Colonic & Rectal Cancers

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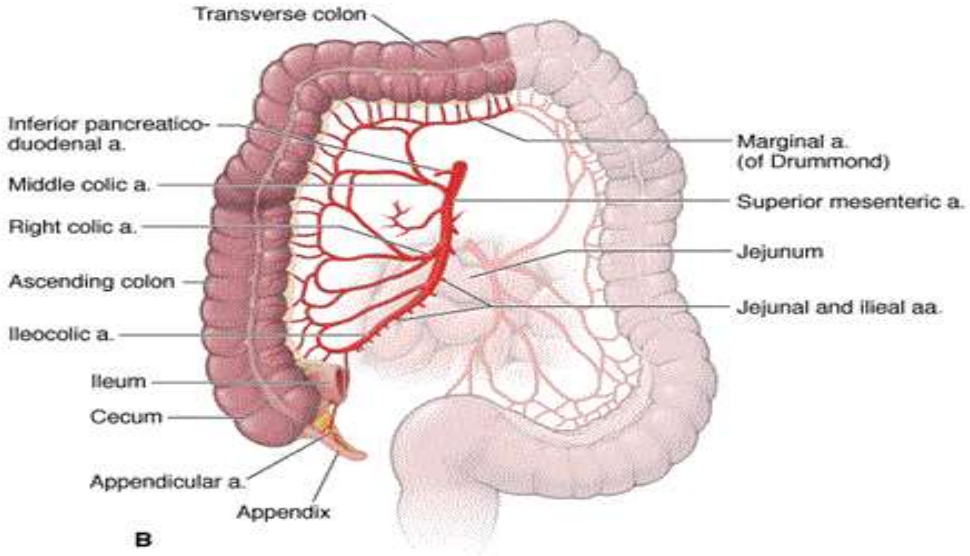
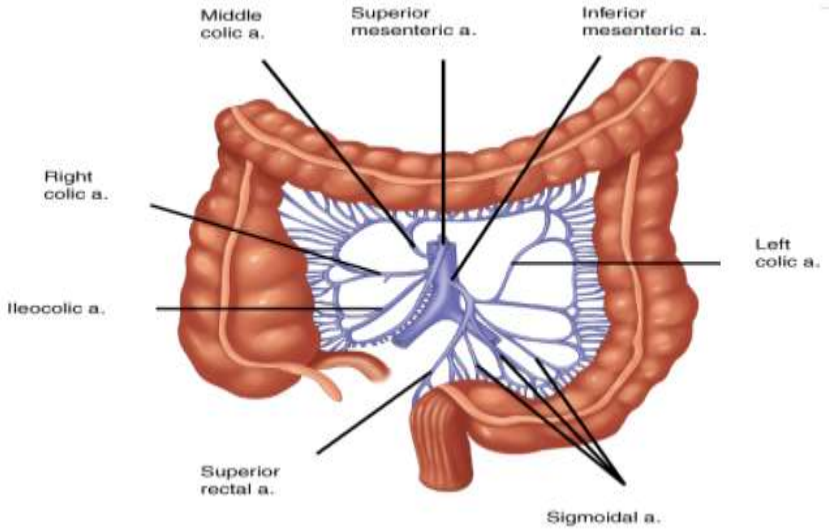
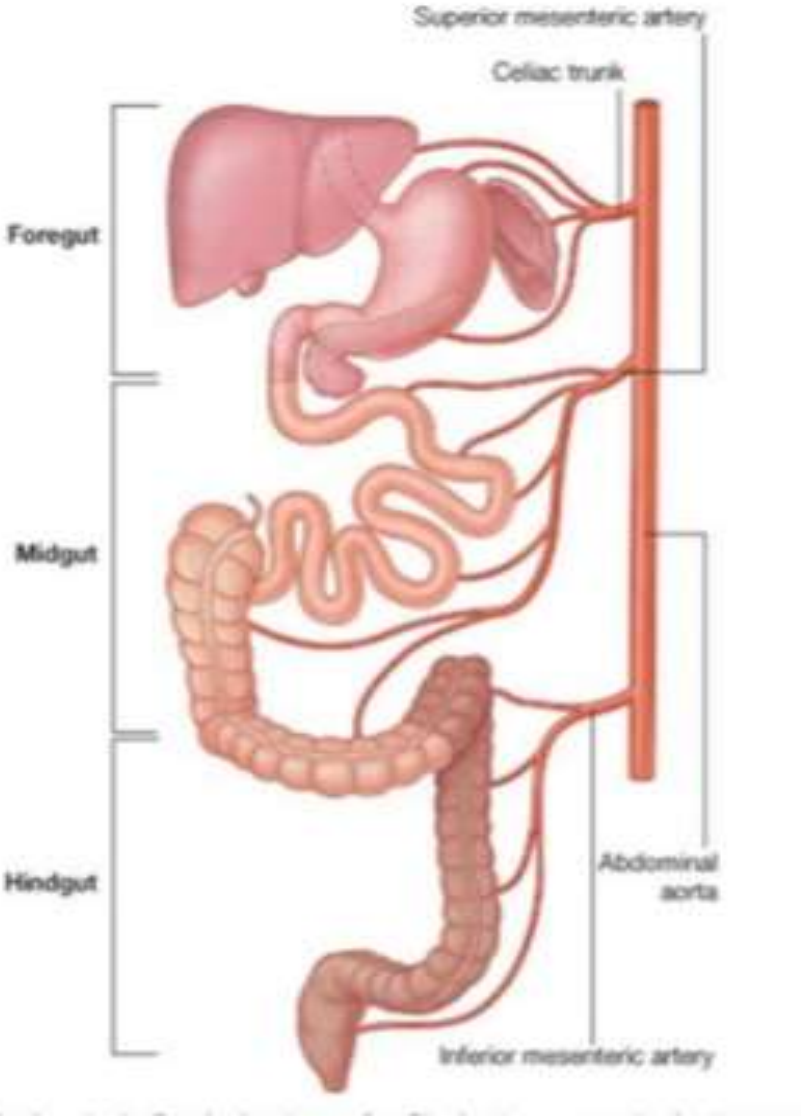
Embryology

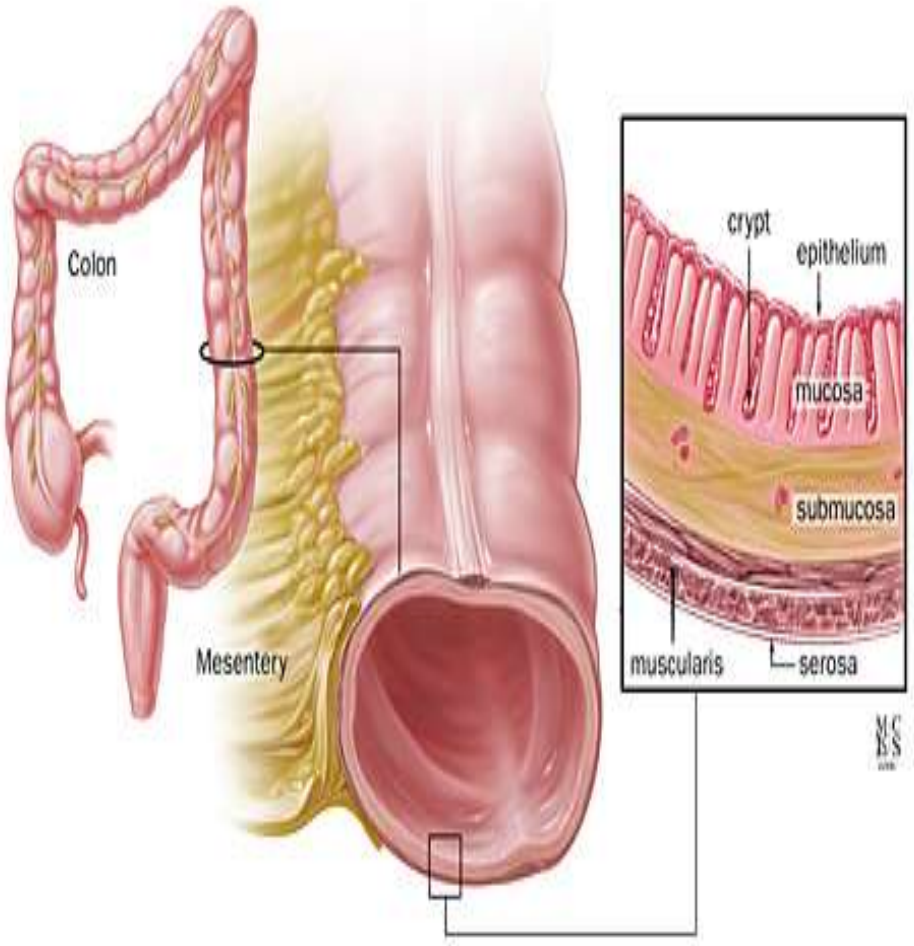

Transverse colon
Inferior pancreatico-duodenal a.
Middle colic a.
Right colic a.
Ascending colon
Ileocolic a.
Ileum
Cecum
Appendicular a.
Appendix
B
Marginal a. (of Drummond)
Superior mesenteric a.
Jejunum
Jejunal and ilieal aa.


Middle colic a.
Superior mesenteric a.
Inferior mesenteric a.
Right colic a.
Ileocolic a.
Left colic a.
Superior rectal a.
Sigmoidal a.


Superior mesenteric artery
Celiac trunk
Foregut
Midgut
Hindgut
Abdominal aorta
Inferior mesenteric artery

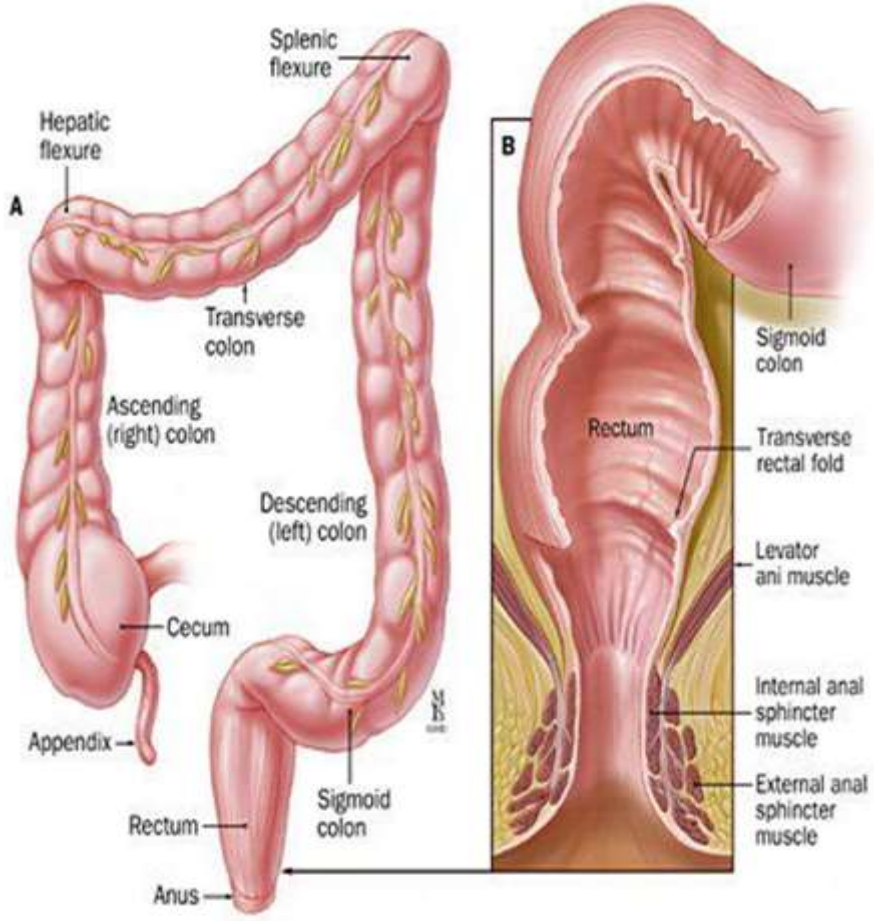
Anatomy

- The wall of the colon and rectum comprise five distinct layers: mucosa, submucosa, inner circular muscle, outer longitudinal muscle, and serosa.
- In the colon, the outer longitudinal muscle is separated into three teniae coli , which converge proximally at the appendix and distally at the rectum, where the outer longitudinal muscle layer is circumferential.

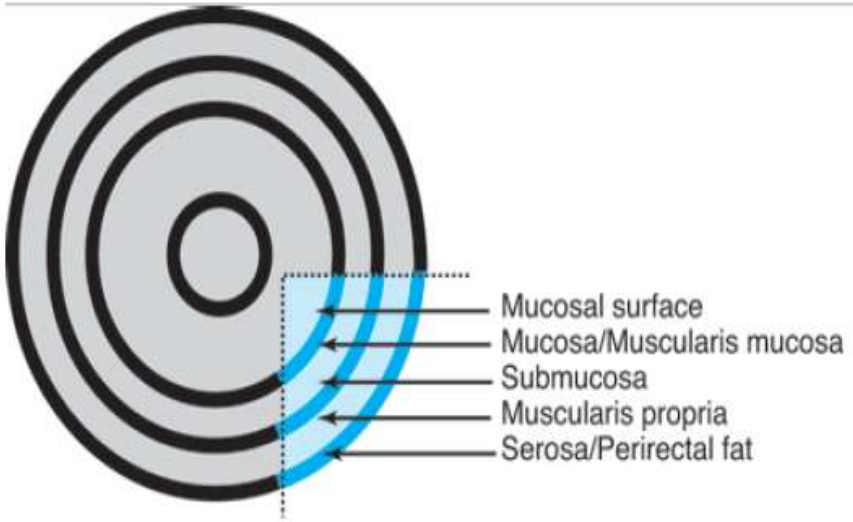


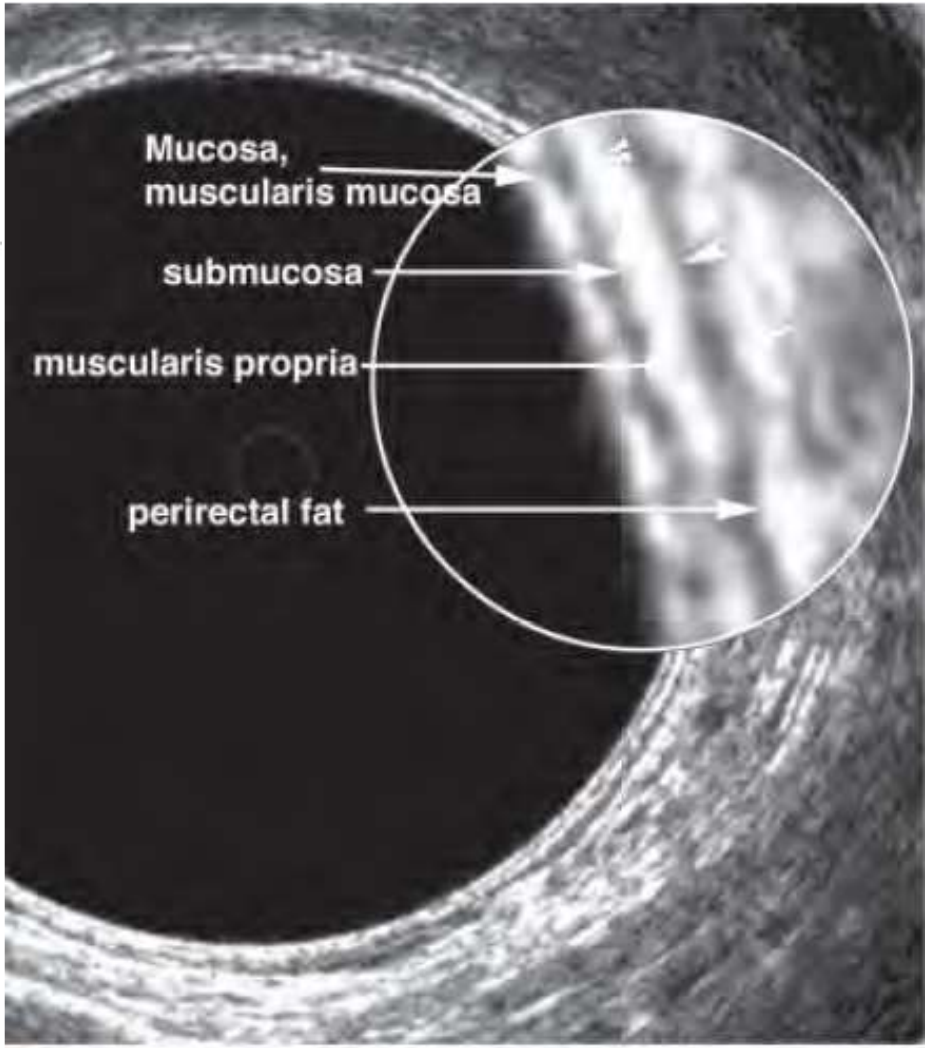
Anatomy

- In the distal rectum, the inner smooth muscle layer coalesces to form the internal anal sphincter.
- The intraperitoneal colon and proximal one third of the rectum are covered by serosa; the mid and lower rectum lack serosa.
- The rectum is approximately 12- 15cm in length. Three distinct submucosal folds, the *valves of Houston* , extend into the rectal lumen.



Anatomy by EUS

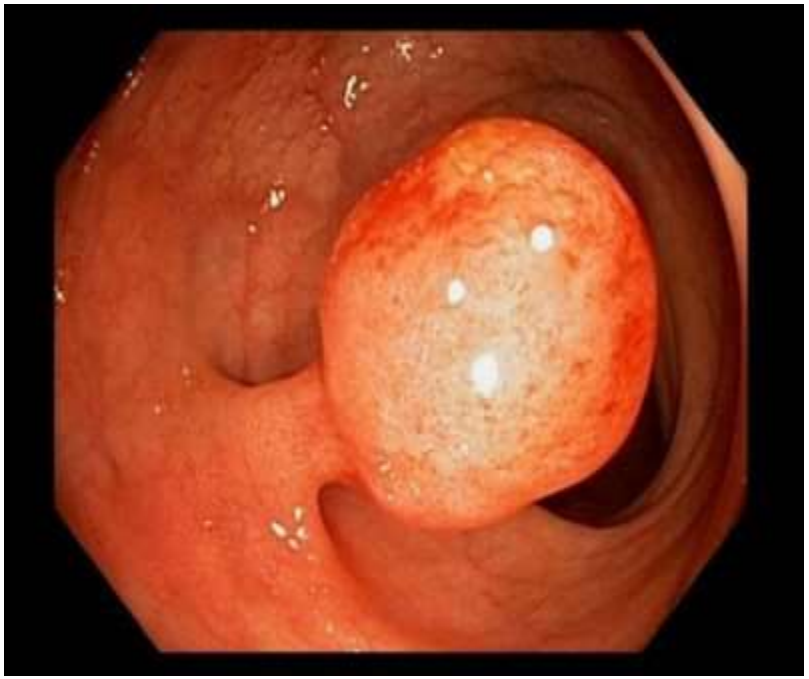




Colorectal Polyp

- None specific clinical term that describes any projection from the surface of the intestinal (colorectal) mucosa into the lumen regardless of the histologic nature

- A variety of polyp types have been described, all with different biologic behaviors



Epidemiology

- Approximately 50% occur in the rectosigmoid region, and 50% are multiple.
- Distinguishing among polyp types is important because some types are clearly associated with carcinoma of the colon.
- Approximately 5% of all barium enema and 5% to 10% of colonoscopic studies show polyps.

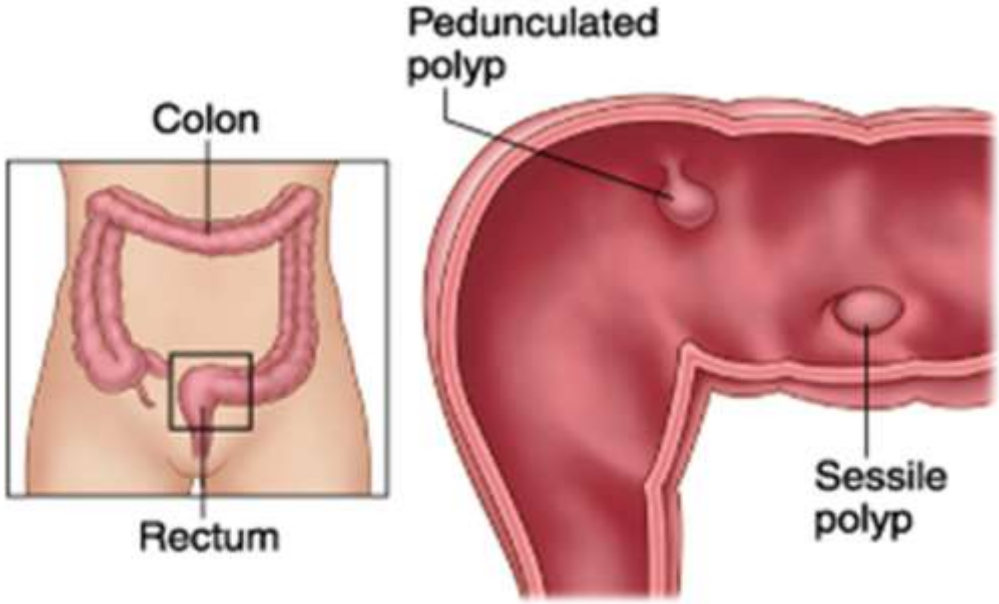
Classification of Colorectal Polyps

- Neoplasic (Adenomatous)
- Hamartomatous (Juvenile, Peutz-Jeghers, Cronkite-Canada)
- Inflammatory (Pseudopolyp)
- Hyperplastic (Hypertrophic)

Type		Frequency	Location	Malignant Potential	Treatment
Adenoma (Neoplastic)	Tubular	Common: 10% of adults	Rectosigmoid in 20%	7% malignant	Endoscopic excision
	Villous	Fairly common, especially in the elderly	Rectosigmoid in 80%	33% malignant	Endoscopic excision / Surgical removal
Hamartoma		Uncommon. eg: Peutz–Jeghers polyp. Juvenile polyp	Small bowel mainly	Low; uncommon	Excise for bleeding or obstruction
Inflammatory		Uncommon, except in IBD - common	Colon and rectum	None, except in IBD esp. UC (low)	Observation (often spontaneously regress or autoamputate) / Endoscopic excision
Hyperplastic		Fairly common	Stomach, colon, and rectum	None	Observation / Endoscopic excision

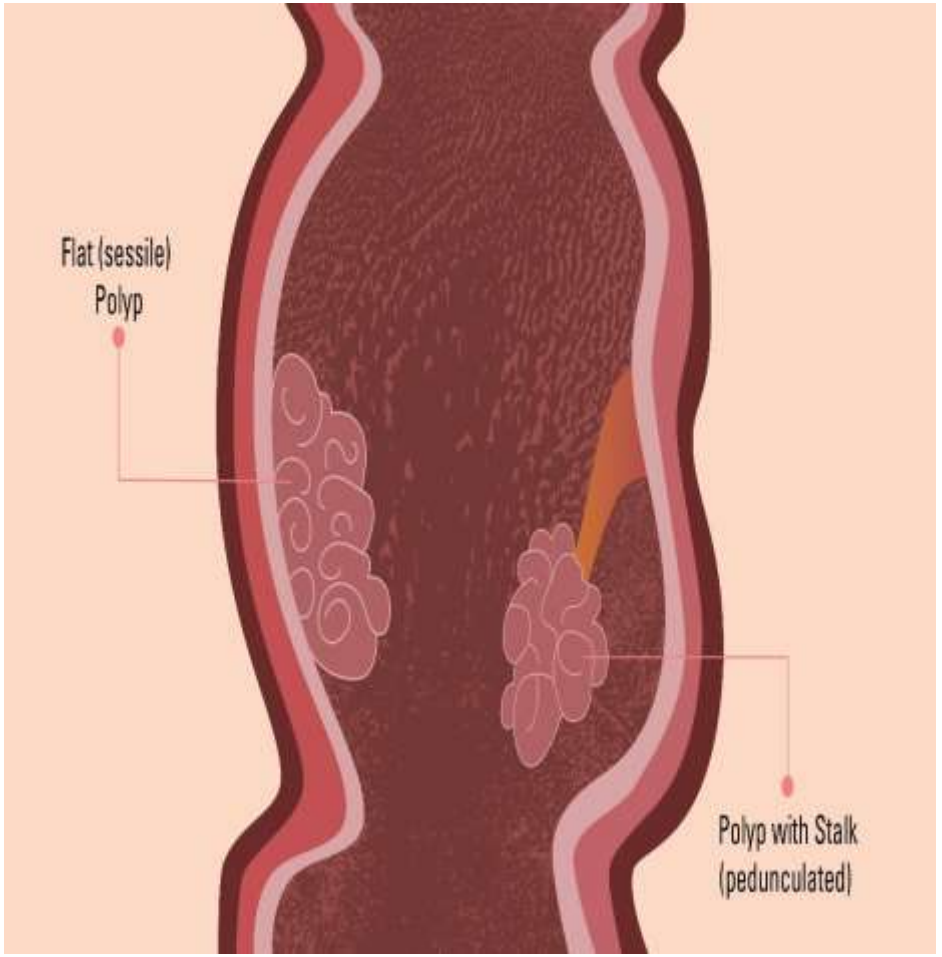
Neoplasic (Adenomatous)

- Premalignant
- Divided into
  - Tubular
  - Tubulovillous
  - Villous
- Morphology
  - Sessile (flat & intimately attached to mucosa)
  - Pedunculated (rounded and attached to mucosa by a long thin neck



Adenoma

- Tubular and tubulovillous adenomas are more commonly pedunculated
- Villous adenomas are more commonly sessile.



Polyps & Malignant potential

- Approximately 7% of tubular, 20% of tubulovillous, and 33% of villous adenomas become malignant.

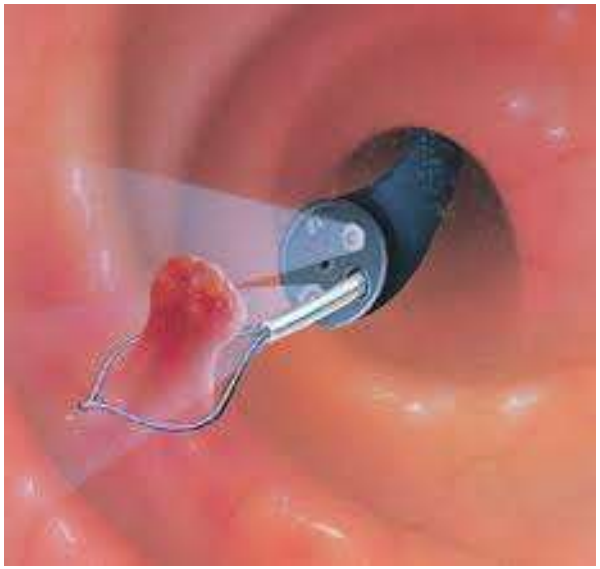
- Villous adenomas >3 cm in diameter have a greater probability of malignancy.

Polyps Clinical Presentation

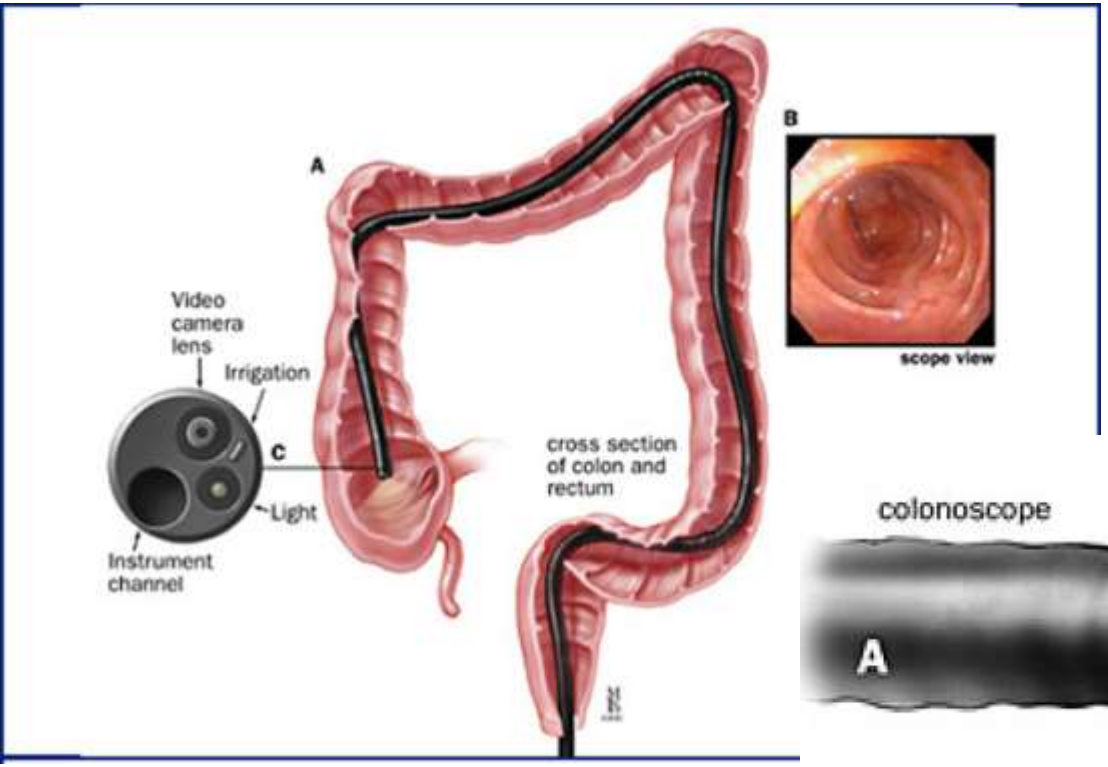
- Usually asymptomatic
- Occasionally bleed
- Most commonly detected during routine endoscopic surveillance
- Patients with family history of polyps usually they seek endoscopic screening

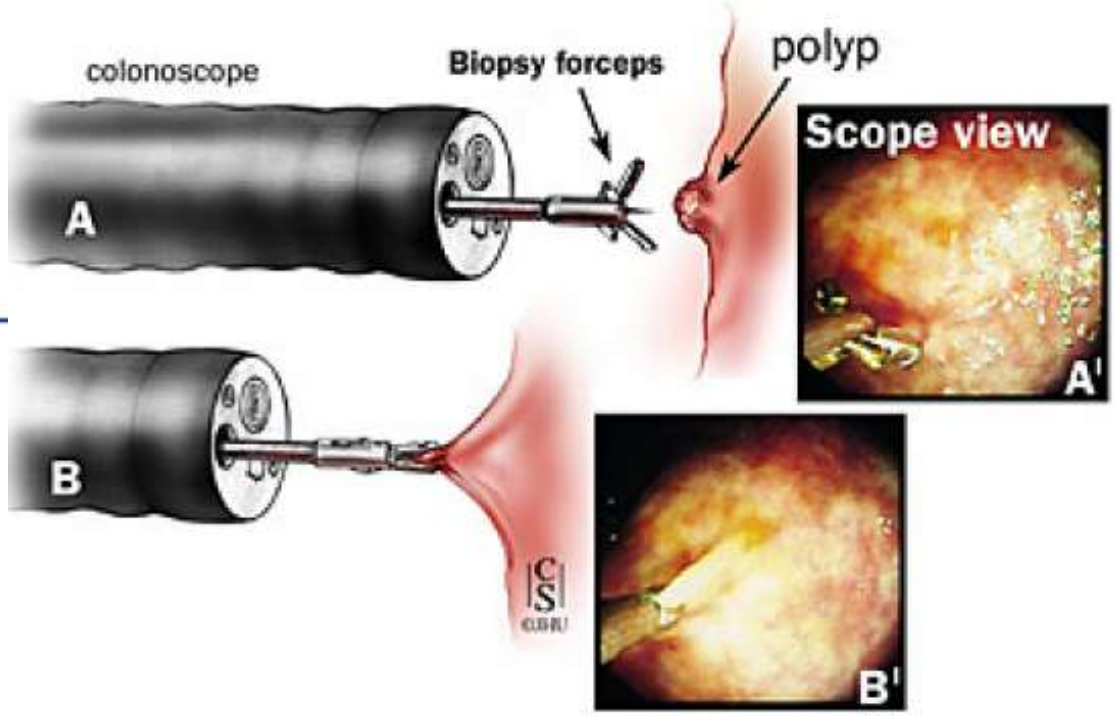
Polyps Treatment

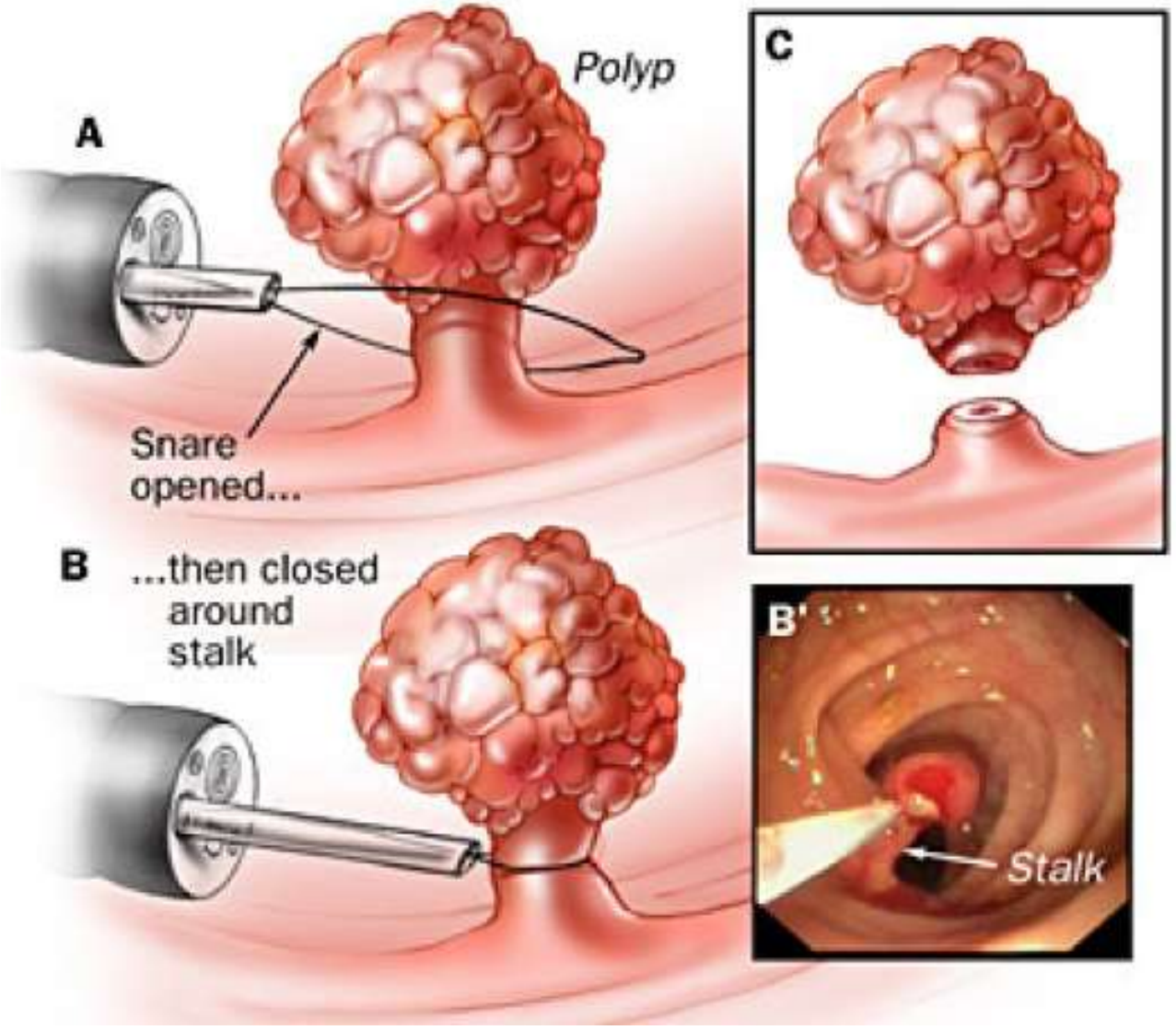
- Treatment of adenomatous polyps involves colonoscopic polypectomy.
- If some cannot be safely removed colonoscopically, biopsy should be performed and a segmental resection of the colon done if the lesion is:
  - **Villous adenoma** or
  - **Large**, **ulcerated**, **dysplastic**, or
  - **Indurated**.

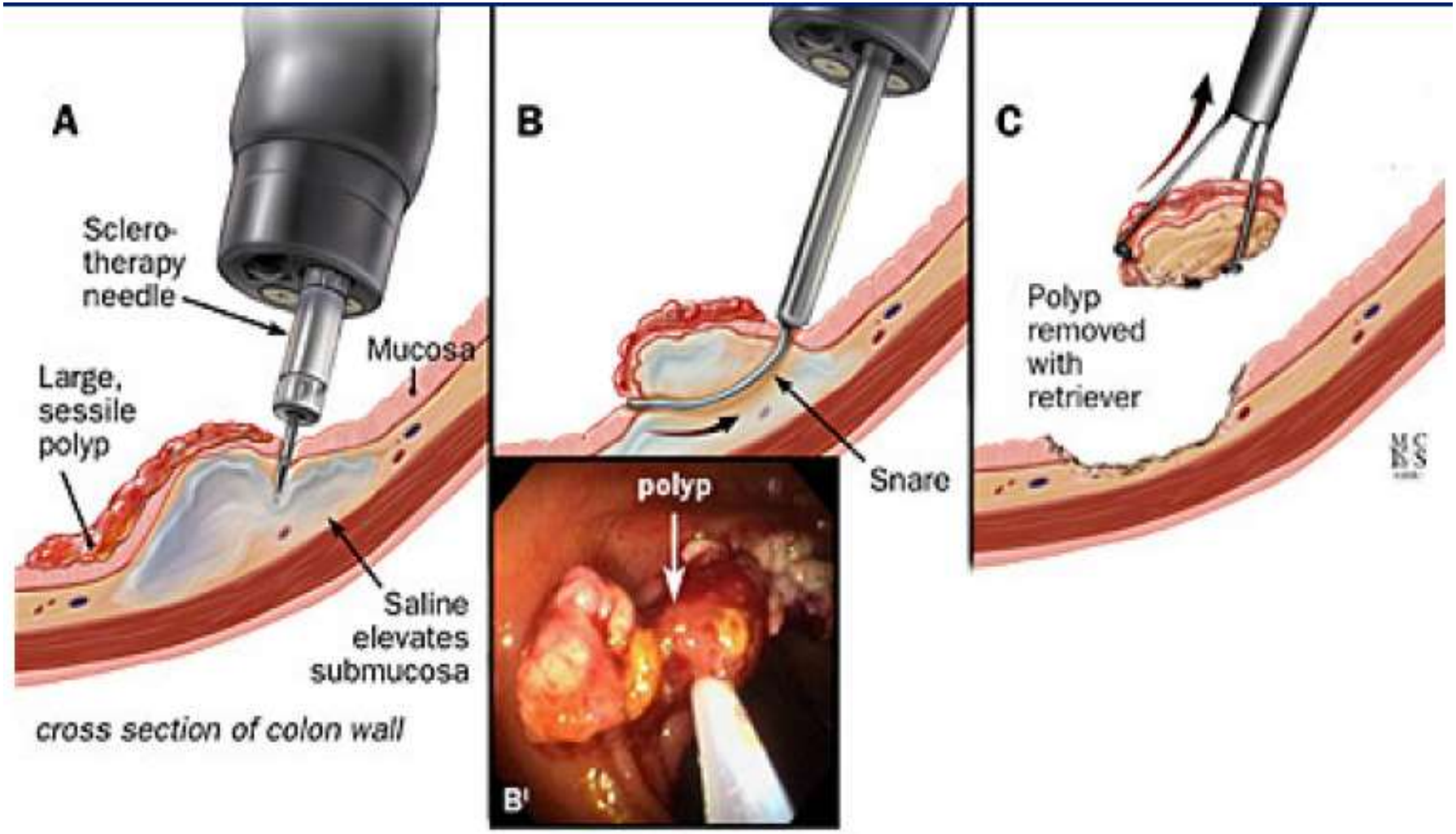


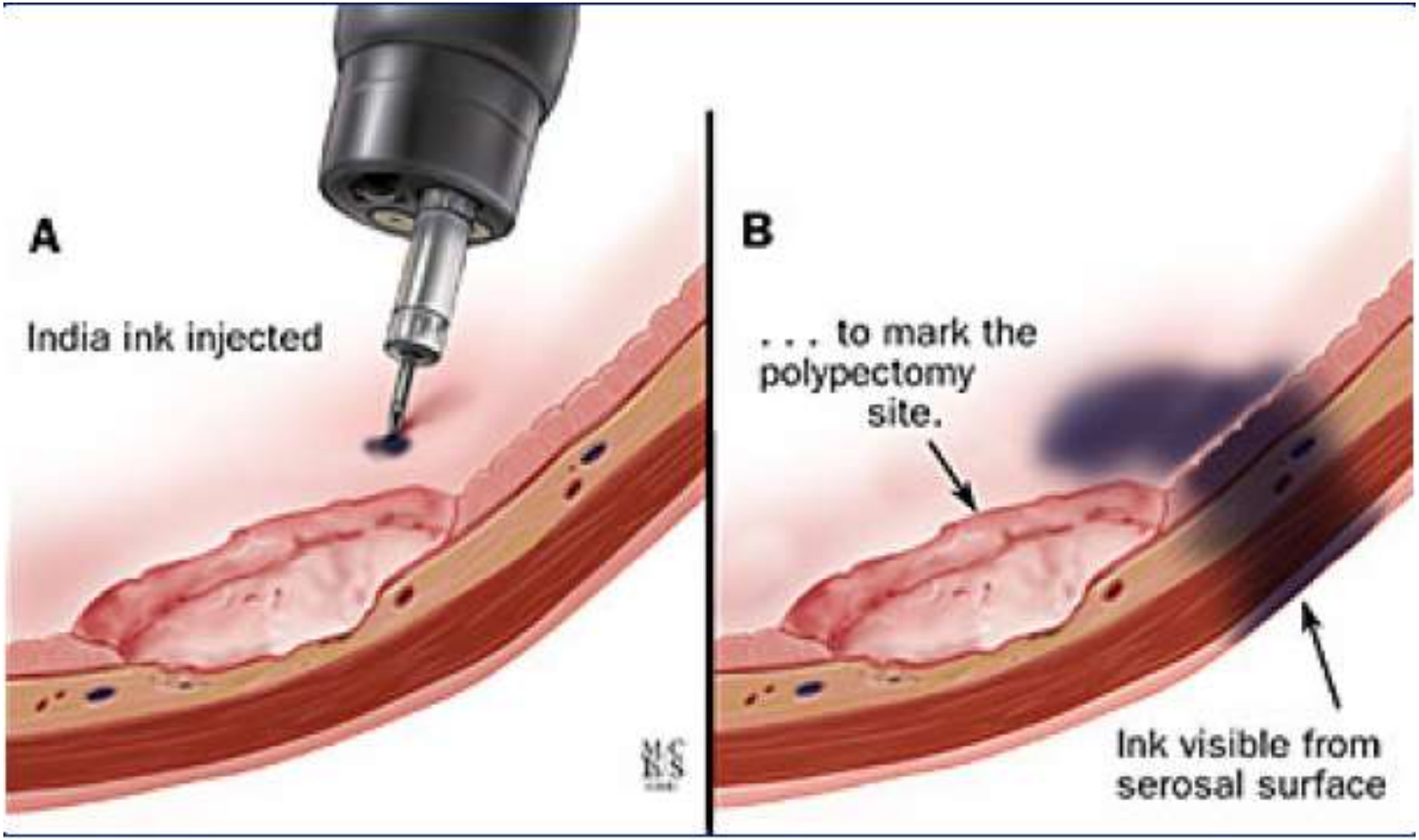
Treatment


A
B
scope view
Video camera lens
Irrigation
C
Light
Instrument channel
cross section of colon and rectum


colonoscope
Biopsy forceps
polyp
A
Scope view
A'
B
B'

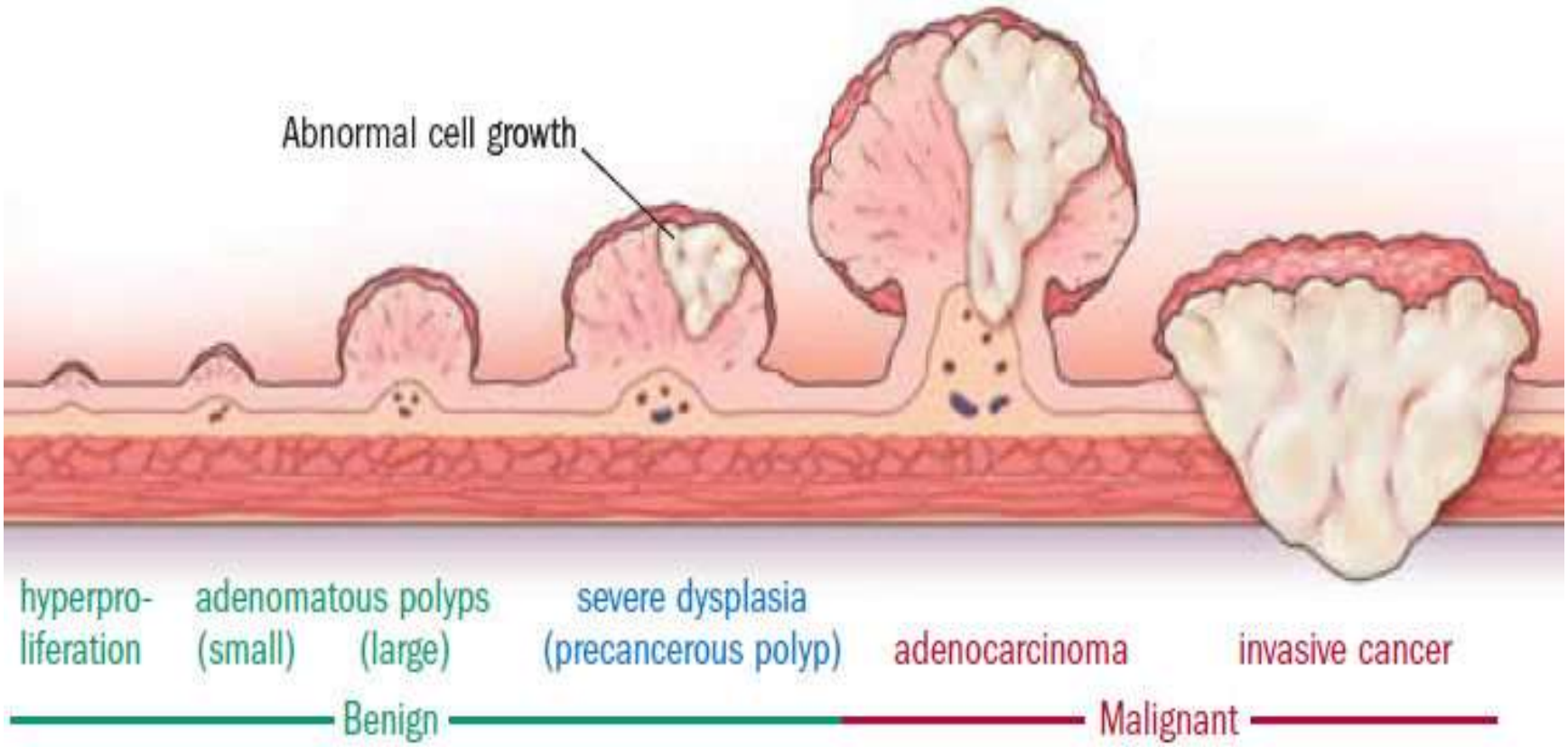

Polyp
A
Snare
opened...
B
...then closed
around
stalk
C
B'
Stalk


A
Sclero-
therapy
needle
Mucosa
Large,
sessile
polyp
Saline
elevates
submucosa
cross section of colon wall
B
Snare
polyp
B'
C
Polyp
removed
with
retriever


A
India ink injected
B
. . . to mark the polypectomy site.
Ink visible from serosal surface

Questions ?

Adenoma–Carcinoma Sequence

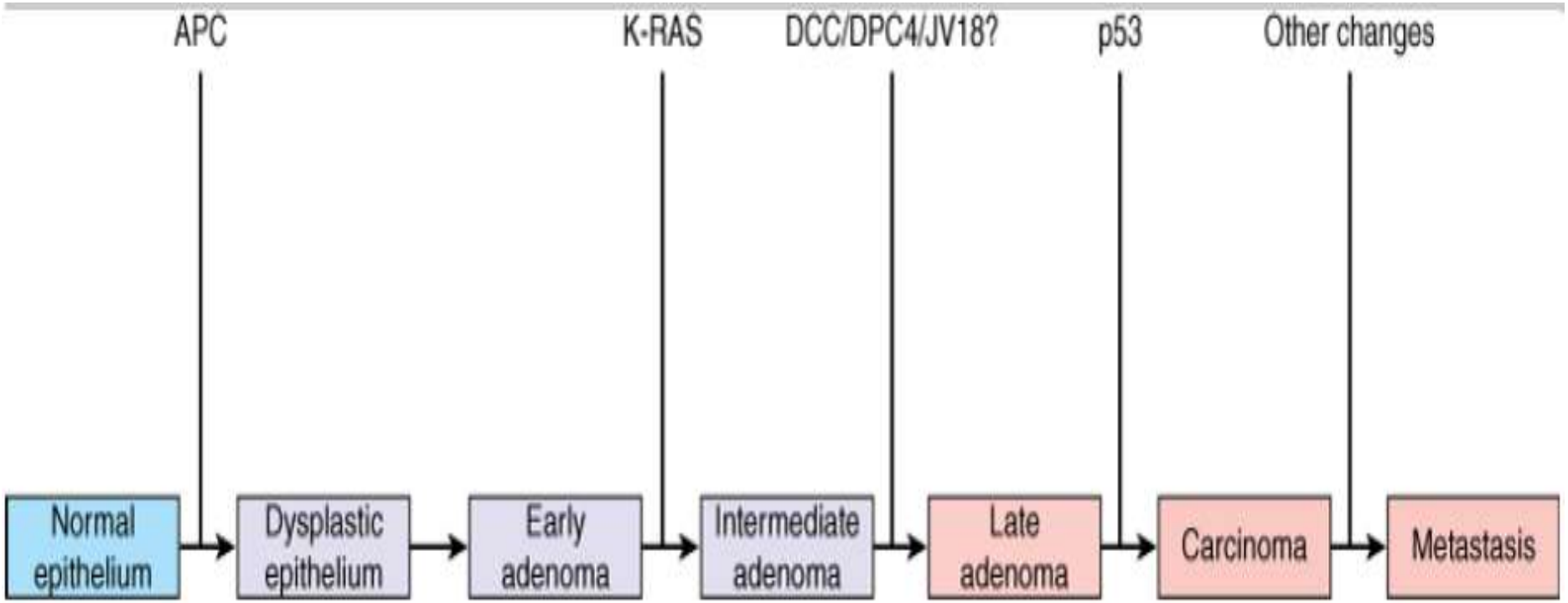


Adenoma–Carcinoma Sequence

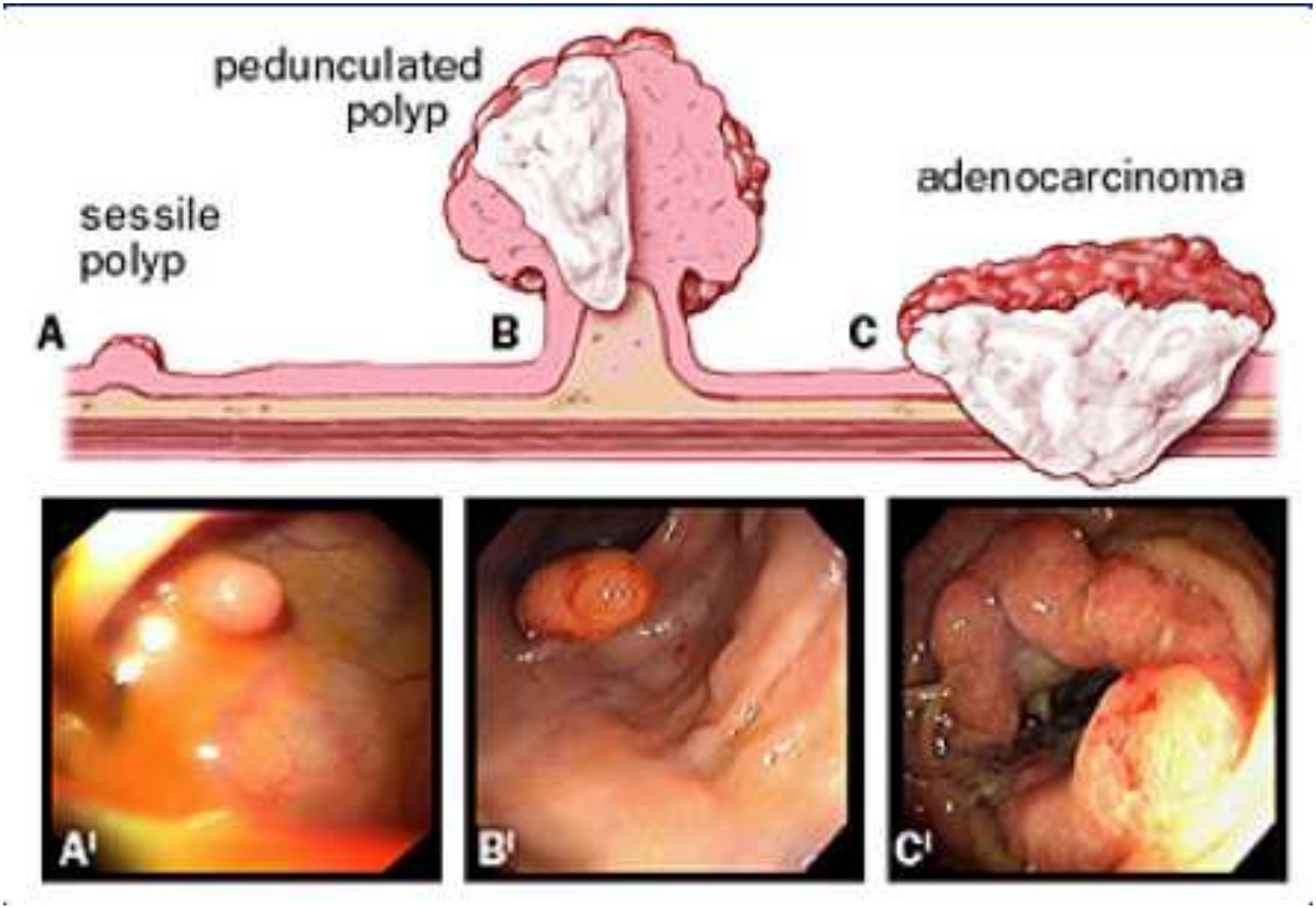
- Evidence for adenoma–carcinoma sequence
  - The prevalence of adenomas and carcinomas is very similar – carcinoma patients are about five years older.
  - The distribution of adenomas in the colon is the same as that of cancers (70 per cent left sided)
  - When small cancers are studied, they almost always have adjacent adenomatous tissue
  - Adenomas are found in a third of specimens resected for colorectal cancer
  - Sporadic adenomas are identical to the adenomas of familial adenomatous polyposis, which is associated with a 100 per cent chance of colorectal adenocarcinoma unless treated
  - Larger adenomas are more likely to be dysplastic and to have higher grades of dysplasia than small adenomas.
  - Incidence of colorectal cancer falls within a screening programme that involves colonoscopy and polypectomy

Polyp – Cancer sequence

- Colorectal carcinoma is thought to develop from adenomatous polyps due to accumulation of these mutations

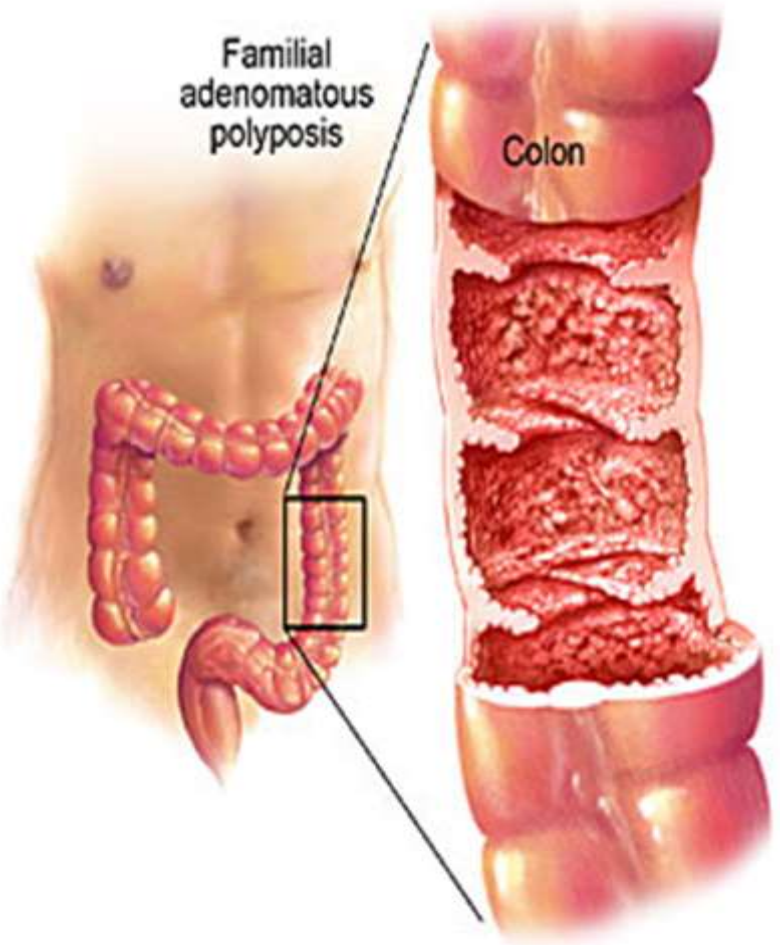


Polyp – Cancer sequence



Familial Adenomatous Polyposis FAP

- Autosomal dominant inherited disease due to mutation of **Adenomatous Polyposis Coli (APC) gene** on the short arm of chromosome 5.
- >80% positive family history.
- >100 colonic adenomas are diagnostic.
- The risk of colorectal cancer is 100%.
- Prophylactic surgery is indicated to prevent colorectal cancer.
- Accounts for =/<1% of all colon cancer.
- Characterised by extraintestinal manifestations.
- Polyps and malignant tumours can develop in the duodenum & small bowe
- FAP is less common than hereditary non-polyposis colon cancer (HNPCC).



Extra-colonic manifestations of FAP

■ **Endodermal derivatives:**
Adenomas and carcinomas of the duodenum, stomach, small intestine, thyroid and biliary tree.
Fundic gland polyps
Hepatoblastoma

■ Ectodermal derivatives:
Epidermoid cysts (Gardner's syndrome)
Pilomatrixoma Congenital hypertrophy of the retinal pigment epithelium (CHRPE)
Brain tumours

■ Mesodermal derivatives:
Desmoid tumours
Osteomas
Dental problems

Screening policy for FAP

- At-risk family members are offered genetic testing in their early teens.
- At-risk members of the family should be examined (endoscopies) at the age of 10–12 years, repeated every year.
- If there are no polyps at 20 years, continue with five-yearly examination until age 50 years.

Treatment of FAP

- The aim of surgery is to prevent the development of colorectal cancer. The surgical options are:
  - Colectomy with ileorectal anastomosis (IRA);
  - Restorative proctocolectomy (RPC) with an ileal pouch-anal anastomosis, the anastomosis may be defunctioned with a loop ileostomy;
  - Total proctectomy and end ileostomy (normally reserved for patients with a low rectal cancer).

Postoperative surveillance for FAP

- Because of the risk of further tumour formation, follow up is important and takes the form of rectal/pouch surveillance, with biopsy of the pouch-anal anastomosis.
- Gastroscopies are also carried out to detect upper gastrointestinal tumours.
- Even with prevention of colorectal cancer, FAP patients have reduced life span due to the development of duodenal and ampullary cancers and the complications of desmoid tumours.

Hereditary non-polyposis colorectal cancer (Lynch syndrome)

- Characterised 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%, and the mean age of diagnosis is 45 years.
- Most cancers develop in the proximal colon.
- Females with HNPCC have a 30–50 % lifetime risk of developing endometrial cancer

Diagnosis (HNPCC)

- By genetic testing or the Amsterdam II criteria:
  - three or more family members with an HNPCC-related cancer (colorectal, endometrial, small bowel, ureter, renal pelvis), one of whom is a first-degree relative of the other two;
  - two successive affected generations;
  - at least one colorectal cancer diagnosed before the age of 50 years;
  - FAP excluded;
  - tumours verified by pathological examination.

  Patients with HNPCC are subjected to regular (every one to two years) colonoscopic surveillance.

Questions ?

Carcinoma of Colon & Rectum

- Cancer of the colon and rectum is a major cause of death worldwide.
- Its etiology centers on the impact of intraluminal chemical carcinogenesis.
- These carcinogens are **ingested** or are the result of **biochemical processes** that occur intraluminally from existing substances that are found normally in the fecal stream.

Carcinoma of Colon & Rectum

- Approximately 80% 0f colorectal cancers occur sporadically, while 20% arise in patients with a known family history of colorectal cancer.
- Genetic defects and molecular abnormalities associated with the development and progression of colorectal adenomas and carcinoma
- Mutation causing

  (1)activation of oncogenes (K-ras) and/or

  (2)inactivation of tumor suppressor genes (APC, DCC, p53)

Carcinoma of Colon & Rectum

- **Synchronous** (simultaneously occurring or within 6months) tumors develop in 5% of patients,

- 3% to 5% of patients have **Metachronous** tumors (a second tumor developing after resection of the first).

Risk Factors

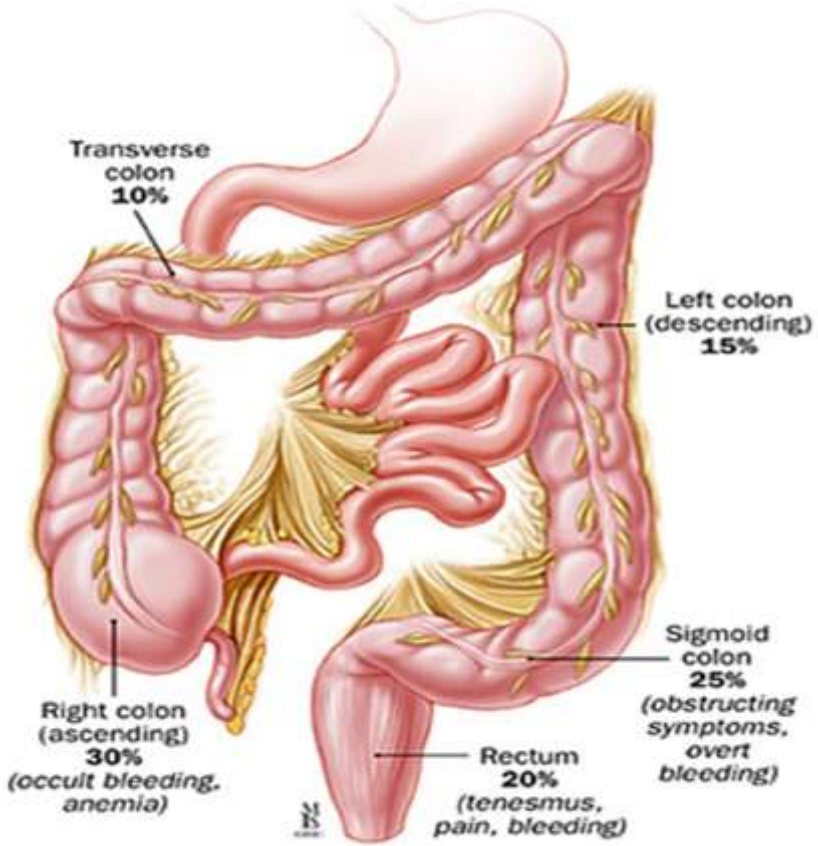
- Other predisposing diseases include Ulcerative Colitis, Crohn's colitis, lymphogranuloma venereum, and certain polyps (described previously).
- The peak incidence of colon cancers occurs at approximately 70 years of age, but the incidence begins to increase in the fourth decade of life.

Protective Factors

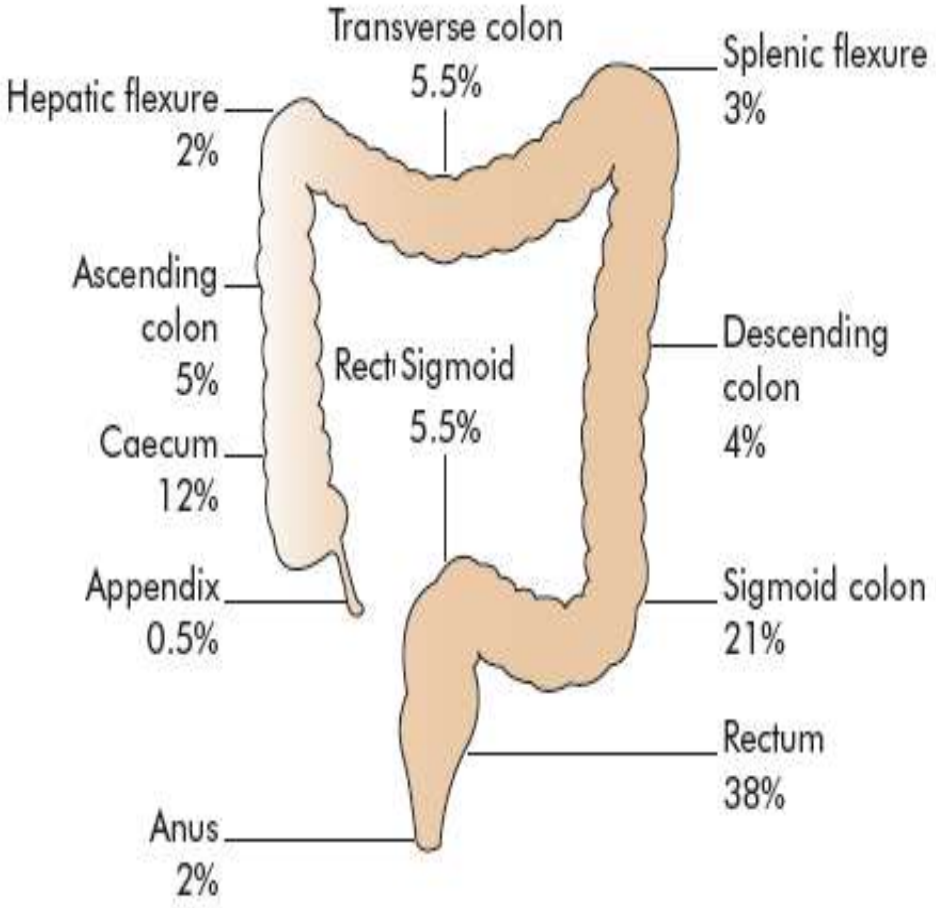
- Certain health agencies promote a **low-fat, high-fiber diet** as protective against cancer of the colon and rectum.
- Chemoprevention by ingestion of such agents as carotenoids and other antioxidants has been suggested, but the efficacy of this measure is unproven.
- There is good evidence that prostaglandin inhibitors such as **aspirin** and sulindac significantly lower the risk of polyp formation and colon cancer when taken on a regular basis.

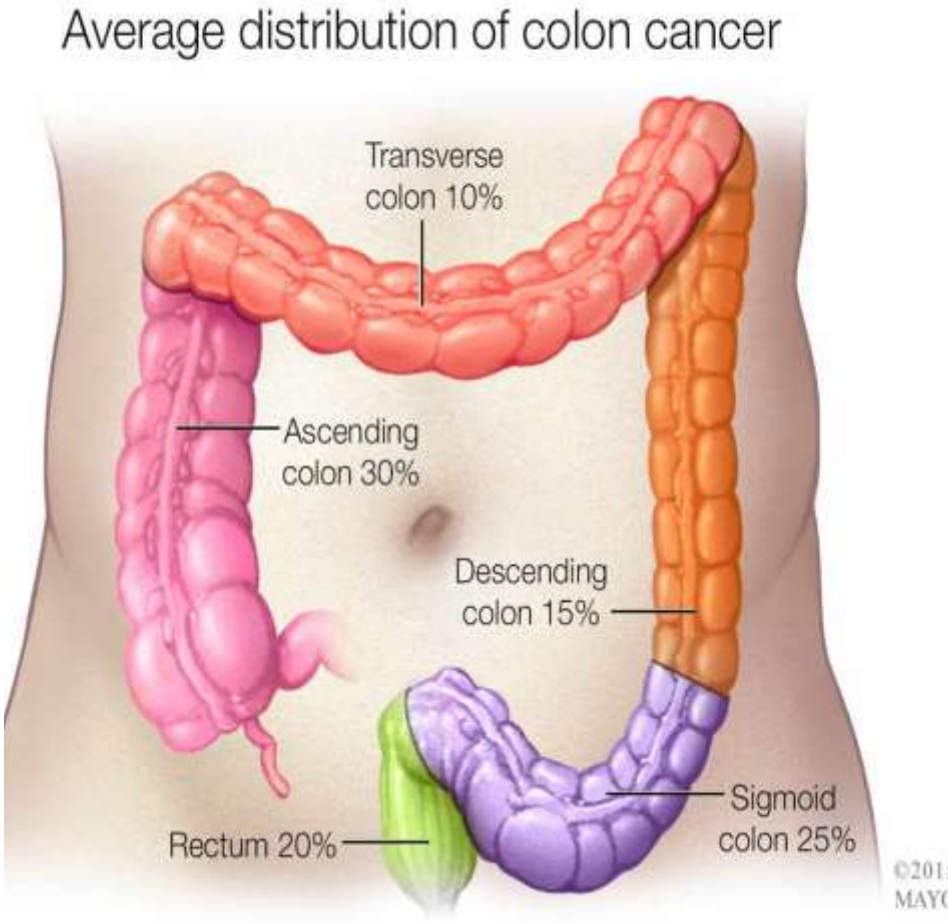
Clinical Presentation

- The clinical signs and symptoms of colorectal cancer are determined largely by the anatomic location.
- Cancers of the **right** colon are usually exophytic lesions associated with **occult blood loss**, resulting in **iron deficiency anemia**
- At advanced stages of the disease, patients may have a palpable **right** lower abdominal **mass**.



Clinical Presentation





Clinical Presentation

- Cancers that arise primarily in the **left** and sigmoid colon are more frequently annular and invasive, resulting in **obstruction** and macroscopic rectal **bleeding**.
- Cancers of the **rectum** also cause a symptom complex of rectal **bleeding**, obstruction, and, occasionally, **alternating** diarrhea and constipation.
- **Tenesmus** occurs with far advanced disease.

Clinical Presentation

- Any patient older than 30 with a change in bowel habits, iron deficiency anemia, or rectal bleeding should undergo a complete examination of the colon and rectum by **colonoscopy.**

  (symptoms → colonoscopy)
- If rectal bleeding occurs, workup for a possible malignancy should be initiated, even if the apparent source is a benign lesion (e.g., hemorrhoid) unless the patient is younger and rapidly responds to treatment.

Investigations

- Total Colonoscopy lies in its ability to detect the 3% to 5% of patients with synchronous colon cancers, allowing better planning of surgical therapy.
- CT of the Chest, Abdomen and pelvis is performed for staging and before surgery.
- Preoperative blood tests should evaluate the patient's overall nutritional status and should include liver function tests and carcinoembryonic antigen (CEA) study as a baseline for follow up post op.
- Endorectal ultrasound or MRI is used to stage the depth of penetration of the tumor in the **rectal** wall.

Tumor Markers

Carcinoembryonic antigen (CEA) may be elevated in 60 to 90% of patients with colorectal cancer.

Despite this, CEA is **not** an effective screening tool for this cancer.

Serial CEA levels are done after curative intent surgery to detect early **recurrence** of colorectal cancer.

However, this tumor marker is nonspecific, and no survival benefit has yet been proven.

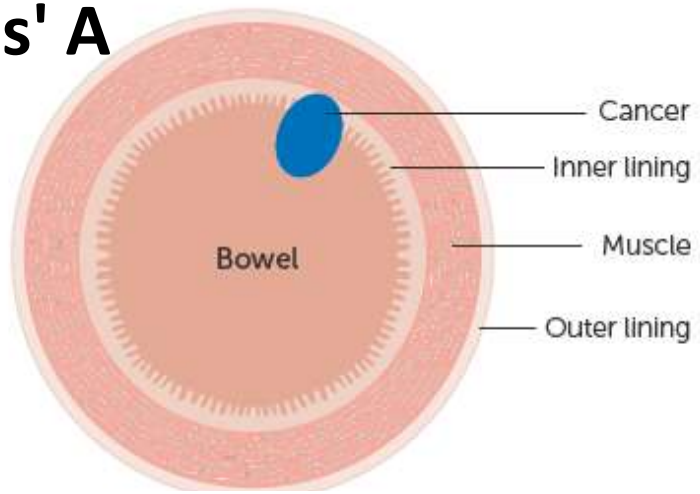
Other biochemical markers (ornithine decarboxylase, urokinase) have been proposed, but none has yet proven sensitive or specific for detection, staging, or predicting prognosis of colorectal carcinoma.

Spread of colorectal cancer

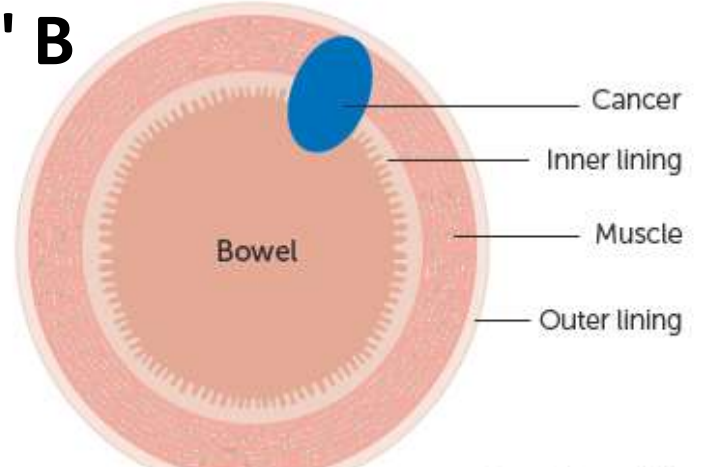
- Direct spread (locally)
- Via lymphatics
- Via bloodstream (liver ‘most common’ - lung)
- Transcoelomically around the peritoneum (including Blumer’s shelf on rectal examination).

Dukes' staging system

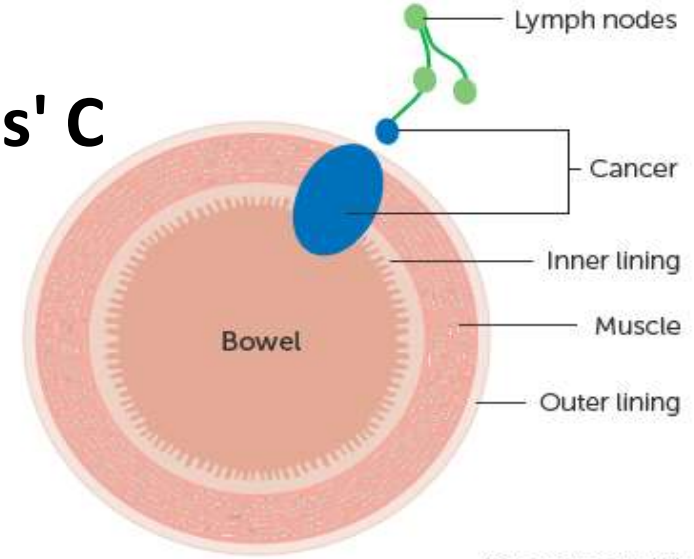
Dukes' A



Dukes' B

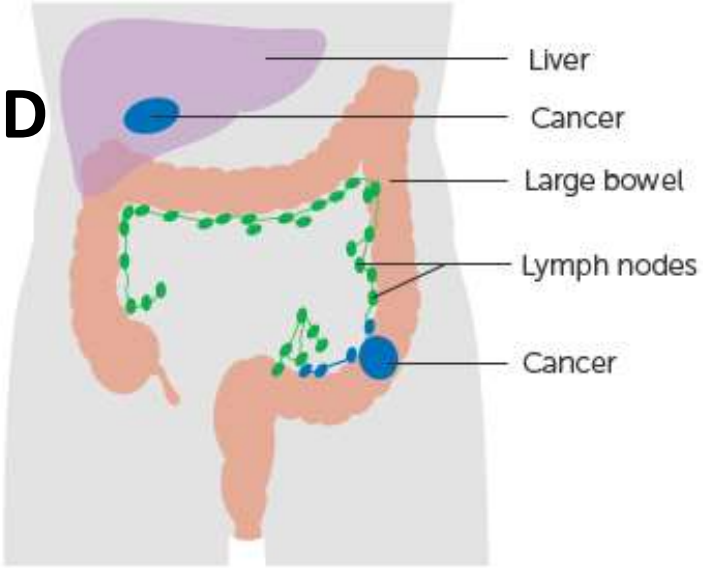


Dukes' C



Cancer Research UK

Dukes' D



Cancer Research UK

Dukes' staging (Astler-Coller modification)

Carcinoma in situ (may be referred to as high grade dysplasia) – Intramucosal carcinoma that does not penetrate the muscularis mucosae.

Stage A – tumors invade through the muscularis mucosae into the submucosa but do not reach the muscularis propria.

Stage B1 – tumors invade into the muscularis propria.

Stage B2 – tumors completely penetrate the smooth muscle layer into the serosa.

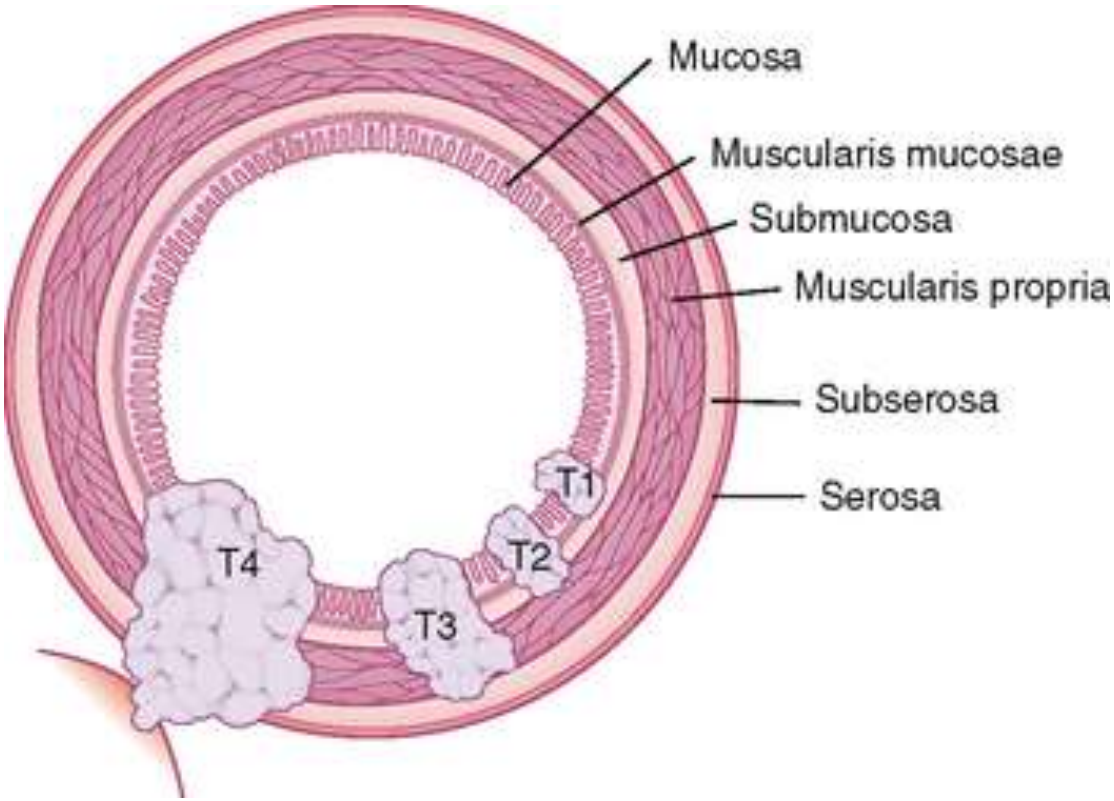
Stage C – tumors encompass any degree of invasion but are defined by regional lymph node involvement.

Stage C1 – tumors invade the muscularis propria with less than 4 positive nodes.

Stage C2 – tumors completely penetrate the smooth muscle layer into the serosa with 4 or more involved nodes.

Stage D – lesions with distant metastases.

TNM Classification



TNM Classification

TX – primary tumor cannot be assessed.

T0 – no evidence of primary tumor.

Tis – carcinoma in situ: intraepithelial or invasion of lamina propria.

T1 – tumor invades submucosa.

T2 – tumor invades muscularis propria.

T3 – tumor invades through muscularis propria into subserosa or into nonperitonealized pericolic or perirectal tissues.

T4 – tumor directly invades other organs or structures and/or perforates visceral peritoneum.

NX – regional lymph nodes cannot be assessed.

N0 – no regional lymph node metastasis.

N1 – metastasis in 1–3 regional lymph nodes.

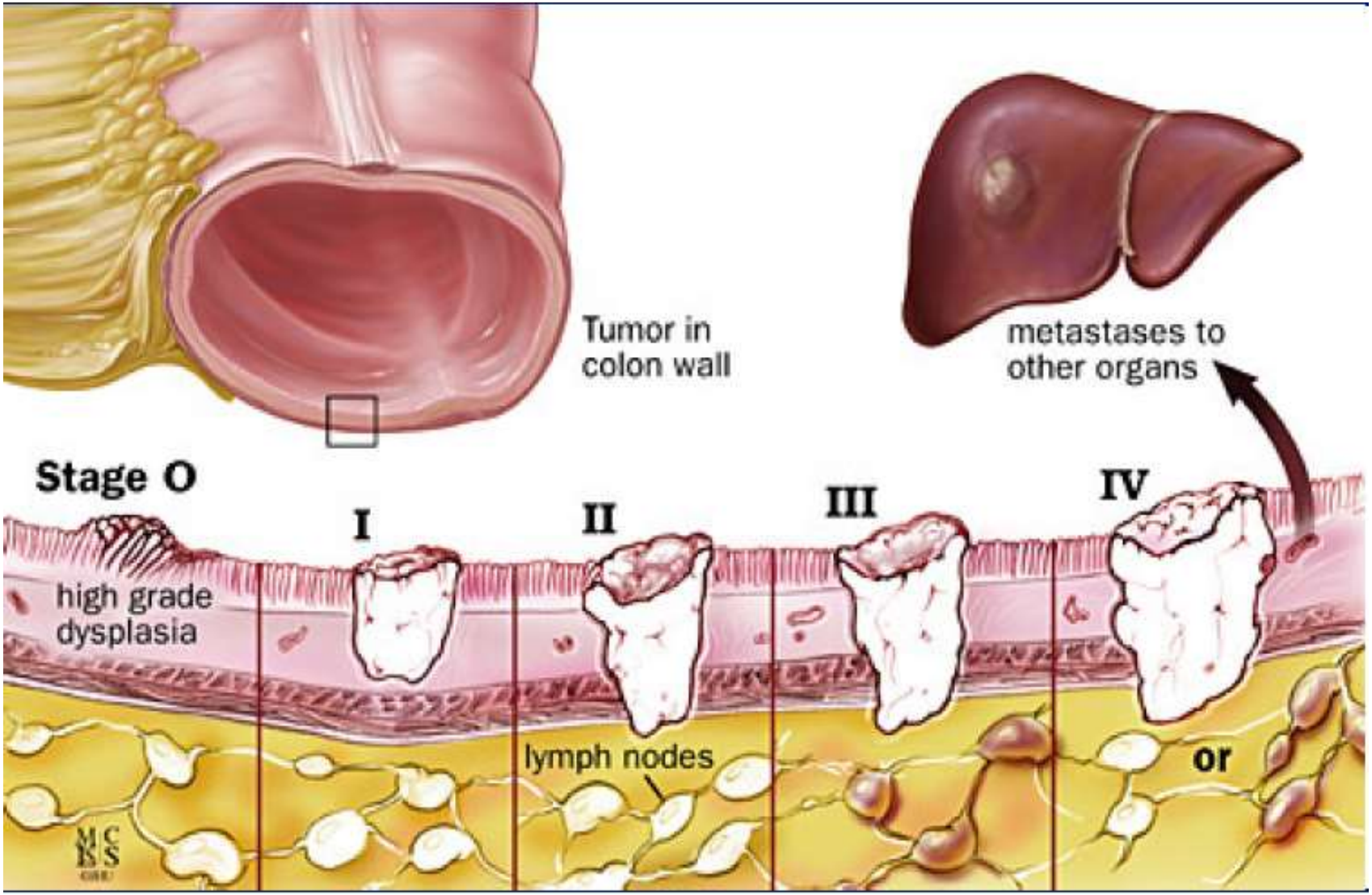
N2 – metastasis in 4 or more regional lymph nodes.

MX – distant metastasis cannot be assessed.

M0 – no distant metastasis.

M1 – distant metastasis.

TNM staging


Tumor in
colon wall
metastases to
other organs
Stage 0
I
II
III
IV
high grade
dysplasia
lymph nodes
or

TNM Classification (American Joint Commission on Cancer)				Dukes' Classification
Stages	T	N	M	Stages
Stage 0	Tis	N0	M0	
Stage I	T1	N0	M0	A
	T2	N0	M0	B1
Stage II	T3	N0	M0	B2
	T4	N0	M0	B2
Stage III	T1, T2	N1 or N2	M0	C1
	T3, T4	N1 or N2	M0	C2
Stage IV	Any T	Any N	M1	D

Prognosis

- Of all GI cancers, more progress has been made in improving cure rates in colorectal cancer than any other.
- **The most important prognostic variable is lymph node involvement.**
- Other prognostic factors (e.g., tumor markers, size of the lesion and depth of invasion)

Prognosis

TNM stage		Dukes's stage	Description	5-year survival
0	TIS, N0, M0		Growth limited to the mucosa	> 95%
I	T1-2, N0, M0	A	Growth limited the wall of the intestine	85-100%
II	T3-T4, N0, M0	B	Penetration through muscularis propria.	50-75%
III	T1-4+any T, N1-2, M0	C	Regional lymph node metastasis	30-50%
IV	Any T, any N, M1	D	Distant metastasis to other organs	< 5%

Questions ?

Screening

- ‘Screening’ is for asymptomatic people.
- If symptoms present → No screening, but ‘Colonoscopy’

Screening

- This improvement in prognosis is the direct result of two factors:

  (1) an effective screening instrument, colonoscopy, and

  (2) effective strategies for screening based on risk factors.
- Mild risk factors include age, diet, physical inactivity, obesity, smoking, race, and alcohol.
- Intermediate risk groups include those with a personal history of colorectal cancer or adenoma, as well as those with a strong family history.

Screening

- Individuals with **one first-degree relative** with colorectal cancer have a **twofold** increased risk of colorectal cancer
- Those with **two first-degree relatives** have a **six fold** increased risk.
- Patients at high risk for developing colorectal cancer are those with:
  - **Familial colorectal cancer syndromes** (familial polyposis, Gardner's, and HNPCC) and
  - patients who have had **Ulcerative** or **Crohn's colitis** for more than 10 years.

Screening

- Screening Genetic studies are offered to individuals with positive family history for inherited sydromes.
- For patients with average risk, the guidelines recommend that, beginning at age 50, both men and women should follow one of these screening options:

Screening

A – Stool Tests

1. **Guaiac-based Fecal Occult Blood Test (gFOBT)** uses the chemical guaiac to detect blood in the stool. It is done once a year. At home, a stick is used to obtain a small amount of stool.
2. **Fecal Immunochemical Test (FIT)** uses antibodies to detect blood in the stool. It is done once a year in the same way as a gFOBT.
3. **FIT-DNA test (also referred to as the stool DNA test)** combines the FIT with a test that detects altered DNA in the stool. For this test, an entire bowel movement is sent to a lab, where it is checked for cancer cells. It is done once every one or three years.

Colonoscopy should be done if test results are positive.

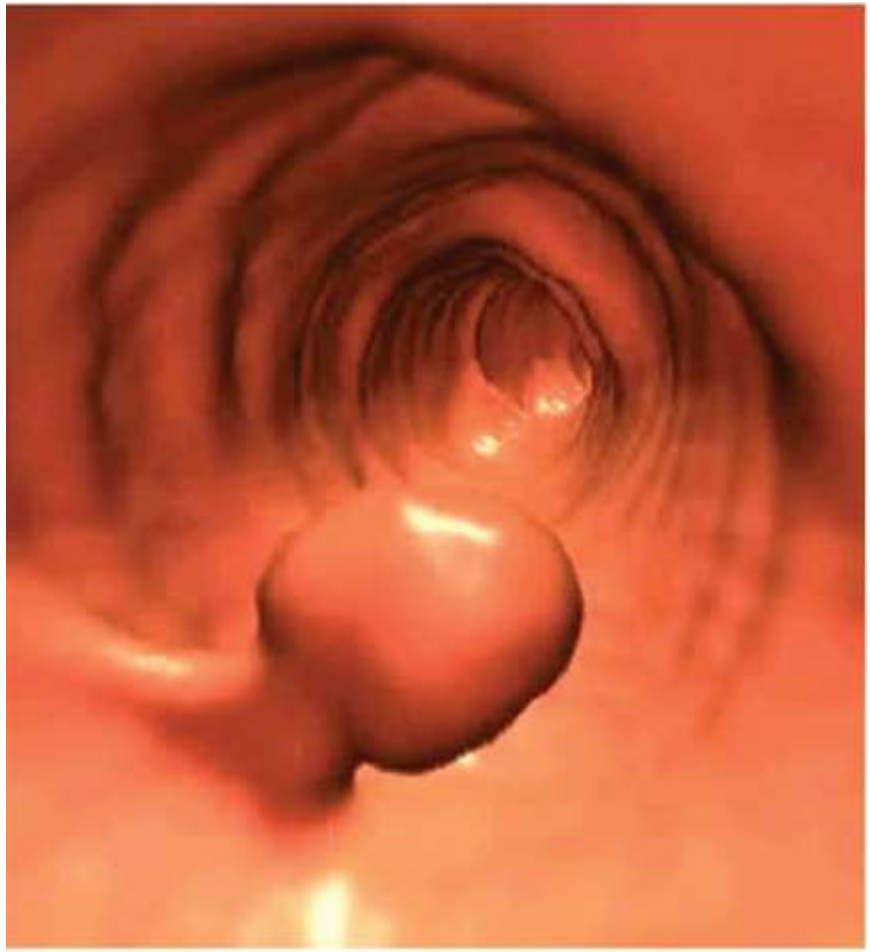
Screening

B – Scopes / Imaging Tests

1. **Flexible sigmoidoscopy every 5 years***
2. **Colonoscopy every 10 years**
3. **Double-contrast barium enema every 5 years***
4. **CT colonography (virtual colonoscopy) every 5 years***

Screening

- CT colonography is a newer test and the final results for its use as a screening method remain unclear.
- However, it is an important tool in a patient that can’t be evaluated by colonoscopy or barium enema.



Screening

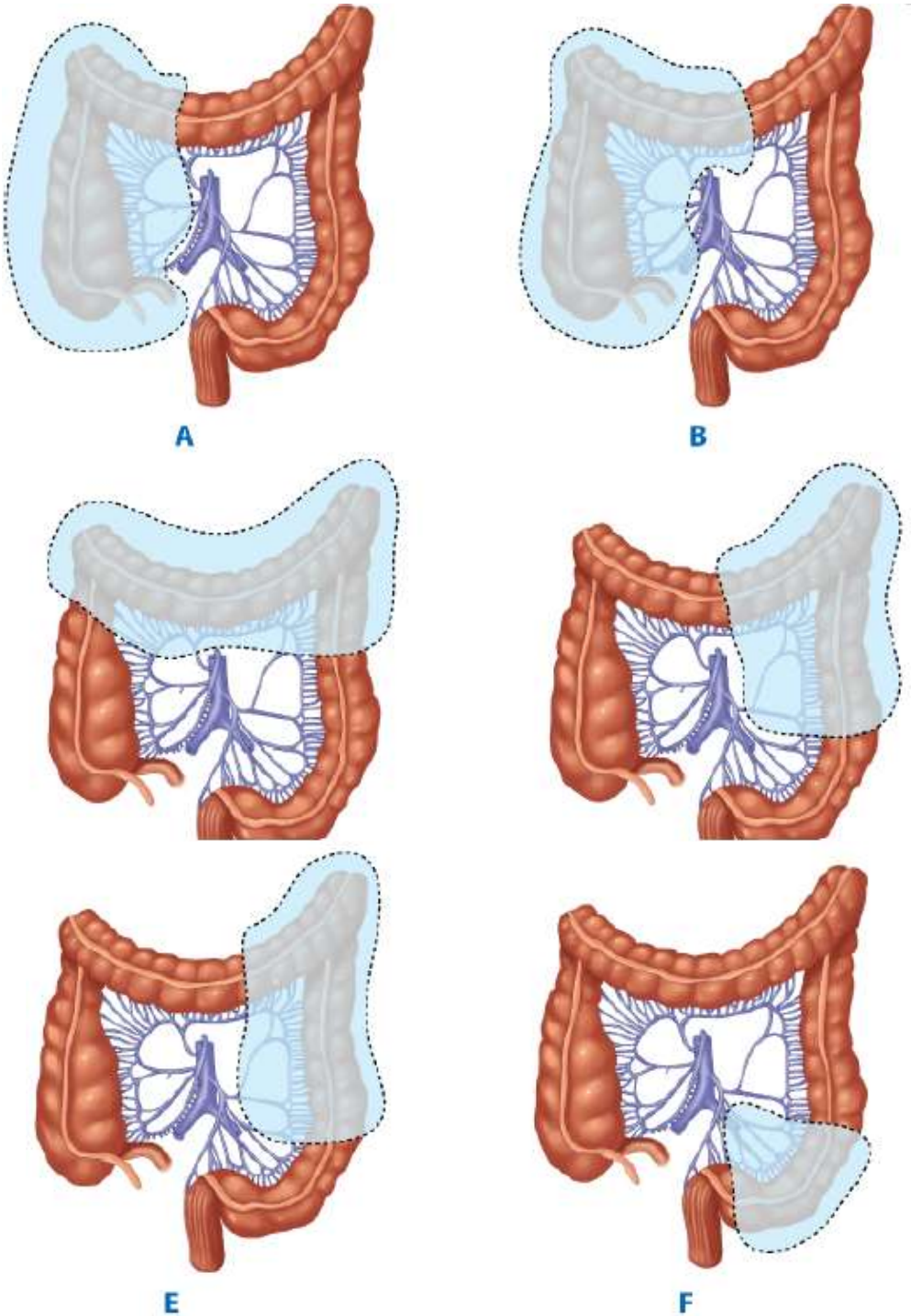
- If the patient has intermediate risk, the screening should begin at age 40 and be done more frequently than every 10 years (e.g., every 3 to 5 years).

Screening

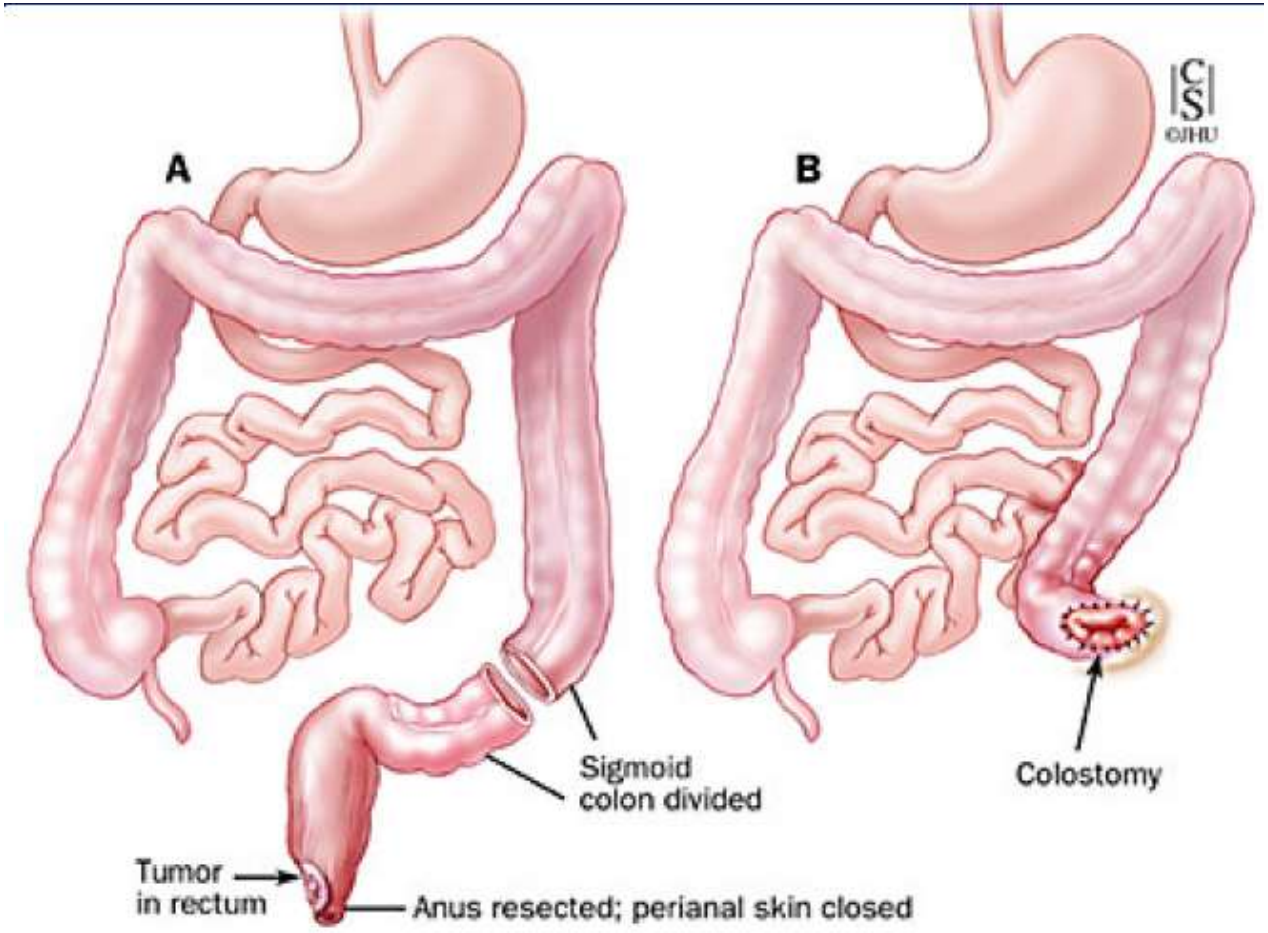
- Patients who have had ulcerative or Crohn's colitis for 10 years or more should begin annual colonoscopic surveillance with biopsies.
- Serious consideration should be given to prophylactic total colectomy with ileoanal pull-through in both high-risk groups before an invasive cancer develops.

Treatment

- The surgical treatment used by most surgeons includes adequate local excision of the tumor, with a **length of normal bowel on either side, and resection of the potentially involved lymph node** draining basin found in the mesentery that is determined by the vascular supply.



Abdomino-Perineal Resection with colostomy



Chemotherapy

Adjuvant Therapy for *Colon* Cancer

In cases of Stage III and some Stage II cancers, postoperative adjuvant chemotherapy is often recommended.

Randomized trials have demonstrated statistically improved survival in such patients.

Typically, combination chemotherapy is administered (e.g. 5-fluorouracil '5-FU' plus leucovorin or levamisole) for a six-months following surgery.

It is administered on an outpatient bias, most patients do not lose their hair, and are able to continue usual daily activities.

This therapy is usually well tolerated, however, common complications of adjuvant chemotherapy include diarrhea, neutropenia, and stomatitis.

Chemotherapy

- The recent development of a new chemotherapeutic agent, oxaliplatin, appears to be twice as effective as 5-FU alone in reducing cancer recurrence in high-risk patients as well as treating patients with metastatic colorectal cancer.
- FOLFOX (5-FU, leucovorin and oxaliplatin) is the standard treatment regimen currently.
- A number of new biologic agents show promise in achieving even better results especially with hepatic metastases (bevacizumab, cetuximab).

Chemotherapy / Radiotherapy

Adjuvant Therapy for *Rectal* Cancer

Postoperative chemotherapy combined with radiation therapy has been shown to improve outcomes in patients with transmural or node positive rectal cancers.

Combined chemotherapy and radiation improve local control and increase overall survival.

This combined therapy is the standard of care for patients with Stage II and III rectal cancers.

Radiation therapy is typically administered with chemotherapy for a six-week period. Chemotherapy alone is then administered for an additional four months.

Chemotherapy / Radiotherapy

NeoAdjuvant Therapy for *Rectal* Cancer

Preoperative chemoradiation therapy is being used with increased frequency in patients with rectal cancer.

The advantages of preoperative therapy include increased sphincter preservation, less small intestinal radiation injury, and improved bowel function.

Typically, preoperative therapy is reserved for those tumors with evidence of nodal or transmural disease.

The combined chemoradiation is given for six weeks, followed by a break of 4–10 weeks before surgery.

An additional four months of chemotherapy is given postoperatively.

Surveillance (post-op follow up)

- A frequently used approach is a **clinic visit** every 3 months for 2 years, every 6 months for 3 years, and then yearly until 5 or 10 years postresection.
- Visits include physical examination and measurement of **carcinoembryonic (CEA)** levels.
- **Colonoscopy** is usually performed at 1 and 2 years postoperatively and then every 2 to 3 years after.
- Most recurrences occur in the first 18 to 24 months.

Surveillance (post-op follow up)

- The use of CEA is well established, with recurrence suggested not only by the absolute level of this antigen, but also by a progressive rise.
- A progressive rise mandates a complete evaluation of the patient, colonoscopy, including CT of the chest, abdomen, and pelvis.

Surveillance (post-op follow up)

- A potentially important new diagnostic modality used to detect widespread metastases in colorectal cancer is the positron emission tomography (PET) scan.

- It is currently the most sensitive test to detect recurrent colorectal cancer.

Colonic Stents

It is used in obstructing colorectal tumors.

It is only useful in tumors located in the left colon (i.e. splenic flexure to rectosigmoid junction).

Colonic stents may be placed endoscopically in a relatively short period of time with very few complications.

Colonic Stents

It can be used either as a palliative or as a bridge to surgery:

Palliative: Stenting is a useful therapeutic modality in non-operative patients to preserve quality of life and keep these patients out of the hospital.

Bridge to surgery: Preoperative colonic decompression with an expandable stent often allows postponement of surgery until there can be adequate bowel cleansing and/or stabilization of the patient's general health.

Questions ?

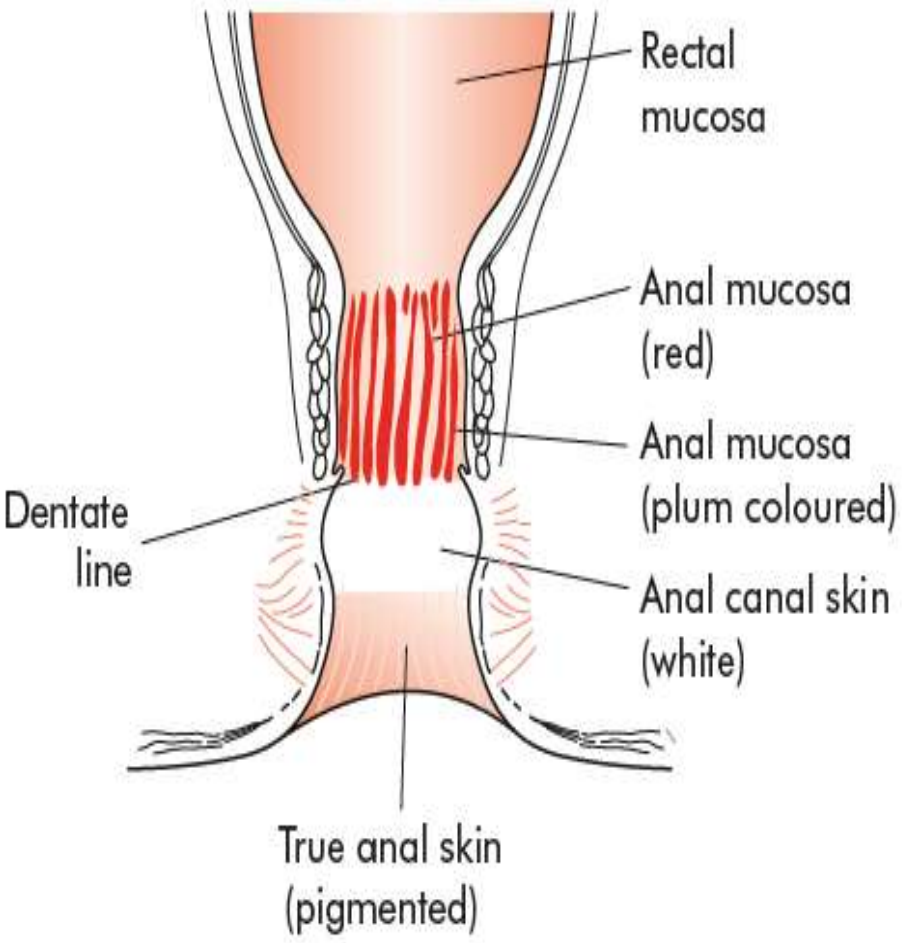
Anal Canal

The epithelium

The **pink columnar** epithelium lining the **rectum** extends through the anorectal ring into the surgical anal canal.

Passing downwards, the mucous membrane becomes **cuboidal** and **redder** in colour, whereas above the anal valves it is **plum** coloured. Just below the level of the anal valves there is an abrupt, albeit wavy, transition to **stratified squamous** epithelium, which is the colour of **parchment**. This wavy junction constitutes the dentate line.

The **dentate line** is a most important landmark both morphologically and surgically, representing the site of the **crypts of Morgagni** (synonym: anal crypts, sinuses).



Arterial supply

The historical description of the blood supply to the upper anal canal is bifurcation of the main trunk of the superior rectal artery into right and left branches and with subsequent division of the former into anterior and posterior divisions thereby determining the sites of haemorrhoids around the anal circumference

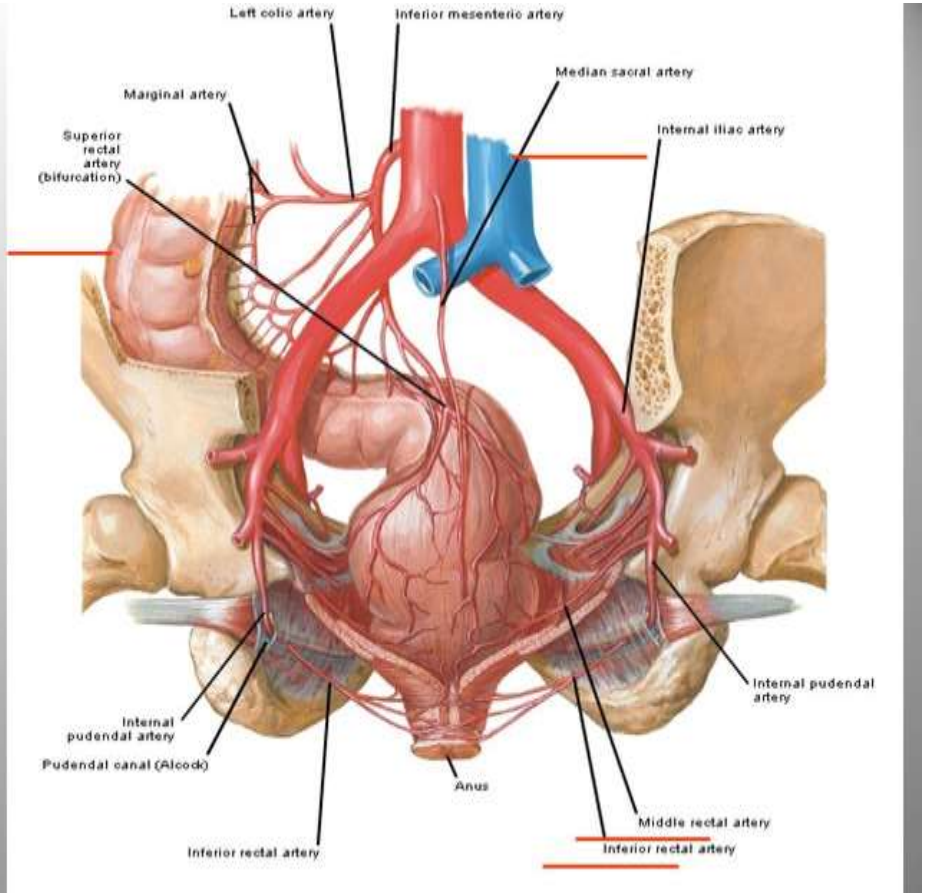
Arterial supply of the rectum and anal canal

median sacral artery

superior rectal artery (inferior mesenteric)

middle rectal artery (internal iliac)

inferior rectal artery (internal pudendal)

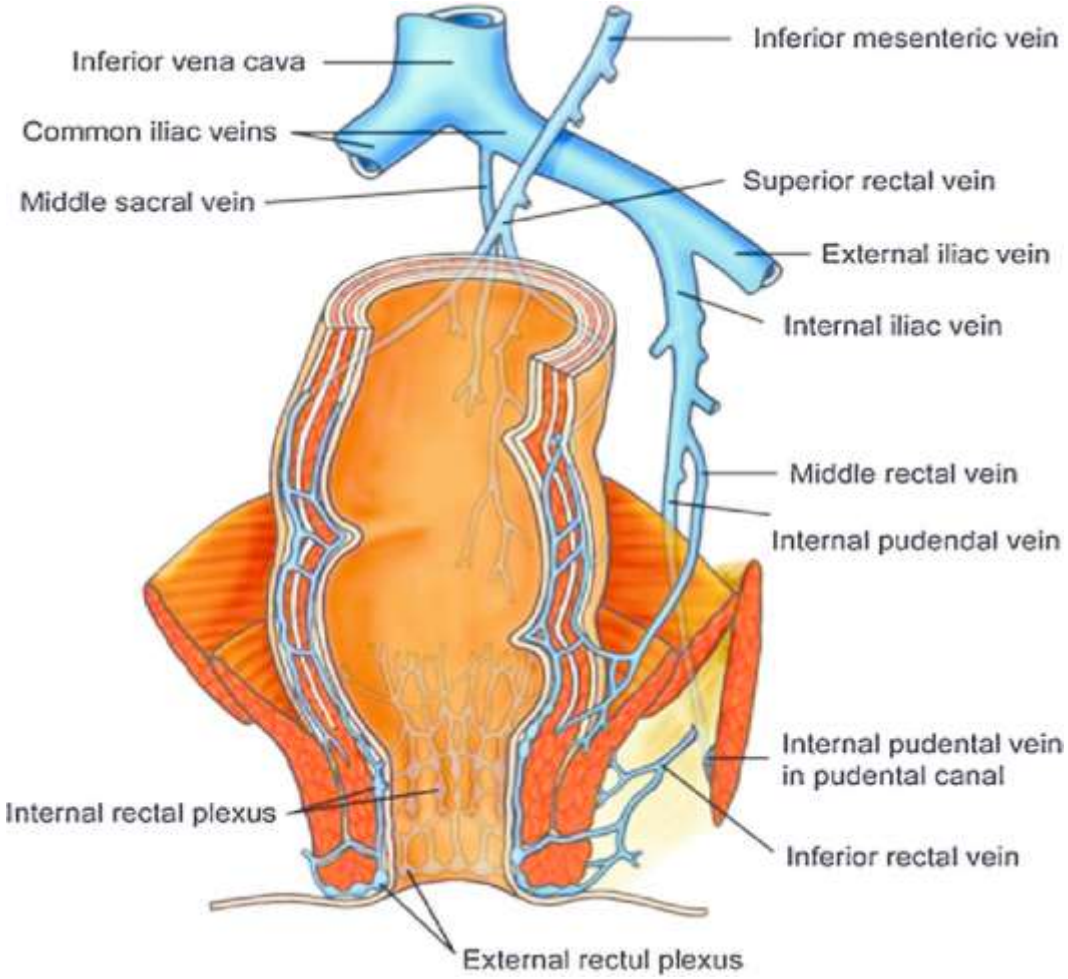


Venous drainage

The anal veins are distributed in a similar fashion to the arterial supply.

The upper half of the anal canal is drained by the superior rectal veins, tributaries of the inferior mesenteric vein and thus the portomesenteric venous system, and the middle rectal veins, which drain into the internal iliac veins.

The inferior rectal veins drain the lower half of the anal canal and the subcutaneous perianal plexus of veins: they eventually join the internal iliac vein on each side.

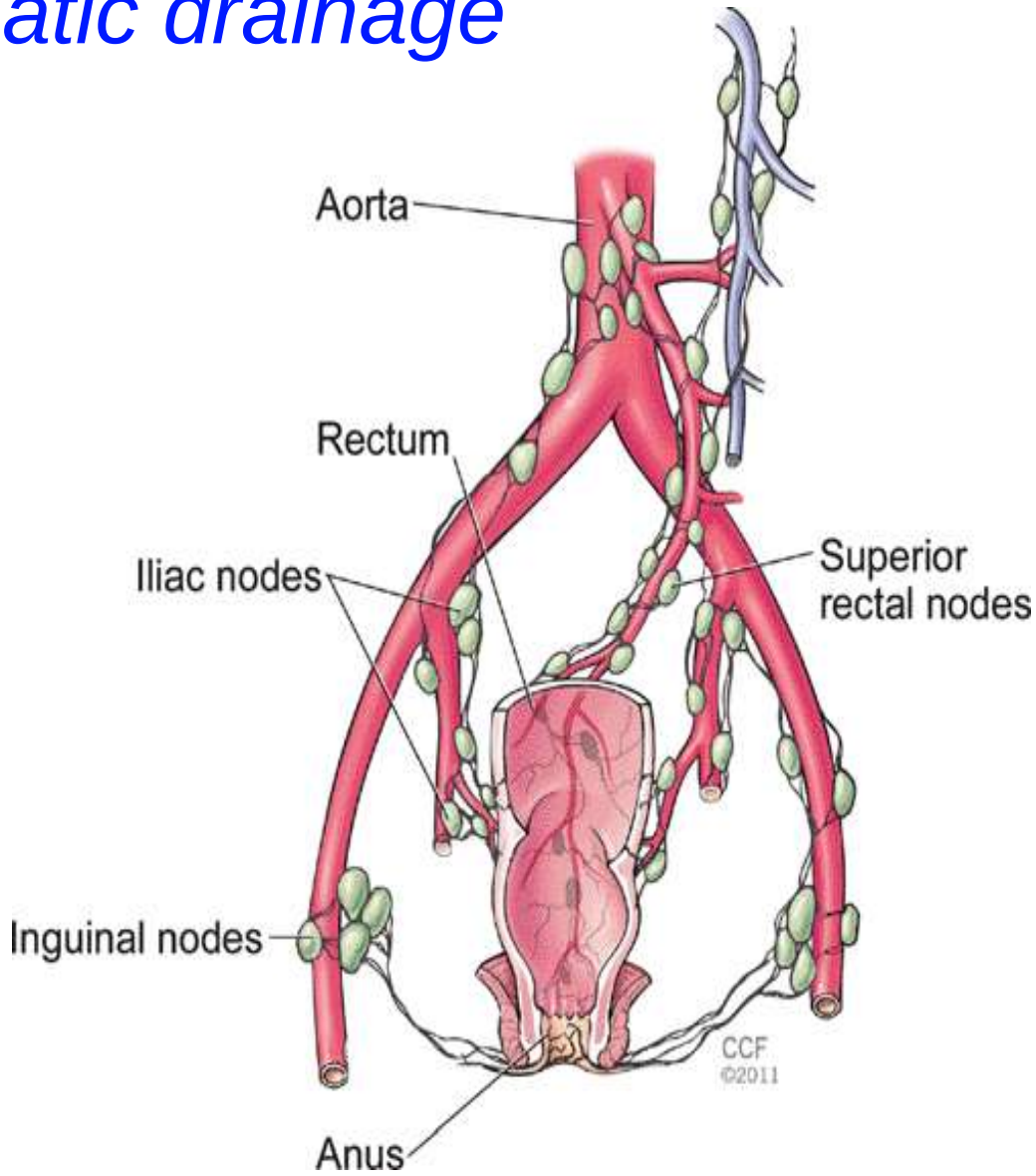


Lymphatic drainage

Lymph from the **upper half** of the anal canal flows upwards to drain into the **postrectal lymph nodes** and from there goes to the **para-aortic nodes** via the **inferior mesenteric** chain.

Lymph from the **lower half** of the anal canal drains on each side first into the superficial and then into the **deep inguinal** group of lymph glands.

However, if the normal flow is blocked, e.g. by tumour, the lymph can be diverted into the alternative route



Anal Cancer

- Rare (0.65/100000)
- <2% of all large bowel cancers.
- The overall 5-year survival rate for anal cancer is about 65%
- **SCC** is by far the most common type of anal cancer.

Anal Cancer

- Those arising below the dentate line are usually ***squamous***:
  - Associated with human papilloma virus (HPV),
  - More prevalent in patients with HIV infection
- Adenocarcinoma is the next most common (usually extension from above dentate line).
- Other tumours include melanoma (usually affects the anal verge), lymphoma and sarcoma.

Clinical Presentation

- 20% are asymptomatic.
- Symptoms are frequently late, nonspecific, and generally relate to the size of the tumor and extent of infiltration.
- Tumors are most frequently associated with rectal bleeding (45%), pain or the sensation of a rectal mass is present in 30%.
- Other manifestations include pruritus, discomfort when sitting, change in bowel habits, incontinence due to sphincter involvement, discharge, fissure, and fistula.

- Gynecologic examination including screening for cervical cancer is suggested for women, due to its association with anal cancer and HPV.

Staging

- Routine imaging with a CT scan of the chest, abdomen, and pelvis is recommended.
- MRI of the pelvis may be indicated in select cases for superior discernment of adjacent structure involvement.
- PET/CT scan does not replace a CT scan, but can provide additional information

Management

- Definitive radical surgical resection of anal cancer by abdominoperineal resection (APR) was the standard of care prior to the 1980s. paradigm shift toward organ and sphincter preservation occurred after reported complete responses to neoadjuvant chemoradiation with 5-fluorouracil (5-FU), mitomycin C (MMC), and radiation, which wad confirmed by multiple randomized trials.

- Surgical resection major ablative surgery (APR) is performed in patients who did not appear to have responded to the treatment

TREATMENT SEQUELAE: SHORT- AND LONG-TERM RISKS OF PELVIC IRRADIATION

- Any normal tissue within the pelvis can be affected by radiotherapy.
- Short-term: patients most commonly report possible nausea, vomiting, diarrhea, bladder irritation manifested by dysuria, abdominal cramping, rectal urgency/frequency/pain, and skin effects ranging from erythema to moist desquamation.
- Long-term pelvic radiation can be associated with sexual and gastrointestinal impairment. Given the young age of many patients with anal cancer, fertility preservation is often an issue. sperm cryopreservation for male patients and embryo or oocyte cryopreservation for women are suggested.

THANK YOU

Add:

- Genetic studies for determining the post-op chemo therapy specially in stage II