COMMONLY USED TERMINOLOGY

- **Mechanical obstruction**
  There is a physical barrier which prevents the abnormal progress of intestinal contents.

- **Paralytic ileus**
  There is no physical barrier but failure of peristalsis to propel intestinal contents due to neurogenic causes.

- **Simple obstruction**
  It refers to obstruction to lumen only (early cases)

- **Strangulated obstruction**
  It refers to obstruction with impairment of blood supply to the gut.

- **Closed loop obstruction**
  In this condition, the intestine is occluded in two places. More chances of gangrene and perforation are present, e.g. volvulus.

- **Pseudo-obstruction**: No mechanical cause.

II. Depending on the blood supply

A. **Simple obstruction**: Blood supply is not seriously impaired.
B. **Strangulated obstruction**: Blood supply is seriously impaired.
C. **Closed loop obstruction**: It means both proximal and distal ends are blocked. This occurs in carcinoma of the right colon with constrictive lesions. If the ileocaecal valve is competent and the obstruction is total, the intraluminal pressure within the colon increases. As a result of this, the caecum may perforate. Thus, closed loop obstruction can be dangerous (Fig. 30.2). Another example is sigmoid volvulus.

Pathophysiology (Fig. 30.5)

- As a result of obstruction, the proximal bowel undergoes hyperperistalsis which is responsible for colicky pain abdomen. The peristalsis may continue for a few days and later the intestine may be paralysed and flaccid. After 3–4 hours, distal to the obstruction, all physiological activities of the bowel are stopped. Intestine becomes contracted, pale and does not exhibit peristalsis. After a few hours, the proximal bowel gets dilated secondary to obstruction.
- The causes of distension of intestinal loop are:
  A. Gaseous distension
     - Swallowed air (70%). Because of colic and anxiety, the swallowed air is increased. Oxygen is absorbed and nitrogen remains as it cannot be absorbed. This results in distension.
     - Diffusion of air from the blood into bowel lumen increases carbon dioxide which diffuses very rapidly.
     - Gas due to bacterial activity releases H₂S, NH₃, etc.
**COMMON CAUSES OF ILEAL OBSTRUCTION**

- Adhesions
- Obstructed hernia
- Stricture
- Intussusception
- Ileocaecal tuberculosis
- Bands
- Worm ball—in children
- Ileal atresia—in children

**COMMON CAUSES OF COLONIC OBSTRUCTION**

- Carcinoma colon
- Sigmoid volvulus
- Faecal impaction
- Mesenteric ischaemia
- Hirschsprung's disease
- Anorectal malformations
- Stricture colon—rare

**COMMON CAUSES OF GANGRENE**

- Volvulus
- Intussusception
- Obstructed hernia
- Mesenteric vascular occlusion
- Twisting around a band
- Necrotising enterocolitis

Fig. 30.4: Differential diagnosis of intestinal obstruction—diagrammatic representation
OBSTRUCTION
- Distension
- Venous compression
- Congestion and oedema bowel wall turns purple
- Progressive arterial compromise
  blackish discoloration
  Twisting around band
  Gangrene
  Proliferation of bacteria
  Toxins
  Transmigration
  Peritoneal cavity
  Septic shock

Fig. 30.5: Pathophysiology of intestinal obstruction

B. Distension due to fluids
- 1500 ml of saliva
- 2 litres of gastric juice
- 3 litres of intestinal secretions
- 1 litre of bile and pancreatic juice

Normally, all this fluid is absorbed in the bowel. In cases of intestinal obstruction, this fluid absorption is delayed. It accumulates in the intestinal loop. Excretion of water and electrolytes into the lumen is also increased.

C. Role of nitric oxide
Activated neutrophils and macrophages accumulate within the muscular layer of the bowel wall due to dilatation and inflammation of the bowel wall. This damages the secretory and motor processes by release of reactive proteolytic enzymes and cytokines. Net result is increase in the local release of nitric oxide, itself a potent inhibitor of smooth muscle tone. It further aggravates the intestinal dilatation.

D. Role of bacteria
- Bacterial colony count increases following obstruction resulting in stasis. From less than 10⁶ in jejunum and from 10⁴ in ileum, counts increase.
- Bacterial translocation can occur even in simple obstruction without strangulation. Thus, bacteria can enter into lymph nodes and into systemic circulation. Abdominal distension, hypovolaemia, renal failure and sepsis set in.

In addition to these changes, diaphragm gets elevated, respiration is impaired which result in respiratory complications such as atelectasis and basal pneumonia.

- In doubtful cases of viability, if facilities are available, a test called fluorescein test can be done. 1000 mg of fluorescein is injected into peripheral vein and bowel is inspected under Wood light. If loops are nonviable, resection and anastomosis is done.

Strangulation (Fig. 30.6 and Key Box 30.2)
- Interference with blood supply: As the tension within the loops becomes more and more, venous congestion takes place resulting in oedema of the bowel wall.
- If the obstruction is not relieved, capillary rupture and haemorrhage into bowel may ensue. In cases of volvulus and intussusception, the arterial supply gets compromised rapidly causing gangrene of bowel wall very early. Bacterial proliferation takes place and endotoxins are released.
- Transmigration (translocation) of gram-negative organisms, anaerobes and gram-positive organisms through the gangrenous bowel results in peritonitis.
- The organisms release powerful endotoxins which are absorbed from the peritoneal surface and cause gram-negative shock or septic shock. It carries very high mortality rate (30%).
- Early gangrene without obstruction is a feature of mesenteric thrombosis or embolism.
- Loss of blood volume is an important feature of massive gangrene.

Fig. 30.6: Gangrene of the intestine due to bands

FACTORS PREDISPOSING ISCHEMIA
- Volvulus
- Mesenteric ischaemia
- Necrotising enterocolitis
- Intussusception
- Progressive distension
- Extrinsic compression by adhesions, bands, etc.
Features of septic shock—fever, hypothermia, renal failure, respiratory failure (Key Box 30.5).

Rebound tenderness: It is called Blumberg’s sign. It is a classical sign of peritonitis.

Guarding and rigidity of the abdominal wall.

Absent bowel sounds because rest of the bowel loops undergo paralytic ileus.

Sudden symptoms—spasmodic pain (due to peristalsis) and continuous pain suggest strangulation (Fig. 30.11).

Features of strangulation and perforation occur quickly in cases of closed loop obstruction (Figs 30.8 to 30.11 and Key Box 30.6).

**KEY BOX 30.5**

**FEATURES OF STRANGULATION**

- Tachycardia
- Tenderness
- Temperature—fever
- Acidosi

**CLOSED LOOP OBSTRUCTION**

- This occurs when the bowel is obstructed at both proximal and distal points.
- Proximal bowel is not distended as much in this condition
- Gangrene and perforation can occur fast.
- Retrograde thrombosis of mesenteric vein, can result in distension of the bowel.
- A few examples of closed loop obstruction include sigmoid volvulus, strangulated hernia, carcinoma right colon.

**Rectal examination**

- In small bowel obstruction, rectum is empty and is often ballooned out.
- Carcinomatous growth with or without stools can be felt.
- The finger may be stained with blood.
**PEARLS OF WISDOM**

Vomiting of faeculent contents indicates terminal ileal obstruction.

3. Distension of the abdomen: It may be central abdominal distension as seen in ileal obstruction, peripheral abdominal as in large bowel obstruction, or localised to one or two quadrants as in sigmoid volvulus.

4. Constipation occurs because the distal bowel does not move. Constipation to faeces and flatus is called obstipation. Exceptions are given in Key Box 30.4.

**Signs**

1. General signs of dehydration such as dry skin, dry tongue, sunken eyes, feeble pulse, low urinary output are seen. Dehydration occurs due to persistent vomiting and sequestration of fluid and electrolytes. Hypokalaemia is an important finding.

2. Abdominal findings:
   - Distension, tympanitic note on percussion
   - Step ladder peristalsis is seen in terminal ileal obstruction.
   - Right to left colonic peristalsis is seen in left-sided colonic obstruction, large bowel obstruction.
   - On auscultation—loud, noisy intestinal sounds are heard. They are called borborygmi.
   - Hernial orifices have to be examined, especially for a femoral hemia in females.

**Signs of strangulation**

It should be suspected when features of obstruction are present along with features of shock.

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**KEY BOX 30.3**

**CARDINAL FEATURES OF INTESTINAL OBSTRUCTION**

- Colicky abdominal pain
- Abdominal distension
- Vomiting
- Absolute constipation

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**Table 30.1**

<table>
<thead>
<tr>
<th>High (jejunum)</th>
<th>Distal (ileum)</th>
<th>Low (colon)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting</td>
<td>Frequent, bilious</td>
<td>Moderate bilious, faeculent</td>
</tr>
<tr>
<td>Distension</td>
<td>No</td>
<td>Moderate distension</td>
</tr>
<tr>
<td>Pain</td>
<td>Intermittent, not crescendo type</td>
<td>Intermittent, crescendo type, colicky</td>
</tr>
<tr>
<td>Constipation</td>
<td>Not initially</td>
<td>Not initially</td>
</tr>
<tr>
<td>Peristalsis</td>
<td>Not seen</td>
<td>Step ladder peristalsis</td>
</tr>
</tbody>
</table>
• The small intestine is considered dilated if loops of bowel measure more than 3 cm in diameter. Measurements for the large bowel vary among different anatomic segments, with a relative threshold of 9 cm in diameter for the proximal colon and 5 cm for the sigmoid colon.

INVESTIGATIONS

• Complete blood picture: Low Hb% indicates underlying malignancy. Increased total WBC count indicates infection and sepsis (perforation).
• Electrolytes: Most of the electrolytes are low in cases of intestinal obstruction and require correction preoperatively. Strangulation may be associated with deranged potassium, amylase or lactic dehydrogenase.
• Plain X-ray abdomen in the erect position may show multiple gas fluid levels. Gas levels appear earlier than fluid level. Normally, two insignificant fluid levels can be present, one in the terminal ileum and one in the first part of the duodenum (Key Box 30.7). Supine films indicate the distal limit of obstruction (Figs 30.12 to 30.16).

PEARLS OF WISDOM

Enteroclysis is rarely performed in acute intestinal obstruction but it has greater sensitivity in the detection of partial small bowel obstruction (Fig. 30.17).

• Ultrasound/CT scan—See Fig. 30.18, Key Boxes 30.8 and 30.9

KEY BOX 30.7

PLAIN X-RAY FINDINGS
UPRIGHT AND SUPINE

• First get supine films. They indicate distal limit of obstruction. Erect films are asked if any doubt exists about obstruction.
• Jejunum is characterised by regularly placed mucosal folds called valvulae conniventes (Fig. 30.14) placed opposite to each other (Herring bone pattern). They are produced by valves of Kerckring.
• Large bowel is characterised by haustations (Fig. 30.15). Incomplete, large mucosal folds, not placed opposite to each other.
• Caecum has no haustations. It appears as a round gas shadow in the right iliac fossa.
• Ileum has no characters—characterless loop of Wangensteen.
• Plain X-ray may demonstrate gall stone ileus or foreign body.
Gas is absent in the small bowel as in mesenteric vascular ischaemia.
Sigmoid volvulus appears as a large dilated loop—inverted ‘U’ shape.
CT SCAN IN INTESTINAL OBSTRUCTION

- Can detect dilated intestines proximally and collapsed bowel distally.
- If bowel wall is thick and air is present (pneumatosis), strangulation is likely.
- It can detect portal venous gas (suggesting gangrene).
- CT can detect mass lesions—carcinoma sigmoid, caecum or ileocaecal mass (TB).
- CT has low sensitivity in detecting low grade or partial small bowel obstruction. Sensitivity increases in total obstruction.

MANAGEMENT

Preoperative preparation includes correction of dehydration, electrolytes and broad spectrum antibiotics. Principles in the management of intestinal obstruction are as follows:

A. **Aspiration** with Ryle's tube. This is the most important step in the management of intestinal obstruction. It helps in decreasing the distension and also prevents vomiting. This will help in preventing respiratory complications, such as aspiration following general anaesthesia.

B. **Bowel prep**. No purgatives because purgation can cause perforation.

C. **Charting** (temperature, pulse, respiration and intake-output chart). In cases of conservative management such as obstruction due to adhesions, change in temperature and increasing pulse rate suggests perforation or gangrene. These cases have to be explored immediately.

D. **Drugs** to cover gram-positive, gram-negative and anaerobic organisms.

E. **Exploratory laparotomy** is done and depending upon the findings, obstruction is treated. A few examples are given in Key Box 30.10 and Fig. 30.19.
**Intestinal Obstruction**

**F. Fluids** should be given before, during and after surgery. It forms the most important treatment of intestinal obstruction.

**KEY BOX 30.10**

**PRINCIPLES OF EXPLORATORY LAPAROTOMY**

- Ideally done within 6-8 hours
- Long midline incision
- Resection of gangrene and anastomosis
- Adhesion—release
- Bands—divide
- Gall stone ileus—remove the stone
- Volvulus—untwist or resection
- Obstructed hernia—reduce
- Gangrene—resect
- Stricture—resection or stricturoplasty
- Advanced malignancy—bypass

**Fig. 30.19:** Principles of management of intestinal obstruction

**APPRAOCH TO THE MANAGEMENT OF INTESTINAL OBSTRUCTION**

Ask the following questions to yourself and proceed.

1. What is the probable cause of obstruction?
2. Is it small bowel obstruction at laparotomy?
3. Is it large bowel obstruction at laparotomy?
4. Is it simple obstruction?
5. Is it strangulation?
6. Is it some kind of a surprise or a difficult case?
7. Can I manage conservatively?

1. **Probable cause of obstruction**

   - A previous laparotomy scar may indicate that it could be an adhesive obstruction (most common).
   - An obvious obstructed hernia (inguinal or femoral) can be managed with inguinal approach.
   - An elderly man, hypertensive and atherosclerotic, with features of blood in the stools and acute abdominal pain may be having superior mesenteric ischaemia.
   - A constipated, elderly man in poor health, with acute or chronic obstruction may be having carcinoma of the colon.

2. **Diagnosis of small bowel obstruction at laparotomy**

   - Caecum is collapsed
   - Dilated loops of small intestine are present.
   - A stricture or a mass lesion may be obvious at laparotomy.

3. **Diagnosis of simple obstruction**

   - It is done when bowel is not gangrenous.
   - In doubtful cases, because of long-standing ischaemia, wrapping the bowel with warm and moist pack and administration of pure oxygen may help the bowel to recover from ischaemia.

4. **Diagnosis of large bowel obstruction at laparotomy**

   - Caecum is distended.
   - A growth may be palpable and obvious in the transverse colon or in the hidden colon, i.e. splenic flexure.
   - It is very important to examine the entire colon (synchronous carcinoma is more common).

5. **Diagnosis of strangulation**

   (Fig. 30.20 and Key Box 30.11)

   - Black, dark, foul-smelling bowel is seen as soon as laparotomy is done.
   - Peritoneal fluid contains blood-stained fluid.
   - Precautions must be taken not to contaminate peritoneal cavity when gangrenous segment is removed.

   **Do not hesitate to take the help of** senior experienced surgeons in treating an uncommon situation such as massive ischaemia and gangrene of small bowel and colon (due to mesenteric vascular occlusion), synchronous carcinoma and ileo-sigmoid knotting, etc.

**KEY BOX 30.11**

**Viable bowel—features**

- Normal peristalsis
- Normal peritoneal sheets are present
- Normal pulsations are visible or felt at the mesentery
- Normal pink colour is present

**Fig. 30.20:** Viable and nonviable bowel
6. It is a surprise
- Surprises are well known in intestinal obstruction. Congenital bands, foreign bodies, internal herniation, lymphomatous strictures are a few examples.
- The detailed management of individual cases is discussed below.

7. Can I manage conservatively?
- In these cases, a long intestinal tube called Miller-Abbott tube can be passed to decompress intestines (Key Box 30.12).

**KEY BOX 30.12**

**INTESTINAL OBSTRUCTION**

**CONSERVATIVE TREATMENT**

1. **Partial small bowel obstruction** mostly due to adhesion: Wait for 48 to 72 hours. They may show improvement. If not, surgery is required.
2. **Early postoperative obstruction**: It rarely progresses to strangulation. Hence, nonoperative management can be extended to many days (3–7) provided there is no evidence of peritonitis.
3. **Intestinal obstruction in Crohn’s disease**: Aim in Crohn’s disease is to ‘preserve’ bowel as it may respond to medications.
4. **Carcinomatosis**: Disseminated malignancy with obstruction. The aim is nonoperative treatment as nothing much can be achieved with laparotomy.

**DIFFERENTIAL DIAGNOSIS OF INTESTINAL OBSTRUCTION**

**VOLVULUS OF THE SIGMOID COLON**

- Common in North India (Punjab), Eastern Europe, Uganda.
- In certain parts of India as mentioned above, it is one of the common surgical emergencies in elderly population (Key Box 30.13).

**KEY BOX 30.13**

**CERTAIN FACTS ABOUT SIGMOID VOLVULUS**

- More common in males
- 2/3 of the cases are sigmoid volvulus and 1/3 are caecal volvulus.
- Common in middle age and > 60 years
- It consists about 10 to 15% of cases of intestinal obstruction in India.
- More common in rural population
- It is not an uncommon event during pregnancy

**Precipitating factors** (Fig. 30.21)

1. Long mesentery of the pelvic colon
2. Narrow attachment at the base

**Fig. 30.21**: Causes of sigmoid volvulus (see text for numbers)

3. Long, redundant, pendulous sigmoid
4. Loaded colon due to high residue diet
5. Diverticulitis with a band, or adhesions

**Sigmoid volvulus** is a definite occurrence in mentally disturbed patients, hypothyroidism, Parkinson’s disease, multiple sclerosis, etc. probably due to severe constipation due to medications.

**PEARLS OF WISDOM**

*Ogilvie’s syndrome precipitates volvulus.*

**Clinical features**

1. **Acute sigmoid volvulus (fulminant)** presents as intestinal obstruction. It starts usually after straining at stools. Volvulus is usually in an antihorwwise direction and after one and a half turns, the entire loop becomes gangrenous.

   Enormous distension of the abdomen takes place, which gives a tympanic note all over the abdomen. It is due to diffusion of CO₂ (Fig. 30.22). Due to gross distension of the loop, there may be acute circulatory failure.

**Fig. 30.22**: Uneven distension due to sigmoid volvulus
Motility
Colon has 4 types of motility. Propulsive, repulsive, mass peristalsis and gastrocolic reflex. Thus, contents travel aborally. Retropulsive activity is more in the right colon, these allowing the contents to ‘churn’ more and more. Mass contractions are found more in left colon—specially after meals.

Factors which stimulate the colonic motility
- Dietary fat
- Rich fibre diet
- Physical activity—walking, change in posture, exercises
- Emotional activity
- Less water intake

Constipation (Key Box 29.1)
- It depends upon several factors such as food habits, genetic, social customs.
- Generally a patient is said to have constipation if he passes less than 2 stools per week.
- In addition to the low fibre diet, emotional feelings and many rectal diseases also cause constipation. Example—prolapsed rectum, solitary rectal ulcer syndrome. Colonic disease such as megacolon—Hirschprung disease is an important cause of constipation in children.
- Increasing constipation in elderly patient suggest carcinoma in left colon. Needs to be evaluated by colonoscopy.

Recycling
- Recycling of various nutrients takes place in the colon. Examples: Fermentation of carbohydrates, short-chain fatty acids and urea cycling.
- Butyrate is the main product of bacterial fermentation. It is required mainly as a fuel for colonic epithelium.
- To accomplish this, the colon depends highly on its bacterial flora, especially for degeneration and fermentation ability.

Pearls of Wisdom
More distal the colon—more is the protein metabolism and putrefaction resulting in carcinogens and greater exposure to colonic mucosa. Hence, two-thirds of colonic cancer occur in the left colon.

Key Box
Constipation
- Digestion and absorption
- In a child—Hirschsprung disease
- Adult women—idiopathic following child birth
- Middle aged women—following hysterectomy
- Elderly man—canceroma left colon
- Constipation with severe pain—anal fissure
- Depression patient—psychotropic drugs—used to treat schizophrenia, antidepressants and antiepileptic drugs.
Colonic bacteria

- Anaerobic bacteria: They constitute more than 99%. The most common pathogen is Bacteroides fragilis (10^{10}/g of faeces). Other organisms are clostridia, cocci, etc.
- Aerobic bacteria: Escherichia coli is the most common organism about 10^{7}/g of faeces. Other organisms are Klebsiella, Proteus and Enterobacter.
- Normal function: Bacteria degrade bile pigments thus resulting in brown coloured stools. They also help in colonic motility and absorption. Fatty acids produced by bacteria supply nutrition to colonic epithelium. Bacteria also supply vitamin K to the host.

Prebiotics and probiotics

- Prebiotics are non-digestible food ingredients that stimulate the growth and/or activity of bacteria in the digestive system in ways claimed to be beneficial to health. Traditional dietary sources of prebiotics include soybeans, inulin sources (such as Jerusalem artichoke, jicama, soya and chicory root), raw oats, unrefined wheat, unrefined barley, and yacon.
- Probiotic is defined as a “live microbial feed supplement which beneficially affects the host animal by improving its intestinal microbial balance”. They are non-degradable oligosaccharides. They stimulate the growth of beneficial intestinal bacteria.
- Probiotics are dietary supplements which contain live cultures of bacteria and yeast that are beneficial to colonic and host function. The common species used as probiotics are Lactobacillus and Bifidobacterium. Probiotics stimulate immune function, exhibit anti-inflammatory property and suppress pathogenic organism.
- Clinical application: When a person takes antibiotics, both the harmful bacteria and the beneficial bacteria are killed. Bacterial change in flora alters carbohydrate metabolism with decreased short-chain fatty acid absorption and result in osmotic diarrhoea. In a similar fashion, antibiotic therapy causes increase in the growth of Clostridium difficile. Thus, probiotics have been recommended in antibiotic induced diarrhoea. Also, they have been used to treat diarrhoeas in ulcerative colitis, in pouchitis (inflammation of the pouch after total proctocolectomy for ulcerative colitis) and in necrotising colitis in children.

**TUMOURS OF THE LARGE INTESTINE**

Benign tumours (Fig. 29.6) are usually referred to as polyp, which means elevated from the surface. They are as follows:

**ADENOMATOUS POLYP** (Key Box 29.2)

- It may be a villous adenoma which is a flat lesion or a tubular adenoma having a pedicle. Tubular is more common.
Symptoms and signs of polyps: Bleeding per rectum is the most common symptom. Fresh bleeding is seen in rectal polyps. Typically it is painless. It is intermittent. If it is associated with change of bowel habits means probably a malignant change. These changes include mucus discharge, tenesmus, sometimes constipation. In children, polyp may project outside the anus. In such cases, it has to be distinguished from prolapsed rectum.

Treatment
- Colonoscopy and polypectomy is the standard treatment.
- If specimen shows invasive carcinoma, radical surgery needs should be done.

PEARLS OF WISDOM
Although most neoplastic polyps do not evolve to cancer, most colorectal cancers originate as a polyp.

HAMARTOMATOUS POLYP (JUVENILE POLYP)
- This can occur in the colon as in Peutz-Jeghers syndrome. Risk of malignancy is very limited. Symptomatic polyps need to be treated.
- Juvenile polyps are usually single and occur in children. They give rise to bleeding and are easily resected. They do not have malignant potential.

FAMILIAL POLYPOSIS COLI (FPC) OR FAMILIAL ADENOMATOUS POLYPOSIS (FAP)
- FAP is a genetic disorder inherited as a Mendelian dominant. The gene APC (adenomatous polyposis coli) is located on the short arm of chromosome 5. Prevalence: 1 in 10,000. It is clinically defined by the presence of more than 100 colorectal adenomas (Figs 29.8 and 29.9).
- It is transmitted from both sexes. The incidence is same in either sex.
- When it is associated with desmoid tumour, craniofacial osteoma, epidermoid cysts, congenital hypertrophy of retinal pigment epithelium, it is described as Gardner's syndrome (Key Box 29.3).
- When familial polyposis coli is associated with central nervous system tumour and glioblastoma, it is called Turcot's syndrome.
- 50% of them have benign gastric polyps and 90% of them have duodenal polyps.

Clinical features
- Runs in families, other members of the family are affected.
- Manifests at the age of 20 in the form of blood and mucus in the stool, loose stools, etc. It produces crampy lower abdominal pain.
- Anaemia, weight loss and protein malnutrition occur slowly.
- Mean age of development of carcinoma is 39 years.

Complications of FAP
- Malignancy (100% risk)

Investigations
- Colonoscopy—details in page 707

Treatment
- NSAID: Sulfasalazine 300 mg, twice a day and aspirin 325 mg once a day have been found to decrease the size of polyps.

FAMILIAL POLYPOSIS COLI—SUMMARY
- Polyps are more than 100 (colorectal adenomas)
- Other mesodermal tumours—desmoid tumours, osteoma, epidermoid cysts can be present (Gardner's syndrome).
- Large bowel is predominantly involved.
- Mean age of development of carcinoma—mean age 39 years.
- Polyposis is due to autosomal dominant APC gene.
- Other syndrome—Turcot.
- Barium enema from age of 15 at intervals is the investigation of choice.
- Ileoanal anastomosis with pouch—restorative proctocolectomy—advisable above age of 30.
- Surgery is the only means of preventing colonic cancer.

Remember as POLYPOSIS
- Patients with FAP who are above the age of 30 have high chances of having a carcinoma in the colon. Hence, even when there is no malignancy, surgery is advisable.

**Types of surgery** (Figs 29.10 and 29.11)
- Many patients do not like ileostomy. Hence, a subtotal colectomy with ileorectal anastomosis can be done. This is done provided that rectum is examined frequently and endoscopic snaring of the polyps is done regularly, especially in a young patient.
- **Restorative proctocolectomy** with ileal pouch anal anastomosis by using a pouch is another alternative. However, it is a major surgical procedure and should be undertaken only by an experienced surgeon.

**Screening**
Starts from the age of 15 years using sigmoidoscopy.

**Pearls of Wisdom**

*If there are no adenomas by the age of 30 years, FAP is unlikely.*

**Metaplastic Polyp**

Also called hyperplastic nodules. They are of viral aetiology. They do not have malignant potential.

---

**Figs 29.10 and 29.11:** Familial adenomatous polyposis with two malignancies—lower rectum and hepatic flexure. This patient was being treated for chronic diarrhoea for 7–8 years with various medications. He underwent colonoscopy for the first time in our hospital. Total proctocolectomy specimen (Courtesy: Dr Challa Srinivas Rao, Professor, Dept of Surgery, and Dr Ravi, Konaseema Institute of Medical Sciences (KIMS), Amalapuram—Andhra Pradesh)

**HNPPC (Hereditary Nonpolyposis Colorectal Cancer)**

- Autosomal dominant, no polyps
- Lynch's syndrome I: Site specific colorectal cancer.
- Lynch’s syndrome II: Cancer family syndrome—they have extracolonic cancers such as endometrial cancer, ovarian cancer, transitional cell cancer, etc.
- Lifetime risk of developing colorectal cancer is 80%.
- Synchronous carcinoma means more than one cancer at the time of diagnosis. Metachronous carcinoma means appearance of second carcinoma after 6 months can occur here (Fig. 29.12).

**Diagnostic criteria (Amsterdam criteria II)**

1. At least 3 members in a family should have colorectal cancer—two of whom are first degree relations.
2. At least two consecutive generations
3. At least one relative should have colorectal cancer by less than 50 years of age.
4. Exclusion of FAP

**Buttock examination**

Increased incidence of proximal colorectal cancer.

**Anorectoscope:** One can examine up to 10–12 cm of anal canal and rectum. Rubber band ligation (for piles) and polypectomy can be done with this instrument.

**Flexible sigmoidoscope:** The scope measures about 60 cm in length. One can easily reach up to splenic flexure. Bowel wash or an enema is given before the procedure. No sedation is required (Figs 29.13 and 29.14).
CLINICAL NOTES

A 68-year-old lady was admitted with large bowel obstruction. Plain X-ray abdomen showed intestinal obstruction. Exploratory laparotomy was done. A 3 cm constricting growth was identified at the rectosigmoid junction and high anterior resection and anastomosis was done. On the 4th postoperative day, the patient was allowed liquid diet. Distention increased. For another 3 days, distension went on increasing. Plain X-ray abdomen revealed obstruction with more gas than before. The patient was having colicky abdominal pain. Exploratory laparotomy was done. Findings at 2nd laparotomy—anastomosis was intact. Transverse colon was hugely dilated. Careful palpation of splenic flexure revealed one more growth. A resection and anastomosis was done again. Patient was discharged after 10 days. The first surgeon agreed that after finding out the rectosigmoid growth, he did not look for any other lesions (mistake). This was obviously a case of synchronous carcinoma.

METACHRONOUS COLONIC CANCER

- Metachronous cancer was defined as those cancer occurring more than 6 months following resection of one malignancy.
- A few examples of metachronous site are: Colorectum, breast, kidney.
- Family history of hereditary, nonpolyposis colorectal cancer (HNPCC or Lynch syndrome), an autosomal dominant disease, also can present with both synchronous or metachronous colorectal cancers.
- It is more common in females
- Common usually at young age.
- The associated genetic defect lies at the mismatch repair genes, responsible for the correction of DNA bases mismatch.
- The coexistence of adenomatous polyps is also considered a risk factor for the development of metachronous lesions. Thus, after treating one carcinoma, example— carcinoma sigmoid, annual colonoscopy is recommended. If polyps are detected, patients have to be informed about the polyps and their potential of malignancy.
- Survival is better

FAMILIAL COLORECTAL CANCER

- All these have a carrier gene and thus, run in families. Malignancies occur in young age group. Often they are synchronous. Metachronous lesions are not uncommon.

- Certain criteria have been laid upon for the diagnosis of these conditions which have been discussed already.
- Genetic instability is the chief factor responsible. The instability can be at chromosomal level called chromosomal instability or at DNA level called microsatellite instability—MSI. As a result of this after the cell division by duplication, mismatched genes develop. These genes cannot be repaired. This predisposes to mutation, results in a cancer gene.
- Familial polyposis coli accounts for about 1% of colorectal cancers. However, incidence of malignancy is 100%. Gardner’s and Turcot’s syndrome are the variants of FPC.
- HNPCC accounts for about 5 to 10% of colorectal cancers. They also have extracolonic cancers such as endometrial, ovarian and urinary bladder cancers.
- Other familial syndromes are Cronhite Canada syndrome. It is more common in females. Multiple polyps develop in stomach, duodenum and in the colon. Diarrhoea is the clinical presentation. Other features include pigmentation, alopecia, loss of weight and cachexia. Chances of developing malignancy is about 15%.

Precancerous conditions (Key Box 29.5)
1. Polyp: Environmental and genetic factors favour the development of colonic polyps and their transformation into malignancy. The incidence of malignancy is increased when the polyp is more than 1 cm, polyps are multiple or flat (Table 29.2).

<table>
<thead>
<tr>
<th>Type</th>
<th>Cause</th>
<th>Malignant potential</th>
<th>Features/syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Adenomatous polyp</td>
<td>Benign tumour</td>
<td>1-10%</td>
<td>Hypokalaemia, diarrhoea</td>
</tr>
<tr>
<td>2. Hamartomatous polyp</td>
<td>&quot;Misfire&quot;</td>
<td>Negligible</td>
<td>Peutz-Jeghers syndrome</td>
</tr>
<tr>
<td>3. Familial polyposis coli</td>
<td>Genetic disorder</td>
<td>100%</td>
<td>Gardner’s and Turcot’s syndrome</td>
</tr>
<tr>
<td>4. Metaplastic polyps</td>
<td>Hyperplasia</td>
<td>Nil</td>
<td>Asymptomatic</td>
</tr>
</tbody>
</table>
Fibreoptic colonoscopy can assess the entire colon. It is 100–160 cm in length. Usually there will be multiple polyps varying from a few millimetres to centimetres. Biopsy has to be taken. Polyps are visible after 15 years and certainly by the age of 30 years.
- It is the investigation of choice in most of the large intestinal lesions.
- It permits examination of entire colon and terminal ileum.
- Colon is prepared by polyethylene glycol given orally.
- Risk of perforation of colon is less than 0.1%.

**Indications**

**Diagnostic**
- Lower gastrointestinal bleeding
- Inflammatory bowel diseases
- Abnormal finding in barium enema
- Family history of colorectal cancers
- Biopsy of caecum/ileum in suspected cases of cancer.
- Ileoceleal tuberculosis—to take biopsy

**Therapeutic**
- Control of bleeding—coagulation or injection sclerotherapy.
- Snaring of polyps
- Removal of foreign body
- Detorsion of volvulus
- Decompression of pseudo-obstruction

**Carcinoma Colon**

**Introduction**

It is the second most common cancer and cancer related death cases in the western world next only to lung cancer. The incidence increases with age. Multiple synchronous lesions (more than 1 malignancy at the time of diagnosis) is found in about 5% of the patients. Colon is also one of the sites of metachronous cancer (new malignancy appearing after 6 months of curative surgery). More than 95% are adenocarcinoma and surgery remains the most effective treatment. Survival has improved because of early diagnosis and multimodality of the treatment.

Over a period of years the understanding of development of carcinoma has changed more and more molecular biology of colonic cancer is being discussed. The **Fearon-Vogelstein adenoma-carcinoma multistep model of colorectal neoplasia** represents one of the best-known models of carcinogenesis (Key Box 29.4).

**KEY BOX 29.4**

<table>
<thead>
<tr>
<th>Normal tissue</th>
<th>Normal colonic epithelium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumour initiation</td>
<td>APC</td>
</tr>
<tr>
<td>Tumour progression</td>
<td>Dysplastic aberrant foci</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Early adenoma</td>
</tr>
<tr>
<td></td>
<td>K-RAS</td>
</tr>
<tr>
<td></td>
<td>Intermediate adenoma</td>
</tr>
<tr>
<td>p53</td>
<td>Late adenoma</td>
</tr>
<tr>
<td>Carcinoma</td>
<td>p53</td>
</tr>
</tbody>
</table>

APC, K-RAS, DCC and p53 are oncogenes that convert normal cell to cancerous cell

- Terminology: Before we start the discussion on carcinoma colon, we shall study a few terminologies used in carcinoma colon. They are synchronous carcinoma, metachronous carcinoma, familial colorectal carcinoma.

**Synchronous Carcinoma**

- Moertel’s definition: Synchronous cancers as those occurring within 6 months of the first primary cancer, or two or more histologically distinct simultaneously detected malignancies or more than one malignancy at the time of initial diagnosis. This will happen especially in cases of colon and upper aerodigestive tract wherein the stimulus or aetiological factor for malignancy affects different parts of the organ.
- Colon, head and neck, esophagus are the site of synchronous carcinomas.
- In cases of carcinoma colon with specific aetiological factors such as familial polyposis coli, ulcerative colitis, hereditary non-polyposis cancer, often carcinoma is synchronous.
- Thus, it is important to do a complete colonoscopy when a patient comes with colonic carcinoma because he/she may be having another synchronous carcinoma elsewhere. See the case report in the next page.
2. Inflammatory bowel disease

A. Ulcerative colitis is a definite precancerous condition. Presence of dysplasia diagnosed by colonoscopic biopsy is an indication for colectomy.

B. Crohn's involving colon also has a mildly increased risk of developing carcinoma when compared to ulcerative colitis.

C. Schistosomal colitis: The risk of colorectal cancer is increased in patients with long-standing schistosomal colitis. Long-standing cases are associated with mild to severe grades of colonic epithelial dysplasia. Thus ulcers or pseudo-polyps can occur. These dysplastic changes are considered as premalignant.

D. Radiation exposure: Usually it is mucin secreting adenocarcinoma with poor prognosis.

E. Ureterosigmoidostomy increases risk of colonic cancer over 100-500 times.

Aetiological factors

1. SAD factors: It is sad to know that SAD factors are responsible for carcinoma colon. They are S—Smoking, A—Alcohol, D—Dietary factors. Diet rich in red meat has high animal fat. This alters intestinal bacteria, which convert primary bile acids into secondary bile acids. This is the beginning of formation of carcinogenic polycyclic aromatic compounds. After cholecystectomy, there is increase in free bile acid concentration, thus increasing the risk of colonic cancer. Thus increased roughage is associated with increased transit time which in turn reduces exposure of mucosa to carcinogens.

- Increase incidence is found in western countries wherein diet rich in animal fat is consumed in large quantity. Incidence is more after the age of 50 years. Obesity, poor exercise and smoking are the contributing factors.
- Some interesting observations are also found in females with colonic cancer which have been depicted in Key Box 29.6.

KEY BOX 29.6

WOMEN AND COLONIC CANCER

- Caecum is more commonly involved than other parts.
- Women with breast cancer have increased incidence of colonic cancer.
- Women smokers have increased chances of colonic cancer.
- Women who have undergone cholecystectomy have increased chances of colonic cancer.
- Metastasis to ovary is mostly haematogenous (1 to 10%).

PEARLS OF WISDOM

Familial polyposis coli has 100% chance of carcinoma.

PEARLS OF WISDOM

Calcium salts are protective. They form insoluble bile salt complexes, thus reducing the concentration of bile acids in the colon.

Pathological types (Fig. 29.15)

- It is an adenocarcinoma—columnar. Rectum (40%) and sigmoid (20%) take a major share in colorectal carcinoma followed by caecum (12 to 15%). Multiple synchronous cancers are also common in the colon.

A. Annular stricture: Common in left colon (splenic flexure, pelvic colon).

B. Tubular stricture: Common in left colon and at the rectosigmoid junction.

C. Ulcerative lesion: Ascending colon or caecum.

D. Proliferative growth: More in right colon, the least malignant, fleshy and bulky polyloid lesion (Fig. 29.16).

- It is a columnar cell adenocarcinoma. In about 5% of cases, it undergoes mucoid degeneration. Such tumours carry poor prognosis. They spread to the liver very fast and secondaries produce mucoid material.

Figs 29.15a to D: Types of carcinoma colon

Fig. 29.16: Ulceroproliferative growth in the descending colon
Clinical features of carcinoma colon (Figs 29.17A to C and Table 29.3) (Mnemonic TMA Pai)

1. **Tumour:** The mass produced by carcinoma caecum and even hepatic flexure is palpable. It is firm to hard, irregular and with or without fixity.
   - Occasionally, growth at pelvirectal junction can be felt on rectal examination.
   - However, on left-sided constrictive lesions, growth is not often felt. It is the hard faecal matter and lymph nodes which are felt as a mass.

2. **Metastasis:** 5–10% of the patients present with metastasis to liver (mucoid adenocarcinoma), ascites, etc. Distant metastasis is not common (Fig. 29.18).

3. **Anaemia** is an important feature of carcinoma caecum. It may be due to blood loss or a proliferative growth secreting toxins causing suppression of bone marow. Asthaemia and anorexia are the other features.

4. **Pain abdomen:** Dull aching pain may be present. Colicky pain is due to chronic obstruction as in left-sided growths (napkin ring stricture).

5. **Alteration in the bowel habits:** A recent constipation, increase in the dose of laxatives followed by attacks of diarrhoea can be due to carcinoma colon. Diarrhoea is due to hard faecal balls, irritating the colonic mucosa resulting in increased secretion of mucus produced by proximal colon.

6. **Intestinal obstruction** is caused by constricted left-sided lesions (Fig. 29.19). On the left side, diameter of the colon is narrow, contents are solid and growth is constrictive. Lower abdominal distension, right to left peristalsis are the

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1Late Padmashree Dr TMA Pai, was the founder of Kasturba Medical College which he started in 1953. He was also a banker, educationist. He is called ‘Modern Architect of Manipal’, where ‘Manipal University’ is situated.
29.21: Pallor disproportionate to the blood loss—typical of carcinoma caecum

late features. Carcinoma sigmoid can cause colovesical fistula.

To summarise
- Early cases: It can be easily missed—such as change in bowel habits like diarrhoea, vague ill health, weakness (due to anaemia), intermittent bleeding per rectum, often attributed to piles or some other cause. Mass is usually not palpable.

Late cases: It can present with obstruction (rectosigmoid junction growth), perforation, intussusception (right-sided tumours), mass abdomen, secondaries in the liver, left supraclavicular nodes (Troisier's sign), etc.

See Key Boxes 29.7 to 29.9.

<table>
<thead>
<tr>
<th>Table 29.3 Carcinoma right colon and carcinoma left colon</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Carcinoma right colon</strong></td>
</tr>
<tr>
<td>Presentation</td>
</tr>
<tr>
<td>Bleeding</td>
</tr>
<tr>
<td>Abdominal discomfort</td>
</tr>
<tr>
<td>Incidence</td>
</tr>
<tr>
<td>Frequency</td>
</tr>
<tr>
<td>Pathology</td>
</tr>
<tr>
<td>Investigation</td>
</tr>
<tr>
<td>Complication</td>
</tr>
<tr>
<td>Unexplained weakness, anaemia</td>
</tr>
<tr>
<td>Occult blood in stools</td>
</tr>
<tr>
<td>Right side and dyspeptic symptoms also</td>
</tr>
<tr>
<td>More common in women</td>
</tr>
<tr>
<td>About 10-20%</td>
</tr>
<tr>
<td>Ulcerative/proliferative lesion</td>
</tr>
<tr>
<td>Colonoscopy</td>
</tr>
<tr>
<td>Obstruction—less common</td>
</tr>
</tbody>
</table>

PEARLS OF WISDOM
Obstructed and perforated carcinoma colon have poor prognosis.

Spread
1. Local: For a long time, the lesion is confined to mucosa and submucosa. They grow in annular fashion and later...
longitudinally. Once serosa is involved, spread occurs rapidly into neighbouring structures such as ureter, bladder, uterus, etc. The involvement of these structures is not a contraindication for surgery (TNM staging).

- Local perforation may result in pericolic abscess.
- Hollow viscus perforation results in internal fistula.

2. Lymphatic spread (see page 702)

3. Blood spread: It occurs late, resulting in secondaries in the lungs, liver, etc. Cannon ball in lung nodule in the liver.

PEARLS OF WISDOM

Because of the drainage into the portal system, colonic cancers spread to the liver first. On the other hand, rectal cancers spread to the lungs because of drainage into inferior vena cava.

STAGING/CLASSIFICATIONS

There are many classifications and staging for carcinoma colon/rectum. They are not important. A few important ones have been given here.

I. Dukes’ staging for colorectal cancer

- **Stage A**: Invasion of but not breaching the muscularis propria.
- **Stage B**: Breaching the muscularis propria but not involving the lymph nodes.
- **Stage C**: Lymph nodes are involved.
  
  Few authors describe a stage D for metastatic disease. Since it has not been described by Duke, it is called modified Dukes’ staging.

II. : Astler-Coller’s modification of Dukes’ staging

Stages (Fig. 29.29)

- **A** Limited to mucosa—no nodes
- **B1** Extension into muscularis propria—no nodes
- **B2** Extension into entire bowel wall—no nodes
- **B3** Extension into adjacent organs—no nodes
- **C1** Extension into muscularis propria—positive nodes
- **C2** B2 + Lymph nodes
- **C3** B3 + Lymph nodes
- **D** Distant metastasis

III. WHO classification—it is based on histology

- Majority are adenocarcinoma—90%
- Mucinous adenocarcinoma—5–10%
- Signet ring cell carcinoma
- Small cell carcinoma
- Squamous cell carcinoma
- Undifferentiated carcinoma

IV. TNM staging (see text on the right side)

A few clinical photograph, staging pictures and operative pictures have been given in next page.
Fig. 29.22: A 34-year-old lady presented to the hospital with mass in the right iliac fossa with slight flexion of the right hip. Mass was hard and irregular. She also had anaemia.

Fig. 29.23: Exploration of the mass (caecum). It was mobile.

Fig. 29.24: Right hemicolectomy specimen—a case of carcinoma caecum.

Fig. 29.28: Intestinal obstruction due to rectosigmoid stricture—a common complication. Tumour rarely goes beyond 2 cm from the edge of the tumour unless there is concomitant spread to lymph nodes.

Fig. 29.25: Carcinoma hepatic flexure with partial obstruction.

Fig. 29.26: Extended right hemicolectomy specimen.

Fig. 29.27: Rectosigmoid stricture. Patient underwent high anterior resection. 5 cm proximal margin is enough for radical cure.

Fig. 29.30: Limited colectomy leak—managed by refashioning of the stoma followed by ileostomy.