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THE SMALL AND LARGE INTESTINES

Anatomy

- **Small intestine**
  - The length of the small bowel may vary from 300 to 850 cm between the duodenojejunal (DJ) flexure to the ileocecal valve.
  - The jejunum tends to have a wider diameter and a thicker wall, with more prominent mucosal folds (valvulae conniventes).
  - The ileum has a thicker, more fatty mesentery with more complex arterial arcades. The ileum also contains larger aggregates of lymph nodes (Peyer's patches), which can occasionally become lead points in intussusception in childhood.

- **Large intestine**
  - The large intestine begins at the ileocecal valve and extends to the anus. It is approximately 1.5 m long.
  - The large intestine is less mobile than the small bowel as the ascending and descending colon are fixed to the retroperitoneum. The colon is also distinguished by having fat-filled peritoneal tags known as ***appendices epiploicae*** and the presence of ***taeniae***. The taenia coli are three flat bands of longitudinal muscle that run the length of the large intestine from the appendix base to the rectosigmoid junction and they act to pull the colon into its typical sacculated state, producing a series of haustrations.

Physiology

- **Small intestine**
  The jejunum is the principal site for digestion and absorption of fluid, electrolytes, iron, folate, fat, protein and carbohydrate, but the absorption of bile salts and vitamin B12 occurs in the terminal ileum where there are specific transporters. If the jejunum is resected, the ileum can assume all the required absorptive functions, but resection of the terminal ileum will result in a diminished bile salt pool, B12 deficiency and may lead to deficiency of the fat-soluble vitamins A, D and K.

- **Large intestine**
  The principal function of the colon is absorption of water; 1000 mL of ileal contents enter the cecum every 24 hours of which only about 150–250 mL is excreted as feces. Fermentation of dietary fiber in the colon by the normal colonic microflora leads to the generation of short chain fatty acids (SCFAs) such as butyrate, which is an important metabolic fuel for the colonic mucosa and may also contribute to normal daily energy requirements.

Inflammatory Bowel Disease

By definition, the term 'inflammatory bowel disease' is reserved for conditions characterized by the presence of idiopathic intestinal inflammation, i.e. ulcerative colitis (UC) and Crohn's disease (CD). Although the availability of population genetics and molecular biology has contributed to our understanding of the pathogenesis of inflammatory bowel disease, the cause of these conditions ***remains unknown***.

Ulcerative Colitis

Ulcerative colitis is a disease of the rectum and colon with extraintestinal manifestations. It is most commonly diagnosed between the ages of 20 and 40. There is a marked geographical distribution of the disease which is far more common in the United States and Western Europe, but relatively rare in the Far East and the Tropics.

- **Pathological features**
  - Rectum, always involved unless already on topical treatment. Disease spreads proximally with increasing extent of disease.
  - Acute neutrophil infiltration of the colonic mucosa and submucosa; mucosal crypt abscesses with goblet cell mucin depletion.
  - With more severe inflammation, there are multiple aphthous ulcers, which may become confluent with only islands of inflamed mucosa and granulation tissue remaining (pseudopolyposis).
  - Chronic "burnt-out" disease leads to a pale, featureless, ahaustral pattern to the colon.
- **Clinical features**
  - ***Proctitis:*** Commonest presentation. Symptoms of urgency and frequency of defecation due to rectal irritability; bloody mucus mixed with loose stools.
  - ***Left-sided colitis:*** Disease up to the splenic flexure. Symptoms of rectal irritation plus extensive bloody mucus in stools, often leading to bloody diarrhea; mild associated systemic features.
  - ***Pancolitis:*** Disease involving the entire colon. May be associated with mild secondary inflammation of the terminal ileum (backwash ileitis). Bloody diarrhea predominant feature; systemic features common (fever, malaise, anorexia, tachycardia). May be associated with anemia (due to blood loss), hypoalbuminemia, and hypokalemia (due to mucus loss).
- **Extraintestinal manifestations**
  Sacroiliitis and ankylosing spondylitis are 20 times more common in patients with UC than the general population and are associated with HLA-B27. Sclerosing cholangitis is associated with UC and can progress to cirrhosis and hepatocellular failure. The skin lesions erythema nodosum and pyoderma gangrenosum normally get better with good colitis control. The eyes can also be affected with uveitis and episcleritis.
- **Investigations**
  - ***Endoscopy:*** Rigid/flexible sigmoidoscopy can detect proctitis in the outpatient setting. Colonoscopy and biopsy has a key role in diagnosis and management:
    - to establish the extent of inflammation.
    - to distinguish between UC and Crohn's colitis.
    - to monitor the response to treatment
    - to assess long-standing cases for malignant change.
  - ***Radiology:*** A plain abdominal X-ray is valuable in demonstrating the development of toxic megacolon. Barium enema has been replaced by computed tomography (CT), although a contrast study will give an excellent view of loss of haustra, especially in the distal colon, pseudopolyps and in chronic cases a narrow, featureless, shortened 'hosepipe' colon. CT findings in pancolitis may show significant thickening of the colonic wall, as well as inflammatory stranding in the colonic mesentery.
  - ***Bacteriology:*** A stool specimen should be sent for microbiology analysis when UC is suspected, in order to exclude infective colitides.

- **Treatment**
  Effective treatment of UC requires a multidisciplinary approach to management.
  - ***Medical treatment:*** Principles are to reduce inflammation and prevent complications. Acute derangements in blood results should be corrected (e.g. blood transfusion for severe anemia, potassium supplementation, nutritional support for hypoalbuminemia).
    Medical therapy is based on anti-inflammatory agents. The 5-aminosalicylic acid (5-ASA) derivatives can be given topically (per rectum) or systemically. They can be used long term as maintenance therapy. Corticosteroids are the mainstay of treatment for any 'flare up', either topically or systemically and have a widespread anti-inflammatory action. The immunosuppressive drugs azathioprine and cyclosporin can be used to maintain remission and as 'steroid-sparing' agents.
  - ***Surgical treatment:*** The greatest likelihood of a patient with UC requiring surgery (colectomy) is during the first year after diagnosis. Indications for surgery in UC are:
    - severe or fulminating disease failing to respond to medical therapy.
    - chronic disease with anemia, frequent stools, urgency and tenesmus.
    - steroid-dependent disease (remission cannot be maintained without substantial doses of steroids).
    - inability of the patient to tolerate medical therapy required to control the disease.
    - neoplastic change: patients who have severe dysplasia or carcinoma on review colonoscopy.
    - extraintestinal manifestations.
    - rarely, severe hemorrhage or stenosis causing obstruction.
- **Complications**
  - ***Acute***
    - Acute fulminant colitis and Toxic dilatation: Around 5 per cent of patients present with severe acute (fulminant) colitis characterized by frequent bloody diarrhea, weight loss and dehydration. Intensive medical treatment and fluid resuscitation leads to remission in 70 per cent, but the rest will require urgent surgery. Toxic dilatation should be suspected in patients with active colitis who develop severe abdominal pain and confirmed by the presence on a plain abdominal radiograph of colon with a diameter of more than 6 cm.
    - Perforation
    - Hemorrhage
  - ***Chronic***
    - Cancer: The risk of cancer in ulcerative colitis increases with duration of disease. At ten years from diagnosis, it is around 1 per cent. This increases to 10–15 per cent at 20 years and may be as high as 20 per cent at 30 years.
    - Extra-alimentary manifestations.

Crohn's Disease (Regional Enteritis)

It is characterized by a chronic full thickness inflammatory process that can affect any part of the gastrointestinal tract from the lips to the anal margin. It is most common in North America and Northern Europe It is slightly more common in women than in men, and is most commonly diagnosed in young patients between the ages of 25 and 40 years. There does, however, seem to be a second peak of incidence around the age of 70 years.

- **Pathological features**
  - The terminal ileum is most commonly involved, either in isolation or in combination with colonic disease. Colitis alone occurs in up to a third of cases and the remainder are patients with more proximal small bowel involvement. The stomach and duodenum are affected in around 5 per cent, but perianal lesions are common, affecting up to 50–75 per cent of patients.
  - CD is characteristically discontinuous, with inflamed areas separated from normal intestine, so-called 'skip' lesions.
  - Affected bowel looks blue-grey, thickened, with spiral surface vessels and encroachment of the mesenteric fat around the bowel (fat wrapping).
  - Transmural inflammation in the form of lymphoid aggregates, particularly in the subserosal tissues (Crohns rosary), mucosal crypt ulceration, and fissuring ulceration.
  - Non-caseating giant cell granulomas are found in 60 per cent of patients and clearly define Crohn's disease.
  - Mucosal thickening and serpiginous longitudinal ulceration combine to give the appearance of cobblestoning.
  - Perforation, fistulation, and abscess formation are occasional fistulizing sequelae of transmural inflammation.
  - Extensive fibrosis and smooth muscle hyperplasia may occur, giving rise to stenosis.
- **Clinical features**
  - Inflammatory features: Fever, malaise, abdominal pain (often RIF), change in bowel habit (usually diarrhea without blood), and weight loss. Children and adolescents may have failure to thrive or have retarded growth. Rectal bleeding is rare except in Crohns colitis.
  - Fistulizing features: Para-enteric abscess formation often with a tender abdominal mass, fistula formation (ileocolic, ileoileal, ileocutaneous); rarely free perforation with features of peritonitis.
  - Stenosing features: Colicky abdominal pain, weight loss due to poor food intake (food fear), palpable or visible distended small bowel loops.
  - Anal disease: Atypical severe anal fissures, fistula in ano, anal mucosal thickening, and discoloration.
- **Investigations**
  - In acute presentations, an abdominal CT may show an inflammatory mass, abscess formation, localized or free perforation.
  - In subacute or chronic presentations, small bowel disease may be shown by a small bowel contrast study (shows mucosal irregularity and narrowing).
  - Crohns colitis is diagnosed by endoscopy and biopsy.
  - Anal disease may require anal ultrasound or MRI scanning for assessment.
  - OGD and biopsies may show features of Crohns in gastric mucosa.
- **Treatment**
  - ***Medical treatment:*** Principles are to reduce inflammation and control complications. Acute derangements in blood results should be corrected.
    - Systemic (5-ASA) drugs are first-line acute and long-term treatment.
    - Systemic steroids (hydrocortisone, prednisolone) control acute exacerbations of inflammation and steroids with very high first pass metabolism (budesonide) can be used chronically.
    - Immunosuppressives (azathioprine, 6-mercaptopurine) are used as maintenance therapy and anti-TNFA antibodies (infliximab) may be effective in fistulizing complications.
    - Dietary manipulation (elemental diet) may reduce inflammatory factors.

- ***Surgical treatment:*** Surgical resection *will not cure CD*. Surgery therefore focuses on the complications of the disease. Principles are to deal with septic complications, relieve significant bowel obstruction, and remove as little bowel as possible. Complications of the disease which are indications for surgery include the following:
  - recurrent intestinal obstruction
  - bleeding
  - perforation
  - failure of medical therapy
  - intestinal fistula
  - fulminant colitis
  - malignant change
  - perianal disease.

Infections of the Small and Large Intestine

Intestinal amebiasis

Amebiasis is an infestation with *Entameba histolytica*. This parasite has a worldwide distribution and is transmitted mainly in contaminated drinking water. It can cause colonic ulcers, which are described as 'bottlenecked' because they have considerably undermined edges. The ulcers typically also have a yellow necrotic floor, from which blood and pus exude. In the majority of cases, they are confined to the distal sigmoid colon and the rectum. Clinically amebiasis can mimic UC, most commonly causing bloody diarrhea but more severe colonic complications can occur, including severe hemorrhage, stricture formation or perforation. A pericolitis is not uncommon and results in adhesions and may cause intestinal obstruction. Amebiasis may cause liver abscesses or an amebic mass (ameboma) of the cecum or sigmoid which is difficult to distinguish from a carcinoma. Surgery is fraught with danger as the bowel is extremely friable. Endoscopic biopsies or fresh hot stools are examined to look for the presence of amebae. It is especially important to exclude amebic infection in patients suspected of having UC. Treatment is by metronidazole in the acute setting, three times daily for 7–10 days. Diloxanide furoate is effective against chronic infections associated with the passage of cysts in stools.

Typhoid fever

Typhoid fever is caused by *S. typhi* and presents with fever and abdominal pain after an incubation period of 10–20 days. Over the next week, the patient can develop distension, diarrhea, splenomegaly and characteristic 'rose spots' on the abdomen caused by a vasculitis. Diagnosis is confirmed by *culture of blood or stool*. Treatment is by antibiotics.

Perforation of a typhoid ulcer usually occurs during the third week and is sometimes the first sign of the disease. The ulcer is parallel to the long axis of the gut and is usually situated in the distal ileum. Perforation requires surgery to wash out and close the perforated ulcer; resection is usually avoided. In unstable patients, notably with evidence of septic shock, the bowel should be exteriorized and the perforation closed after recovery.

Tuberculosis of the intestine

Tuberculosis, like Crohn's disease, can affect any part of the gastrointestinal tract from the mouth to the anus. The sites affected most often are the ileum, proximal colon and peritoneum.
There are two principal presentations.

- **Ulcerative tuberculosis**
  Ulcerative tuberculosis is secondary to pulmonary tuberculosis and arises as a result of swallowing tubercle bacilli. Multiple ulcers, lying transversely, develop in the terminal ileum and the overlying serosa is thickened, reddened and covered in tubercles. Patients typically present with diarrhea and weight loss, although subacute obstruction and even local perforation and fistula formation can occur. A barium follow-through or CT examination show absent filling of the lower ileum, caecum and the ascending colon as a result of narrowing of the ulcerated segment.
  A course of chemotherapy (antituberculous antibiotics) usually leads to cure, provided the pulmonary tuberculosis is adequately treated. Surgery is usually undertaken only in the rare event of a perforation or complete intestinal obstruction.
- **Hyperplastic tuberculosis**
  This is caused by the ingestion of *Mycobacterium tuberculosis* by patients with a high resistance to the organism. The infection usually occurs in the ileocaecal region. The infection establishes itself in lymphoid follicles, and the resulting chronic inflammation causes thickening of the intestinal wall and narrowing of the lumen. There is early involvement of the regional lymph nodes, which may caseate. Unlike in Crohn's disease, abscess and fistula formation is rare. Patients usually present with attacks of abdominal pain and intermittent diarrhea. There is incomplete ileal obstruction, leading to stasis and bacterial overgrowth. This in turn causes steatorrhea, anemia and loss of weight. Patients may present with a mass in the right iliac fossa and vague ill health. The differential diagnosis is that of an appendix mass, lymphoma, carcinoma of the caecum, CD, tuberculosis or actinomycosis. A barium follow-through or small bowel enema will show a long narrow filling defect in the terminal ileum. CT will also demonstrate the narrowed segment with proximal distension and can also demonstrate the lymphadenopathy. When the diagnosis is certain and the patient has not yet developed obstructive symptoms, treatment with chemotherapy is advised and may be curative. Where obstruction is present, surgery with ileocecal resection is often required.

Pseudomembranous colitis

Clostridium difficile is a toxin producing Gram-positive bacillus. Although normally present in around 2 per cent of the population, it seems to proliferate after antibiotic treatment (especially cephalosporins) and can cause antibiotic-associated diarrhea and pseudomembranous colitis. Clinically, *C. difficile* infection presents with diarrhea, abdominal pain and fever. It may progress to pseudomembranous colitis, so called because on visualization of the bowel, plaques of inflammatory exudate between edematous mucosa are seen. Treatment is by metronidazole or vancomycin alongside supportive care. If the colitis does not settle, an emergency subtotal colectomy and ileostomy may be necessary.

Tumors of the Small Intestine

Small bowel tumors are rare and in total account for less than 10 per cent of gastrointestinal neoplasia.

- **Benign**
  The majority of small bowel neoplasms are benign, comprising adenomas, lipomas, hemangiomas and neurogenic tumors. They are frequently asymptomatic and identified incidentally, but can present with intussusception, small bowel obstruction and bleeding that may cause anemia or may even be overt.

- **Malignant**
  These are rare and classically present late, most often diagnosed after surgery for small bowel obstruction. Four types account for over 99 per cent of small bowel malignancies: adenocarcinoma, carcinoid tumors, lymphomas and mesenchymal tumors (gastrointestinal stromal tumors (GIST)).

  - **Adenocarcinoma**
    Small bowel adenocarcinoma is more often found in the jejunum than the ileum and though the etiology is unknown they are more common in patients with Crohn's disease, coeliac disease and familial adenomatous polyposis (FAP) and Peutz-Jeghers syndrome. They present with anemia, overt gastrointestinal bleeding, intussusception or obstruction. Prognosis is poor.

  - **Carcinoid tumor**
    These neuroendocrine tumors occur throughout the gastrointestinal tract, most commonly in the appendix, ileum and rectum in decreasing order of frequency. Appendicular carcinoid tumors are most commonly noted as an incidental finding at appendectomy. Carcinoid tumors arise from Kulchitsky cells at the base of intestinal crypts (of Lieberkuhn). The tumors can produce a number of vasoactive peptides, most commonly 5-hydroxytryptamine (serotonin), but also histamine, prostaglandins and kallikrein. When they metastasize to the liver, the *carcinoid syndrome* can become evident, because the vasoactive substances escape the filtering actions of the liver. The clinical syndrome itself consists of reddish-blue cyanosis, flushing attacks, diarrhea, borborygmi, asthmatic attacks and, eventually, pulmonary and tricuspid stenosis. Classically, the flushing attacks are induced by alcohol. Surgical resection is usually sufficient for patients with primary disease, but the incidence of recurrence is significant. The extent of disease can be assessed preoperatively using octreotide scanning, which may detect otherwise clinically apparent primary and secondary tumors. Plasma markers of tumor bulk, such as chromogranin A concentrations, may be useful markers of disease recurrence, as well as of prognostic value.
    In patients with metastatic disease, hepatic resection can be carried out. The treatment has been transformed by the use of octreotide (a somatostatin analogue), which reduces both flushing and diarrhea, and octreotide cover is usually used in patients with a carcinoid syndrome who have surgery to prevent a carcinoid crisis. Carcinoid tumors are not usually sensitive to chemo- or radiotherapy.

  - **<u>Primary</u> lymphoma**
    Arise from the lymphoid tissue of the small bowel wall. It is more common in patients with Crohn's disease and immunodeficiency syndromes. Almost always non-Hodgkin's; commonest are B-cell lymphomas arising from the mucosa-associated lymphoid tissue (MALTomas). Presents with malaise, abdominal pain, diarrhea; may present with acute perforation or small bowel obstruction. The *mainstay of treatment for these conditions is chemotherapy*; however surgery may be required for obstruction, perforation or bleeding.

- **Gastrointestinal Stromal Tumors (GISTs)**
  GISTs arise from interstitial cells of Cajal (ICC) and are distinct from leiomyoma and leiomyosarcoma, which arise from smooth muscle. Prognosis in patients with GIST tumors depends mostly on tumor size and mitotic count, and metastasis, when it occurs, is typically by the hematogenous route. Any lesion >1 cm can behave in a malignant fashion and may recur. Thus, all GISTs are best resected *along with a margin of normal tissue*. Almost all GISTs (and almost no smooth muscle tumors) express c-KIT (CD117) as well as CD34; almost all smooth muscle tumors (and almost no GISTs) express actin and desmin. These markers can often be detected on specimens obtained by fine-needle aspiration and are useful in differentiating between GIST and smooth muscle tumor histopathologically. Lesions that are definitively leiomyoma by current histopathologic criteria are adequately treated by *enucleation*. Lesions that are definitively GIST or leiomyosarcoma are best treated by *resection with negative margins*. They occur most commonly in the 50- to 70-year age group. Patients may be asymptomatic. Symptoms include lethargy, pain, nausea, hematemesis or melena. Surgery is the most effective way of removing GISTs, as they are radioresistant. Glivec (imatinib) is a tyrosine kinase inhibitor that has been shown to be effective in advanced cases and may also have an adjuvant role.

Tumors of the Large Intestine

Colorectal polyps

The term 'polyp' is a clinical description of any protrusion of the mucosa. It encompasses a variety of histologically different tumors. Polyps can occur singly, synchronously in small numbers or as part of a polyposis syndrome. It is important to be sure of the histological diagnosis because colonic *adenomas* have significant malignant potential.

- **Hyperplastic Polyps**
  Hyperplastic polyps are extremely common in the colon. These polyps usually are small (<5 mm) and show histologic characteristics of hyperplasia without any dysplasia. They are not considered premalignant. In contrast, large hyperplastic polyps (>2 cm) may have a slight risk of malignant degeneration. Moreover, large polyps may harbor foci of adenomatous tissue and dysplasia.
- **Hamartomatous Polyps**
  - ***Juvenile polyp*** is a bright-red glistening pedunculated sphere ('cherry tumor'), which is found in infants and children. Occasionally, it persists into adult life. It can cause bleeding, or pain if it prolapses during defecation. It often separates itself, but can be removed easily with forceps or a snare. A solitary juvenile polyp has virtually no tendency to malignant change, but should be treated if it is causing symptoms
  - ***.Familial juvenile polyposis*** is an autosomal dominant disorder in which patients develop hundreds of polyps in the colon and rectum. Unlike solitary juvenile polyps, these lesions may degenerate into adenomas and, eventually, carcinoma. Annual screening should begin between the ages of 10 and 12 years. Treatment is surgical and depends, in part, upon the degree of rectal involvement.

- ***Peutz-Jeghers syndrome*** is an autosomal dominant disease characterized by melanosis of the mouth and lips and multiple hamartomatous polyps in the small bowel and colon. Melanin spots can also occur on the digits and perianal skin. It is associated with increased risk of a wide range of cancers. It is logical to perform regular colonic surveillance and encourage female patients to attend breast and cervical screening. Malignant change in the polyps rarely occurs and, in general the polyps can be left alone. Resection may be indicated for heavy and persistent or recurrent bleeding or intussusception.

- **Adenomatous polyps**

  True neoplastic polyps formed by excessive growth of the colorectal epithelium; divided by the morphology of the glandular tissue into tubular, tubulovillous, and villous types. May be sessile, pedunculated or mixed. Thought to be the precursor of most colorectal cancers; the risk of cancerous change within an adenomatous polyp increases with size (particularly >1cm), villous morphology, and sessile form. Majority are sporadic (either isolated or in small numbers), although occasionally part of a hereditary syndrome. Solitary adenomas are usually found during the investigation of colonic symptoms and may be the cause of rectal bleeding. Villous tumors can cause diarrhea, mucous discharge and, occasionally, hypokalemia and hypoalbuminemia.

  *** Familial adenomatous polyposis (FAP)**

  FAP caused by an autosomal dominant defect in the APC gene on chromosome 5 and characterized by between dozens and thousands of adenomatous polyps in the colorectum and an increased risk of polyp formation in the stomach and duodenum. The risk of cancerous transformation in any given polyp is similar to that in normal polyps, but the overall risk is very high due to the vastly increased number present. Associated with:

  - Desmoid formation, particularly in the abdominal tissues.
  - Multiple osteomata, fibromata, and thyroid inflammation (called Gardner's syndrome).

  FAP is usually treated by proctocolectomy.

  *** Hereditary non-polyposis colorectal cancer (HNPCC). Lynch syndrome**

  This syndrome is characterized by increased risk of colorectal cancer and also cancers of the endometrium, ovary, stomach and small intestines. It is an autosomal dominant condition that is caused by a mutation in one of the DNA mismatch repair genes. The most commonly affected genes are MLH1 and MSH2. The lifetime risk of developing colorectal cancer in Lynch syndrome is 80 per cent, and the mean age of diagnosis is 45 years. Most cancers develop in the proximal colon. Females with HNPCC have a 30–50 per cent lifetime risk of developing endometrial cancer. Patients with HNPCC are subjected to regular colonoscopic surveillance.

Classification of intestinal polyps		
Inflammatory	Inflammatory polyps (pseudopolyps in ulcerative colitis)	
Metaplastic	Metaplastic or hyperplastic polyps	
Harmartomatous	Peutz–Jeghers polyp Juvenile polyp	
Neoplastic	Adenoma	Tubular Tubulovillous Villous
	Adenocarcinoma	
	Carcinoid tumor	

Colorectal cancer (CRCa)

In the UK, colorectal cancer is the second most common cause of cancer death; but seems to occur less frequently in the developing world than in industrialized countries.

- **Pathology**
  - The accepted model of colorectal cancer development is that it arises from adenomatous polyps after a sequence of genetic mutations influenced by environmental factors (adenoma–carcinoma sequence)
  - The predominant type is adenocarcinoma.
  - Predisposing factors include:
    - Polyposis syndromes (including FAP, HNPCC, juvenile polyposis).
    - Strong family history of colorectal carcinoma.
    - Previous history of polyps or CRCa.
    - Chronic ulcerative colitis or colonic Crohn's disease.
    - Low dietary fibers.
  - CRCa may occur as a polypoid, ulcerating, stenosing, or infiltrative tumor mass. The majority lie on the left side of the colon and rectum. Three to five per cent have a synchronous carcinoma at time of diagnosis.
- **Clinical features**
  - Rectal location
    - *PR bleeding*. Deep red on the surface of stools.
    - *Change in bowel habit*. Difficulty with defecation, sensation of incomplete evacuation, and painful defecation (tenesmus).
  - Descending-sigmoid location
    - *PR bleeding*. Typically dark red, mixed with stool, sometimes clotted.
    - *Change in bowel habit*. Typically increased frequency, variable consistency, mucus PR, bloating, and flatulence.
  - Right-sided location
    - Typically present late with iron deficiency anemia or a mass.
  - Emergency presentations
    - Up to 40% of colorectal carcinomas will present as emergencies.
    - Large bowel obstruction (colicky pain, bloating, bowels not open).
    - Perforation with peritonitis.
    - Acute PR bleeding.
- **Diagnosis and investigations**
  - ***Elective diagnosis*** by PR examination or rigid sigmoidoscopy for rectal carcinoma. Colonoscopy is the preferred diagnostic investigation (alternatives are barium enema and CT colonography).
  - ***Emergency presentations*** commonly diagnosed by abdominal CT scan. Single contrast enema may be used when the diagnosis of large bowel obstruction is possible and CT scanning is unavailable. Acute PR bleeding is sometimes investigated by urgent colonoscopy.
  - ***Staging investigations***
    - Assessment of the presence of metastases (liver, lung, or para-aortic): Thoracoabdominopelvic CT scanning is gold standard; CT PET scan may be used to evaluate equivocal lesions.
    - Assessment of local extent: For colonic carcinoma, CT scanning is adequate; for rectal cancer, pelvic MRI and transrectal US are commonly used.

- Assessment of synchronous tumors: If not diagnosed by colonoscopy or barium enema, one of these two tests is usually performed to identify synchronous tumors.
- Tumor marker (CEA) is of no use for diagnosis or staging, but can be used to *monitor disease relapse* if raised at diagnosis and falls to normal after resection.

Dukes' staging for colorectal cancer
A , confined to bowel wall only
B , through bowel wall
C , any with +ve lymph nodes
D , any with metastases

TNM classification for colonic cancer
T1–4 , stages of tumor invasion of bowel wall
N, Nodal stage
N0, No nodes involved
N1, 1–3 nodes involved
N2, Four or more nodes involved
M, Metastases: M0, No metastases**,** M1, Metastases

- **Treatment**
  - ***Potentially curative treatment***
    Suitable for technically resectable tumors with no evidence of metastases (or metastases potentially curable by liver or lung resection).
    • Surgical resection (with lymphadenectomy) is the only curative treatment.
    • Preoperative (***neo***adjuvant) chemoradiotherapy may be used to increase the chance of curative resection.
    • Adjuvant chemotherapy (5-FU based) is offered for tumors with *positive lymph nodes or evidence of vascular invasion*.
    • Hepatic or lung resection may be offered to patients with suitable metastases and a clear resected/resectable primary tumor.

  - ***Palliative treatment***
    For unresectable metastases or unresectable tumors.
    • Chemotherapy may effectively extend life expectancy with a good quality of life.
    • Obstructing tumors may be endoluminally stented with self-expanding metal stents or transanally ablated (e.g. with laser) if rectal.
    • *Palliative* surgery reserved for untreatable obstruction, bleeding, or severe symptoms.

Intestinal Diverticula

Diverticula (hollow out-pouching) are a common structural abnormality that can occur from the esophagus to the rectosigmoid junction (but not usually in the rectum). They can be classified as:

- Congenital. All three coats of the bowel are present in the wall of the diverticulum, e.g. Meckel's diverticulum.
- Acquired. There is no muscularis layer present in the diverticulum, e.g. sigmoid diverticula.

Jejunal diverticula

These arise from the mesenteric side of the bowel as a result of mucosal herniation at the point of entry of the blood vessels. They can vary in size and are often multiple. They are most often asymptomatic and an incidental finding at surgery or on radiological imaging; however, they can result in malabsorption, as a result of bacterial stasis, or present as an acute abdominal emergency if they become inflamed or perforate. Bleeding from a jejunal diverticulum is a rare complication.

Meckel's diverticulum

A Meckel's diverticulum is a persistent remnant of the vitellointestinal duct and is present in about 2 per cent of the population. It is found on the antimesenteric side of the ileum, commonly at 60 cm from the ileocecal valve and is classically 5 cm long. A Meckel's diverticulum contains all three coats of the bowel wall and has its own blood supply. It is vulnerable to obstruction and inflammation in the same way as the appendix; indeed, when a normal appendix is found at surgery for suspected appendicitis, a Meckel's diverticulum should be looked for by examining the small bowel particularly if free fluid or pus is found. In around 20 per cent of cases, the mucosa of a Meckel's diverticulum contains heterotopic epithelium of gastric, colonic or pancreatic type. A Meckel's diverticulum can present clinically in the following ways:

- **Hemorrhage**. If heterotopic gastric mucosa is present, peptic ulceration can occur and present as painless maroon rectal bleeding or melaena. If the stomach, duodenum and colon are cleared by endoscopy, radioisotope scanning with technetium-99m may demonstrate the heterotopic tissue in a Meckel's. (A Meckel's is notoriously difficult to see with contrast radiology.)
- **Diverticulitis**. Meckel's diverticulitis presents like appendicitis, although if perforation occurs the presentation may resemble a perforated duodenal ulcer.
- **Intussusception**. A Meckel's can be the lead point for ileoileal or ileocolic intussusception.
- **Chronic ulceration**. Pain is felt around the umbilicus, as the site of the diverticulum is midgut in origin.
- **Intestinal obstruction**. A band between the apex of the diverticulum and the umbilicus (also part of the vitellointestinal duct) may cause obstruction directly or by a volvulus around it.
- **Perforation** with peritonitis.
- The vast majority of Meckel's are **asymptomatic**.

When found in the course of abdominal surgery, a Meckel's can safely be left alone provided it has a wide mouth and is not thickened. When there is doubt, it can be resected. The finding of a Meckel's diverticulum in an inguinal or femoral hernia has been described as *Littre's* hernia.

Diverticular disease of the large intestine

Colonic diverticula are acquired outpouchings of colonic mucosa and overlying connective tissue through the colonic wall. Tend to occur along the lines where the penetrating colonic arteries traverse the colonic wall between the taenia coli. Associated with hypertrophy of the surrounding colonic muscle with thickening of the colonic mucosa. This is probably due to the underlying pathological process, which is high pressure contractions of the colon, causing chronic pressure on the colonic wall. Peak age of presentation is 50‑70y. Diverticular disease is ***rare*** in Africa and Asia where the diet is high in natural fibers.

Vascular Diseases of the Intestine

Mesenteric ischemia

Mesenteric ischemia can present as one of two distinct clinical syndromes: acute mesenteric ischemia and chronic mesenteric ischemia.

Acute mesenteric ischemia

Four distinct pathophysiologic mechanisms can lead to acute mesenteric ischemia:

1. ***Arterial embolus:*** Embolus is the most common cause of acute mesenteric ischemia. The embolic source is usually in the heart; most often the left atrial or ventricular thrombi or valvular lesions. Embolism to the superior mesenteric artery accounts for 50% of cases; most of these emboli become wedged and cause occlusion at branch points in the mid- to distal superior mesenteric artery, usually distal to the origin of the middle colic artery.
2. ***Arterial thrombosis:*** In contrast, acute occlusions due to thrombosis tend to occur in the proximal mesenteric arteries, near their origins. Acute thrombosis is usually superimposed on pre-existing atherosclerotic lesions at these sites.
3. ***Vasospasm*** (also known as non-occlusive mesenteric ischemia): is the result of vasospasm and usually is diagnosed in critically ill patients receiving vasopressor agents.
4. ***Venous thrombosis:*** Mesenteric venous thrombosis accounts for 5 to 15% of cases of acute mesenteric ischemia and involves the superior mesenteric vein in the majority of cases. The inferior mesenteric vein is only rarely involved. Mesenteric venous thrombosis is classified as primary if no etiologic factor is identifiable, or as secondary if an etiologic factor, such as heritable or acquired coagulation disorders, is identified.

Regardless of the pathophysiologic mechanism, acute mesenteric ischemia can lead to intestinal mucosal sloughing within 3 hours of onset and full-thickness intestinal infarction by 6 hours.
Severe abdominal pain, out of proportion to the degree of tenderness on examination, is the hallmark of acute mesenteric ischemia, regardless of the pathophysiologic mechanism. The pain typically is perceived to be colicky and most severe in the mid-abdomen. Associated symptoms can include nausea, vomiting, and diarrhea. Physical findings are characteristically absent early in the course of ischemia. With the onset of bowel infarction, abdominal distention, peritonitis, and passage of bloody stools occur.

Chronic mesenteric ischemia

In contrast, chronic mesenteric ischemia develops insidiously, allowing for development of collateral circulation, and, therefore, rarely leads to intestinal infarction. Chronic mesenteric arterial ischemia results from atherosclerotic lesions in the main splanchnic arteries (celiac, superior mesenteric, and inferior mesenteric arteries). A chronic form of mesenteric venous thrombosis can involve the portal or splenic veins and may lead to portal hypertension, with resulting esophagogastric varices, splenomegaly, and hypersplenism.
Chronic mesenteric ischemia presents insidiously. Postprandial abdominal pain is the most prevalent symptom, producing a characteristic aversion to food ("food-fear") and weight loss. These patients are often thought to have a malignancy and suffer a prolonged period of symptoms before the correct diagnosis is made.

Angiodysplasia

Vascular lesions of unknown etiology, most frequently found in the right colon, occasionally associated with cutaneous and oral lesions. They occur with increasing age and present with bleeding that may be torrential, but more often as a series of small bleeds.

Stomas

A colostomy (or ileostomy) stoma is an artificial opening made in the colon (or small intestine) to divert feces and flatus outside the abdomen where they can be collected in an external appliance. Depending on the purpose for which the diversion has been necessary, a stoma may be temporary or permanent. Temporary or defunctioning stomas are usually fashioned as loop stomas, while end stomas usually as a result of surgical removal of distal bowel.

Ileostomy: Formed from any part of the mid- or distal small bowel. Ileostomies (loop or end) are usually spouted, have prominent mucosal folds, tend to be dark pink/red in color, and are most common in the right side of the abdomen. Ileostomy effluent is usually liquid; patients are more likely to develop fluid and electrolytes problems.

Colostomy: Formed from any part of the large bowel. Colostomies (loop or end) are usually flush, have flat mucosal folds, tend to be light pink in color. A colostomy effluent is usually solid and they are most common in the left side of the abdomen.

Stoma complications

- Skin irritation	- Retraction	- Stenosis	- Bleeding
- Prolapse	- Ischemia	- Parastomal hernia	- Fistulation

Enterocutaneous Fistula

An abnormal connection between *small bowel* and skin can occur in fistulating Crohn's disease or as a result of radiotherapy or abdominal trauma, but most commonly follows a surgical complication – either a leak from an anastomosis or an inadvertent enterotomy during dissection. This can be very challenging to manage in patients with a high-output fistula (>500 mL/day). Low-output fistulae (<500 mL/day) can be expected to heal spontaneously, provided there is no distal obstruction.
Reasons for failure of spontaneous healing also include:

- Epithelial continuity between the gut and the skin.
- The presence of active disease where, for example, there is Crohn's disease or carcinoma at the site of the anastomosis or in the fistula track.
- An associated complex abscess.

The management of high output fistulae is based on well-established principles (SNAP), as an *early return* to theatre to try and fix the problem in a septic, malnourished patient is doomed to failure.

Principles of management of enterocutaneous fistulae (SNAP)

_ S, elimination of ***S***epsis and skin protection
_ N, ***N***utrition – a period of parenteral nutrition may well be required
_ A, ***A***natomical assessment
_ P, definitive ***p***lanned surgery