SMALL BOWEL NEOPLASMS

Benign neoplasms account for 30 to 50% of small bowel tumors and include adenomas, lipomas, hamartomas and hemangiomas. Primary small bowel cancers are rare with adenocarcinoma comprise 35 to 50% of all cases. Carcinoid tumors 20 to 40% and lymphoma 10%. Gastrointestinal stromal tumors (GISTs) are the most common mesenchymal tumors arising in the small intestine and forms 10 to 15% of all small bowel malignancies. GISTs comprise the tumors that were formerly classified as leiomyomas, leiomyosarcomas, and smooth muscle tumors of the intestine.

Small intestine can be affected by metastasis from other site and melanoma is associated with a propensity for this metastasis.

Most patients with small intestinal cancers are in their fifth or sixth decade of life. Risk factors for developing small intestinal cancers include consumption of red meat, ingestion of smoked foods, Crohn's disease, familial adenomatous polyposis (FAP), and Peutz-Jeghers syndrome.

Peutz–Jeghers syndrome

This is an autosomal dominant disease.

The gene STK11 on chromosome 19 has been found in a proportion of patients with this condition. This consists of:

• Intestinal hamartomatosis is a polyposis affecting the whole of the small bowel, (Fig. 1) and colon, where it is a cause of hemorrhage and often intussusception; these polyps can contain adenomatous foci that can undergo malignant transformation.

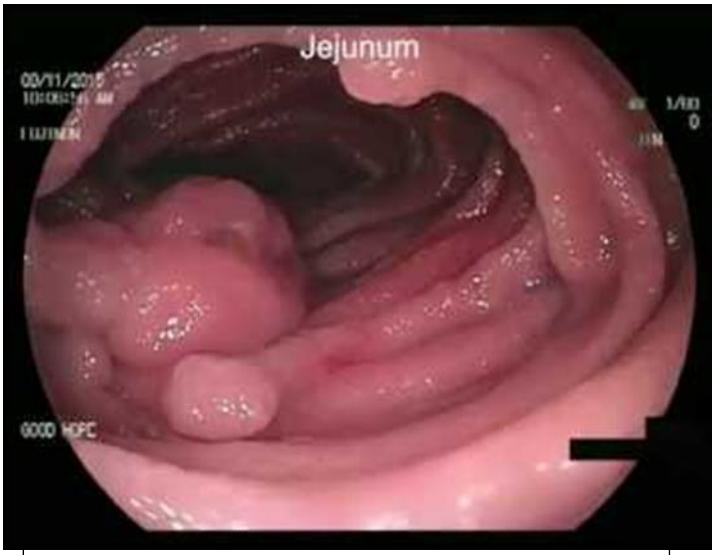

Figure 1: hamartomtous polyp of small intestine

• Melanosis of the oral mucous membrane and the lips. The melanosis takes the form of melanin spots sometimes present on the digits and the perianal skin, but pigmentation of the lips is the sine qua non (Fig. 2).

Figure 2: melanin spots on Melanin spots on the lips of a patient afflicted with Peutz–Jeghers syndrome

Long-term follow-up of patients with Peutz–Jeghers syndrome has shown reduced survival secondary to complications of recurrent bowel cancer and the development of a wide range of cancers. These include colorectal, gastric, breast, cervical, ovarian, pancreatic and testicular cancer. It is therefore important to keep these patients under surveillance. This can be done by endoscopy or contrast examinations every 3 years to detect early gastrointestinal cancers. It is also important to make sure that female patients attend cervical and breast screening programs.

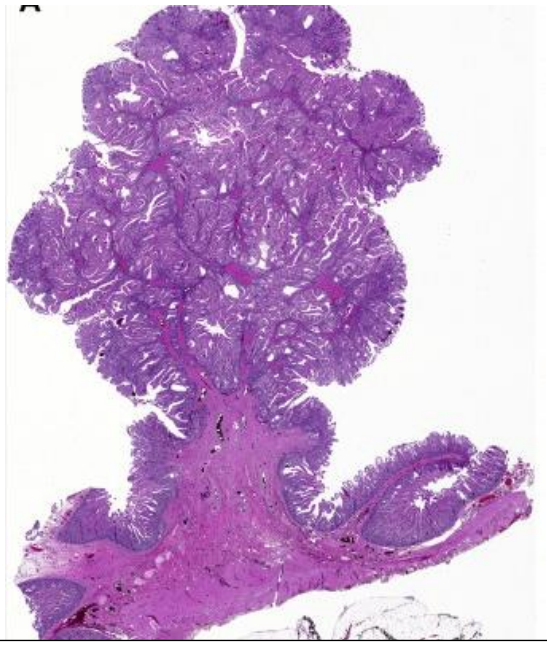

Figure 3: the treelike branching with central smooth muscle bundles

Histology

The polyps can be likened to trees. The trunk and branches are smooth muscle fibers and the foliage is virtually normal mucosa. (Fig.3)

Treatment

As malignant change rarely occurs, resection is only necessary for serious bleeding or intussusception. Large single polyps can be removed by enterotomy, or short lengths of heavily involved intestine can be resected. The incidence of further lesions developing problems in the future can be reduced by thorough intraoperative examination at the time of the first laparotomy. Using on-table enteroscopy, polyps suitable for removal can be identified. Those lesions within reach can be snared by colonoscopy.

Pathology

Small intestinal adenocarcinomas are believed to arise from preexisting adenomas in a way similar to the development of colorectal cancer. They are histologically classified as tubular, villous, and tubulovillous. Tubular adenomas have the least aggressive features. Villous adenomas have the most aggressive features and usually present in the second portion of the duodenum. Malignant degeneration present in up to 45% of villous adenomas.

Clinical presentation

Most small intestinal neoplasms are asymptomatic until they become large. Partial small bowel obstruction, with associated symptoms of crampy abdominal pain and obstruction distension, nausea, and vomiting, is the most common mode of presentation. Obstruction can be the result of either luminal narrowing by the tumor itself or intussusception, with the tumor serving as the lead point.
Haemorrahage, usually indolent, is second most common mode of presentation.
Physical examination may be unrevealing. Up to 25% of patients present with palpable abdominal mass. Findings of intestinal obstruction are reported to be present in 25% of patients. Cachexia, hepatomegaly, jaundice, and ascites may be present with advanced disease.
Although the clinical presentation usually is not specific for tumor type, some general comments are appropriate.
Adenocarcinomas, as well as adenomas are most commonly found in the duodenum, except in patients with Crohn's disease, in whom most are found in the ileum. Adenocarcinomas located in the duodenum tend to be diagnosed earlier in

their progression than those located in the jejunum or ileum, which are rarely diagnosed before the onset of locally advanced or metastatic disease.
Carcinoid tumors of the small intestine also are usually diagnosed after the development of metastatic disease. These tumors are associated with a more aggressive behavior than the more common appendiceal carcinoid tumors. Approximately 25 to 50% of patients with carcinoid tumor-derived liver metastasis will develop manifestations of the carcinoid syndrome. These manifestation include diarrhea, flushing, hypotension, tachycardia and fibrosis of the endocardium and valves of the right heart. Tumor derived mediators of the carcinoid syndrome such serotonin, bradykinin, and substance P undergo nearly complete metabolism during first passage through the liver. As a result, symptoms of carcinoid syndrome are rare in the the absence of liver metastasis
Lymphoma may involve the small intestine primarily or as a manifestation of disseminated systemic disease. Primary small intestinal lymphomas are most commonly located in the ileum, which contains the highest concentration of lymphoid tissue in the intestine. Although partial small bowel obstruction is the most common mode of presentation, 10% of patients with small intestinal lymphoma present with bowel perforation.
Sixty to 70% of GISTs are located in the stomach. The small intestine is the second most common site with 25 to 35%. GISTs are associated with hemorrhage more than than other small intestinal malignancies.

Diagnosis

Laboratory tests are nonspecific, with the exception of elevated serum 5-hydroxyindole acetic acid levels in patients with carcinoid syndrome.

Contrast radiography of the small intestine may demonstrate benign and malignant lesion. Enteroclysis or small bowel enema have sensitivity of over 90% in the detection of small bowel tumors. (Fig.4a,b)

CT is helpful in detecting large tumors and in staging.

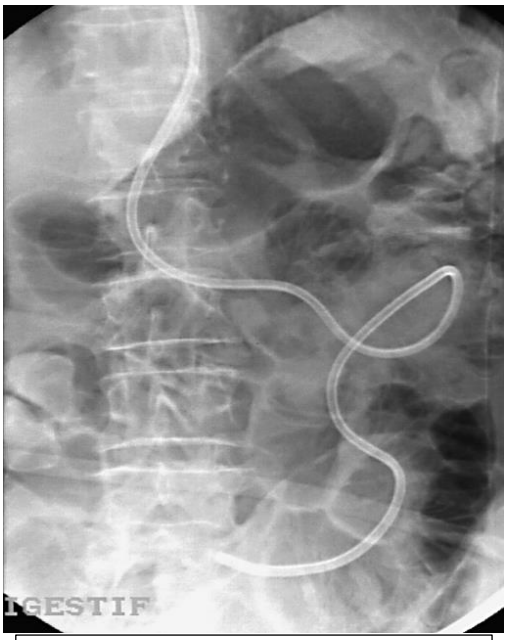

Figure 4a: enteroclysis catheter in jejunum

EGD is useful in detecting duodenal lesions. Capsule endoscopy is recent modality and useful n diagnosis.

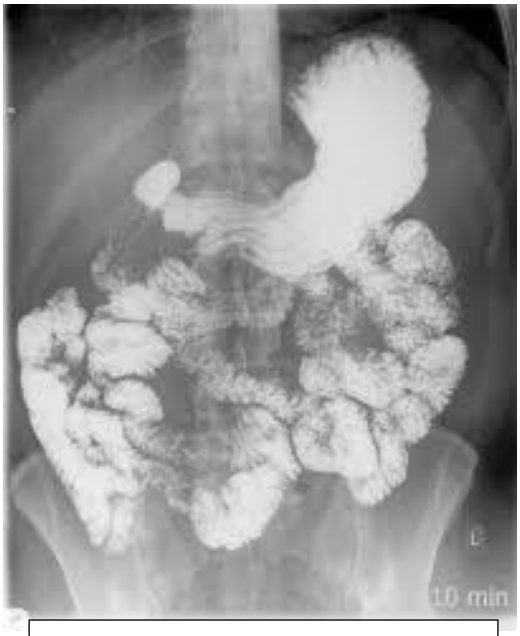

Figure 4b: normal small bowel meal

Treatment

Benign neoplasms of the small intestine that are symptomatic should be surgically resected or removed endoscopically, if feasible.

Tumors located in the duodenum, including asymptomatic lesions, can impose therapeutic challenge. These lesions should be biopsied; symptomatic tumors and adenomas, because of their malignant potential, should be removed.

Surgical therapy of jejunal and ileal malignancies usually consist of wide local resection of the intestine harboring the lesion. Chemotherapy has no role.

The goal of surgical therapy for carcinoids is resection of all visible disease. Localized small intestinal carcinoid tumors should be treated with segmental intestinal resection and regional lymphadenectomy. Nodal metastasis are unusual with tumors less than 1 cm in diameter. In the presence of metastatic disease, tumor debulking should be conducted as it can be associated with long term survival and amelioration of symptoms. Chemotherapy results in response of 30 to 50% patients based on agents used such as doxorubicin, 5-fluorouracil, and streptozocin. Octreotide is the most effective pharmacologic agent for management of symptoms of carcinoid syndrome.

Localized small intestinal lymphoma should be treated with segmental resection of the involved intestine and adjacent mesentery. If the small intestine is diffusely affected by lymphoma, chemotherapy rather than surgical resection should be the primary therapy.

Small intestinal GISTs should be treated with segmental intestinal resection. If the diagnosis is known before resection, wide lymphadenectomy can be avoided as GISTs are rarely associated with lymph node metastasis. GISTs are resistant to conventional chemotherapy agents. A defining feature of GISTs is their gain of function mutation of protooncogene KIT, a receptor tyrosine kinase. Because the interstitial cells of Cajal normally express KIT, these cells have been implicated as the cell of origin for GISTs. Imatinib is a tyrosine kinase inhibitor with potent activity against tyrosine kinase, and is used in those with metastatic disease.

TUMORS OF LARGE INTESTINE

Benign

The term ‘polyp’ is a nonspecific clinical term that describes any projection from the surface of the intestinal mucosa regardless of its histologic nature. It covers a variety of histologically different tumours shown in Table 1. Polyps can occur singly, synchronously in small numbers or as part of a polyposis syndrome. In familial adenomatous polyposis (FAP), more than 100 adenomas are present. It is important to be sure of the histological diagnosis because adenomas have significant malignant potential.

Table 1 Classification of polyps of the large intestine Class Varieties

- Inflammatory — Inflammatory polyps
- Metaplastic — Metaplastic or hyperplastic polyps
- Harmartomatous — Peutz–Jeghers polyp

- Neoplastic
  - Juvenile polyp
  - Adenoma
    - Tubular – Tubulovillous – Villous
  - Adenocarcinoma
  - Carcinoid tumour

Adenomatous polyps

Adenomatous polyps are common. By definition these lesions are dysplastic. The risk of malignant degeneration depends on both the size and type of polyp. Tubular adenomas are associated with malignancy in only 5% of cases, whereas villous adenomas may harbor cancer in up to 40%. Tubulovillous adenomas are at intermediate risk.

Invasive carcinomas are rare in polyps smaller than 1 cm and the incidence increases with size. Although most neoplastic polyps do not evolve to cancer, most colorectal cancers originate as a polyp. It is this fact that forms the basis for secondary prevention strategies to eliminate colorectal cancer by targeting the neoplastic polyp for removal before malignancy develops.

Polyps may be pedunculated or sessile. Most pedunculated polyps are amenable to colonscopic snare excision. Solitary adenomas are usually found during the investigation of colonic bleeding or sometimes incidentaly. Villous tumours more usually give symptoms of diarrhoea, mucus discharge and occasionally hypokalaemia. Huge villous adenomas of the rectum can be difficult to remove even with techniques per anus, and occasionally proctectomy is required; the anal sphincter can usually be preserved.

Familial adenomatous polyposis

FAP is a rare autosomal dominant condition accounts for only about 1% of all colorectal adenocarcinomas. The genetic abnormality is a mutation in the APC gene, located on chromosome 5. (Fig. 5)

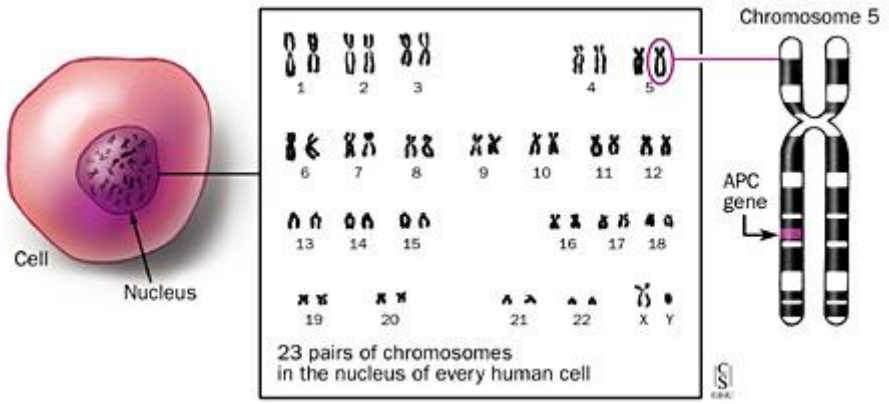

Figure 5: Chromosome 5 for FAP.

Clinically FAP is defined by the presence of more than 100 colorectal adenomas. Over 80% of cases come from patients with a positive family history. However, 20% arise as a result of new mutations of APC gene. It is less common than hereditary non-polyposis colorectal cancer (HNPCC). Although the large bowel is mainly affected, polyps can occur in the stomach, duodenum and small intestine. The main risk is large bowel cancer, but duodenal and ampullary tumours have been reported. The risk of colorectal cancer is 100% in patients with FAP. Males and females are equally affected. It can also occur sporadically without any previous sign or history, presumably by new mutations. There is often, in these cases, a history of large bowel cancer occurring in young adulthood or middle age, suggesting pre-existing adenomatosis.

FAP may be associated with extra intestinal manifestations, such as desmoid tumours, epidermoid cysts and mandibular osteomas (Gardner's syndrome); and central nervous system tumors (Turcot's syndrome).

Desmoid tumors in the abdomen invade locally to involve the intestinal mesentery and, although non-metastasising, they can become unresectable.

Clinical features

Polyps are usually visible on sigmoidoscopy by the age of 15 years and will almost always be visible by the age of 30 years. Carcinoma of the large bowel occurs 10–

20 years after the onset of the polyposis. One or more cancers will already be present in two-thirds of those patients presenting with symptoms.

Symptomatic patients

These are either patients in whom a new mutation has occurred or those from an affected family who have not been screened. They may have loose stools, lower abdominal pain, weight loss, diarrhoea and the passage of blood and mucus. Polyps are seen on sigmoidoscopy, and the number and distribution of polyps, and usually cancers if they are symptomatic, are shown on a double contrast barium enema. If in doubt, colonoscopy is performed with biopsies to establish the number and histological type of polyps. If over 100 adenomas (Fig. 6) are present, the diagnosis can be made confidently, but it is important not to confuse this with non-neoplastic forms of polyposis

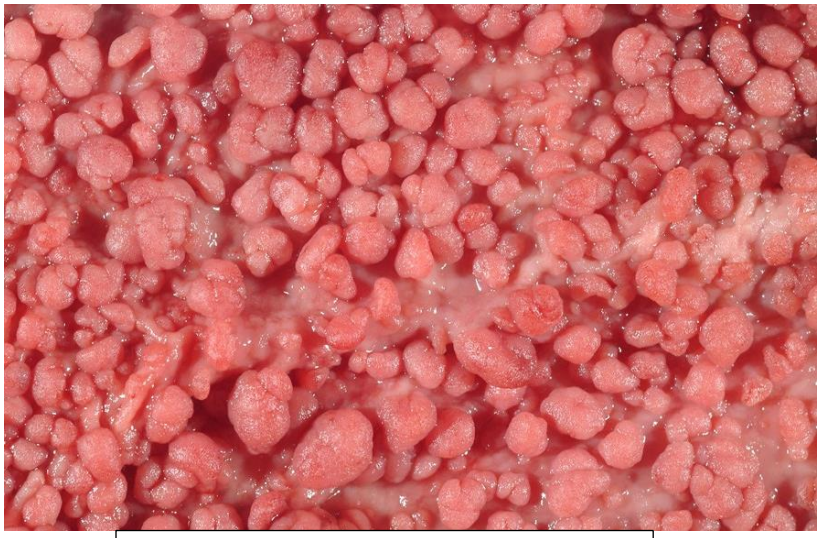

Figure 6: familial adenomatous polyposis

Asymptomatic patients

Direct genetic testing will reveal mutations in 80% of cases. In the presence of an identified mutation in a family with FAP, any resulting negative tests for this can be interpreted to mean that these individuals do not carry the mutation. They can therefore be withdrawn from surveillance programmes and warned that they are at normal population 'risk' of developing colorectal cancer. In those families where a mutation cannot be identified, then surveillance is recommended annually. The site of the mutation within the gene has important effects on the phenotype. Truncations of the carboxy end of the APC protein have a smaller effect on tumour

suppressor function. This results in the attenuated FAP variant. If there are no adenomas by the age of 30 years, FAP is unlikely. If the diagnosis is made during adolescence, operation is usually deferred to the age of 17 or 18 years or when symptoms or multiple polyps develop.

Screening policy

1- At-risk family members are offered genetic testing in their early teens.
2- At-risk members of the family should be examined at the age of 10–12 years, repeated every year.
3- Most of those who are going to get polyps will have them at 20 years, and these require operation.
4- If there are no polyps at 20 years, continue with 5-yearly examination until age 50 years; if there are still no polyps, there is probably no inherited gene. Carcinomatous change may exceptionally occur before the age of 20 years. Examination of blood relatives, including cousins, nephews and nieces, is essential, and a family tree should be constructed and a register of affected families maintained.

Treatment

Colectomy with ileorectal anastomosis has in the past been the usual operation because it avoids an ileostomy in a young patient and the risks of pelvic dissection to nerve function. The rectum is subsequently cleared of polyps by snaring or fulguration. The patients are examined by flexible sigmoidoscopy at 6-monthly intervals thereafter. In spite of this, a proportion of patients develop carcinoma in the rectal stump. The risk of carcinoma in the St Mark's series was 10% over a period of 30 years. The alternative is a restorative proctocolectomy with an ileoanal anastomosis. This has a higher complication rate than ileorectal anastomosis. It is indicated in patients with serious rectal involvement with polyps, those who are likely to be poor at attending for follow-up and those with an established cancer of the rectum or sigmoid. However, it is now used more frequently for less severe cases. There have been reports of cancers developing after stapled anastomosis when a small remnant of rectal mucosa is left behind.

Postoperative surveillance

Because of the risk of further tumour formation, follow-up is important and takes the form of rectal/pouch surveillance. Gastroscopies are carried out to detect upper gastrointestinal tumours.

Hereditary non-polyposis colorectal cancer (Lynch's syndrome)

HNPCC is more common than FAP, but is still rare (1 to 3%) and is characterized by the development of colorectal carcinoma at an early age (40 to 45 years). Cancers appear in the proximal colon more often than in sporadic colorectal cancer and have a better prognosis regardless of stage. HNPCC also may be associated with extra colonic malignancies, including 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 – MLH1, MSH2, MSH6, PMS and PMS2. Most people with this syndrome have mutations in the MLH1 and MSH2 genes. The lifetime risk of developing colorectal cancer is 80%, and the mean age of diagnosis is 44 years.

Diagnosis

HNPCC can be diagnosed by genetic testing or Amsterdam criteria II:

- three or more family members with a HNPCC-related cancer, one of whom is a first-degree relative of the other two;
- two successive affected generations;
- one or more of the HNPCC-related cancers diagnosed before the age of 50 years;
- exclusion of FAP.

ADENOCARCINOMA

Incidence

Colorectal carcinoma is the most common malignancy of the GI tract. The incidence is similar in men and women.

Risk factors

- Aging

  Aging is the most important risk factor for colorectal cancer, with incidence rising steadily after age 50 years. However individuals of any age can develop colorectal cancer, so symptoms such as a significant change in bowel habits, rectal bleeding, melena, unexplained anemia, or weight loss require a thorough evaluation.

- Hereditary Risk Factors

  Approximately 80% of colorectal cancers occur sporadically, while 20% arise in patients with a known family history of colorectal cancer.

- Environmental and Dietary factors

  Diet high in saturated and polysaturated fats increases risk of colorectal cancer while diet high in vegetable fibers appears to be protective. In addition obesity and sedentary life style dramatically increase cancer-related mortality in a number of malignancies including colorectal cancer.

- Inflammatory bowel disease

  In ulcerative colitis, the risk of carcinoma is approximately 2% after 10 years, 8% after 20 years and patients with Crohn's pancolitis have similar risk.

- Other risk factors

  Includes cigarette smoking, ureterosigmoidostomy, acromegaly, and pelvic irradiation.

Genetics

There has been an explosion of information on the molecular genetics of sporadic colorectal cancer. APC mutations occur in two-thirds of colonic adenomas and carcinomas and are thought to present early in the carcinogenesis pathway. K-RAS mutations result in activation of cell signalling pathways. They are more common in larger lesions, thus implying that they are later events in the mutagenesis pathway. Other genes involved include p53. However, it must be noted that the pathway is not one of a simple stepwise progression of mutations but a complicated array of multiple gene changes, which result in an outcome of cancer. No single mutation is a common theme for all colorectal cancer cases. However, knowledge of certain mutations can be used to assess prognosis and direct adjuvant therapy.

Pathology

Microscopically, the neoplasm is a columnar cell carcinoma originating in the colonic epithelium. Macroscopically, the tumour may take one of four forms Fig. 7: (1, 2, 3, and 4).

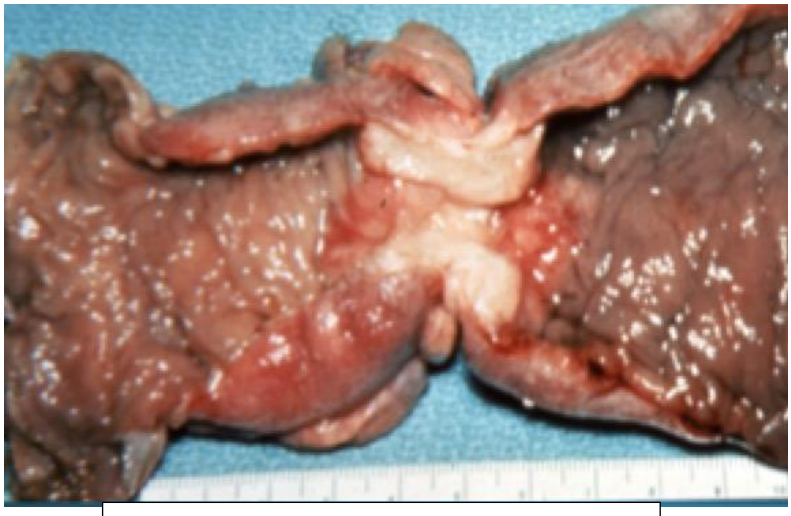

Figure 7: (1) Annular type of colonic tumor

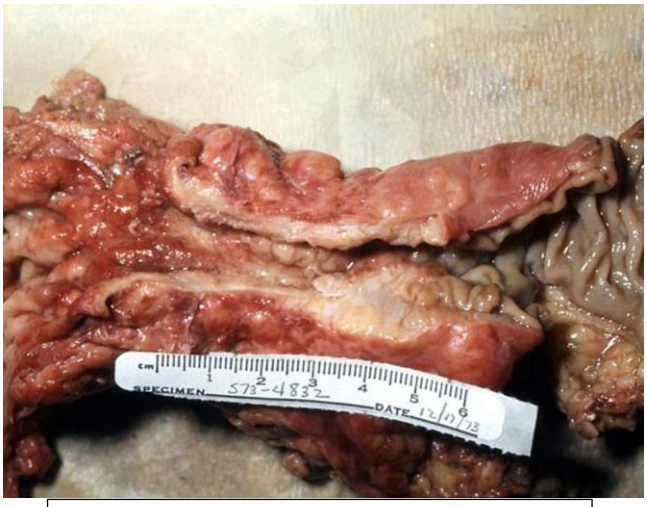

Figure 7: (2) Tubular type of colonic tumor

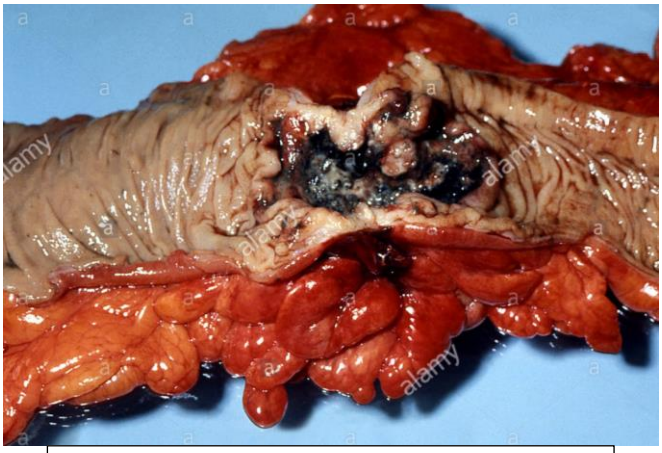

Figure 7: (3) Ulcerative type o colonic tumor

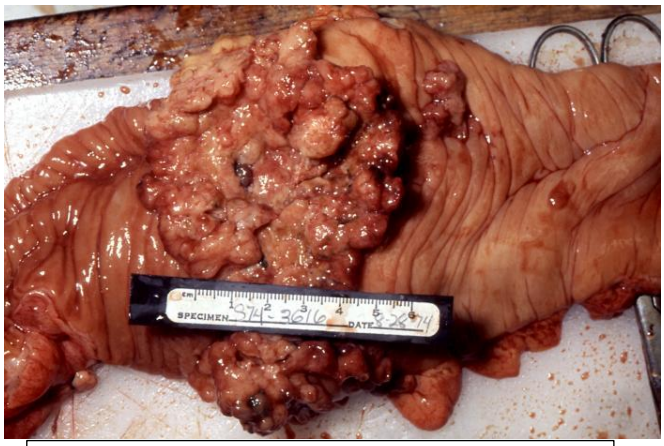

Figure 7: (4) cauliflower type of colonic tumor

Type 4 is the least malignant form. It is likely that all carcinomas start as a benign adenoma, the so called 'adenoma–carcinoma sequence'. This is supported by the observation that the prevalence of adenomas correlates with carcinoma. The distribution of adenoma in the colon also mirrors that of carcinoma. The annular variety tends to give rise to obstructive symptoms, Whereas the others will present more commonly with bleeding. The sites and distribution of cases of cancer are shown in Figure 8. Tumours are more common in the left colon and rectum

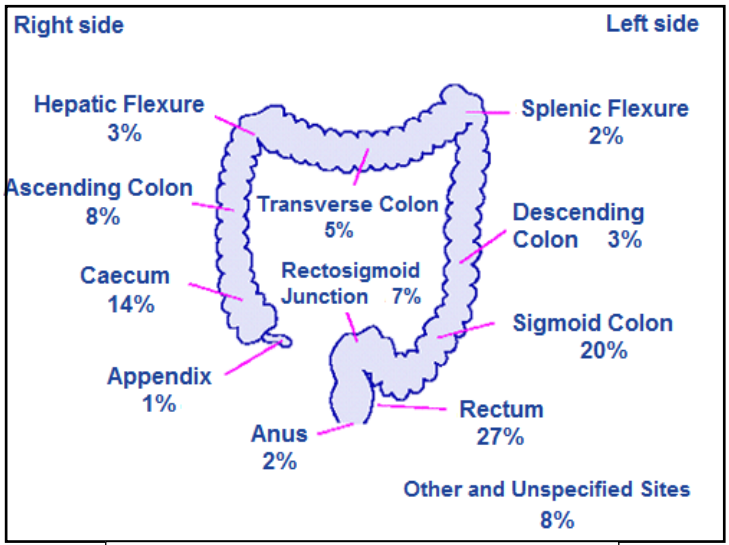

Figure 8: Colorectal cancer distribution

The spread of carcinoma of the colon

Generally this is a comparatively slow-growing neoplasm.

Local spread

The tumour can spread in a longitudinal, transverse or radial direction; it spreads round the intestinal wall and usually causes intestinal obstruction before it invades adjacent structures. The ulcerative type more commonly invades locally, and an internal fistula may result, for example into the bladder. There may also be a local perforation with an abscess or even an external faecal fistula. This type of radial spread to adjacent organs has the largest impact on prognosis. The progression of invasion occurs across the submucosa into the muscularis propria and thence out into the serosa and fat, lymphatics and veins in the mesentery alongside the bowel wall.

Lymphatic spread

Regional lymph node involvement is the most common form of spread of colorectal carcinoma and usually precedes distant metastasis or the development of carcinomatosis (diffuse peritoneal metastasis). Lymph nodes draining the colon are grouped as follows:

N1: nodes in the immediate vicinity of the bowel wall.

N2: nodes arranged along the ileocolic, right colic, midcolic, left colic and sigmoid arteries;

N3: the apical nodes around the superior and inferior mesenteric vessels where they arise from the abdominal aorta. Involvement of the lymph nodes by the tumour progresses in a gradual manner from those closest to the growth along the course of the lymphatic vessels to those placed centrally. The T stage (depth of invasion) is the single most significant predictor of lymph node spread.

Bloodstream spread

This accounts for a large proportion (30–40%) of late deaths. Metastases are carried to the liver via the portal system, sometimes at an early stage before clinical or operative evidence is detected (occult hepatic metastases). The lung is also a site for haematogenous spread but it rarely occur in isolation.

Transcoelomic spread

Rarely, colorectal cancer can spread by way of cells dislodging from the serosa of the bowel or via the subperitoneal lymphatics to other structures within the peritoneal cavity.

Staging colon cancer

There are several staging systems that are used such as Dukes, tumour–node–metastasis (TNM) and Jass. All of them can be used in order to predict prognosis and standardise treatment (Fig. 9). Dukes' classification was originally described for rectal tumours, but has been adopted for histopathological reporting of colon cancer as well. There have been numerous modifications of the original system, leading to some confusion but, in its most basic form, Dukes' classification for colon cancer is as follows:

A: confined to the bowel wall;
B: through the bowel wall but not involving the free peritoneal serosal surface;
C: lymph nodes involved.

Dukes himself never described a D stage, but this is often used to describe either advanced local disease or metastases to the liver.

Dukes	Definition	5-year survival
A	Invasion confined to the mucosa	90%
B	Infiltration through muscle	70%
C	Lymph node involvement	30%
D	Distant metastases	5%

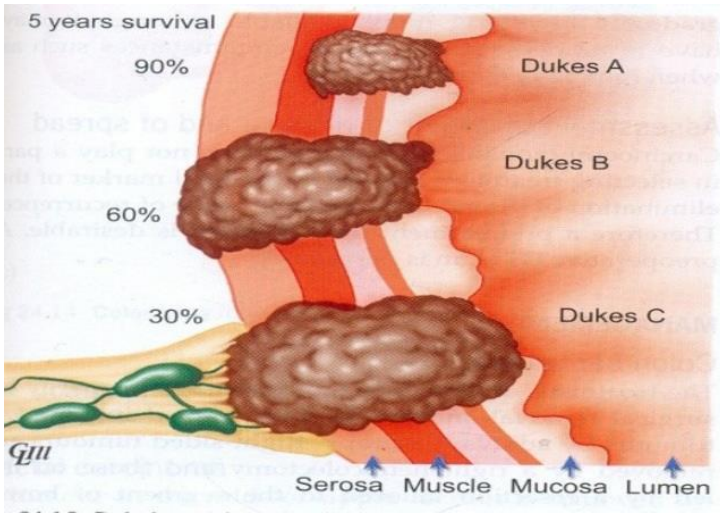

Figure 9: Dukes staging. Table & diagram

TNM classification

The TNM classification is more detailed and accurate but more demanding:

• T Tumour stage;
• T1 Into submucosa;
• T2 Into muscularis propria;
• T3 Into pericolic fat but not breaching serosa;
• T4 Breaches serosa or directly involving another organ;
• N Nodal stage;
• N0 No nodes involved;
• N1 One or two nodes involved;
• N2 Three or more nodes involved;
• M Metastases;
• M0 No metastases;
• M1 Metastases;
• Ly Lymphatic invasion
; • L0 No lymphatic vessels involved;
• L1 Lymphatics involved;
• V Venous invasion;
• V0 No vessel invasion;
• V1 Vessels invaded;
• R Residual tumour;
• R0 No residual tumour;
• R1 Margins involved, residual tumour present

Clinical features

Carcinoma of the colon usually occurs in patients over 50 years of age, but it is not rare earlier in adult life.

The colorectal cancers have wide range of presentations depend on the

- Site of the tumor
- Presence of complications
- Presence of metastasis

Twenty per cent of cases present as an emergency presentations in form of

- Acute intestinal obstruction
- Perforation result in fecal peritonitis
- Sever per rectal bleeding or malena

Metastasis presentation

Patients may present for the first time with liver metastases and an enlarged liver, ascites from carcinomatosis peritonei and, more rarely, metastases to the lung, skin, bone and brain.

Carcinoma of the left side of the colon

Most tumours occur in this location. They are usually of the stenosing variety.

Symptoms The main symptoms are those of increasing intestinal obstruction. This includes lower and left iliac fossa abdominal pain, which may be colicky in nature, and abdominal distension. The patient may have a change in bowel habit with alternating diarrhoea and constipation, and may develop bleeding per rectum.

Carcinoma of the sigmoid

In addition to symptoms of intestinal obstruction, a low tumour may give rise to a feeling of the need for evacuation, which may result in tenesmus accompanied by the passage of mucus and blood. Bladder symptoms are not unusual and, in some instances, may herald a colovesical fistula.

Carcinoma of the transverse colon

This may be mistaken for a carcinoma of the stomach because of the position of the tumour together with anaemia and lassitude.

Carcinoma of the caecum and ascending colon

This may present with the following:

- Anaemia, severe and unyielding to treatment;
- The presence of a mass in the right iliac fossa; colonoscopy may be needed to confirm the diagnosis;
- A carcinoma of the caecum can be the apex of an intussusception presenting with the symptoms of intermittent obstruction

Methods of investigation of colon cancer

- Fecal occult blood testing

Used mainly in screening. However, FOBT is relatively insensitive, missing up to 50% of cancers and the majority of adenomas. Its specificity is low because 90% of patients with positive tests do not have colorectal cancer. A positive FOBT should be followed by colonoscopy.

- Flexible sigmoidoscopy

The 60-cm, fibreoptic, flexible sigmoidoscope is increasingly being used in the out-patient clinic or in special rectal bleeding clinics. The patient is prepared with a disposable enema and sedation is not usually necessary. This is particularly useful in supplementing barium investigations where diagnosis is difficult due to diverticular disease. Patients found to have a polyp, cancer, or other lesion on flexible sigmoidoscopy will require colonoscopy.

- Colonoscopy

This is now the investigation of choice if colorectal cancer is suspected provided the patient is fit enough to undergo the bowel preparation. It has the advantage of not only picking up a primary cancer but also having the ability to detect synchronous polyps or even multiple carcinomas, which occur in 5% of cases. Ideally, every case should be proven histologically before surgery. Full bowel preparation and sedation are necessary. However, one must be aware of a small risk of perforation and haemorrahge, and also the failure to get to the caecum in 10% of cases, even by experienced endoscopists.

- Radiology Double-contrast barium enema

Is used when colonoscopy is contraindicated. It shows a cancer of the colon as a constant irregular filling defect (Fig.10). False positives occur in 1–2% of cases and false negatives in 7–9% of cases.

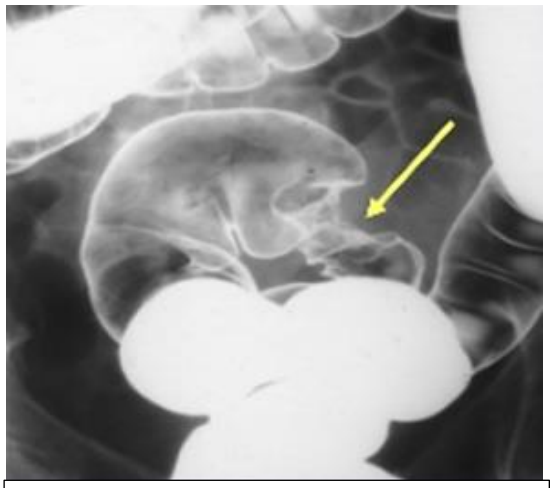

Figure 10: Filling defect by barium enema

- Ultrasonography

  Is often used as a screening investigation for liver metastases over the size of 1.5cm.

- Spiral CT

  CT is used in patients with large palpable abdominal masses (Fig. 11), to determine local invasion, and is particularly used in the pelvis in the assessment of rectal cancer. Spiral CT is particularly useful in elderly patients when contrast enemas or colonoscopy are not diagnostic or are contraindicated. In some centres, it is standard investigation above the age of 80 years. (Fig. 12)

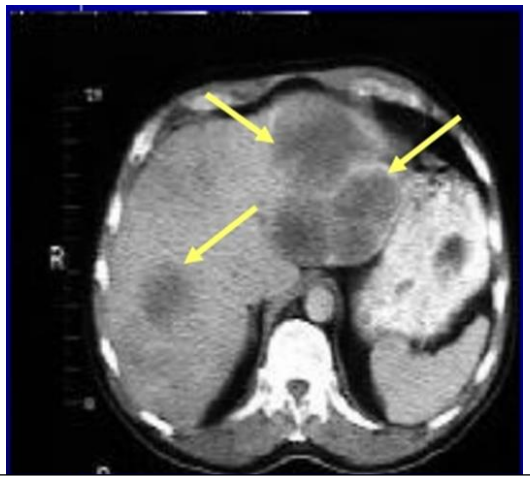

Figure 11: Several low density metastasis from colonic tumor involves both lobes of liver

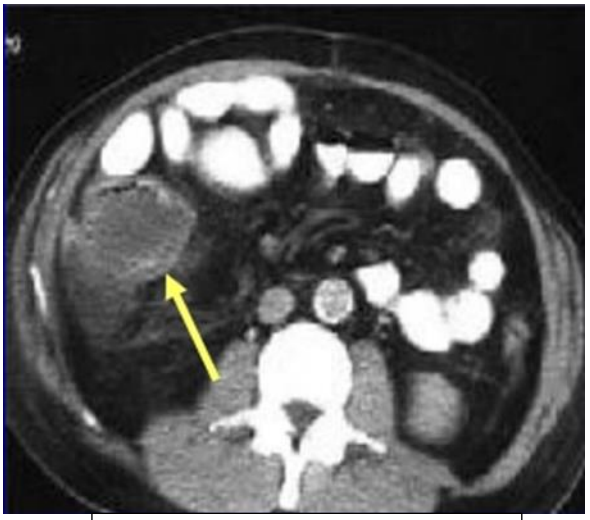

Figure 12: Cecal wall thickening

- Virtual colonoscopy

With the advent of technology in this field, there has been the introduction of virtual colonoscopy, which is effective in picking up polyps down to size of 6mm (Fig. 13). This may even replace colonoscopy as the standard investigation in the future.

- Urograms

Have a role in left-sided tumours where there is evidence of hydronephrosis on CT or ultrasound.

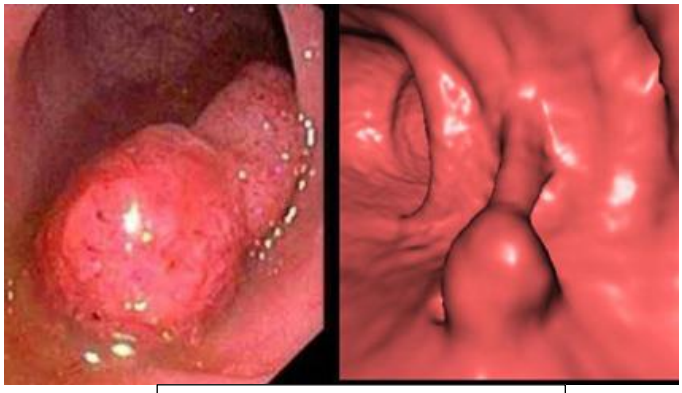

Figure 13: Virtual colonoscopy

TREATMENT

Successful treatment requires a multidisciplinary team of CRC specialists:

- Surgical oncologist
- Medical oncologist
- Radiation oncologist
- Radiologist
- Pathologist
- Oncology nurse specialist
- Social worker
- Nutritionist
- Pharmacist

Preoperative preparation

- Recent literature has suggested that no bowel preparation is safe for right-sided colonic surgery. The most commonly used method is dietary restriction to fluids only for 48 hours before surgery; on the day before the operation, two sachets of Picolax (sodium picosulphate) (Fig. 14) are taken to purge the colon. In addition, a rectal washout may be necessary.
- A stoma site is carefully discussed with the stoma care nursing specialist
- DVT prevention and anti-embolus stockings are fitted; the patient is started on DVT/PE prophylaxis

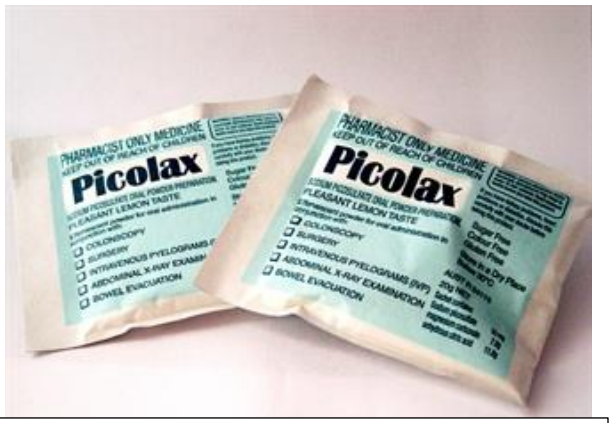

Figure 14: Each Picolax sachet contains the following active ingredients: Sodium picosulfate 10mg Magnesium carbonate 7.9g (equivalent to 3.36g Magnesium oxide (MgO))
Citric acid-anhydrous 11.8gUM PICOSULFATE 10mg: 16g

- Intravenous prophylactic antibiotics are given at the start of surgery.
    - When intestinal obstruction is present, preparation in this way may precipitate abdominal pain, and it may be safer to use an on table lavage technique at the time of the operation.

The test of operability

The abdomen is opened and the tumour assessed for resectability.

1 The liver is palpated for secondary deposits, the presence of which is not necessarily a contraindication to resection because the best palliative treatment for carcinoma of the colon is removal of the tumour.

2 The peritoneum, particularly the pelvic peritoneum, is inspected for signs of small, white, seed-like, neoplastic implantations. Similar changes can occur in the omentum.

3 The various groups of lymph nodes that drain the involved segment are palpated. Their enlargement does not necessarily mean that they are invaded by metastases, because the enlargement may be inflammatory.

4 The neoplasm is examined with a view to mobility and operability. Local fixation, however, does not always imply local invasion because some tumours excite a brisk inflammatory response

Operations

The operations to be described are designed to remove the primary tumour and its draining locoregional lymph nodes, which may be involved by metastases. Lesser resections are indicated, however, should hepatic metastases render the condition incurable surgically. The general principles include:

- Early division of major blood vessels supplying the involved colon (no-touch technique – Turnbull) can slightly improve the number of curative operations.
- Avoidance of contamination by bowel content

The use of stapling and hand-suturing techniques for colonic anastomosis has been compared, and there is little difference in leak rate between the two.

Carcinoma of the caecum

Carcinoma of the caecum or ascending colon is treated when resectable by right hemicolectomy (Fig.15 A). The abdomen is opened, the peritoneum lateral to the ascending colon is incised, and the incision is carried around the hepatic flexure.

The right colon is elevated, with the leaf of peritoneum containing its vessels and lymph nodes, from the posterior abdominal wall, taking care not to injure the ureter, spermatic vessels in the male or the duodenum. The peritoneum is separated medially near the origin of the ileocolic artery, which is divided together with the right colic artery when this has a separate origin from the superior mesenteric. The mesentery of the last 30cm of ileum and the leaf of raised peritoneum attached to the caecum, ascending colon and hepatic flexure, after ligation of the mesenteric blood vessels, is divided as far as the proximal third of the transverse colon. When it is clear that there is an adequate blood supply at the resection margins, the right colon is resected, and an end-to-end anastomosis is fashioned between the ileum and the transverse colon.

Carcinoma of the hepatic flexure

When the hepatic flexure is involved, the resection must be extended correspondingly (Fig. 15B).

Carcinoma of the transverse colon

When there is no obstruction, excision of the transverse colon and the two flexures together with the transverse mesocolon and the greater omentum, followed by end-to-end anastomosis, can be used(Fig.15 C). An alternative is an extended right hemicolectomy.

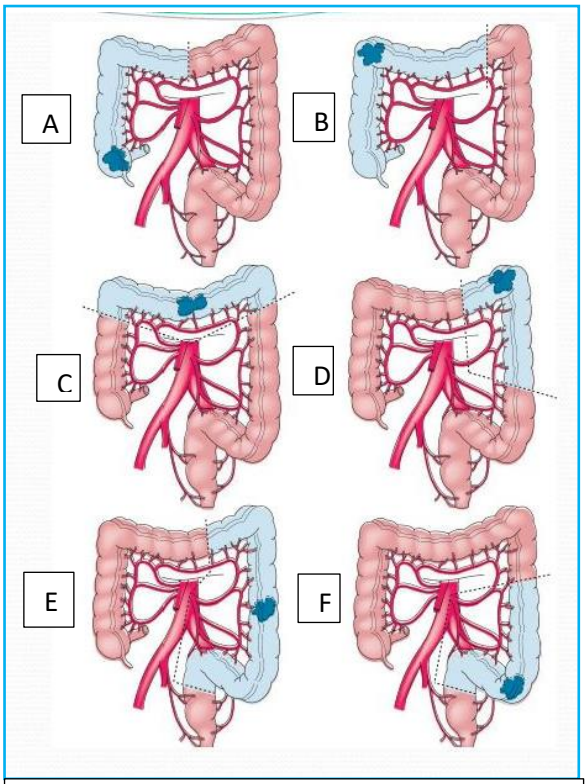

Figure 15: Extent of colonic resection according to site of tumor

Carcinoma of the splenic flexure or descending colon

The extent of the resection is from right colon to descending colon (Fig. 15E, D).
Sometimes, removal of the colon up to the ileum, with an ileorectal anastomosis, is preferable.

Carcinoma of the pelvic colon

The left half of the colon is mobilised completely (Fig. 15F). So that the operation is radical, the inferior mesenteric artery below its left colic branch, together with the

related paracolic lymph nodes, must be included in the resection. This entails carrying the dissection as far as the upper third of the rectum. Many surgeons advocate flush ligation of the inferior mesenteric artery on the aorta (high ligation). Provided that there is no obstruction, primary anastomosis is the rule. Occasionally, a protecting upstream stoma may be necessary.

Laparoscopic surgery

This has been heralded as the next major advance in colorectal surgery. However, its role needs to be accepted with an element of caution. It is technically demanding with a long learning curve.Patients undergoing such surgery need to be entered into clinical trials. Important technical issues are traction and adequate vision, which are vital, as for open conventional procedures. Hand-assisted methods can be used in particularly difficult cases, but this takes away some of the benefits of the minimal approach. Specimen retrieval is via small incisions. Techniques and technology are rapidly evolving.

When a growth is found to be inoperable

In the upper part of the left colon, an ileostomy is performed. In the pelvic colon, a left iliac fossa colostomy is preferable. With an inoperable growth in the ascending colon, a bypass using an ileocolic anastomosis is the best procedure. A total colectomy needs to be considered for multiple tumours. Over 95% of colonic carcinomas can, however, be resected.

Hepatic metastases

It is important not to biopsy hepatic metastases as this may cause tumour dissemination. Hepatic resection is usually performed as a staged procedure after recovery from colonic resection. Most wait 12 weeks before restaging. By doing this, those with aggressive disease are excluded from further drastic surgery. Reports have shown 30% 5-year survival following hepatectomy for colorectal cancer metastases. Radiological imaging will usually correctly identify colorectal metastases and assess patients suitable for liver resection. At present, the criterion for resection is fewer than three lesions in one lobe of liver. Irresectable symptomatic hepatic metastases may be suitable for other treatments including cytotoxic drugs or ablative treatments.