's disease is similar to that in the small intestine (see Chap. 5). Namely surgical intervention is reserved for those in whom medical management has failed (i.e., minimal resection of the diseased segment) or who are suffering complications, e.g., obstruction, pericolic abscess, fistula, etc.

As in Crohn's disease, close liaison between surgeons and physicians is required in the management of ulcerative colitis. Emergency surgery is needed in cases of acute severe colitis and/or toxic megacolon. The procedure of choice is a subtotal colectomy and end ileostomy with the proximal end of the rectum brought to the surface in the form of a mucus fistula. This spares an already sick patient the added trauma of pelvic surgery and, if ulcerative colitis is confirmed by histological examination, allows an ileoanal pouch procedure to be considered in the future. Prior to the mid-1970s, patients with refractory ulcerative colitis underwent a panproctocolectomy (removal of the colon, rectum, and anus) with a permanent end ileostomy. However, in 1976, the procedure of restorative proctocolectomy was introduced and removed the need for a permanent ileostomy in suitable patients. In this procedure, the entire colon and rectum are removed and the mucosa may be stripped from the upper anus above the dentate line (some surgeons prefer to leave this mucosa intact as it is thought to improve future continence). An ileal reservoir (pouch) is formed (Fig. 6.7) and an ileoanal anastomosis is fashioned. A protective loop ileostomy is formed as close to the ileal pouch as possible and this can be closed at a later date (usually 2–3 months) after healing has been completed. A proportion of these patients (approximately 10%) may develop "pouchitis" – increased frequency of stool and feeling generally unwell. The exact etiology of this is unknown but some feel it may be due to bacterial overgrowth in the pouch.

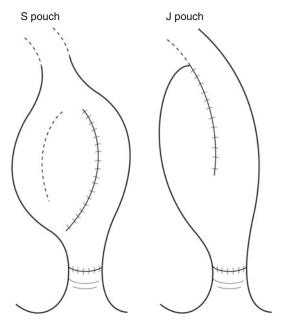


Fig. 6.7 Two popular designs of ileal pouch (Reproduced, with permission, from Allen and Cameron (2004))

Angiodysplasia – If bleeding is severe enough to require surgical intervention, and if conservative treatment such as endoscopic coagulation has been unsuccessful, the procedure of choice will be dictated by the site of the bleeding point(s). However, if the site of bleeding cannot be discovered, a total colectomy with ileorectal anastomosis (or end ileostomy, rectal mucus fistula, and reversal at a later date) may be required.

6.6 Surgical Pathology Specimens: Laboratory Protocols

6.6.1 Biopsy Specimens

For biopsy specimens and local mucosal resections see Chap. 1.

6.6.2 Resection Specimens

Specimen:

 Colorectal specimens are for a range of either specific, ulceroinflammatory, or neoplastic conditions (Table 6.1). These can be complicated by obstruction with or without associated enterocolitis or perforation or show evidence of background disease such as CIBD or FAP. The resection specimen is dictated by the site and nature of the abnormality and extent of any complications or predisposing lesions that are present.

Initial procedure:

- In general, specimens are measured, opened with blunt-ended scissors along the antimesenteric border, and then blocked longitudinally (but see diverticular disease and tumor) following gentle washing out of fecal debris, pinning out with avoidance of unnecessary traction, and immersion in 10% formalin fixative for 48 h. Photographs may be taken before and after dissection.
- When opening avoid areas of perforation or tumor. Tumor segments may either be left unopened for fixation and subsequent transverse slicing or carefully opened – the latter gives better fixation, but the cut should be guided by palpation with the index finger to avoid disturbing the relationship of the tumor to the circumferential margin.

6.6.2.1 Diverticular disease

- Measurements: length × diameter (cm) of the thickened colonic segment
- Inspect and describe: perforation, fistula, pericolonic exudate, or abscess
- Open and fix
- Serially transverse section at 5 mm intervals
- Sample (four blocks minimum) the diverticula, any associated inflammation, or thickened mucosa that might represent crescentic colitis, mucosal prolapse, or tumor
- Sample mesenteric lymph nodes

6.6.2.2 Volvulus, pneumatosis coli, rectal stump, rectal mucosa in prolapse

- Measurements: length × maximum diameter (cm)
- Open and fix
- Inspect and describe
 Volvulus dilatation, thinning, melanosis, stercoral ulceration, ischemia, perforation

Pneumatosis – mucosal cobbling, blebs or gas cysts, inflammation, ulceration, perforation Rectal stump – mucosal granularity, ulceration, polyps, fistulae, tumor Rectal mucosal prolapse – mucosal granular-

Rectal mucosal prolapse – mucosal granularity, thickening, induration, ulceration

- Sample (four blocks minimum) macroscopically normal and abnormal areas as indicated
- · Sample mesenteric lymph nodes

6.6.2.3 Ulceroinflammatory and neoplastic conditions

- Open and inspect
- · Measurements:

Lengths and maximum diameter (cm) of the parts present – terminal ileum, appendix, colon, rectum, anus

Lengths (cm) of ischemic, inflamed, or strictured segments

Maximum dimensions (cm) of any perforation(s), ulcer(s), polyp(s) or tumor(s) Distances (cm) of the abnormality from the proximal and distal resection limits

Distances (cm) of the polyp/tumor/ulcer from the anorectal dentate line and relationship to the peritoneal reflection (above/straddling/ below) and colorectal circumference (anterior/ posterior/right or left lateral)

Distances (mm) of tumor from the nearest aspect of the mesocolic/mesorectal CRM

- Grade the plane of mesorectal excision (mesorectal fascia, intramesorectal, muscularis propria)
- Photograph
- Paint any aspect of the mesocolic/mesorectal margin adjacent to or overlying tumor Gently pin out and fix for 48 h Description:
- Tumor
 - Site

Ileocecal valve/cecum/colon (which segment, flexure)/rectum (above, straddling, or below the peritoneal reflection and upper, mid, or lower, anterior, posterior, or lateral)/anus

Lumenal/mural/extramural/mesenteric

Size
 Length × width × depth (cm) or maximum dimension (cm)

Appearance

Polypoid/nodular – adenoma, carcinoma, carcinoid, multiple lymphomatous polyposis, GIST

Ulcerated/stricture – carcinoma, malignant lymphoma, metastatic carcinoma

Fleshy/rubbery – malignant lymphoma, GIST

Multiple – adenomas, carcinoma (primary or metastatic), malignant lymphoma

- Edge: Circumscribed/irregular
 Perforation
 Adjuvant therapy changes: necrosis, ulceration, fibrosis
- CIBD ulcerative colitis: contiguous/diffuse mucosal distribution, granularity, ulceration (linear/confluent), inflammatory polyps, synechiae, nodular or sessile DALMs, tumor, mucosal reversion with healing and atrophy, backwash ileiitis, treatment-related rectal sparing
 - Crohn's disease: segmental/transmural distribution, cobblestone mucosa, ulceration (aphthous/linear/confluent), stricture, fat wrapping, fistula, polyps or tumor, lymphadenopathy, adhesions, abscess formation, ileal/anal disease
- Ischemia serosal hyperemia/constriction band, mucosal hyperemia/hemorrhage/ulceration/necrosis, wall thinning/perforation/stricture
- Pseudomembranous colitis pseudomembranes (adherent/yellow), mucosal granularity/erosion/ulceration, stricture
- Obstructive enterocolitis ulceration or stricture (contiguous or distant, diffuse or segmental), dilatation, wall thinning, perforation, ileal component

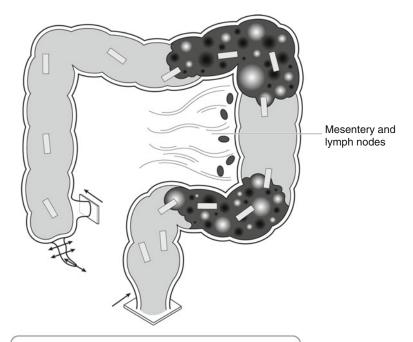
Blocks for histology:

Ulceroinflammatory conditions (Fig. 6.8)

- Sample by circumferential transverse sections the proximal and distal limits of resection
- · Sample macroscopically normal bowel
- Sample representative longitudinal blocks

 (a minimum of four) of any focal abnormality
 that is present to include its edge and junction
 with the adjacent mucosa, e.g., ulceration,
 stricture, fistula, perforation, pseudomembranes,

Fig. 6.8 Ulceroinflammatory colorectal conditions (Reproduced, with permission, from Allen and Cameron (2004))



- 1. Sample the ileal and colorectal resection limits
- 2. Process the appendix as usual
- 3. Sample representative blocks of any abnormality including the junction with adjacent mcosa
- 4. Sequentially sample normal bowel
- 5. Sample mesenteric lymph nodes

inflammatory polyps, serosal adhesions or constriction bands. Also

- CIBD: Sequential labeled samples at 10 cm intervals from cecum to anus and additional blocks from any unusual nodular or sessile abnormality (DALM)
- Ischemia: Sample the mesenteric vessels
- Sample mesenteric lymph nodes and any other structures, e.g., appendix or terminal ileum.

Neoplastic conditions (Fig. 6.9)

- Sample the nearest longitudinal resection margin if tumor is present to within <3 cm of it.
- Sample macroscopically normal bowel and representative blocks of other mucosal lesions that are present, e.g., adenomatous polyps (if multiple particularly those >1 cm diameter).
- Serially section the bulk of the tumor transversely at 3–4 mm intervals.
- Lay the slices out in sequence and photograph.
- Note and measure the relationship of the deep aspect of the tumor to the nearest site-

- orientated point of the serosa and the CRM. Note serosal tumor perforation or CRM involvement (≤1 mm).
- Sample (four blocks minimum) tumor and wall to demonstrate these relationships. With bulky mesentery/mesorectum, the block may have to be split and appropriately labeled for loading in the cassettes.
- Count and sample all lymph nodes and identify a suture tie limit node. Take care to count the nodes in the tumor slices and also those in the mesentery away from the tumor, e.g., sigmoid mesocolon in a rectal cancer.
- Sample multifocal serosal seedlings and omental deposits (pM disease) as indicated by inspection and palpation.

Histopathology report:

Ulcerative colitis – site-related disease activity (healed/quiescent/mild/moderate/severe), rectal sparing, appendiceal and cecal skip lesions, backwash ileiitis, toxic dilatation,

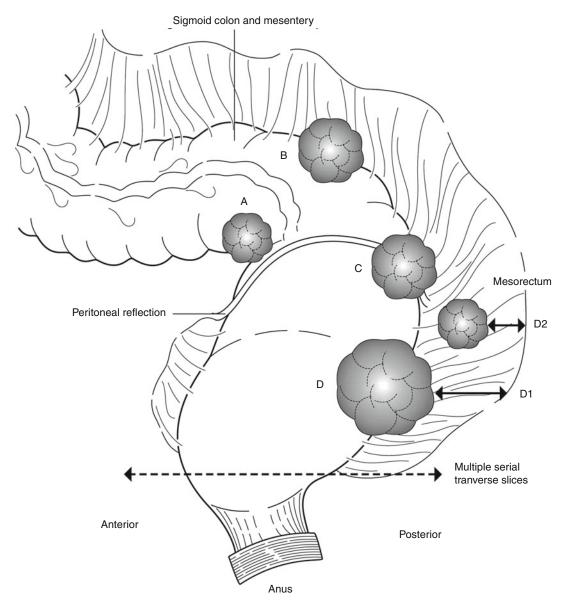


Fig. 6.9 Rectal carcinoma. The upper anterior rectum is invested in peritoneum. The anterior mesorectum is thinner (0.75-1 cm) than the posterior mesorectum (1.5-3 cm). Cut the resection specimen into multiple serial transverse slices about 3–4 mm thick. Blocks for histology are: Above the reflection: A tumor, rectal wall, and serosa; B tumor, rectal wall, and serosa; tumor, rectal wall, and mesentery; At the reflection: C tumor, rectal wall, and

serosa; tumor, rectal wall, and mesorectum; Below the reflection: *D* tumor, rectal wall, and mesorectum; *D1* distance (mm) of the deepest point of continuous tumor extension to the nearest point of the painted CRM; *D2* distance (mm) of the deepest point of discontinuous tumor extension (or in a lymphatic, node, or vessel) to the nearest point of the painted CRM (Reproduced, with permission, from Allen (2000))

- superimposed infection (e.g., CMV), DALMs, carcinoma, or lymphoma
- disease chronic transmural Crohn's inflammation, granulomas, fissures/fistulae, abscess formation, segmental distribution/ appendiceal/ileal disease, malignancy
- Ischemia necrosis (mucosal/transmural/gangrenous), resection limits (ischemic/viable), mesenteric vessels (thrombosis/embolism/vasculitis). miscellaneous (constriction band/volvulus/stricture)
- Pseudomembranous colitis pseudomembranes, ulceration, necrosis, perforation, strictures
- Obstructive enterocolitis note ulceration/ perforation/stricture/distribution and features specific to the etiological abnormality

Neoplastic conditions

- Tumor type adenocarcinoma/malignant lymphoma/other
- Tumor differentiation

pTis

- Adenocarcinoma Well or moderate/poor
- Malignant lymphoma MALT/mantle cell/follicular/Burkitt lymphoma/other

Low grade/high grade

- Tumor edge pushing/infiltrative/lymphoid response
- Extent of local tumor spread (for carcinoma) Carcinoma in situ: intraepithelial (within

r	basement membrane) or invasion of lamina propria (intramucosal) with no extension through muscularis mucosae into submucosa
pT1	Tumor invades submucosa
pT2	Tumor invades muscularis propria
рТ3	Tumor invades through the wall into subserosa or non-peritonealized pericolic/perirectal tissues
pT4	Tumor invades other organs or structures and/ or perforates visceral peritoneum

- Lymphovascular invasion present/not present
- · Regional lymph nodes
 - Pericolic, perirectal, those located along the ileocolic, colic, inferior mesenteric, superior rectal and internal iliac arteries; a regional lymphadenectomy will ordinarily include 12 or more lymph nodes

pN0	No regional lymph node metastasis
pN1	1–3 involved regional lymph node(s)
pN2	4 or more involved regional lymph nodes.

- In the UK, the Royal College of Pathologists currently recommend continuing using TNM 5 due to concerns over data comparison in ongoing clinical trials, the observer reproducibility in applying TNM 6 and TNM 7 rules (the subcategory details of which show some differences to TNM 5) and the lack of an evidence base for the proposed changes.

tumor limited to the wall,
node negative
tumor beyond the wall, node negative
nodes positive, apical node negative
apical node positive
distant metastases

Excision margins

Proximal and distal longitudinal (cm) and mesocolic/mesorectal

Circumferential (mm) limits tumor clearance

Other pathology

Tumor regression grade in response to neoadjuvant therapy, adenoma (s), FAP, ulcerative colitis, Crohn's disease, diverticular disease

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