كلية الطب البشري
Faculty of Medicine

Colorectal Polyps

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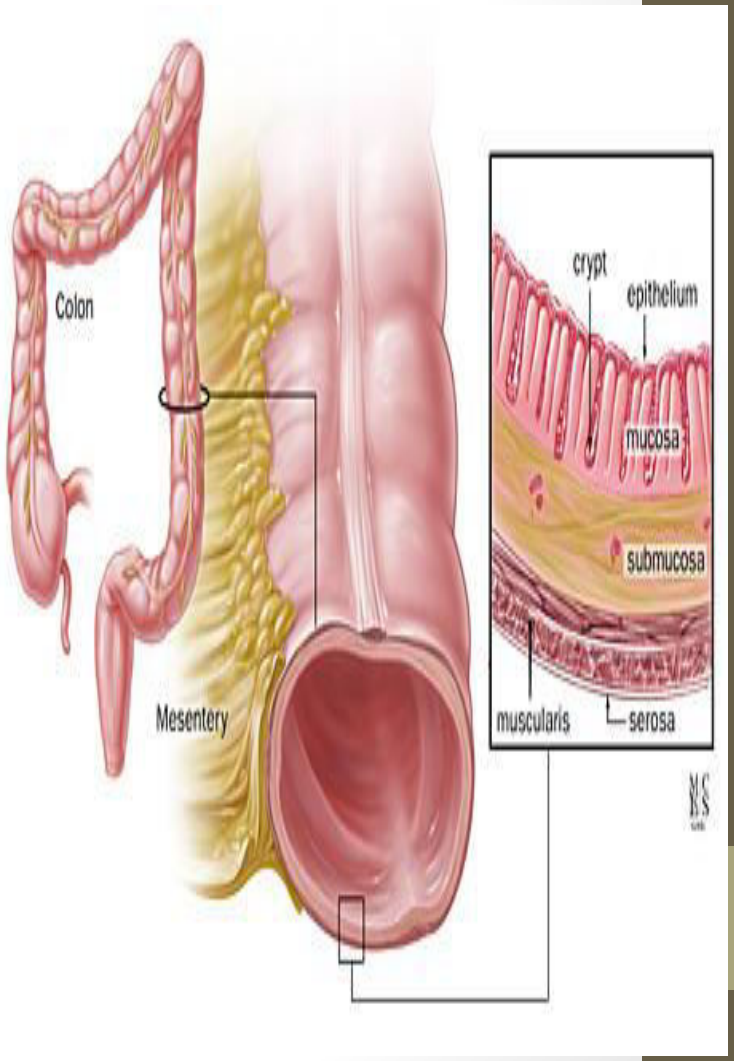
Anatomy

- 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.

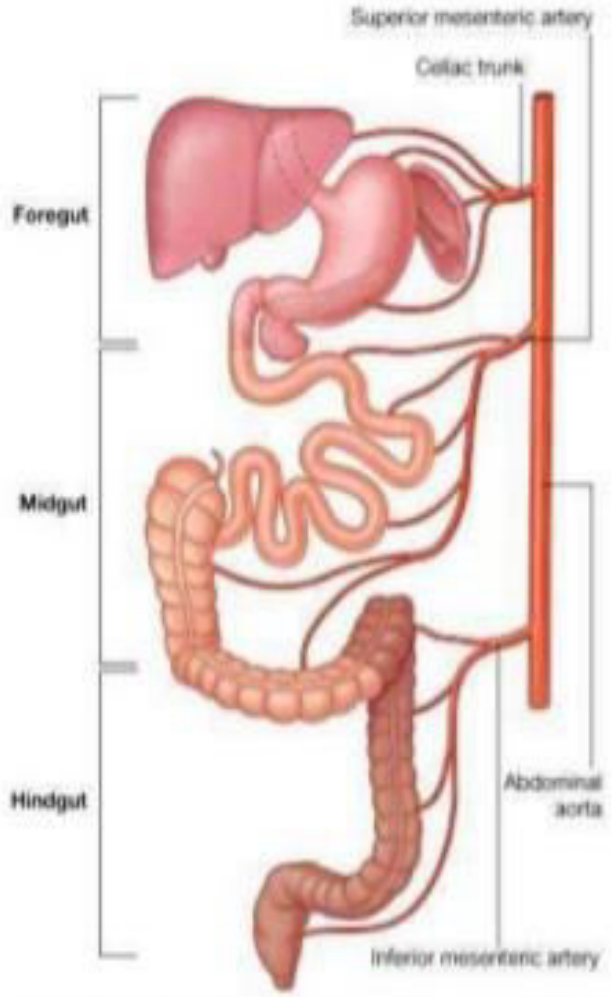


Embryology

1, FOREGUT

2, MIDGUT

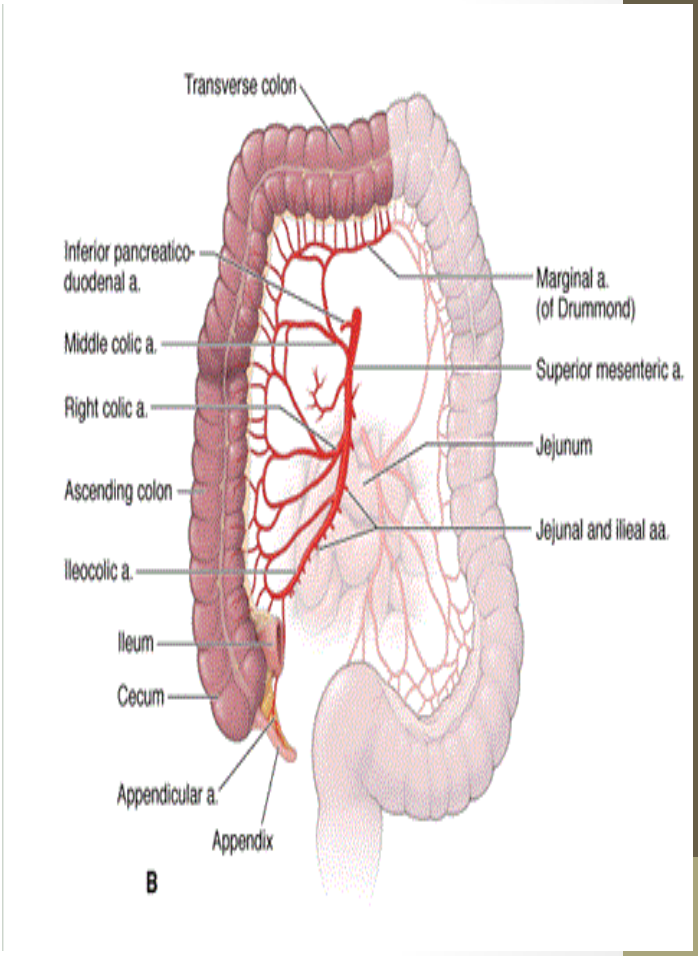
3 ,HINDGUT



Blood supply

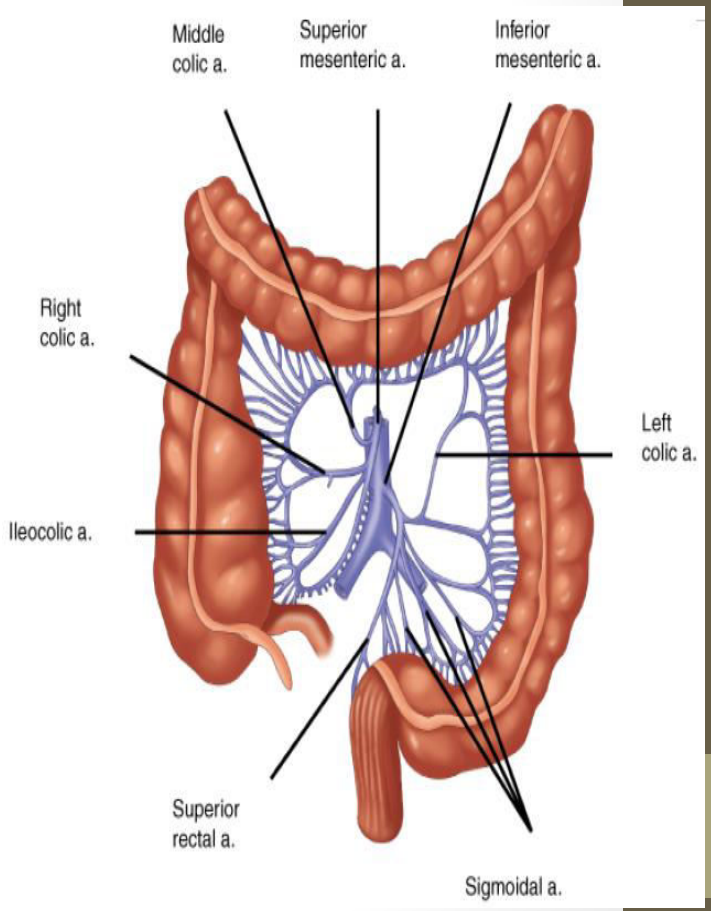
Till the distal transverse colon (midgut), it is supplied by superior mesenteric artery.

Distal to it (hindgut), it is supplied by inferior mesenteric artery



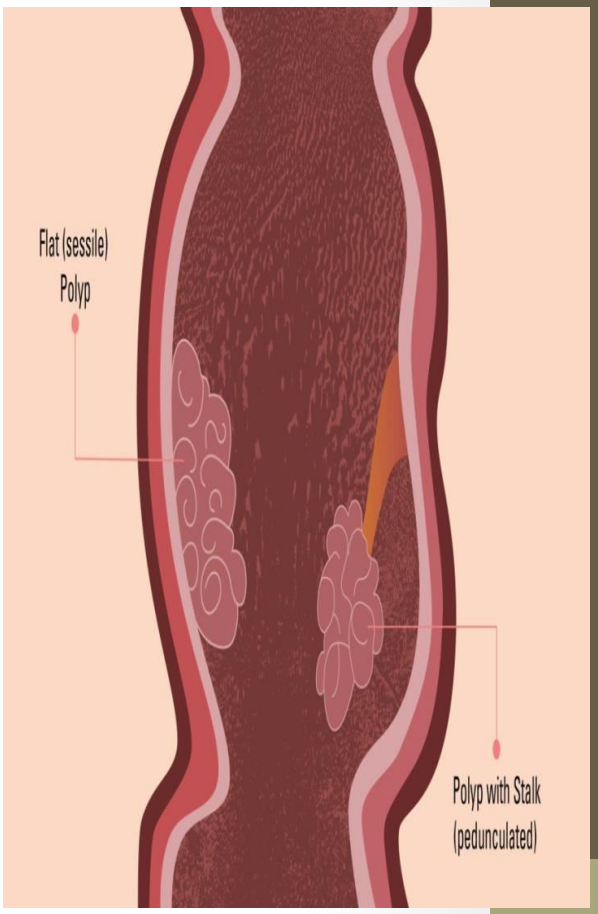
Venous and lymphatic drainage

It is follow the arterial supply and venous drainage is into the portal system.

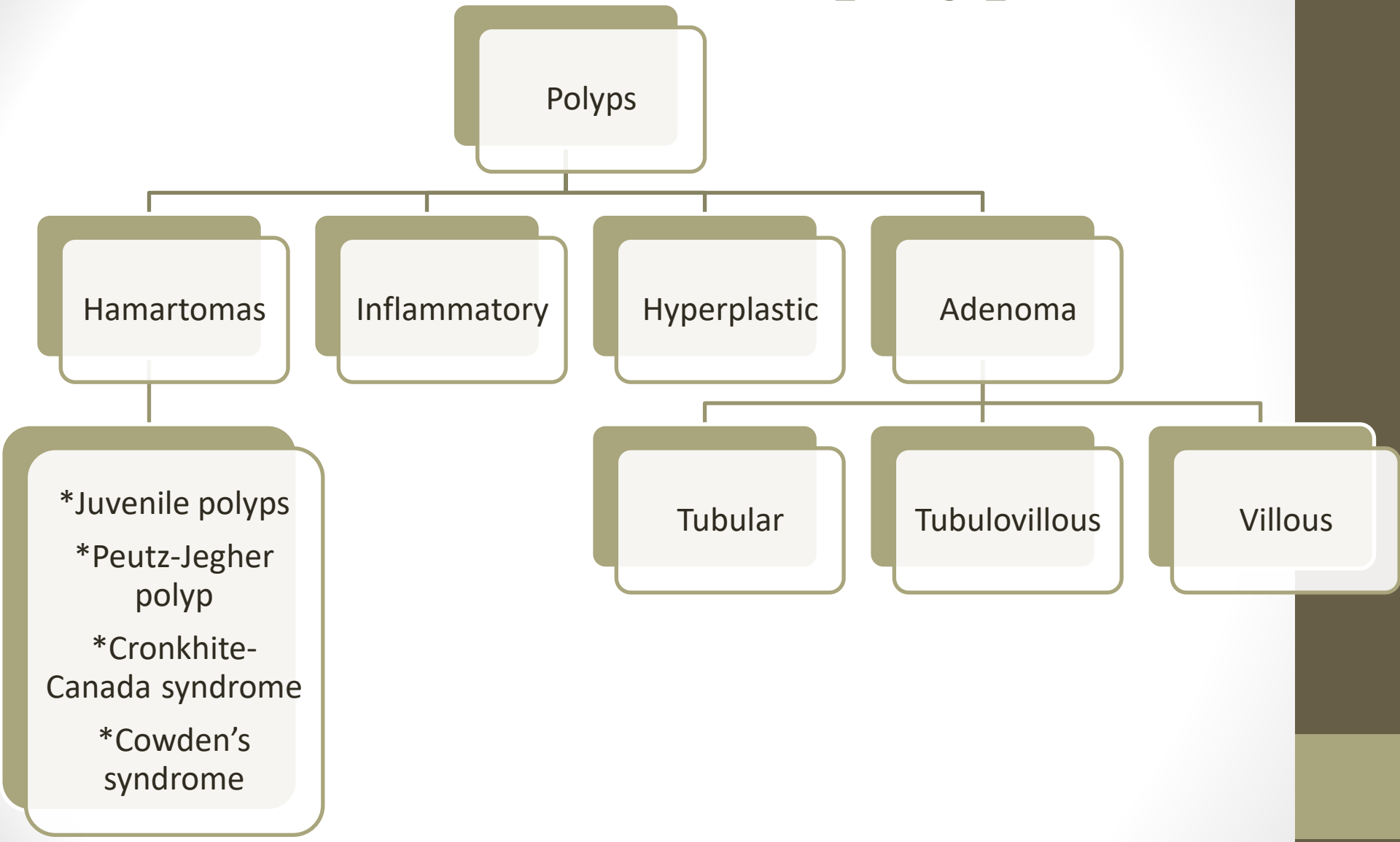


Polyps

- Any projection from the surface of the intestinal (colorectal) mucosa into the lumen regardless of the histological nature.
- Can either be pedunculated (with a stalk) or sessile (flat).
- Approximately 50% occur in the rectosigmoid region, and 50% are multiple



Classification of polyps



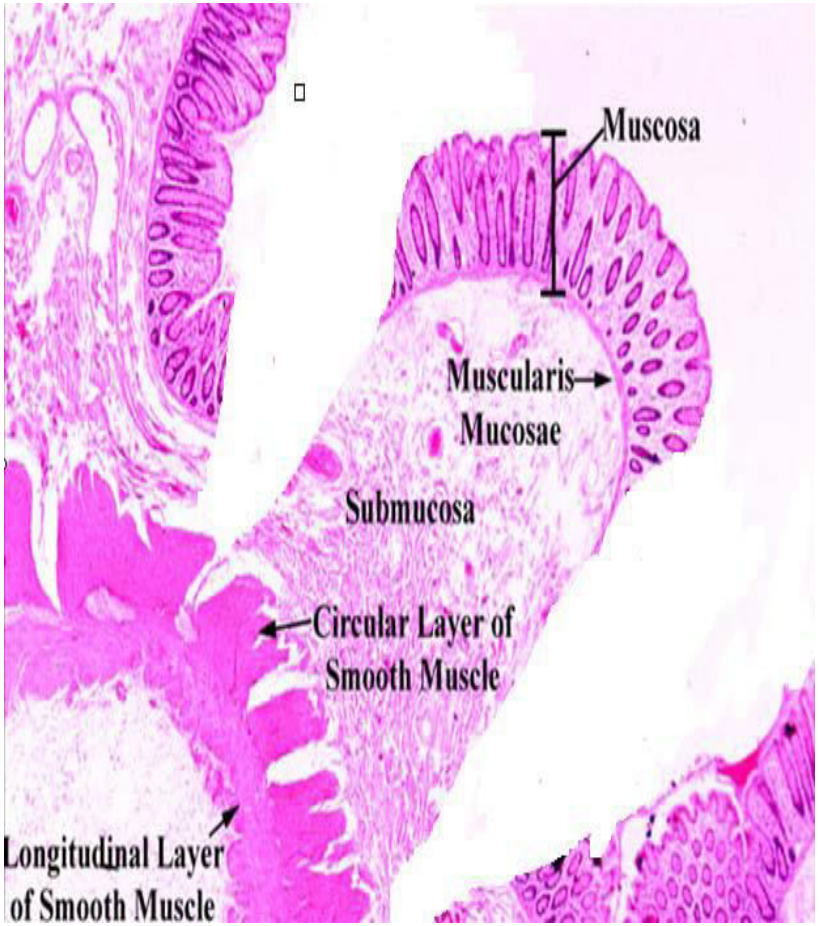
Hyperplastic polyps

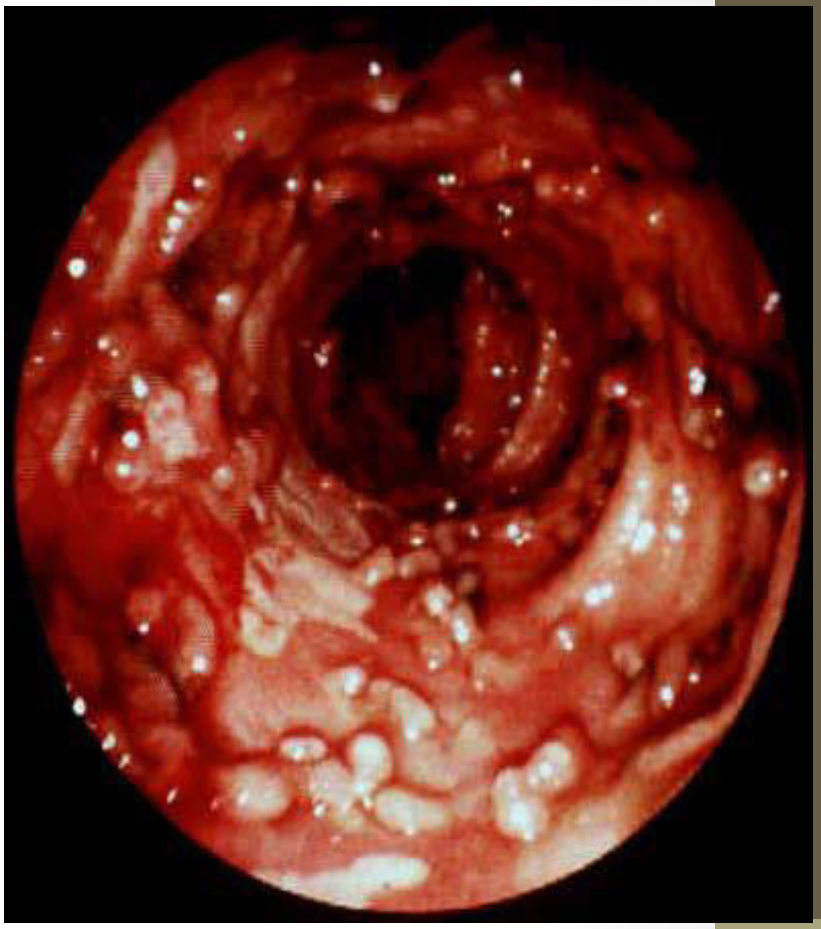
- the **most common non-neoplastic polyp**s in the colon.
- They are typically located in the **rectosigmoid** and are **less than 5 mm in size.**
- They have low malignant potential
- Small hyperplastic polyps are typically **removed (biopsied)** because they can be difficult to distinguish from adenomatous polyps

- they must be distinguished from sessile serrated adenoma / polyp that may demonstrate very similar histopathologic features and are considered to **have malignant potential**.
- **Sessile serrated polyps:** these polyps are bigger and are located on the right side can turn into cancer somewhat quickly.

Inflammatory Polyps

- Non-Neoplastic
- Occur most commonly in the **context of inflammatory bowel disease**, but they may also occur after amebic colitis, ischemic colitis.
- Usually **smaller than 2 cm**
- **Management** do not require excision unless they cause symptoms (eg, bleeding, obstruction). Because they cannot be distinguished from adenomatous polyps based on gross appearance, they should be removed (**biopsied**).


Muscosa
Muscularis
Mucosae
Submucosa
Circular Layer of
Smooth Muscle
Longitudinal Layer
of Smooth Muscle



Hamartomatous

Juvenile polyposis Syndrome	Peutz Jegher Polyp	Cronkhite–Canada syndrome	Cowden Syndrome
• AD , mutation in **BMPR1A and SMAD4** • Multiple polyps throughout the GI tract around age **10 years** • **50%** lifetime risk of colorectal cancer • Colonoscopic surveillance is recommended 1–2 yearly from the age of 15–18 years	• AD • Melanotic pigmentation of face, lips, oral mucosa, and palms • Increased risk for cancer of the pancreas, cervix, lung, ovary, and uterus	• Rare, nonheritable syndrome • Intestinal polyposis with **alopecia**, **nail atrophy** and brown macular hyperpigmentation	• AD • There is an increased risk of colorectal cancer • Benign and malignant disease of the breast and thyroid are the main risks.

Juvenile polyposis Syndrome

- AD , mutation in **BMPR1A and SMAD4**
- Multiple polyps throughout the GI tract around age **10 years**
- • Juvenile polyps are not pre-malignant but patients have an overall increased risk of colon cancer (**50%** lifetime risk of colorectal cancer)
- Colonoscopic surveillance is recommended 1–2 yearly from the age of 15–18 years

Peutz-Jeghers syndrome

- AD
- *LKB1 / STK1* mutations

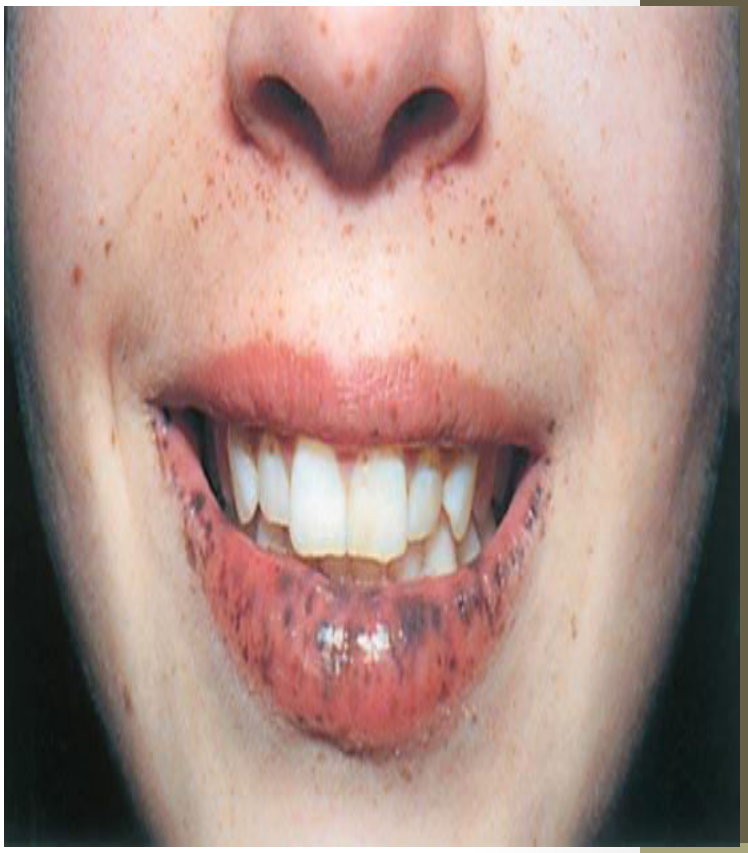
- It is characterized by:

1. polyposis of the **small intestine** and, to a lesser extent, polyposis of the colon
2. Melanotic pigmentation of face, lips, oral mucosa, and palms

- Increased risk **of both gastrointestinal** and nongastrointestinal cancers including breast cancer.

- No indication for prophylactic surgery

- • Managed with close surveillance and polypectomy or surgical resection as needed

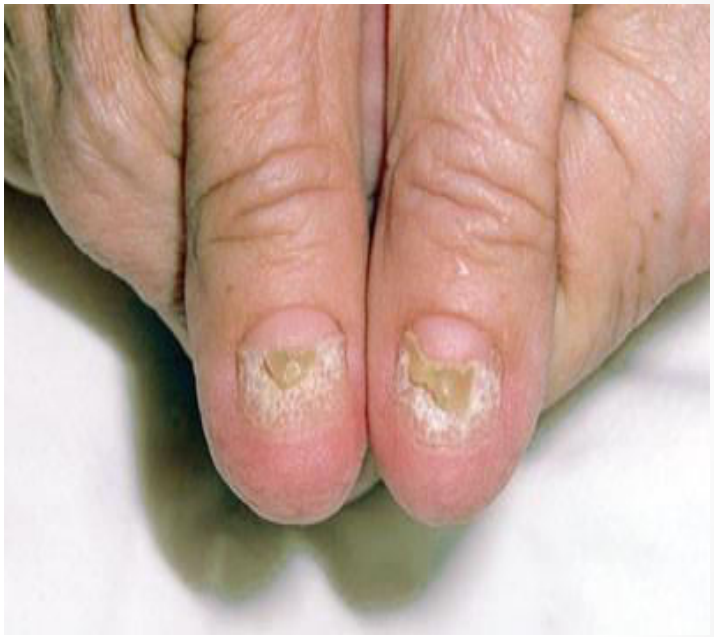


Cronkhite–Canada syndrome

- Rare, nonheritable syndrome
- Sporadic

- Characteristic cutaneous lesions (**oncyholysis , alopecia and hyperpigmentation**) , chronic diarrhea , protein-losing enteropathy and gastrointestinal polyps

- Diarrhea is common and often dictates therapy



Cowden Syndrome

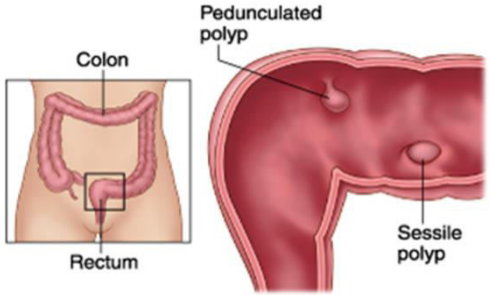
- AD
- Germline mutation in PTEN
- Multiple gastrointestinal hamartomas
- There is an increased risk of colorectal cancer
- Benign and malignant disease of the breast and thyroid are the main risks.

Adenoma

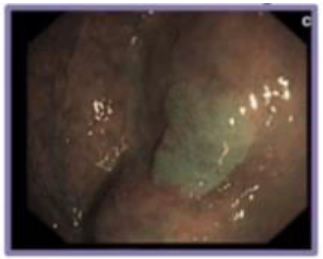
- Adenomatous polyps are common, occurring in up to **25% of the population** older **than 50 years of age** in the United States they are the **most** prevalent neoplastic polyps in the colon.
- Adenomas are generally **asymptomatic** and are most often detected by colon cancer screening tests.

- Based on their **gross** appearance adenomas may be **pedunculated** or **sessile**.

Neoplasic (Adenomatous)



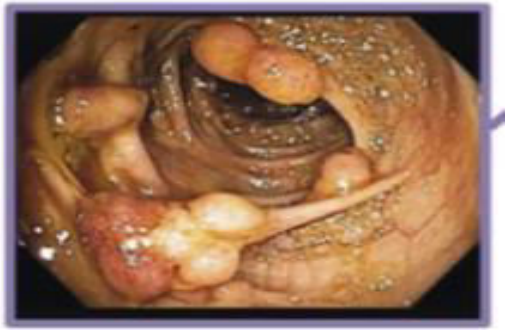
- Premalignant



- Morphology

  - **Sessile** (flat & firmly attached to mucosa), more likely to be malignant

  - **Pedunculated** (rounded and attached to mucosa by a long thin neck (attached to the colon by a stalk)



Adenoma
Histologic features

Tubular

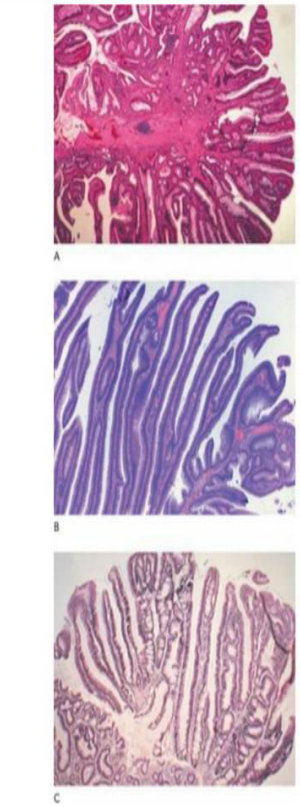
- 75%
- Rectosigmoid in 20%
- 7% malignancy risk
- Mostly pedunculated

Tubulovillous

- 15%
- Both tubular and villous features
- 20% malignancy risk

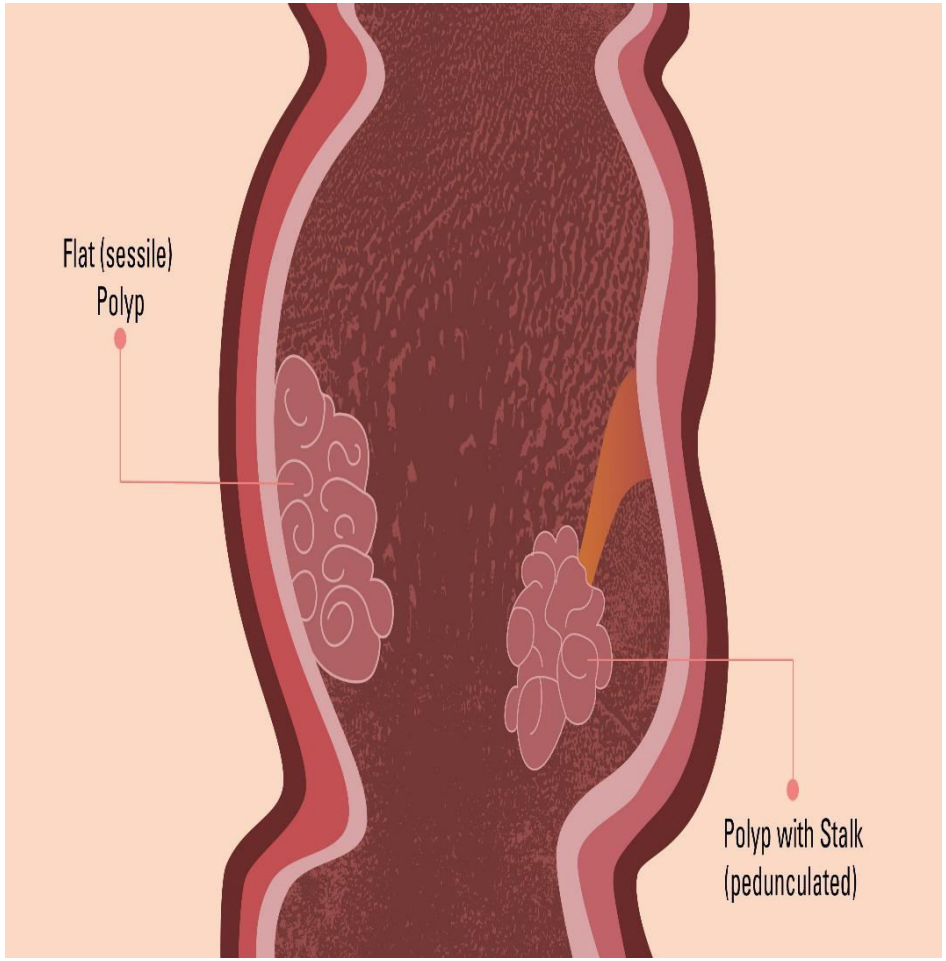
Villous

- 10%
- Rectosigmoid in 80%
- 33% malignancy risk
- Mostly sessile



Adenoma

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



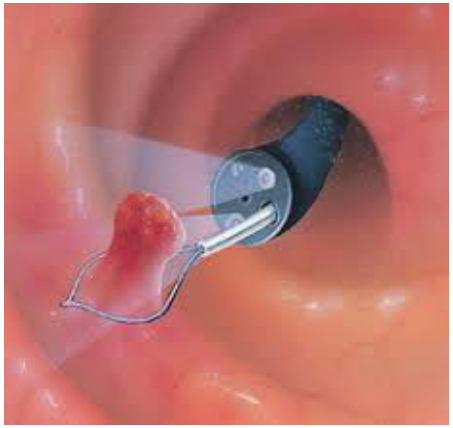
Polyps Clinical Presentation

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

Polyps Treatment

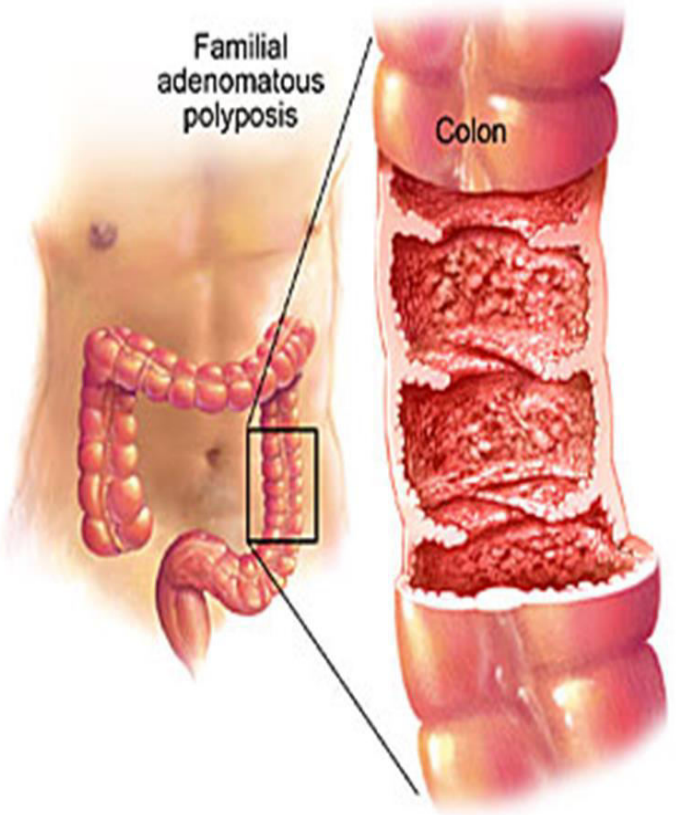
- Treatment of adenomatous polyps involves colonoscopic **polypectomy**.

- 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**.

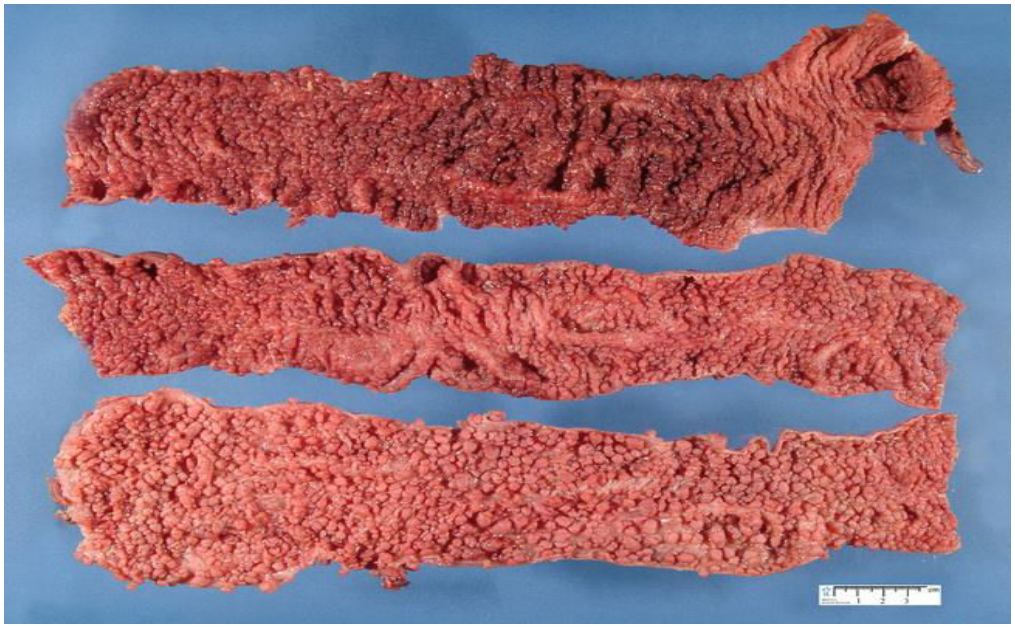


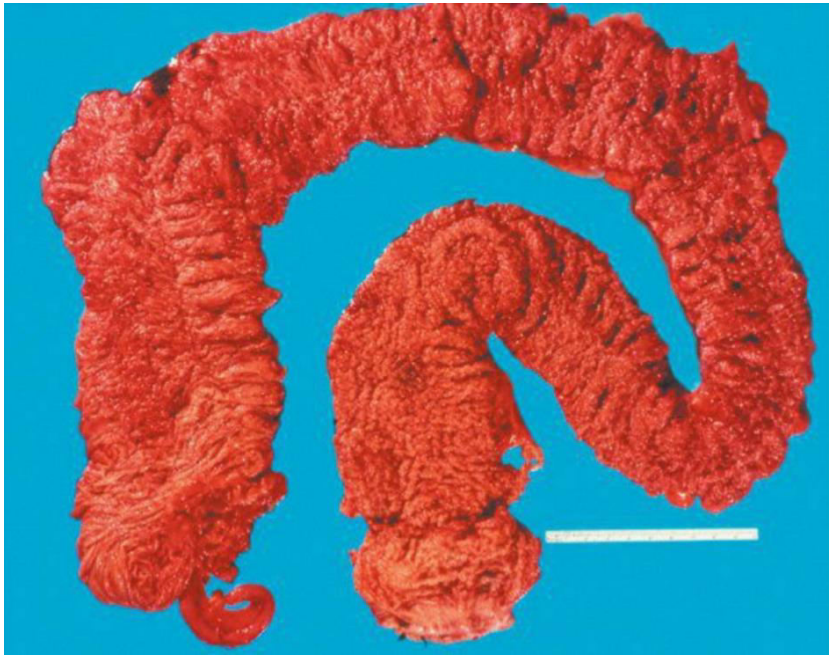
Polyps Treatment

- For disease conditions that are characterized by extensive polyposis (familial polyposis syndrome FAP or Gardner's syndrome), the operation most commonly performed is **total colectomy** and **ileoanal pull-through or pouch.**



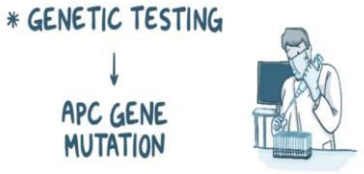
Colorectal Polyps and carcinoma





Familial adenomatous polyposis (FAP)

- Autosomal dominant, germline mutation in APC gene
- 100-1000s of polyps



- An attenuated form of FAP (**AFAP**) caused by a mutation in 5´ end of APC gene manifests with fewer polyps and later onset of cancer

- MYH-associated polyposis is caused by an autosomal recessive mutation in the MYH gene, but patients present similarly with multiple adenomatous polyps in the colon

FORM	COLORECTAL POLYPS
CLASSIC	> 100
ATTENUATED	< 100

* CLASSIC FORM: SCREENING

AGE	TEST	FREQ (yrs)
10	FLEXIBLE SIGMOIDOSCOPY or COLONOSCOPY	1

~ RISK of DEVELOPING CRC ~ 100% → COLECTOMY is RECOMMENDED

* ATTENUATED FORM: SCREENING

AGE	TEST	FREQ (yrs)
25	COLONOSCOPY	1 - 2

~ CRC DEVELOPS LATER in LIFE

Familial adenomatous polyposis (FAP)

- Hundreds to thousands of polyps in the colon Polyps begin on average in the **mid-teens** but can appear as early as **age 7**.
- **Mutation carriers** are screened annually by sigmoidoscopy starting at age **10 to 12**
- Full colonoscopy should be performed when polyps identified
- Without intervention, mutation carriers develop colorectal cancer by age 40

Familial adenomatous polyposis (FAP)

- If left untreated, 100 % of patients develop cancer by the fourth or fifth decade of life.

- Definitive treatment for FAP is **total proctocolectomy** with ileal pouch and anal anastomosis

- **Mucosectomy** of the rectum is controversial—if not performed, strict surveillance of remnant rectal mucosa is required

Familial adenomatous polyposis (FAP)

- **Prophylactic proctocolectomy** is performed
- Immediately for **profuse** polyposis, multiple adenomas **>1 cm**, or adenomas with **villous** histology or high-grade dysplasia
- Patients with sparse, small (<5 mm) adenomas can be followed endoscopically with surgery to accommodate school and work schedules
- **Proctocolectomy should be performed by age 20 for all patients**

Familial adenomatous polyposis (FAP)

- **Extra-intestinal manifestations**

1. Congenital hypertrophy of the retinal pigment epithelium
2. **Osteomas** of skull or mandible (Gardner's syndrome)
3. Supernumerary teeth

Familial adenomatous polyposis (FAP)

- **FAP-associated tumors**

- Desmoid tumors (#1)
- Periampullary tumors
- Medulloblastomas (Turcot syndrome)
- Hepatoblastoma
- Small bowel cancer
- Thyroid cancer
- Adrenal cancer

- Concurrent **duodenal polyps** are common ~95% incidence

- **Endoscopic screening** for polyps should be performed starting at age 25 and every 2 to 3 years thereafter

- Surgery may be required for **extensive** duodenal polyps, **rapid** polyp **growth**, or a lesion demonstrating **high-grade** dysplasia or **ulceration**

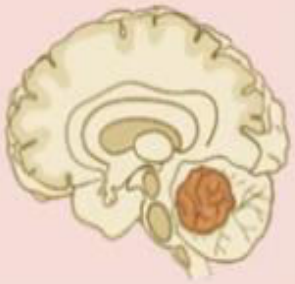
Variant of FAP

GARDNER SYNDROME

* MULTIPLE COLORECTAL POLYPS
* OSTEOMAS
* SOFT TISSUE TUMORS
  - ~ EPIDERMOID CYSTS
  - ~ FIBROMAS
  - ~ DESMOID TUMORS

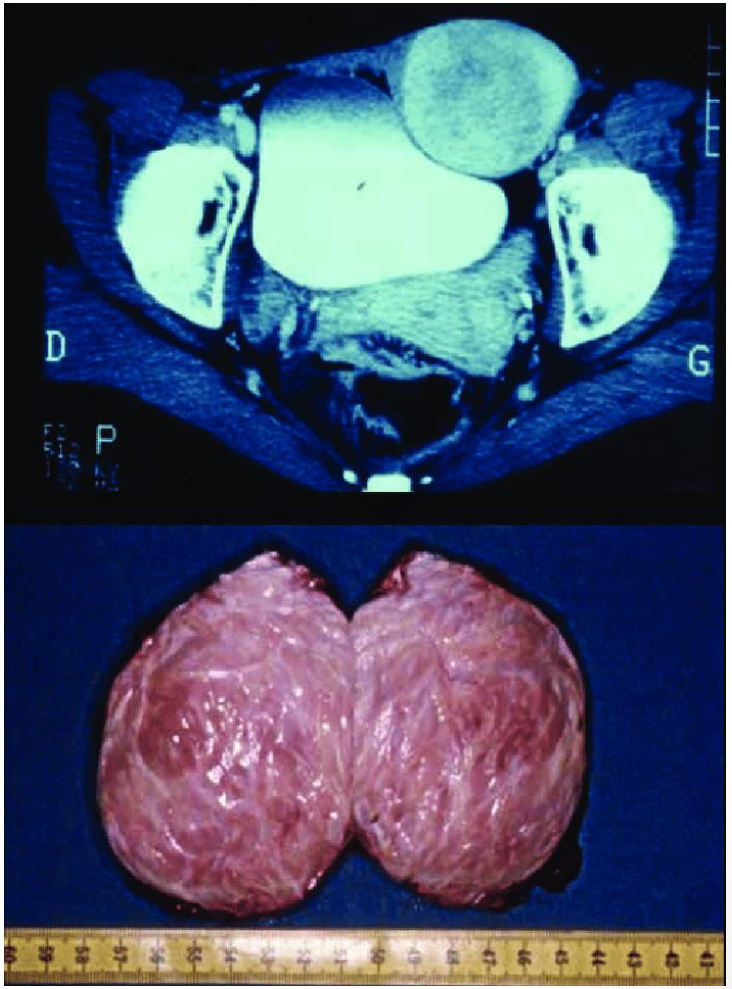
TURCOT SYNDROME

* MULTIPLE COLORECTAL POLYPS
* BRAIN TUMORS
  - ~ MEDULLOBLASTOMA
  - ~ GLIOMA



* ASSOCIATED with LYNCH SYNDROME

Desmoid tumors



Hereditary nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome

- Autosomal dominant
- The genetic defects associated with Lynch syndrome arise from errors **in** ***DNA mismatch repair genes.*** **(MLH or MSH)**
- **Lynch type 1** results in more colonic cancers, often on the **right** side and typically in the 4th decade of life.
- **Lynch type 2** results in more extracolonic cancers.

Hereditary nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome

- Usually right-sided cancers
- Associated with endometrial, ovarian, gastric, and small bowel cancers
- Increased risk of synchronous cancers: Consider subtotal colectomy ± hysterectomy with oophorectomy
- Colon cancers associated with HNPCC still arise from adenomas, but the adenomas tend to be **flat**.
- Also, the adenomas associated with HNPCC are more likely to become malignant and to do so more rapidly (**within 2 or 3 years**)

Hereditary nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome

- **Amsterdam criteria**

- • Three affected relatives (one must be a first-degree relative of one of the others)
- Two generations affected
- One diagnosed before age 50 years

- Revised Bethesda Guideline for testing for MSI in colorectal cancer patients
  - Diagnosis of colorectal cancer at age <50 years
  - Any patient with a synchronous or metachronous colorectal cancer or other HNPCC related cancer, regardless of age
  - Colorectal cancer with HNPCC-like histology (evidence of high MSI) at age <60 years
  - Colorectal cancer or other HNPCC-related tumors in one or more first degree relatives (cancer must be diagnosed at age <50 years or adenoma at age <40 years)
  - Colorectal cancer in two or more first or second degree relatives with HNPCC-related tumors regardless of age

Hereditary nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome

- Surveillance colonoscopy is recommended every 1 to 2 years starting at age 20 to 25 years and annually after age 40 years
- · Women should also undergo an annual transvaginal ultrasound, endometrial biopsy, and CA125 level
- · Women should consider TAHBSO after child bearing is complete
- · Patients can also develop sebaceous tumors (Muir-Torre syndrome)

SCREENING	
TEST	FREQUENCY (YRS)
COLONOSCOPY (start at age 20 - 25)	1 - 2
FEMALES: PEVIC EXAMINATION (look for endometrial or ovarian cancer)	1

The name “hereditary nonpolyposis colorectal cancer” is misnomer since cancer still arises from adenomatous polyps.

Colorectal Cancer

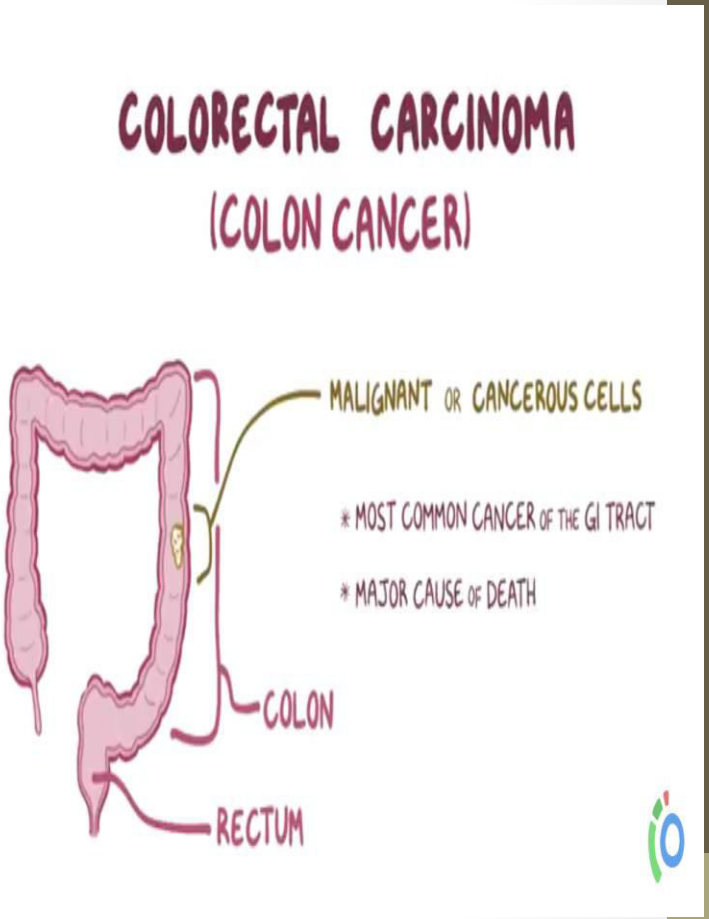
Epidemiology

- The **most common GI cancer**
- 3th most common cancer (after lung& prostate/after lung & breast)
- **2nd most common cause of cancer death (lung canccer is most common)**

- **Nearly equal incidence in both sexes. Some statistics show slight male predominance.**
- Peak incidence is **60-70** years of age

- 95% are adenocarcinomas • Other types include squamous cell, lymphoma, sarcoma, and carcinoid tumors

- **• Must perform colonoscopy to rule out synchronous lesions**



Carcinoma of Colon & Rectum

- Cancer of the colon and rectum is a major cause of death worldwide.
- Although a large number of factors are associated with the development of this disease, theories about its etiology center on the impact of intraluminal chemical carcinogenesis.
- There are various theories as to whether 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.

Epidemiology

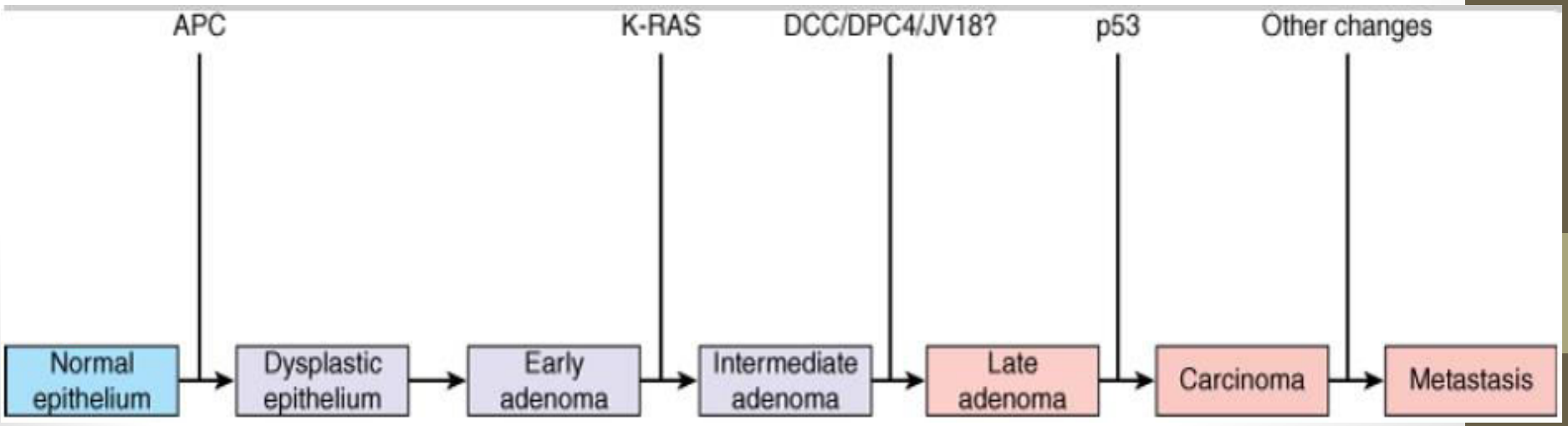
- Approximately 80% 0f colorectal cancers occur sporadically, while 20% arise in patients with a known family history of colorectal cancer.
- Genetic defects and molecular abnormalities associated with the development and progression of colorectal adenomas and carcinoma
- Mutation causing

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

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

Epidemiology

- Adenomatous polyposis coli (APC) gene responsible for FAP
- Colorectal carcinoma is though to develop from adenomatous polyps due to accumulation of these mutations



- Which one of the following is the correct sequence of mutations which lead to colon cancer?

- APC --- K-ras --- TP53
- TP53 --- APC --- K-ras
- K-ras --- TP53 --- APC
- K-ras --- APC --- TP53
- APC --- TP53 --- K-ras

Epidemiology

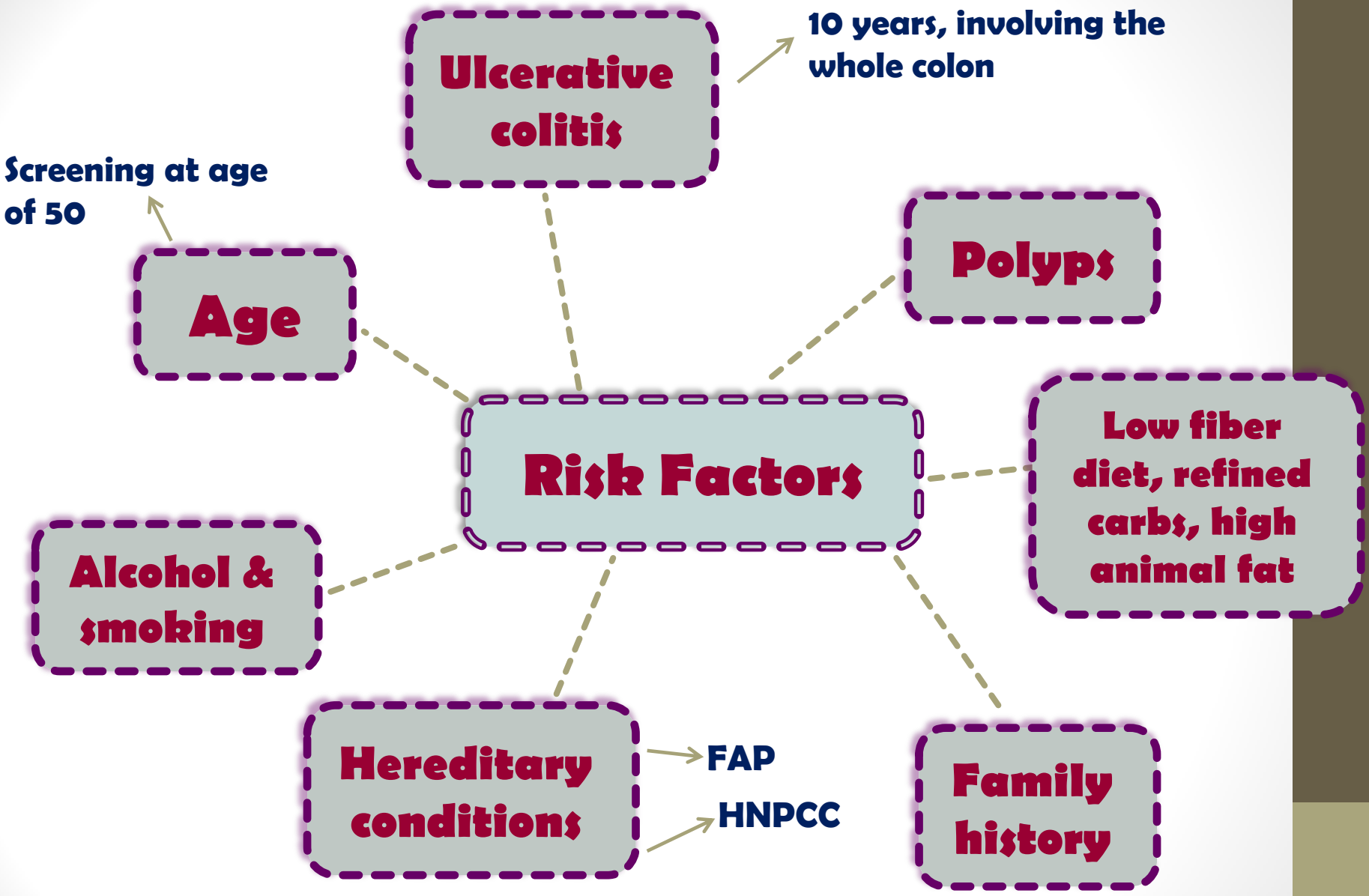
- Certain health agencies promote a **low-fat, high-fiber diet** as protective against cancer of the colon and rectum.
- Chemoprevention by ingestion of such agents as carotenoids and other antioxidants has been suggested, but the efficacy of this measure is unproven.
- There is good evidence that prostaglandin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

Epidemiology

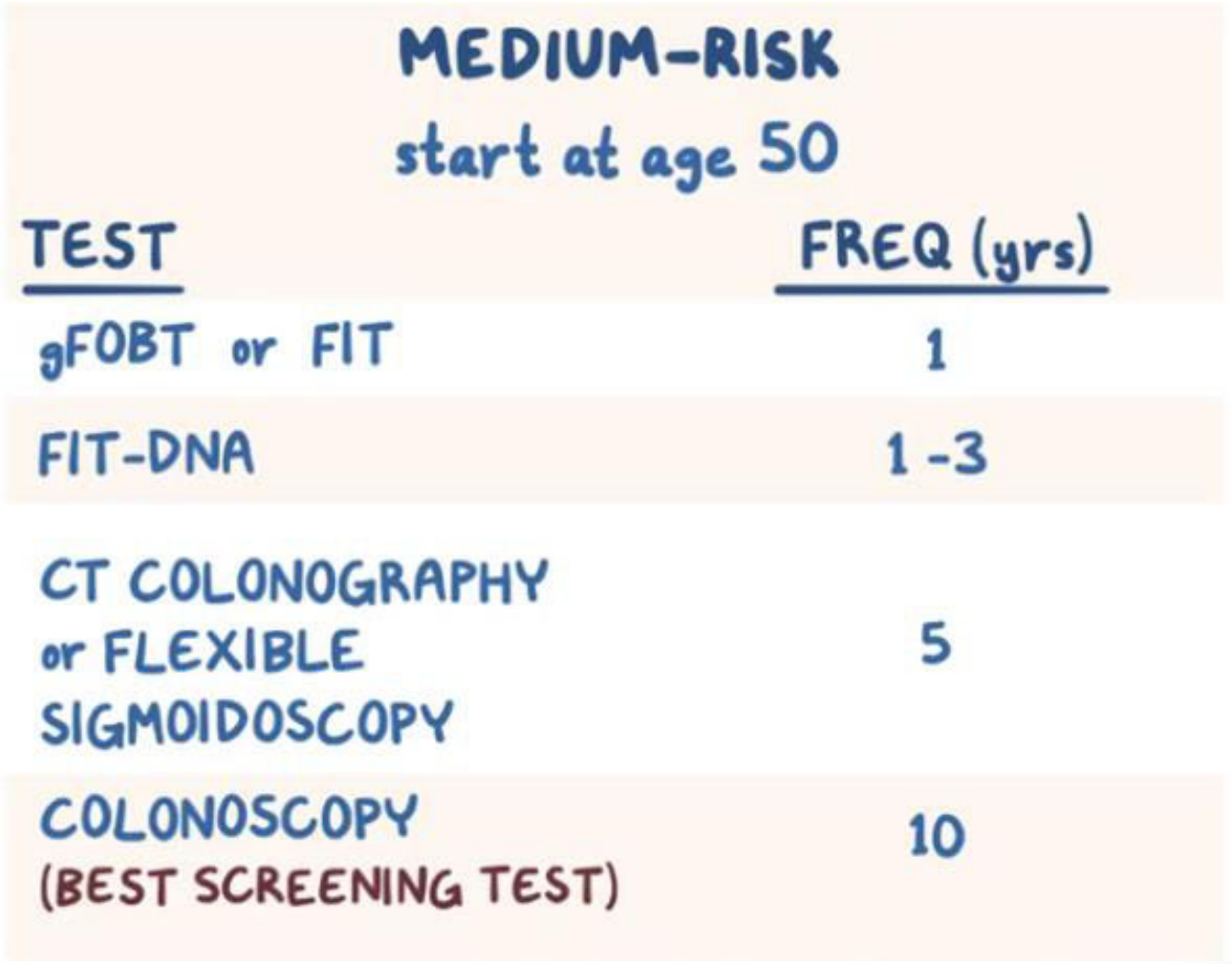
- **Synchronous** (simultaneously occurring) tumors develop in 5% of patients, whereas 3% to 5% of patients have **metachronous** tumors (a second tumor developing after resection of the first).
- A family history of colon cancer is an important risk factor for colon cancer in **first-degree** relatives.

Epidemiology

- Familial polyposis syndrome FAP, Gardner's syndrome, and the cancer family syndrome (hereditary nonpolyposis colon cancer [HNPCC]) clearly show that certain patient subsets are genetically predisposed to cancer of the colon.
- 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.


Ulcerative colitis
10 years, involving the whole colon
Screening at age of 50
Age
Polyps
Risk Factors
Low fiber diet, refined carbs, high animal fat
Alcohol & smoking
Hereditary conditions
FAP
HNPCC
Family history

Screening


MEDIUM-RISK
start at age 50
TEST
FREQ (yrs)
gFOBT or FIT
1
FIT-DNA
1-3
CT COLONOGRAPHY
or FLEXIBLE
SIGMOIDOSCOPY
5
COLONOSCOPY
(BEST SCREENING TEST)
10

HIGH-RISK

family history of sporadic CRC → age 40

TEST	FREQ (yrs)
COLONOSCOPY	5
FIT	1

INFLAMMATORY BOWEL DISEASE

TEST	FREQ (yrs)
COLONOSCOPY	1

* SCREENING STOPS at AGE 75 - 85 DEPENDING on OVER

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 malignancy.

Many practitioners follow serial CEA levels 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.

Clinical Presentation Get the point

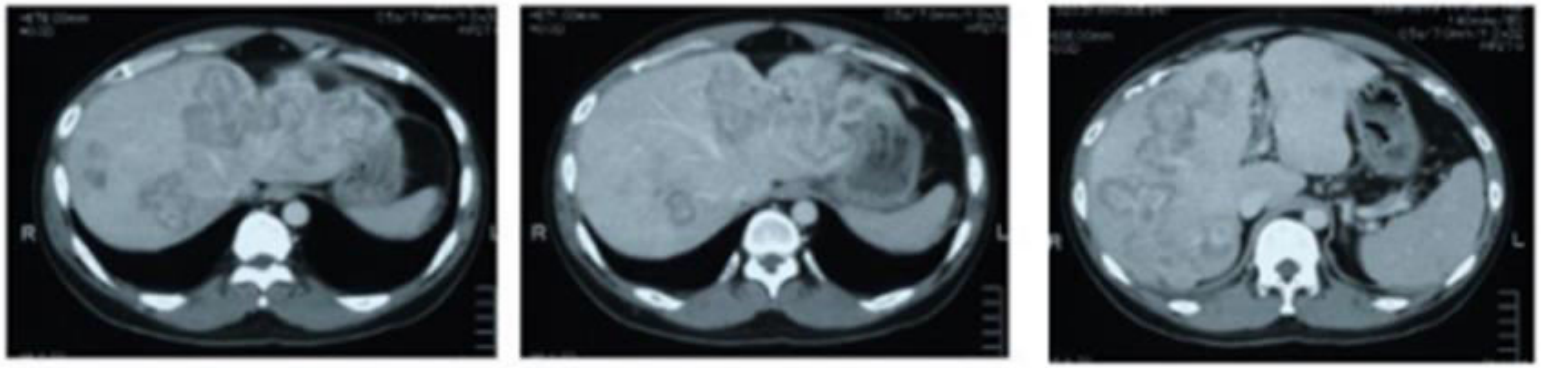
- Left sided lesions are more likely to present with symptoms of obstructions such as constipation or decrease stool caliber (Pencil-shaped stool) >>change in bowel habits

- Right sided lesions are more commonly present with bleeding , Macroscopic or Microscopic Or Iron deficiency anemia

- The reason that right-sided lesions do not cause obstruction is that the diameter of the right colon is greater than the left and the stools have increase water content.

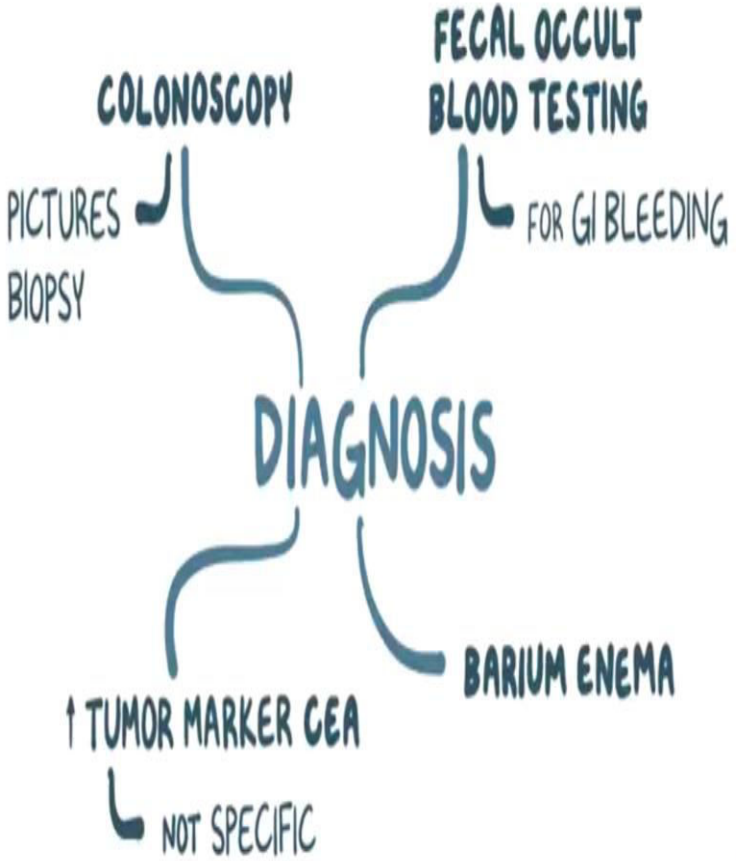
Investigations

- 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 abdomen is frequently performed before surgery.
- Preoperative blood tests should evaluate the patient's overall nutritional status and should include liver function tests (particularly alkaline phosphatase) and carcinoembryonic antigen (CEA) study as a baseline for follow up post op



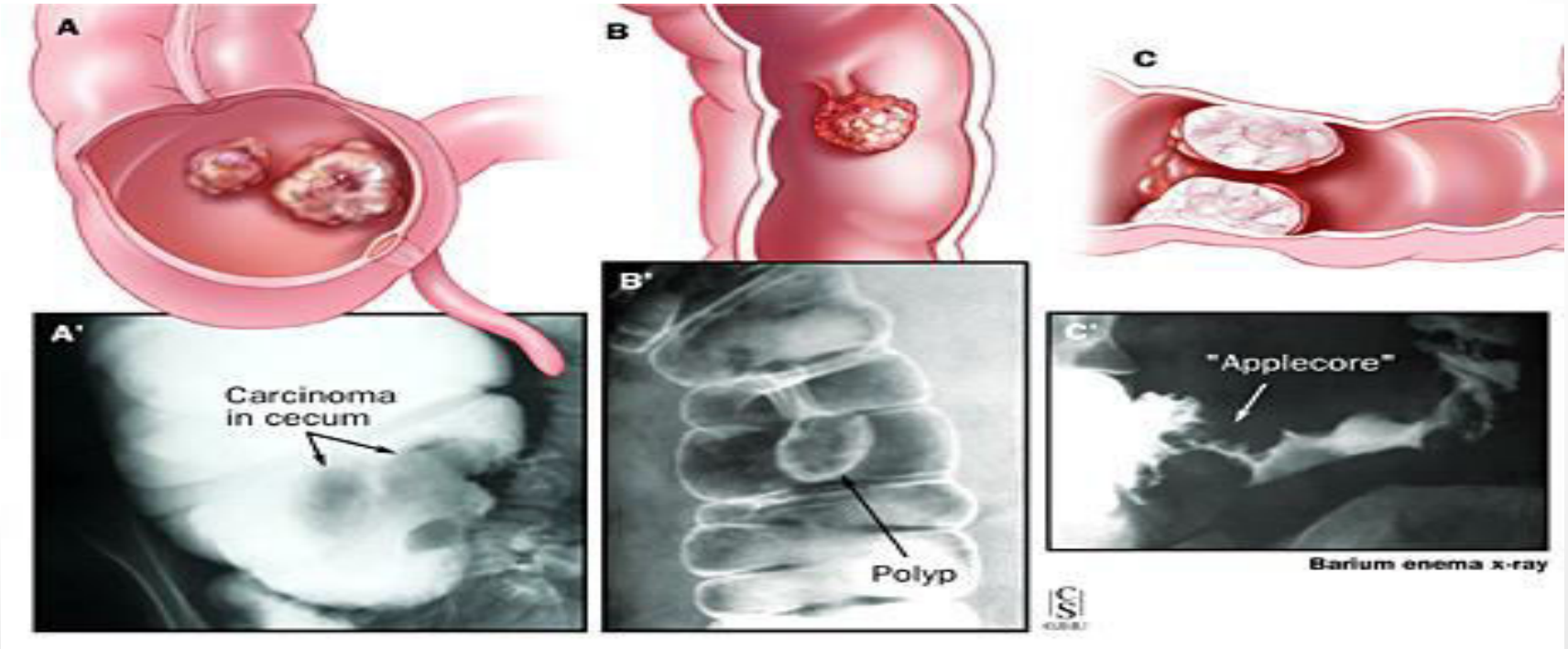
Investigations

- CBC (ANEMIA DUE TO IDA FROM BLOOD LOSS) , LFT (ALT,AST : ELEVATAED WITH LIVER METS)
- BUN+CREATININE : ASSES RENAL FUNCTION DUE TO COMPRESS MASS
- **Fecal occult Blood test**.
- Barium enema. (APPLE CORE SIGN)
- **CT colongraphy** (virtual colonography)
- Lower Endoscopy/ colonoscopy/ sigmoidoscopy
- Endorectal US for rectal Ca
- **CEA Tumor marker**. “Follow up & recurrence”
- CT OF THE CHEST , ABDOMEN , PELVIS (LYMPH NODE INVOLVEMTN OF ANY METS) , MRI (TO LOOK FOR LIVER LESIONS), Chest X-ray, PET scan “metastasis”



Barium Enema

- **filling defects**
- **apple core appearance**



كلية الطب البشري
Faculty of Medicine

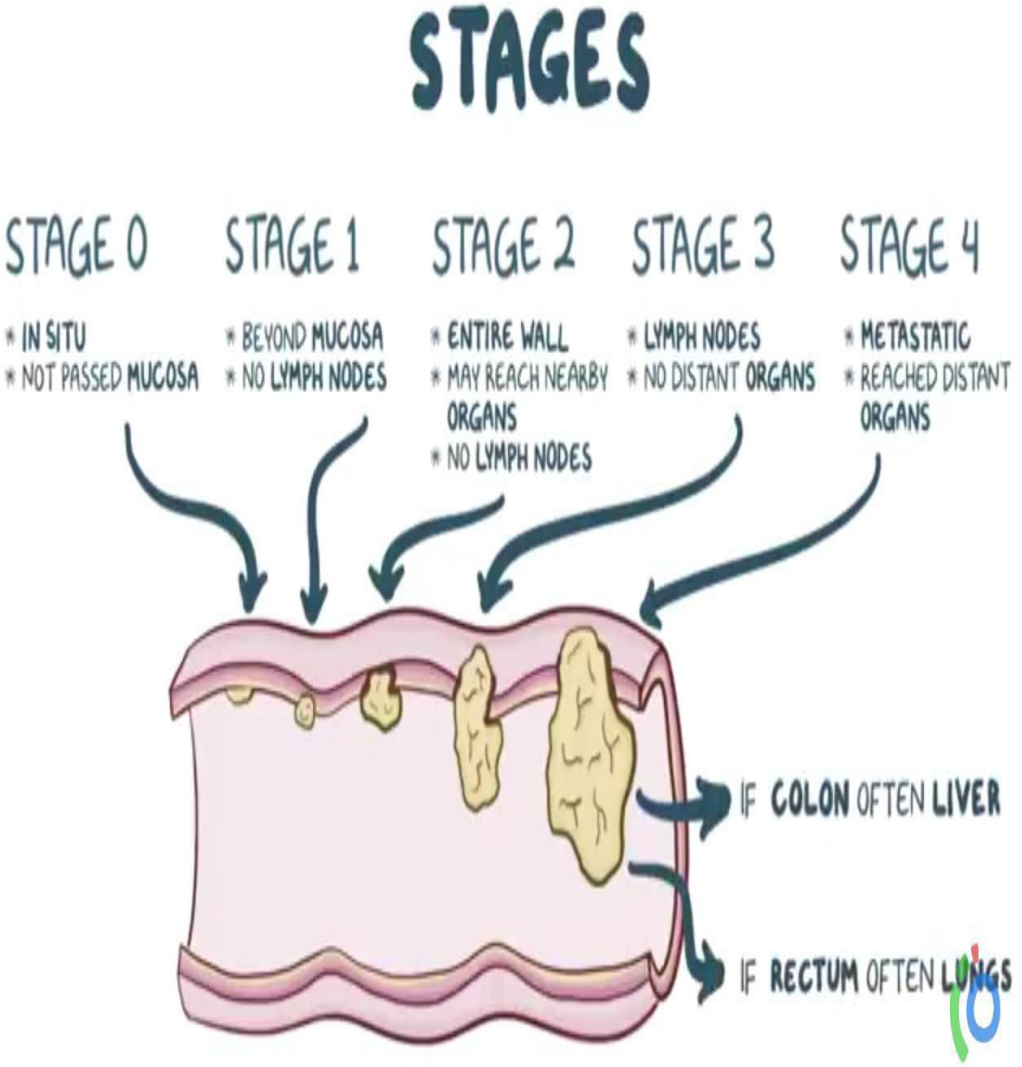
Staging and Treatment of Colorectal carcinoma

Staging

- **TNM**

  - Dukes'

  - modified Dukes



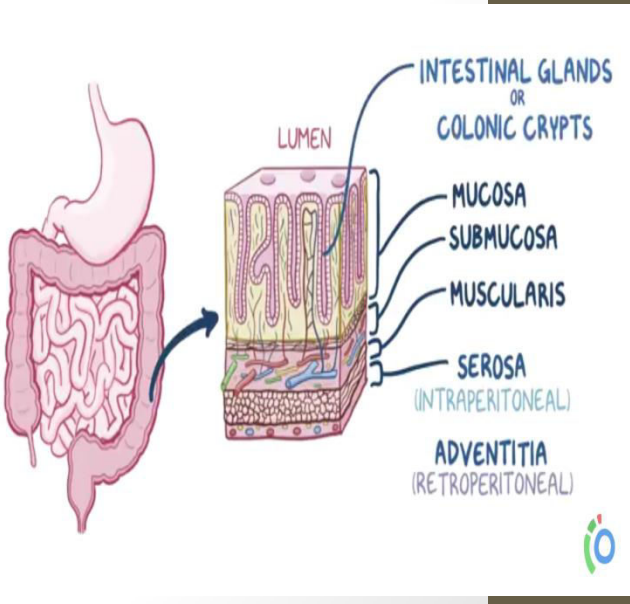
TNM Staging

Primary tumor (T)

- **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 the muscularis propria into the subserosa or into**
- **nonperitonealized pericolic or perirectal tissues**
- **T4 - Tumor directly invades other organs or structures, and/or perforates visceral peritoneum**

- **Regional lymph nodes (N)**
- **NX - Regional lymph nodes cannot be assessed**
- **N0 - No regional lymph-node metastasis**
- **N1 - Metastasis in 1 to 3 regional lymph nodes**
- **N2 - Metastasis in 4 or more regional lymph nodes**

- **Distant metastasis (M)**
- **MX - Distant metastasis cannot be assessed**
- **M0 - No distant**
- **M1 - Distant metastasis**



Dukes staging

- A: tumor within the wall.
- B: tumor through the whole wall.
- C: involvement of lymph nodes (C1,C2)
- D: metastatic spread.

Summary box 69.12

Dukes' staging for colorectal cancer

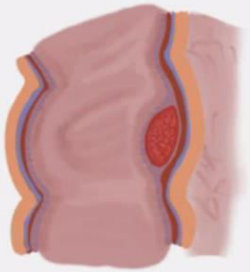
- A, Invasion of but not breaching the muscularis propria
- B, Breaching the muscularis propria but not involving lymph nodes
- C, Lymph nodes involved

Dukes himself never described a stage D, but this is often used to describe metastatic disease

STAGING: TUMOR-NODE-METASTASIS (TNM) SYSTEM

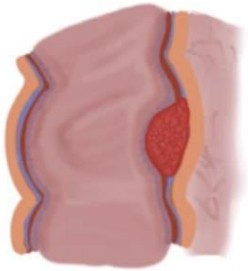
STAGE 0

- carcinoma in situ
- hasn't grown beyond the mucosa



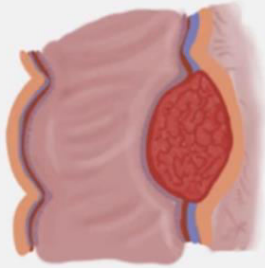
STAGE 1

- tumor has grown beyond the mucosa
- not spread to lymph nodes or distant organs



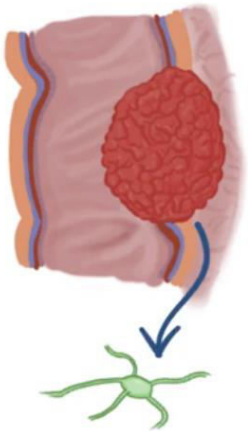
STAGE 2

- may have reached nearby organs/tissues
- not spread to lymph nodes or distant organs



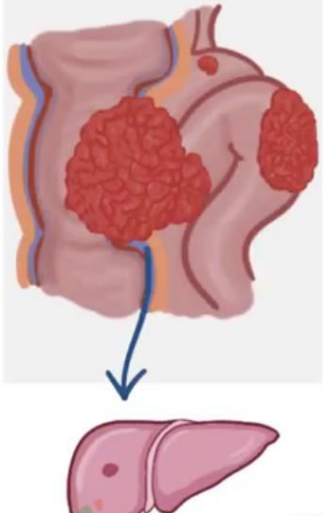
STAGE 3

- tumor has spread to lymph nodes
- hasn't spread to distant organs



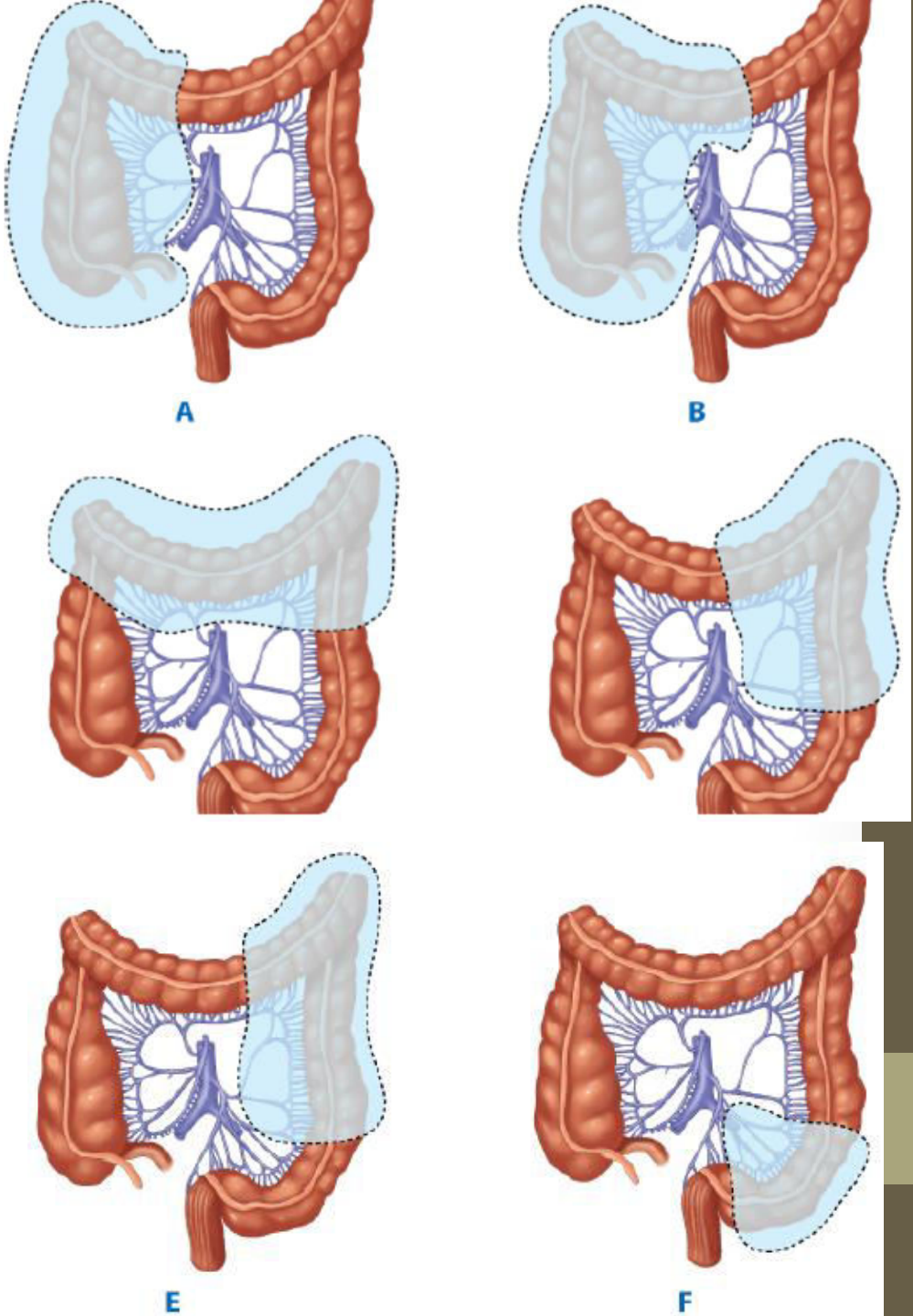
STAGE 4

- metastatic



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.



Treatment

- Removal of the lymphatics that drain the tumor region should be part of the operation because nodal involvement is present in more than 30% of specimens.
- Colorectal cancer may also spread **hematogenously**, **intraluminally**, or by **direct extension** or **peritoneal seeding** (Blumer's shelf on rectal examination).
- The most common organ involved in distant colorectal metastases is the liver followed by lung.

Treatment

- The use of adjuvant therapies in the treatment of colon and rectal carcinoma generated considerable research
- 5-FU used in combination with levamisole or leucovorin lowers the mortality rate in patients with Stage III tumors.

Treatment

- 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.

Treatment

- Because of their significant rate of recurrence (20%) despite the most radical surgical procedure, rectal tumors that have completely penetrated the rectal wall, with or without lymph node metastases, are treated additionally by radiation combined with 5-FU.
- Neoadjuvant chemoradiotherapy is advised for rectal cancer especially in the presence of lymph nodes.

Treatment

- Endorectal ultrasound or MRI is used to stage the depth of penetration of the tumor in the rectal wall.
- Preoperative chemoradiation (neoadjuvant therapy) is generally accepted as the best time to give therapy. This allows shrinkage of the tumor leading to a more complete resection and reduces the complications from postoperative radiation.
- Occasionally, it also causes enough tumor shrinkage in a very low rectal cancer to allow sphincter preservation.

Treatment

- A number of new biologic agents show promise in achieving even better results especially with hepatic metastases (bevacizumab, cetuximab).

Prognosis

- **The most important prognostic variable is lymph node involvement.**
- Other prognostic factors (e.g., tumor markers, size of the lesion and depth of invasion)

Follow up

- A frequently used approach is a 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.

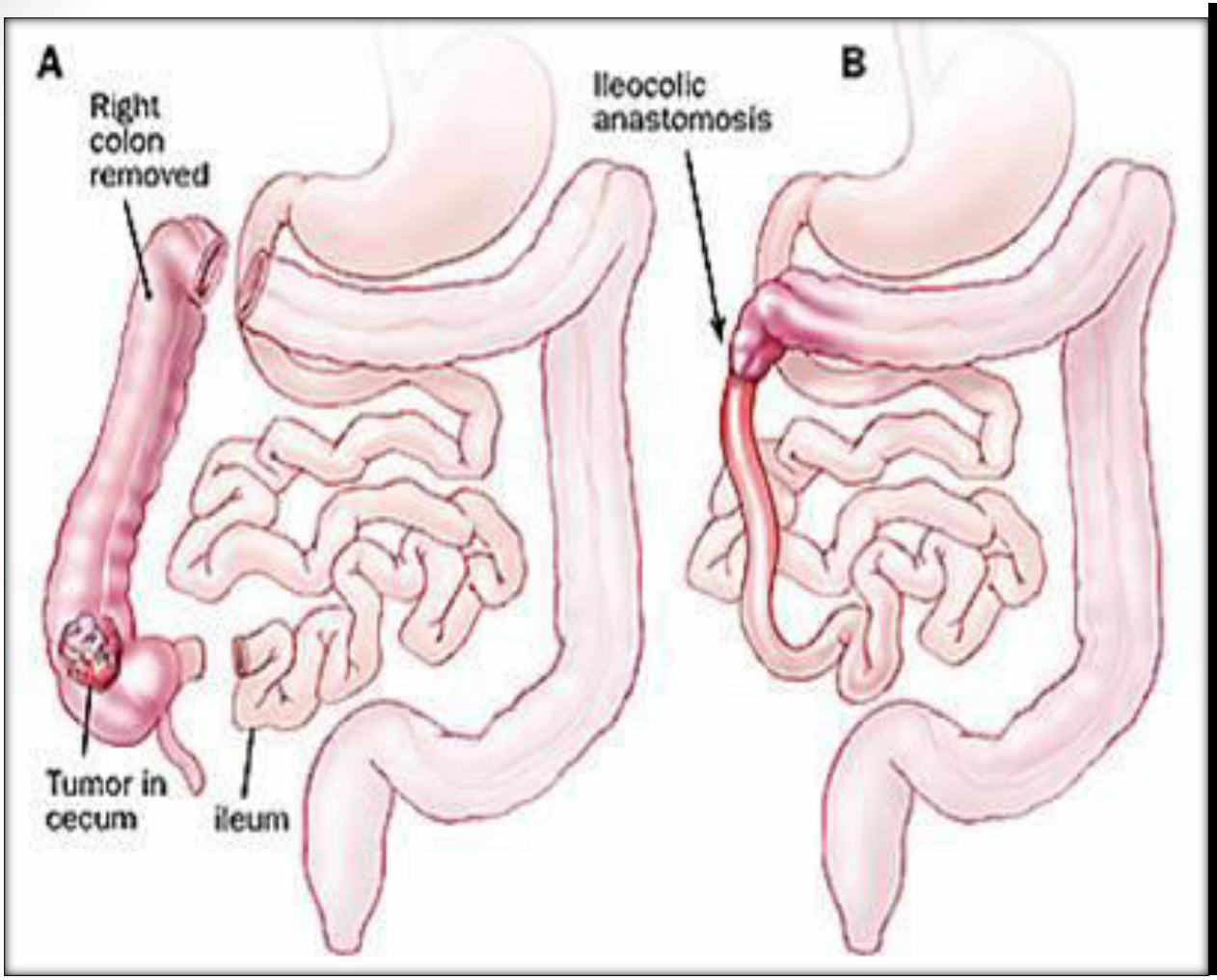
Follow up

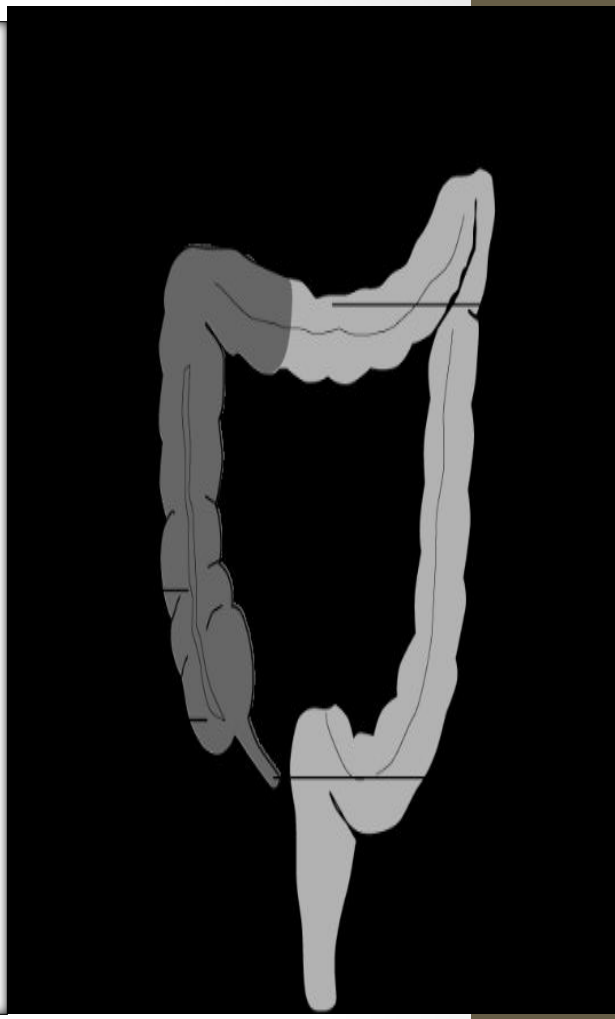
- The use of CEA is well established, with recurrence suggested not only by the absolute level of this antigen, but also by a progressive rise.
- A progressive rise mandates a complete evaluation of the patient, colonoscopy, including CT of the chest, abdomen, and pelvis.

Surgery

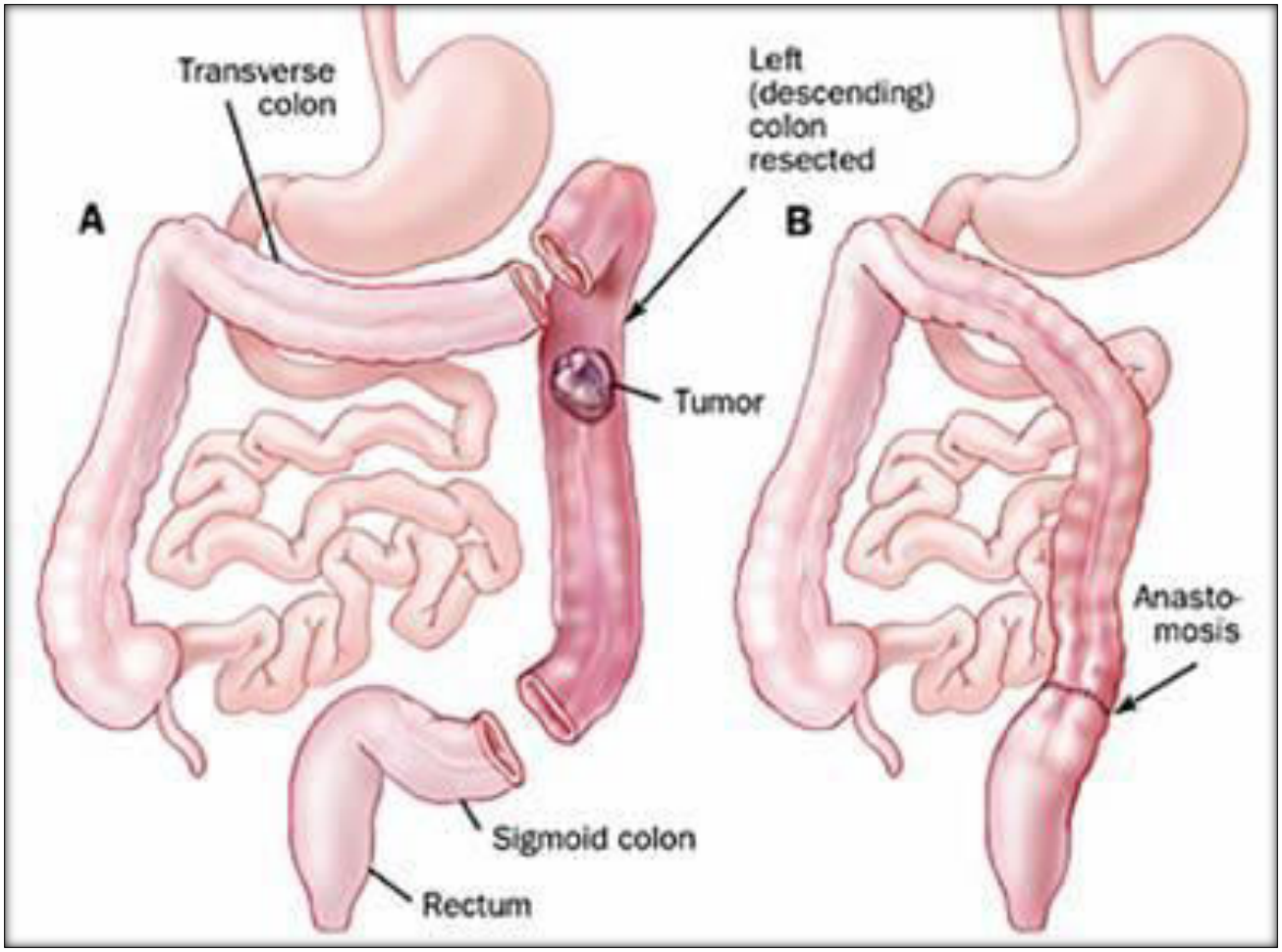
The primary treatment.▪
Curative or palliative.▪
Resection & re- anastomosis as one ▪ stage.
Stoma▪

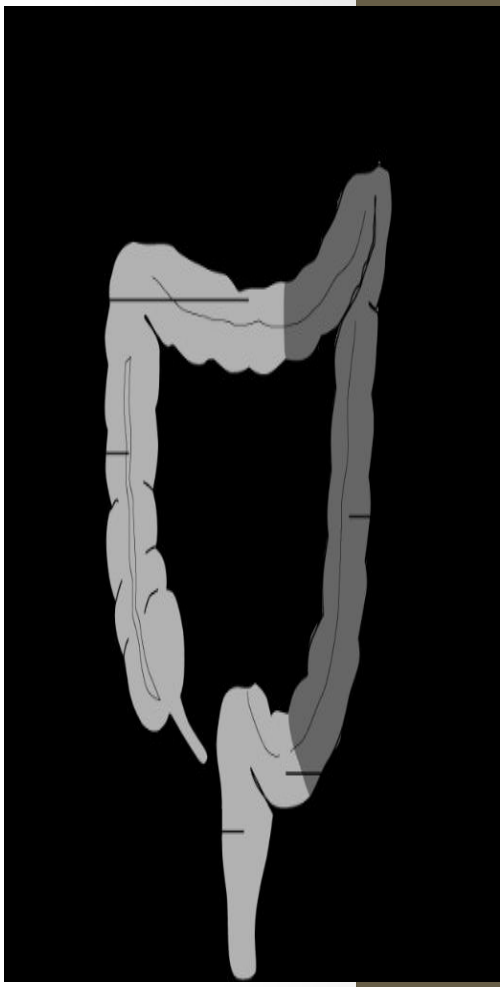
- **Types of surgeries**
- Right hemicolectomy
- Left hemicolectomy
- Total colectomy
- sigmoidectomy
- Anterior resection (removal of the rectum)
- Abdominoperineal resection (removal of the rectum and the anal canal)
- Put colostomy (permanent or temporary)
- Put ileostomy
- Put any other stomy



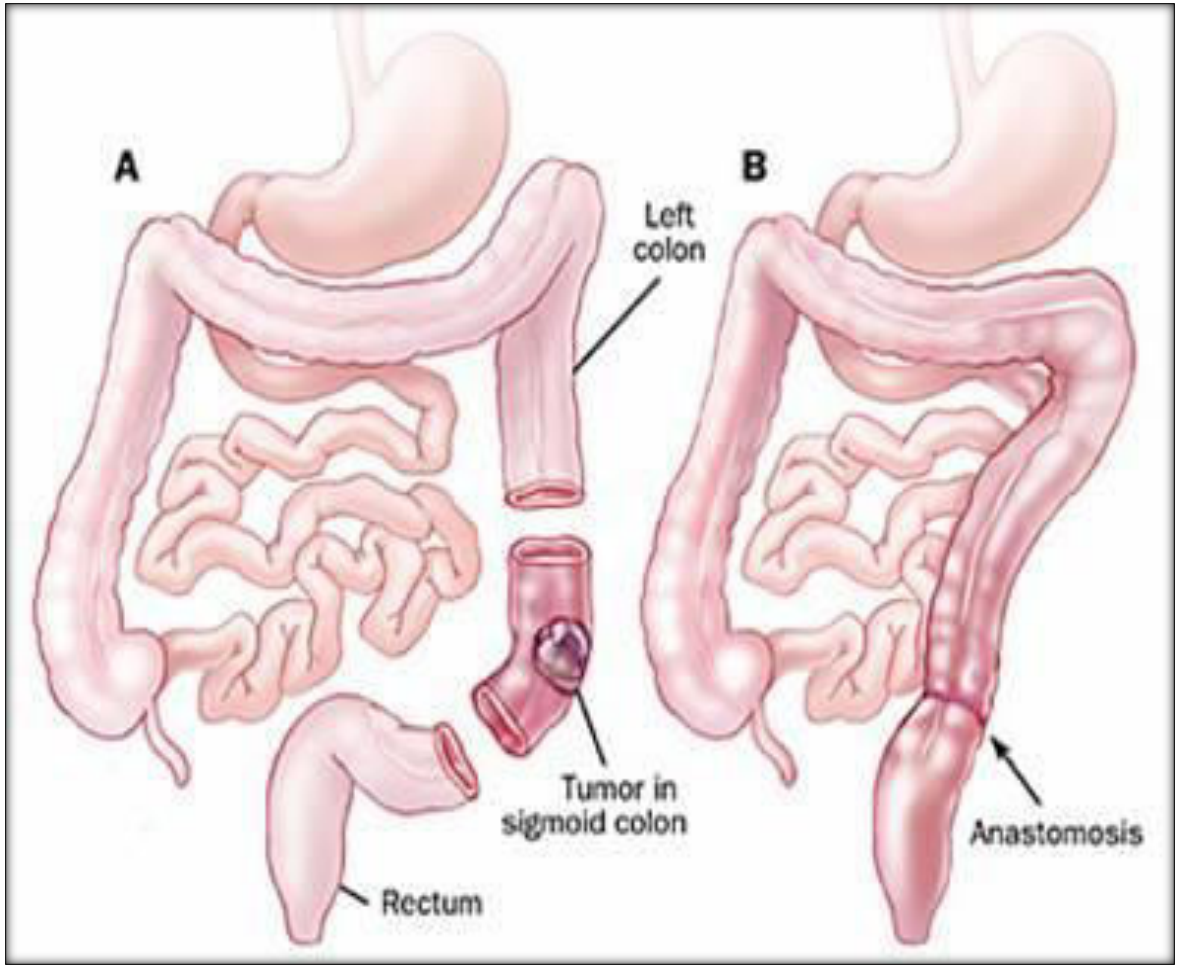


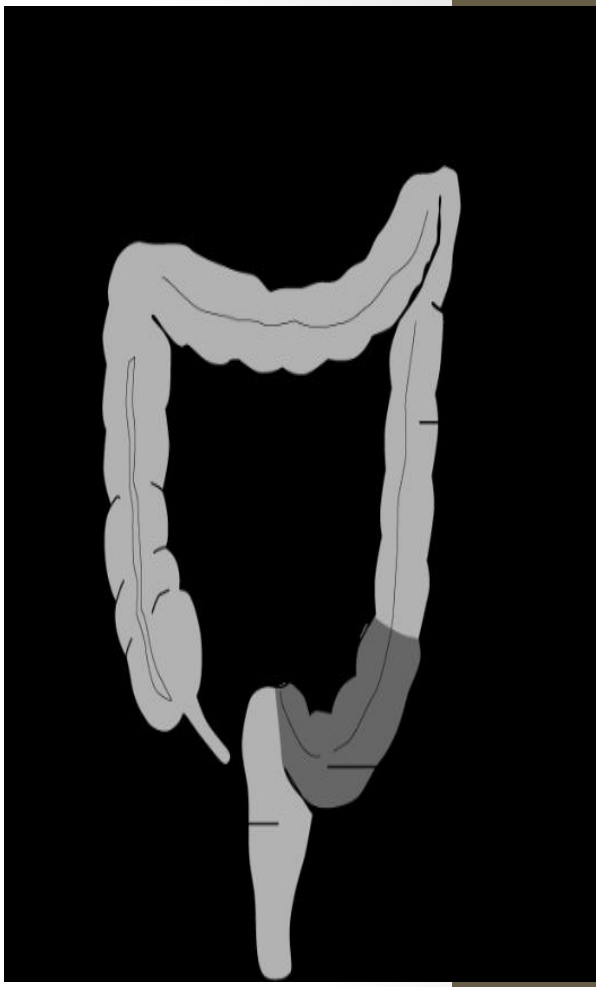
Right Hemicolectomy and ileocolic anastomosis
for Cecal / Right Colon Cancer



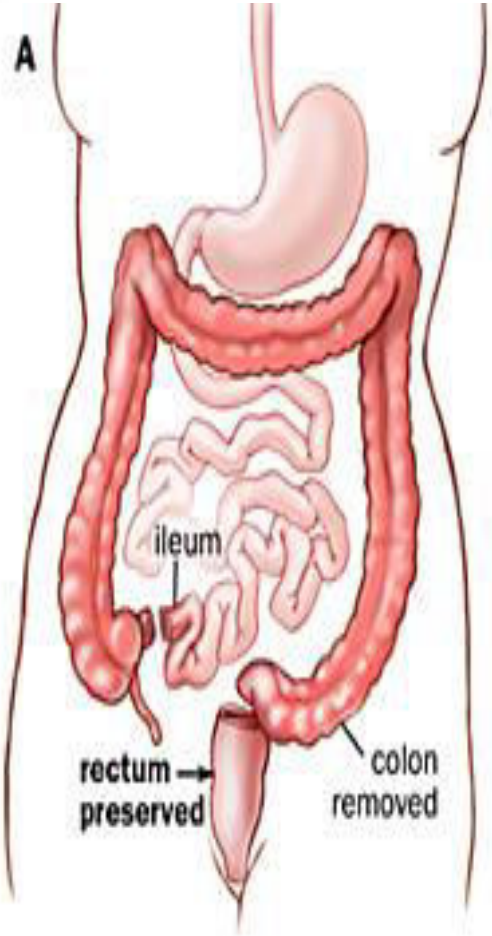


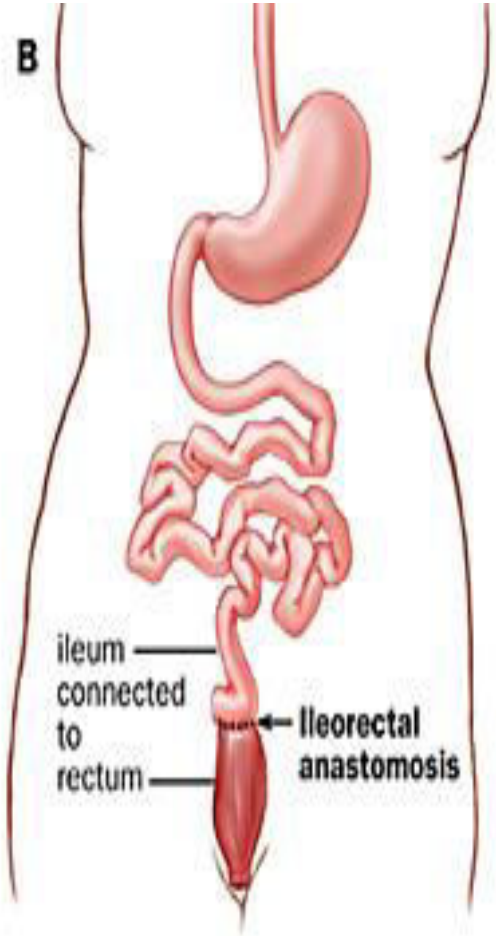
Left Hemicolectomy and anastomosis of colon and rectum for Left Colon Cancer

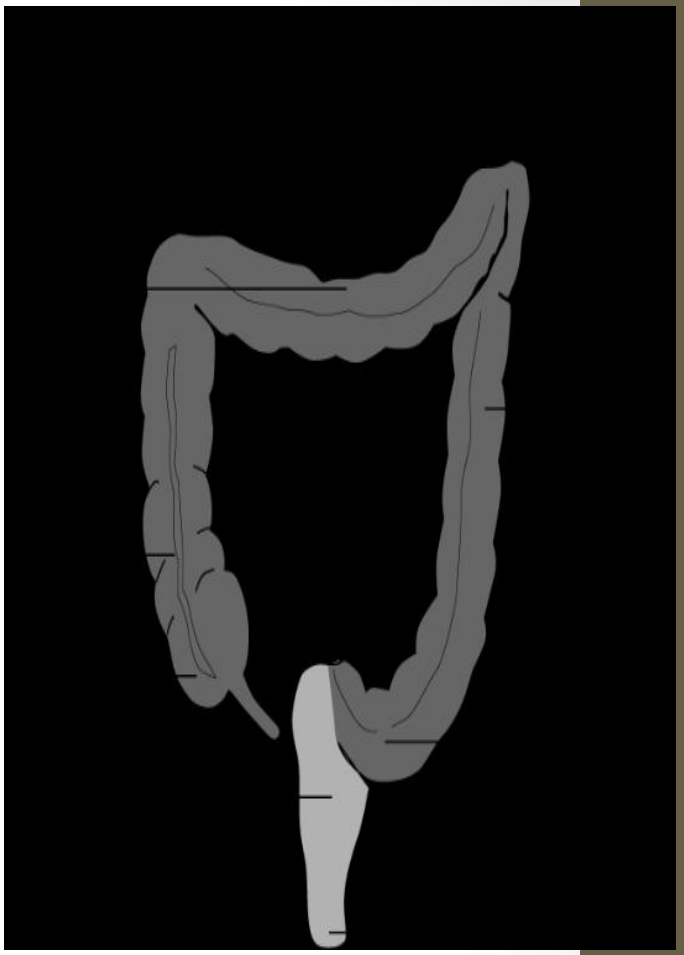




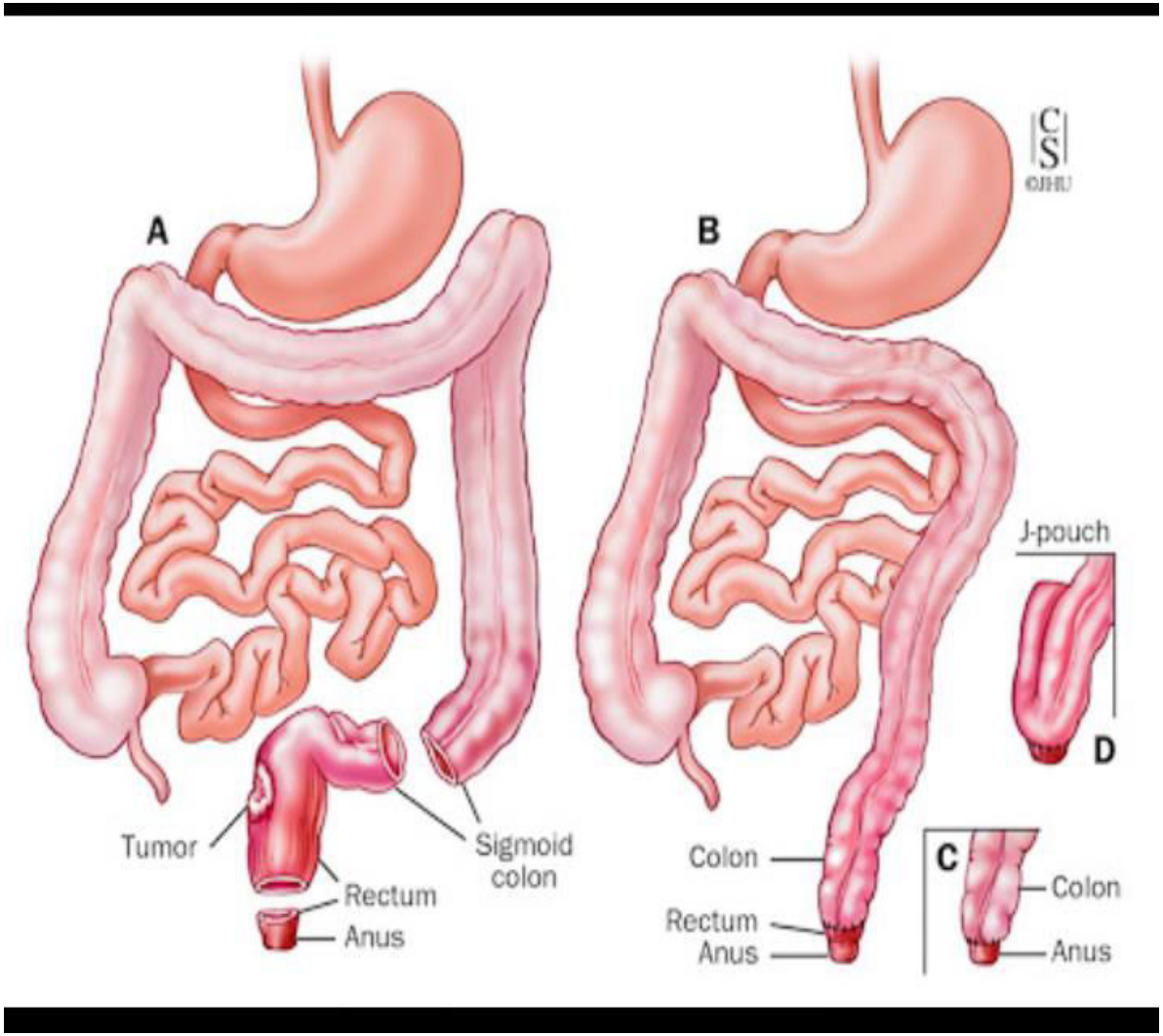
Sigmoid Colectomy and anastomosis of colon and rectum for Sigmoid Cancer

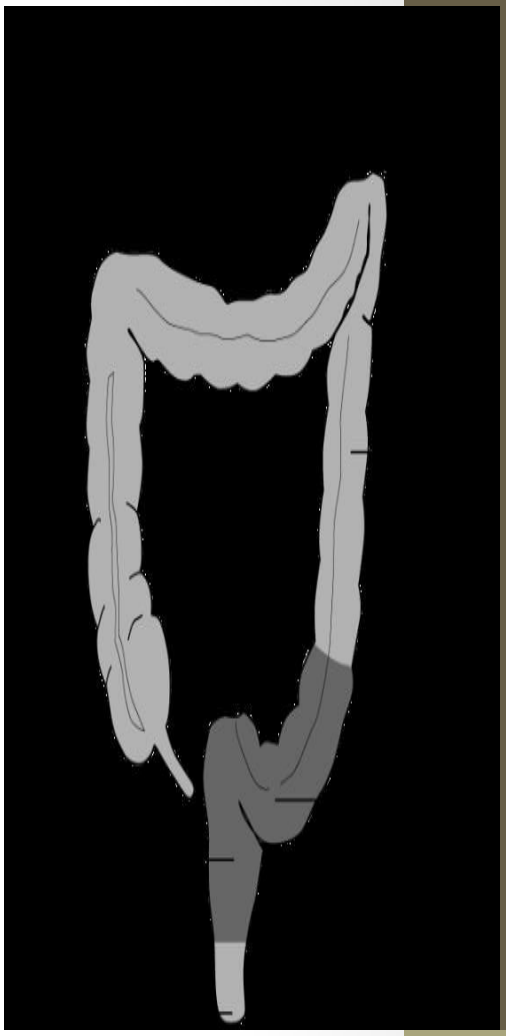




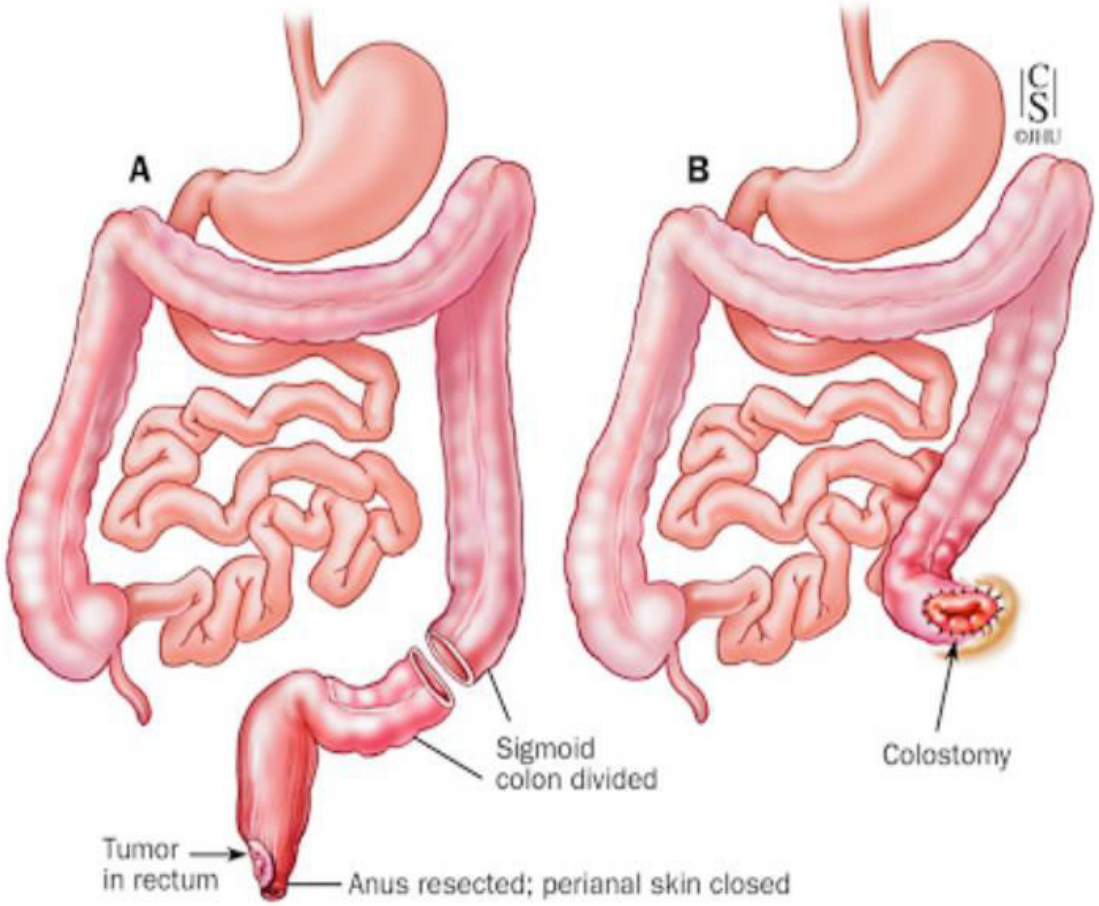


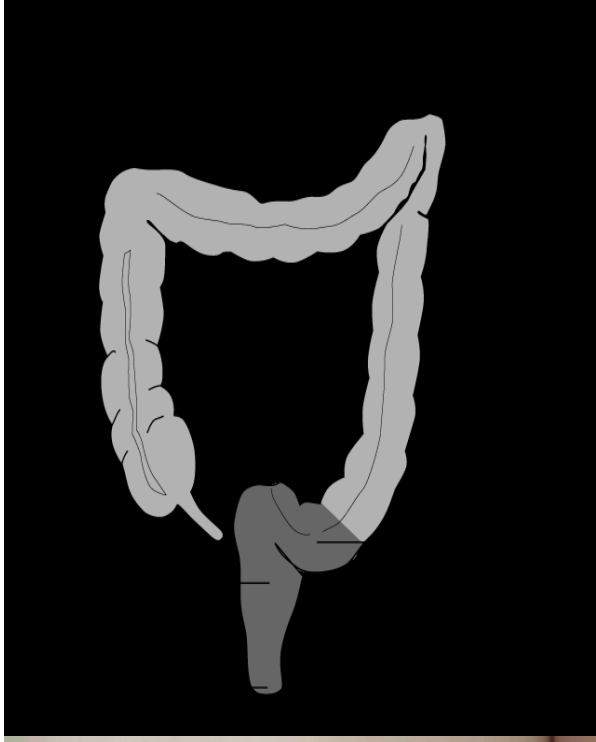
Total colectomy needs to be considered for multiple tumors

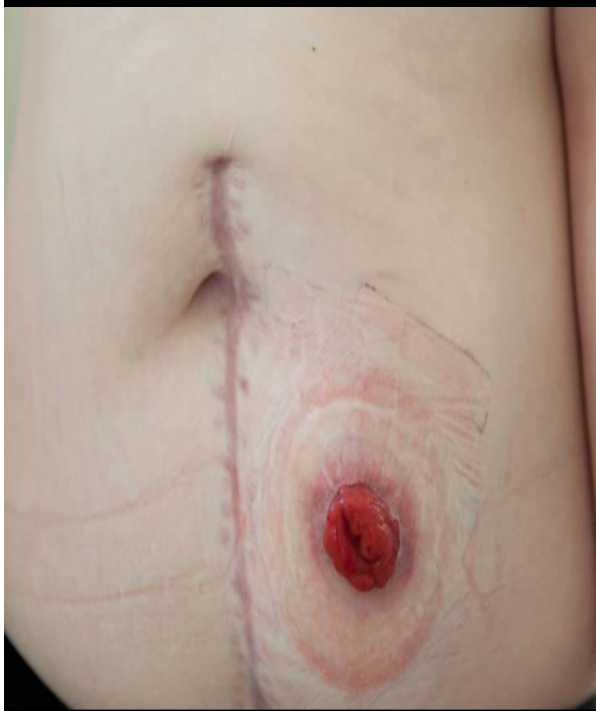




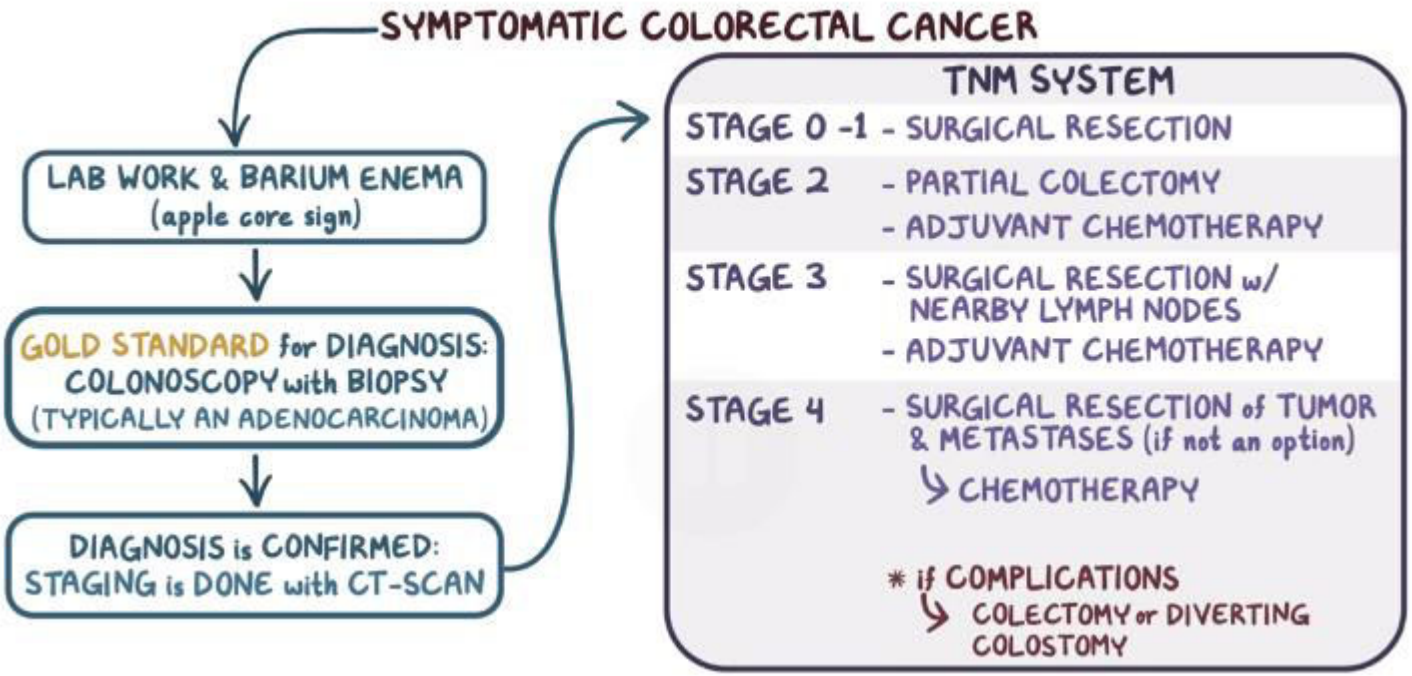
Anterior resection and anastomosis of colon and rectum
for upper and middle rectal tumors







Abdominoperineal resection – resection of anus rectum and part of segmoid colon and leave a permanent colostomy for lower rectal tumors


SYMPTOMATIC COLORECTAL CANCER
LAB WORK & BARIUM ENEMA
(apple core sign)
GOLD STANDARD for DIAGNOSIS:
COLONOSCOPY with BIOPSY
(TYPICALLY AN ADENOCARCINOMA)
DIAGNOSIS is CONFIRMED:
STAGING is DONE with CT-SCAN
TNM SYSTEM
STAGE 0 -1 - SURGICAL RESECTION
STAGE 2 - PARTIAL COLECTOMY
- ADJUVANT CHEMOTHERAPY
STAGE 3 - SURGICAL RESECTION w/
NEARBY LYMPH NODES
- ADJUVANT CHEMOTHERAPY
STAGE 4 - SURGICAL RESECTION of TUMOR
& METASTASES (if not an option)
↳ CHEMOTHERAPY
* if COMPLICATIONS
↳ COLECTOMY or DIVERTING
COLOSTOMY

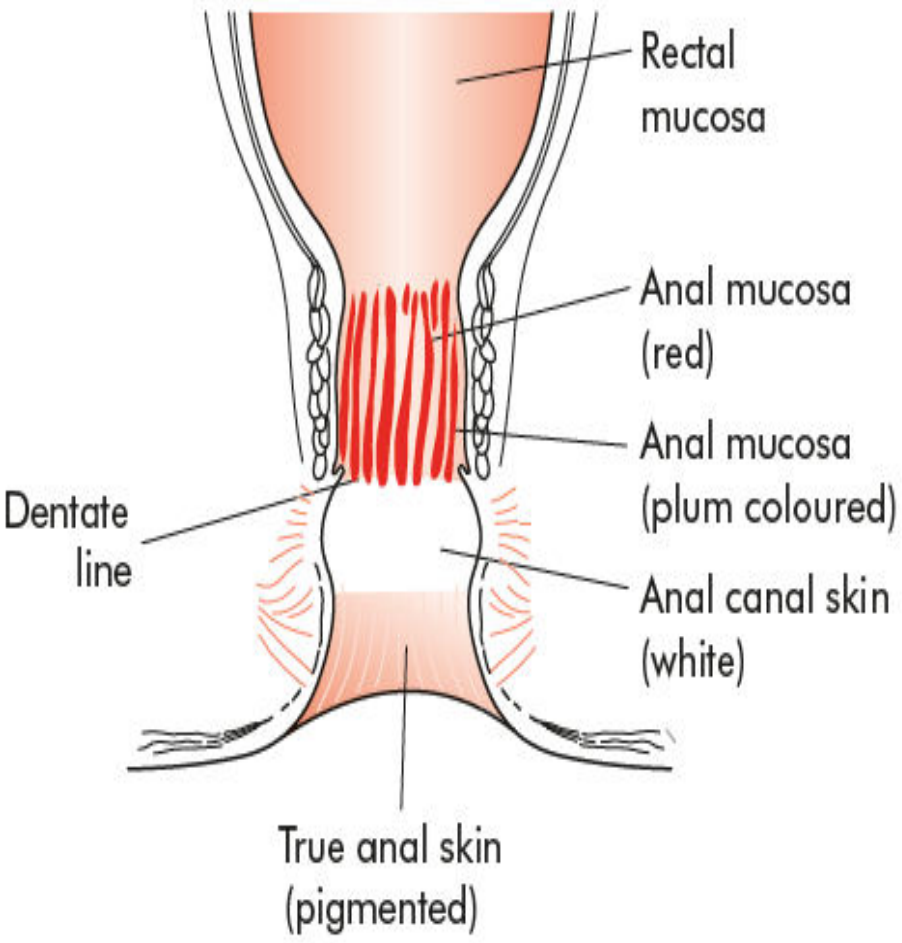
Anal Canal

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).



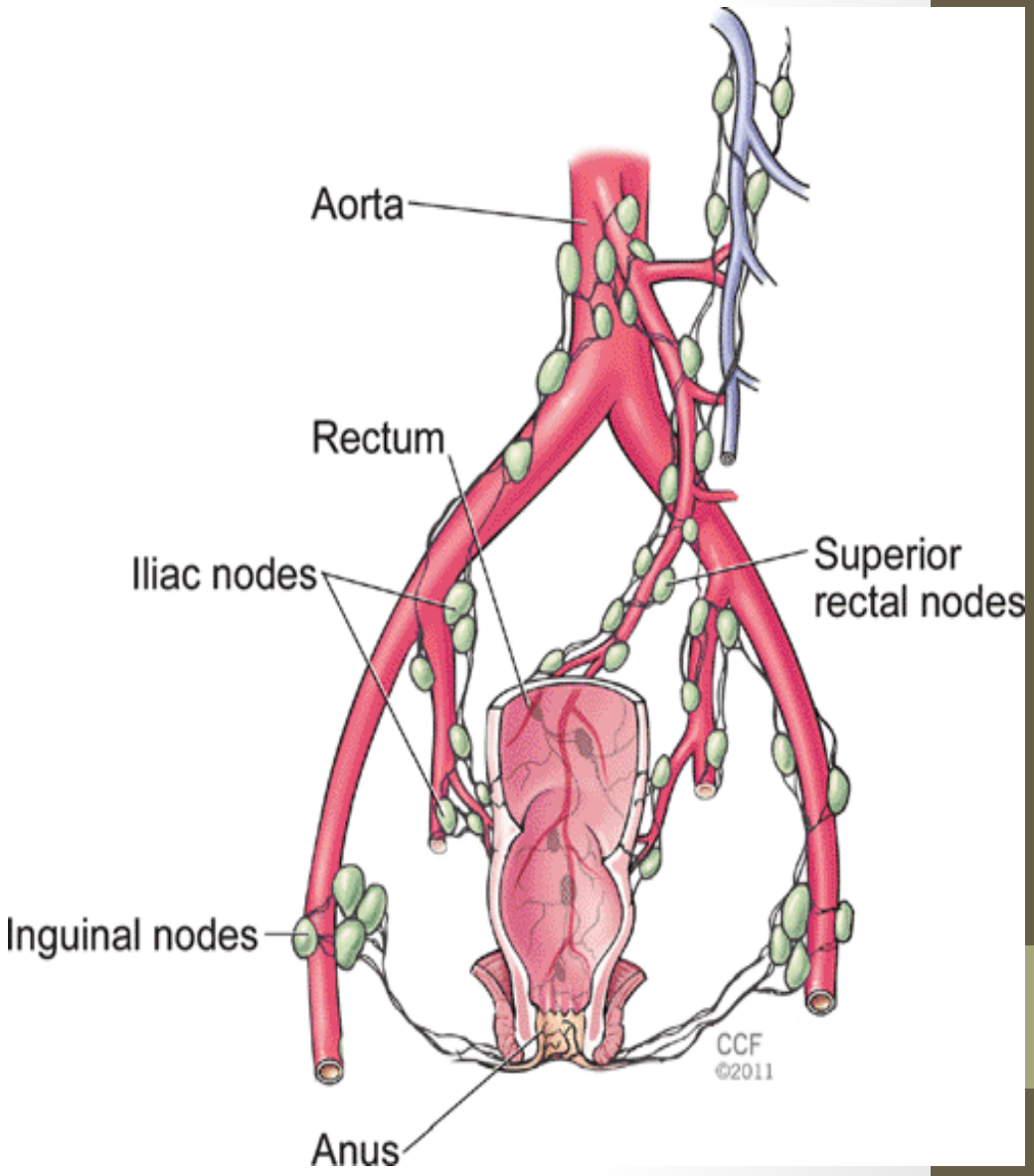
Anal Canal

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 crude incidence rate is 0.65
- Those arising below the dentate line are usually ***squamous***:

  Associated with human papilloma virus (HPV),

  More prevalent in patients with HIV infection

- Adenocarcinomas are the next most common (usually extension from above dentate line).
- Other tumours include melanoma (usually affects the anal verge), lymphoma and sarcoma.

Anal Cancer

- Lymphatic spread is to the inguinal lymph nodes
- Treatment is by chemoradiotherapy in the first instance
- Major ablative surgery (APR) is required if the above fails

Thank you..