

الجامعة السورية الخاصة  
كلية الطب البشري  
قسم الجراحة

الأمراض الجراحية الشائعة في الأمعاء الدقيقة و الغليظة  
Common small and large intestinal surgical diseases

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# Topics

- Bowel obstruction. انسداد الأمعاء
- Small bowel neoplasms.  
الآفات التنشؤية للأمعاء الدقيقة
- Meckle's diverticulum. رتج ميكل
- IBD. متلازمة الكولون المتهيج
- Colorectal cancer. أورام الكولون الخبيثة

# Intestinal Obstruction

- Intestinal obstruction exists when blockage prevents the normal flow of intestinal contents through the intestinal tract.

يحدث الانسداد لأسباب تمنع الجريان الطبيعي ضمن الأنبوب الهضمي

- Two types of processes can impede this flow.

أسباب إعاقة الجريان

**Mechanical.** ميكانيكية

**Functional.** وظيفية

# Intestinal Obstruction

- **Mechanical obstruction:** الإنسداد الميكانيكي  
An intraluminal obstruction ( الانسداد عبر اللمعة ) or a mural obstruction from pressure on the intestinal walls occurs.  
Examples are:
  - intussusception الإنغلاف
  - polypoid tumors and neoplasms البوليبيات السلسلة و الخبيثة
  - Stenosis تضيقات الأمعاء
  - Adhesions الالتصاقات
  - Hernias الفتوق
  - abscesses. الخراجات

# Intestinal Obstruction

- **Functional obstruction:** الإنسداد الوظيفي

The intestinal musculature cannot propel the contents along the bowel.

عندما تعجز الحركات الحوية عن دفع محتوى الأمعاء باتجاه النهاية البعيدة

Examples are:

Amyloidosis

الداء النشواني

Muscular dystrophy

الاعتلالات العضلية

Endocrine disorders such as diabetes mellitus

الأمراض الغدية و الاستقلابية كالداء السكري

Neurologic disorders

الاعتلالات العصبية

# Intestinal Obstruction

The obstruction can be partial or complete.

Its severity depends on:

يمكن للإنسداد أن يكون جزئياً أو كاملاً و تعتمد الخطورة على :

The region of bowel affected

المنطقة المصابة من السبيل الهضمي

The degree to which the lumen is occluded

درجة أو نسبة الانسداد

The degree to which the vascular supply to the bowel wall is disturbed.

درجة تأثر التروية الدموية للجزء المصاب

# Intestinal Obstruction

Most bowel obstructions occur in the small intestine

غالبية الانسدادات تحدث في الأمعاء الدقيقة

**Adhesions** الالتصاقات are the most common cause of small bowel obstruction, followed by **hernias** الفتوق and **neoplasms** الأورام .

Other causes include **intussusception** الانغلاف ,

**volvulus** الانفتال (ie, twisting of the bowel),

and **paralytic ileus** الخذل المعدي المعوي .

About 15% of intestinal obstructions occur in the large bowel; most of these are found in the sigmoid colon

ما يقارب 15% من انسدادات الأمعاء يحدث في الأمعاء الغليظة و غالبيتها في السين الكولوني

# انسدادات الأمعاء الدقيقة

## SMALL-BOWEL OBSTRUCTION

### Epidemiology **المرضية**

The most frequently encountered surgical disorder.

≥75% is due to intra-abdominal adhesions.

تشكل الالتصاقات ضمن جوف البطن و بنسبة تصل لى 75% السبب الجراحي  
الغالب

Other Dx. should be considered:

التشخيصات الأخرى تشتمل على :

Hernias **الفتوق**

Crohn's **disease** **داء كرون**

Intestinal malrotation **عدم دوران الأمعاء**

Mid-gut volvulus **انفتال الجزء المتوسط من الأمعاء**



# انسدادات الأمعاء الدقيقة

## SMALL-BOWEL OBSTRUCTION

Causes can be divided into three categories:

يمكن تصنيف الأسباب إلى :

Extraluminal causes such as adhesions, hernias, carcinomas, and abscesses

أسباب ضاغطة خارج اللمعة المعوية في حالات الالتصاقات و الفتوق و الأورام الخبيثة و الخراجات .

Intrinsic to the bowel wall (e.g., primary tumors)

أسباب ضمن الجدار المعوي كما في الأورام البدئية

Intraluminal obstruction (e.g., gallstones, enteroliths, foreign bodies, and bezoars)

أسباب ضمن اللمعة المعوية ( الحصيات المرارية ، الحصيات البرازية ، الأجسام الأجنبية ، كتل الألياف النباتية و الأشعار )

# انسداد الأمعاء الدقيقة

## SMALL-BOWEL OBSTRUCTION

**PATHOPHYSIOLOGY:** الفيزيولوجيا المرضية

Obstruction onset آلية حدوث الانسداد

Gas and fluid accumulate within the intestinal lumen proximal to the site of obstruction.

يحدث تراكم في السوائل و الغازات ضمن لمعة الأمعاء الدقيقة في الجهة القريبة من منطقة الانسداد .

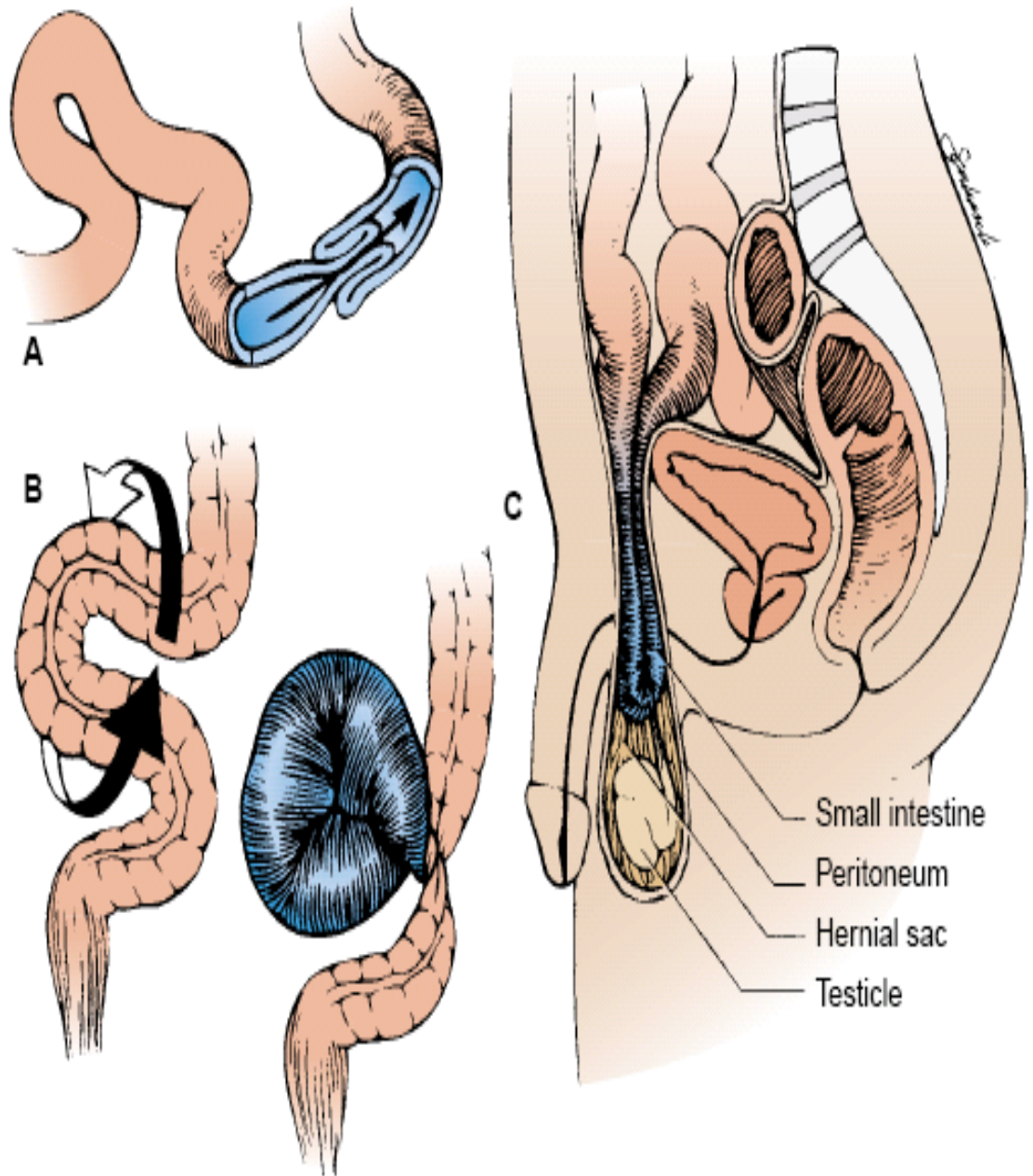
The bowel distends and intramural pressures rise.

توسع في العرى المعوية و ارتفاع في الضغط ضمن اللمعة

Microvascular perfusion to the intestine is impaired, leading to intestinal ischemia, and, ultimately, necrosis. ( strangulating bowel obstruction)

من الممكن أن تؤدي هذه الحالة إلى اضطراب في التروية الدموية ، يعقبها نقص في تروية الأمعاء ، و يمكن أن يؤدي ذلك إلى حدوث نخر بنقص التروية ( انسداد الأمعاء بسبب اختناق الأمعاء )

Progression to strangulation occurs quicker with **complete bowel obstruction** and more rapidly **with closed loop obstruction** which a segment of intestine is obstructed both proximally and distally (e.g., with volvulus).



**FIGURE 38-6** Three causes of intestinal obstruction. (A) Intussusception invagination or shortening of the colon caused by the movement of one segment of bowel into another. (B) Volvulus of the sigmoid colon; the twist is counterclockwise in most cases. Note the edematous bowel. (C) Hernia (inguinal). The sac of the hernia is a continuation of the peritoneum of the abdomen. The hernial contents are intestine, omentum, or other abdominal contents that pass through the hernial opening into the hernial sac.

# BOWEL OBSTRUCTION

## Clinical Presentation

Symptoms:

الأعراض

colicky abdominal pain

آلام بطنية ماغصة

Nausea

غثيان

Vomiting

أقياء لمحتويات المعدة و الأمعاء تختلف طبيعتها حسب مستوى الانسداد

obstipation

انقطاع البراز بحسب مستوى الانسداد

Continued passage of flatus and/or stool beyond 6–12 h after onset of symptoms is characteristic of partial rather than complete obstruction.

في حال استمرار التبرز و طرح الغازات فهذا يرجح وجود اسداد تحت التام

# BOWEL OBSTRUCTION

## Signs

## العلامات

abdominal distention

تطبل البطن

hyperactive bowel sounds. “borborygmi”

أصوات الأمعاء ناشطة

Features of strangulated obstruction include

Tachycardia

تسرع النبض

Localized abdominal tenderness

ألم بطني موضع

Fever

ترفع حروري

Marked leukocytosis

ارتفاع في عدد الكريات البيض

Acidosis

الحماض

# انسداد الأمعاء الدقيقة

## SMALL-BOWEL OBSTRUCTION

### Diagnosis

### التشخيص

The diagnostic evaluation should focus on the following goals:

Distinguishing **mechanical** obstruction from **ileus**

Determining the **etiology** of the obstruction

Discriminating **partial** from **complete** obstruction

Discriminating **simple** from **strangulating** obstruction.

Determining the **site** of obstruction.

# انسداد الأمعاء الدقيقة و الغليظة

## SMALL-BOWEL OBSTRUCTION

- **Diagnosis**

- **Careful history taking:**

- prior Hx of abdominal operations → ? presence of adhesions.
    - Hx of abdominal disorders (e.g., intraabdominal cancer or inflammatory bowel disease).

- **Careful examination:**

- a meticulous search for hernias (particularly in the inguinal and femoral regions) should be conducted.
    - The stool should be checked for gross or occult blood, the presence of which is suggestive of intestinal strangulation.

# LARGE BOWEL OBSTRUCTION :Pathophysiology

- As in small bowel obstruction
  - large bowel obstruction results in an accumulation of intestinal contents, fluid, and gas proximal to the obstruction.
  - Obstruction in the large bowel can lead to severe distention and perforation unless some gas and fluid can flow back through the ileal valve.
  - Large bowel obstruction, even if complete, may be undramatic if the blood supply to the colon is not disturbed.



# LARGE BOWEL OBSTRUCTION :Pathophysiology

- If the blood supply is cut off → intestinal strangulation and necrosis (ie, tissue death) occur; this condition is life threatening.
- dehydration occurs more slowly than in the small intestine because the colon can absorb its fluid contents and can distend to a size considerably beyond its normal full capacity.

# LARGE BOWEL OBSTRUCTION :Clinical Manifestations

- Large bowel obstruction differs clinically from small bowel obstruction in that the symptoms develop and progress relatively slowly.
- In patients with obstruction in the sigmoid colon or the rectum, constipation may be the only symptom for days. loops of large bowel become visibly outlined through the abdominal wall, and the patient has crampy lower abdominal pain.
- Finally, fecal vomiting develops. Symptoms of shock may occur.

# SMALL-BOWEL OBSTRUCTION

- **X-RAY SERIES:**
- Obstruction is usually confirmed with radiographic examination.
- Abdominal series consists of :
  - supine Abdominal X-ray
  - upright Abdominal X-ray
  - Upright Chest X-ray
- The finding most specific for small-bowel obstruction is the triad of
  - dilated small-bowel loops (>3 cm in diameter)
  - air–fluid levels seen on upright films
  - a paucity of air in the colon.
- False negative :
  - Proximal obstruction
  - The bowel lumen is filled with fluid but no gas.

# Assessment and Diagnostic Findings

- Diagnosis is based on symptoms and on x-ray studies.
- Abdominal x-ray studies (flat and upright) show a distended colon.
- Barium studies are contraindicated.



# Plain x-rays



# SMALL-BOWEL OBSTRUCTION

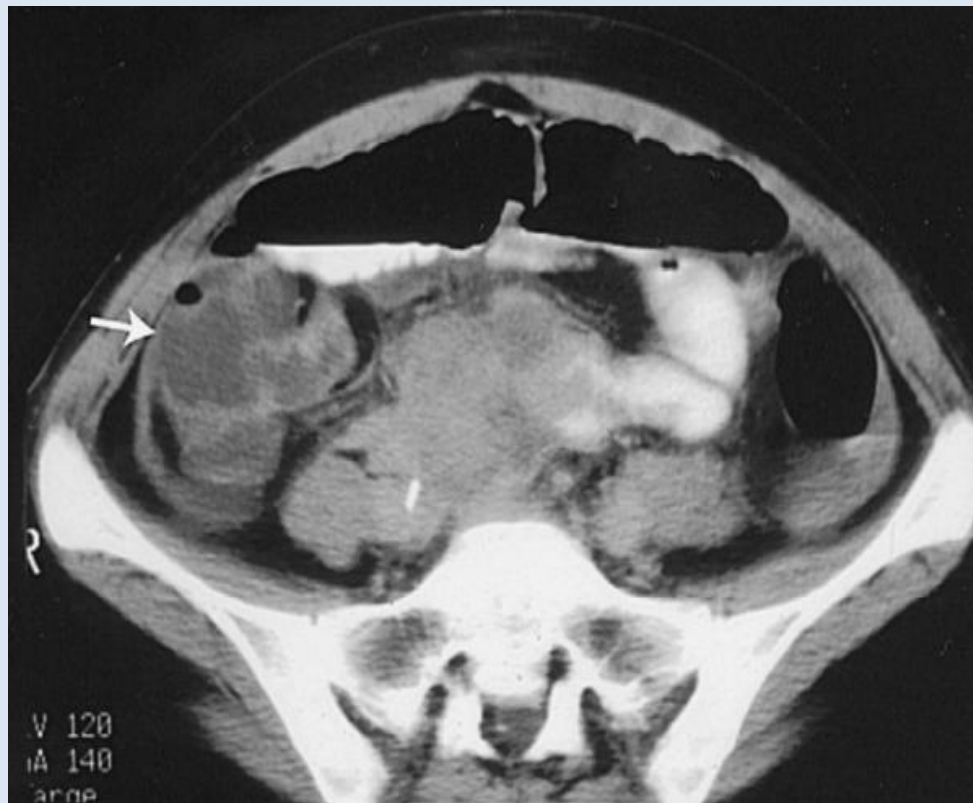
- **CT Abdomen:**
- Findings include:
  - A discrete **transition zone** with dilation of bowel proximally, decompression of bowel distally
  - intraluminal contrast that does not pass beyond the transition zone
  - Colon containing little gas or fluid.
  - **Strangulation** is suggested by:
    - Thickening of the bowel wall
    - Pneumatosis intestinalis (air in the bowel wall)
    - Portal venous gas
    - Mesenteric haziness
    - Poor uptake of intravenous contrast into the wall of the affected bowel.
  - **CT scanning** also offers a global evaluation of the abdomen and may therefore reveal the etiology of obstruction.

# SMALL-BOWEL OBSTRUCTION





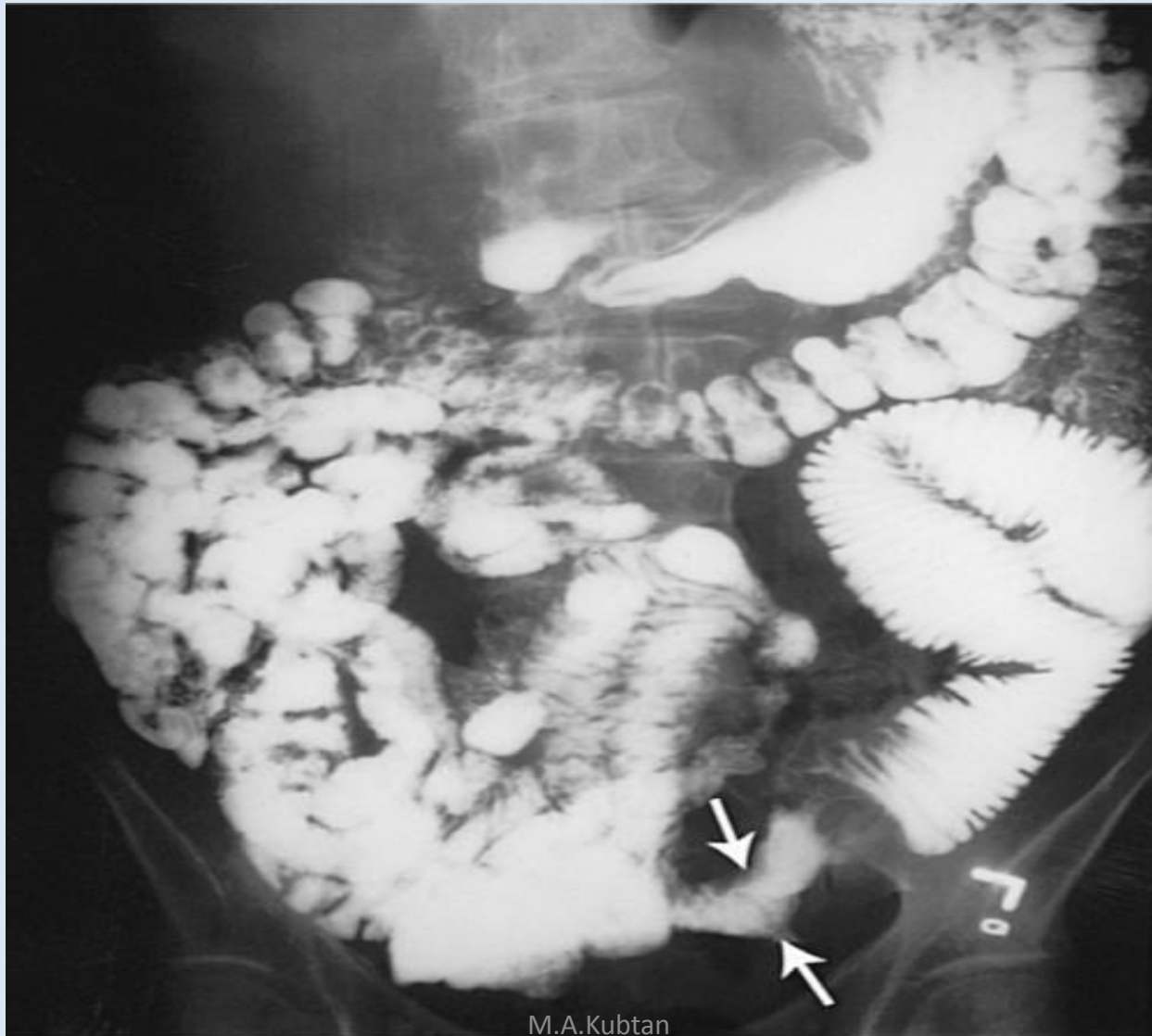




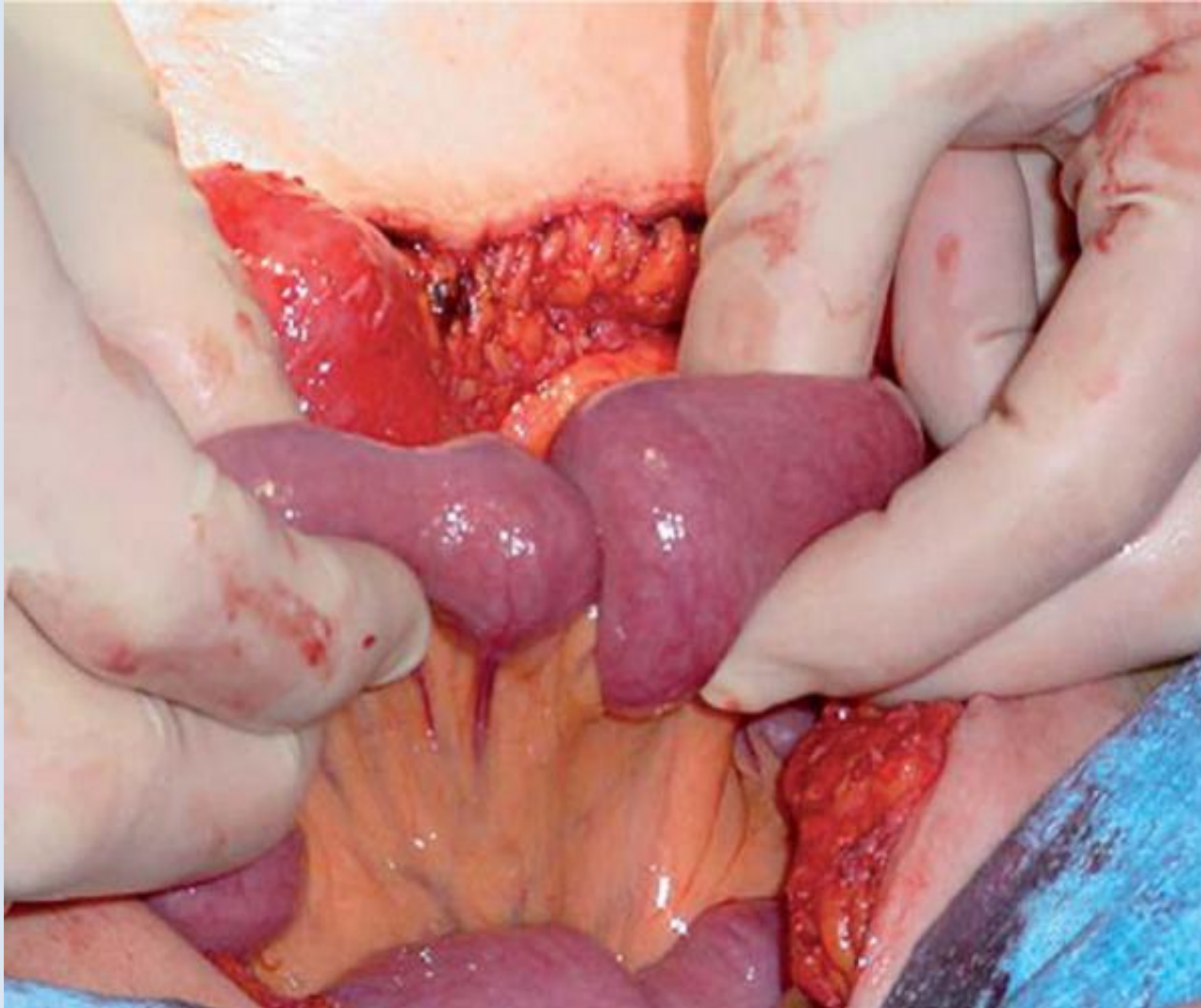


# SMALL-BOWEL OBSTRUCTION

- **SMALL-BOWEL SERIES (SMALL-BOWEL FOLLOW-THROUGH)**
  - can be helpful
  - contrast is swallowed or instilled into the stomach through a nasogastric tube.
  - Barium or water-soluble contrast agents (Gastrografin)
- **ENTEROCLYSIS**
  - 200– 250mL of barium followed by 1–2 L of a solution of methylcellulose in water is instilled into the proximal jejunum via a long nasoenteric catheter.
  - Enteroclysis is rarely performed in the acute setting
  - Offers greater sensitivity for lesions that may be causing partial small-bowel obstruction.







# BOWEL OBSTRUCTION

- **Therapy**
  - Fluid resuscitation.
  - A nasogastric (NG) tube to evacuate air and fluid from stomach.
  - An indwelling bladder catheter to monitor urine output.
  - Central venous or pulmonary artery catheter monitoring may be necessary
  - Broad-spectrum antibiotics
  - The standard therapy for bowel obstruction is expeditious surgery with the exception of specific situations

# MESENTERIC ISCHEMIA

- Therapy
- For embolus or thrombus-induced acute mesenteric ischemia, the standard treatment is surgical revascularization
  - embolectomy/thrombectomy/mesenteric bypass
  - Emergent laparotomy and resection, if signs of peritonitis develop
  - thrombolysis, using agents such as streptokinase, urokinase, or recombinant tissue plasminogen activator, is an alternative therapeutic option in sick, unstable pt.
- The standard treatment of NOMI is selective infusion of a vasodilator, most commonly papaverine hydrochloride, into the superior mesenteric artery.
- Anticoagulation:
  - Heparin administration is associated with reductions in mortality and recurrence rates, and should be initiated as soon as the diagnosis is made.
  - Most patients should be maintained on warfarin to achieve chronic anticoagulation for 6–12 months.



# MESENTERIC ISCHEMIA

- Outcomes
  - Mortality rates among patients with acute arterial mesenteric ischemia range from 59–93%.
  - Mortality rates among patients with acute mesenteric venous thrombosis range from 20–50%.
  - Perioperative mortality rates associated with surgical therapy for chronic mesenteric ischemia range from 0–16%

# Take Home Points

- Careful history (pain, other GI symptoms)
- Remember DDx in **broad** categories
- Narrow DDx based on hx, exam, labs, imaging
- Always perform ABC, Resuscitate before Dx
- If patient's sick or "toxic", get to OR (surgical emergency)
  - Ideally, resuscitate patients before going to the OR
- Don't forget GYN/medical causes, special situations
- For acute abdomen, think of these commonly (below)

Perf DU	Appendicitis +/- perforation	Diverticulitis +/- perforation	Bowel obstruction
Cholecystitis	Ischemic or perf bowel	Ruptured aneurysm	Acute pancreatitis
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# Inflammatory Bowel Disease

- **Disorders:** ulcerative colitis and Crohn's disease
- **Incidence:** 10,000 new cases per year in U.S.
- **Age at onset:** 15 to 40 years old
- **Etiology:** unknown, some familial
- **Pathophysiology**
  - **Ulcerative colitis:** inflammation of colon originates in rectum. Erosion, ulcers, abscesses, and necrosis result.
  - **Crohn's disease:** recurrent inflammatory process in any portion of GI tract. Bowel wall thickened and rigid, lumen narrows, fissures form.

# Crohn's Disease

# Definition

Chronic , transmural, inflammatory condition, which can involve the entire alimentary tract from mouth to anus (usually discontinuously) with involvement of extraintestinal tissue.



# Historical Background

- 1932-Crohn, Ginzburg, Oppenheimer  
“a disease of the terminal ileum, affecting mainly young adults, CHX by a subacute or chronic necrotizing & cicatrizing inflammation
  - They noted that the disease process lead to multiple fistulas.
  - They call it regional ileitis “believed that it involved only the terminal ileum”.
- 1960-Lockhart, Mummery & Morson  
First to report C.D. of the large intestine.

# Incidence and Prevalence of Crohn's Disease in Various Geographic Regions

<b>Author(s)</b>	<b>Country</b>	<b>Annual Incidence/ 100,000</b>	<b>Prevalence/ 100,000</b>
Garland et al. (1981)	Unites states (15 areas)	3.4 – 4.95	
Binder et al. (1982)	Denmark	2.70	34.0
Gollop et al. (1988)	Rochester, Minn., U.S.A.	4.00	
Haug et al. (1989)	Western Norway	5.30	
Stowe et al. (1990)	Rochester, N.Y.,U.S.A	5.00	
Probert et al. (1993)	England		
	Europeans		75.8
	South Asians		33.2
Mate-Jimenz et al. (1994)	Spain	1.61	19.8
	Urban	1.87	
	Rural	0.86	
Odes et al. (1994)	Israel	4.20	50.6
	Asian-African born Jews	4.60	55.0
	European-American born Jews	3.90	58.7
	Bedouin Arabs		8.2
Tsianos et al. (1994)	Greece	0.30	
Anseline (1995)	Australia	2.10	
Lindgren et al. (1996)	Sweden		
Manousos et al. (1996)	Crete, Greece	3.00	34.0
Moum et al. (1996)	Southeastern Norway	5.80	94.0
Tragnone et al. (1996)	Italy	2.30	
Hanauer and Meyers (1997)	United States		

# Epidemiology

- Precise incidence is difficult to determine.
- Russell & Stockbrugger noted rapid increase in freq. of C.D. occurring between 1965 – 1980.
- World wide prevalence is estimated to be 10 –70 cases/100,000 population with incidence of 0.5 – 6.3 cases /100,000 pop./year.
- Highest rates are reported in Scandinavian countries and Scotland followed by England and north America. But are decreased in central and southern Europe.



# Age & Gender

- Bimodal age distribution with a peak onset b/w 15-30 yrs. & second smaller peak b/w 55-80 yrs.
- Female in general 20%-30% greater risk of developing C.D.

# Ethnicity

- Jewish people are afflicted 3-8 times > non Jews
- Jewish people from different countries are not equally affected with remarkable predominance of north American and south African Jewish
- In Israel a study of the Jewish population in Tel-Aviv revealed that Ashkenazi Jews born outside Israel increased 4 times incidence compared with Ashkenazi Jews born in Israel (16.69/100,000 Vs. 4.19/100,000)
- In England Hindus & Sikhs have a decrease incidence of C.D. Than the general population.
- American black & Indian populations are at low risk of IBD.

## Epidemiology of Inflammatory Bowel Disease

Incidence, per 100,000	1-10 (CD) 3-15 (UC)
Prevalence, per 100,000	20-100 (CD) 50-80 (UC)
Geography	Northern Countries > South Countries
Age of onset	Peak: 15-30 Second Peak 50-80 (CD)
Sex	M = F
Race	Whites > Blacks
Ethnic	Jewish > Non-Jewish
Smoking	Associated with CD : Protective in UC
Appendectomy	May be protective in UC
Possible Genetic Associations	Chromosome 16 (CD) Chromosome 3, 7, 12 (UC and CD) TNF- (CD); IL-1 A (CD) HLA-A2; HLA-DR1; DQw5 (CD) HLA-DR2 (UC)

# Etiology & Pathogenesis

UNKNOWN

We know neither its cause nor its cure

# Etiology & Pathogenesis Cont'd

Sartor et al. reviewed 3 most recent theories of etiology of C.D.

1. Specific infectious agent

1. mycobact. Para T.B.
2. Paramoxyvirus & measles
3. Listeria monocytogens

2. Defective mucosal barrier resulting in increase exposure to Ags.

3. Abnormal host response to a ubiquitous Ags

luminal constituents      Dietary Ags.

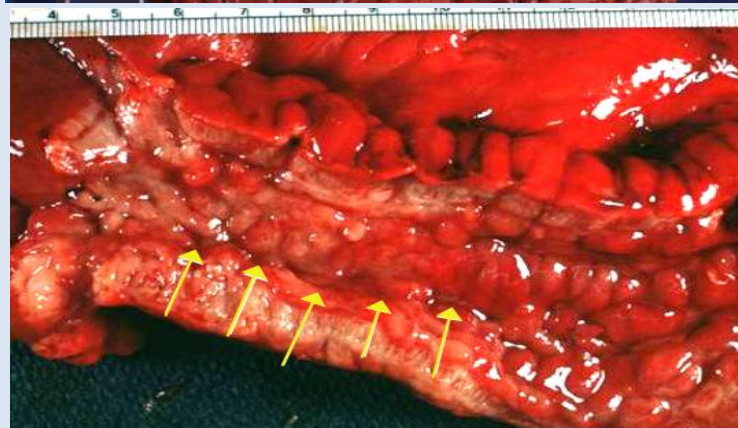
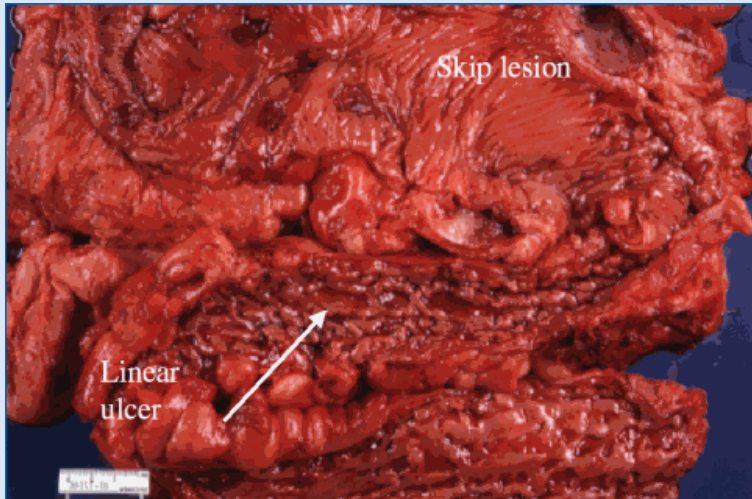
# Other Factors

- Family history
  - 10%-20% increase incidence in close degree relatives
  - Study in Denmark showed 1<sup>st</sup> and 2<sup>nd</sup> generation relatives of Pts. With C.D. have 1-3 folds increase prevalence .
  - Probert et al. -1<sup>st</sup> degree relative of Pt. with C.D. increase risk up to 35 times.
  - Monozygotic twins > dizygotic twins
- Dietary factors
  - Increase intake of refined sugar and starch
  - Decrease intake of fresh fruits
- Smoking
  - Oxford study found R..R.. Of 3.4
  - Increase recurrence rate among smokers
- OCP
  - Use of OCP for 1-3 years has a R..R.. Of 2.5 & 4.3 > 3 years.
  - Risk decrease after D/C OCP

# Pathology

- C.D. can affect any part of GIT
  - Ileocecal region(41%-55%) ,SI (30%-40%) ,colonic (14%-26%)
  - 2/3 of crohn's colitis pts. have total involvement
- **Acute or active phase** is marked by
  - Aphthous mucosal ulcers
  - Lymphoid aggregates
  - Granulomas (2/3 of pts.)
  - Transmural chronic inflammation with fissures & fistulas
- **Healing phase**
  - CHX. By fibrosis with stricture formation & chronic ulcers

# Gross

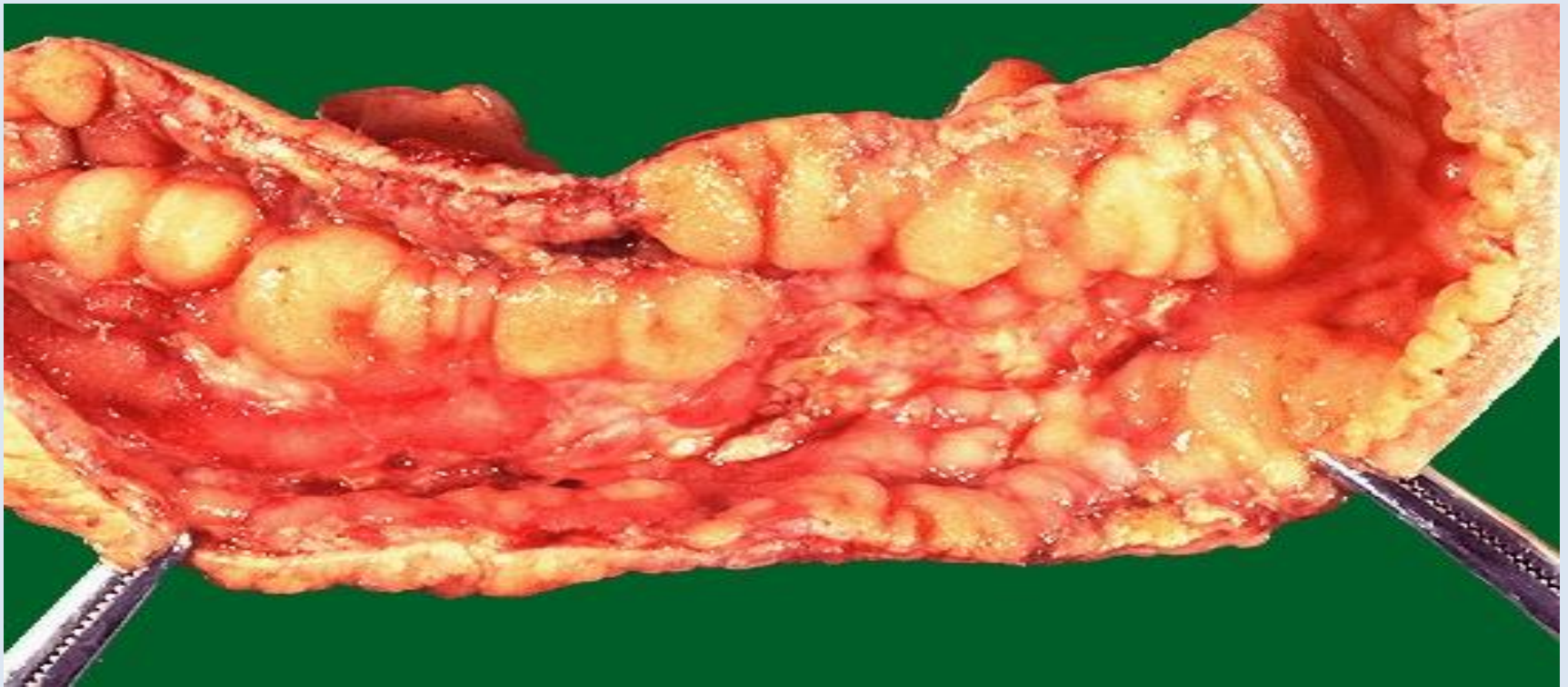


The wall of the ileum severely affected by Crohn's disease is inflamed and red. There are linear mucosal ulcerations present (yellow arrows), which would be focal if you could see the entire ileum. In addition, there is severe thickening of the bowel wall.

## Gross features:

- Skip lesions
- Creeping fat
- Thickened wall +/- structures
- Fissuring ulcers with a linear, serpentine appearance grossly; cobblestoning of mucosa
- Fistulae

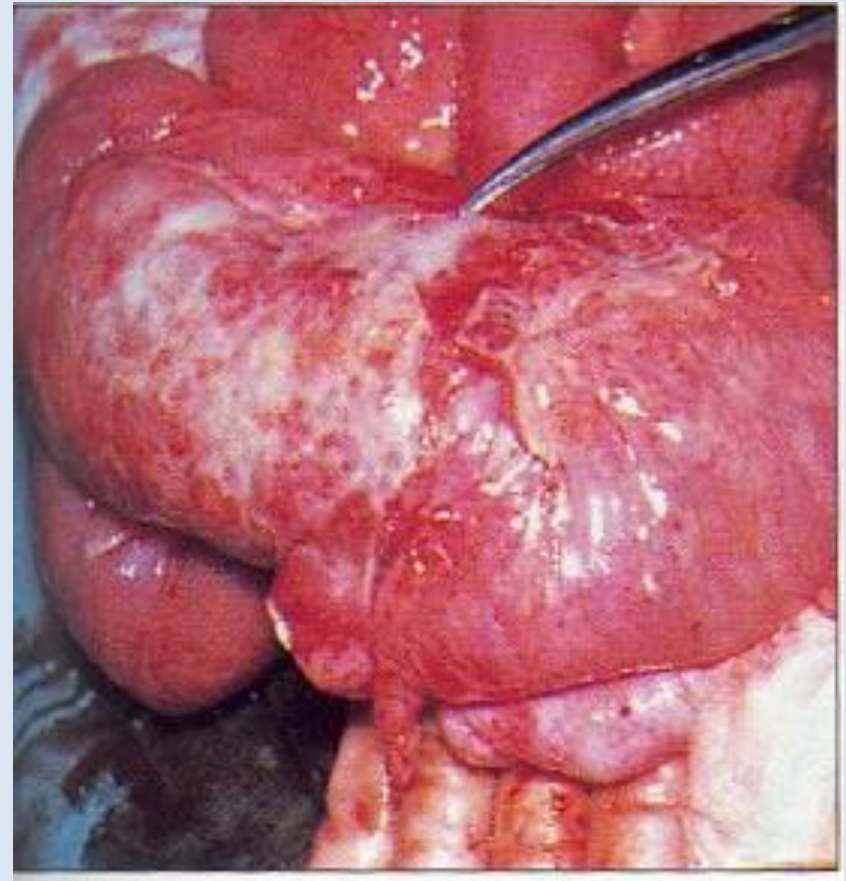




This is another example of Crohn's disease involving the small intestine. Here, the mucosal surface demonstrates an irregular nodular appearance with hyperemia and focal superficial ulceration. The distribution of bowel involvement with Crohn's disease is irregular with more normal intervening "skip" areas.

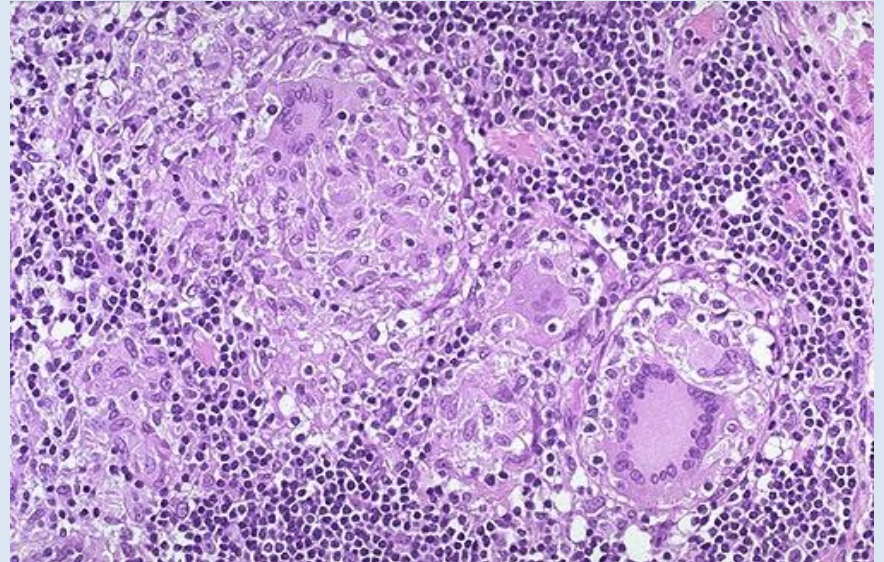
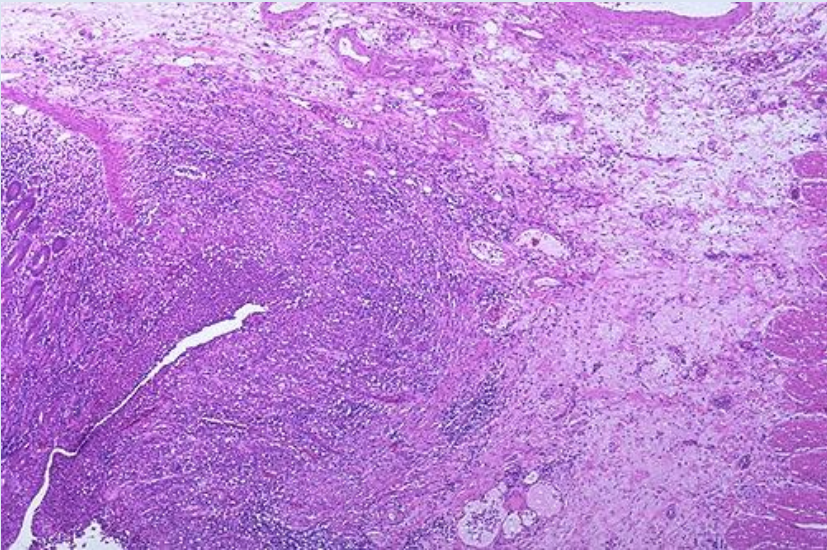
## Perforated terminal ileitis.

Crohn's disease may present in a similar manner to that of acute appendicitis. A 22 year old male has had a history of several bouts of episodic diarrhea associated with recurrent abdominal pain, lassitude and pyrexia. Although the terminal ileum has perforated in the patient, a much more common presentation is that of simple terminal ileitis. Consider a **differential diagnosis** of eosinophilic ileitis, other inflammatory bowel disease or lymphoma.

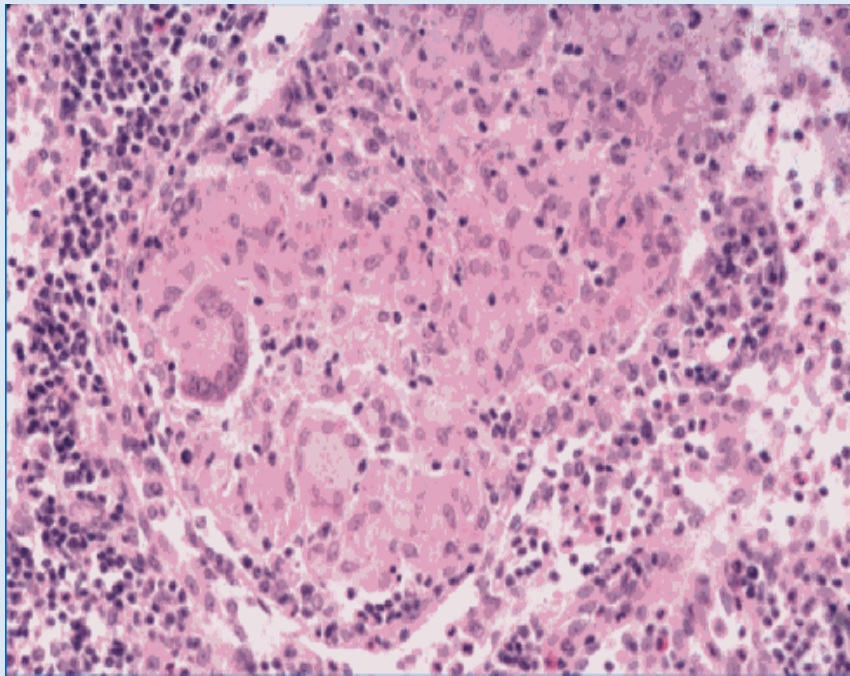




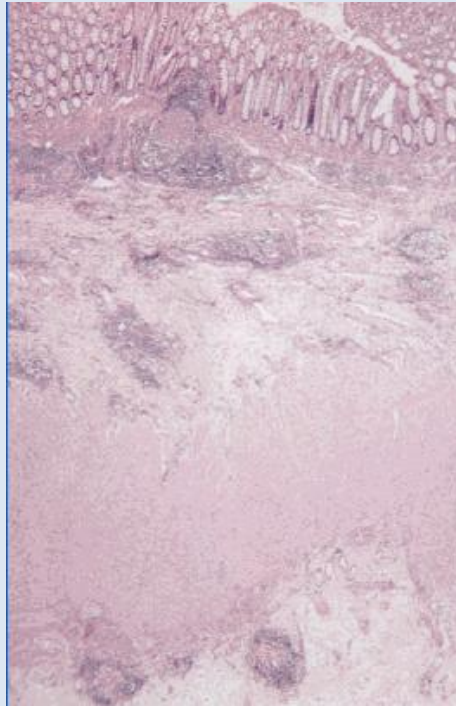
# Crohn's Disease-Microscopic



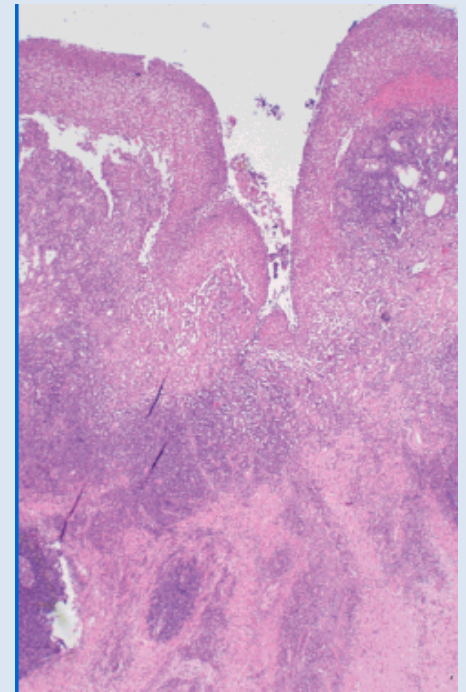
- Aphthous ulcers
- Transmural inflammation
  - Neutrophilic into glands
  - Crypt abscesses
  - +/- granulomas
- Signs of chronic injury: architectural distortion, atrophy, and metaplasia.
- Possible dysplasia late in disease



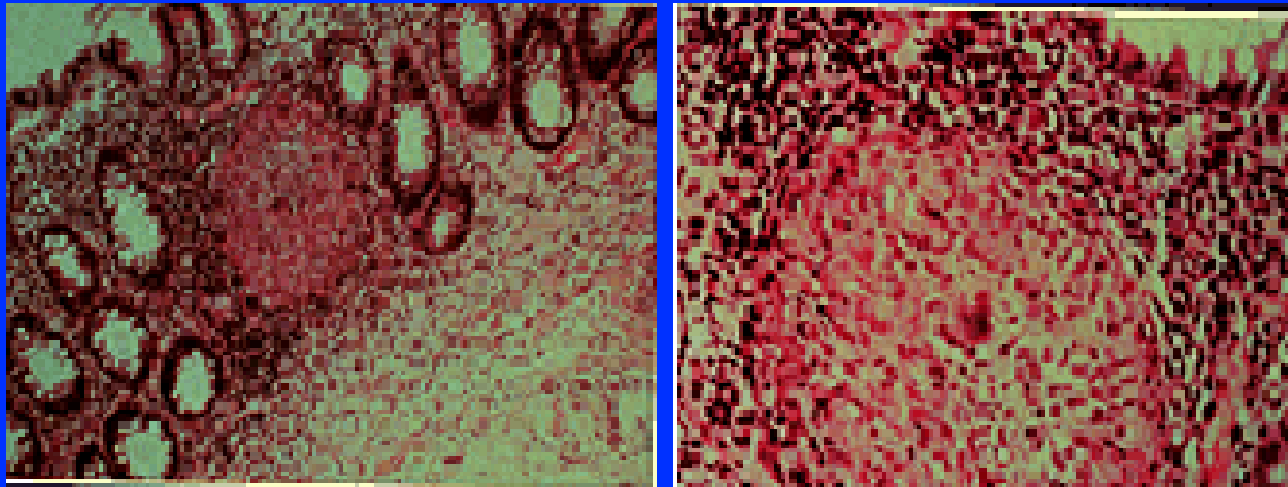
Granulomas are often in C.D. but not in UC.



Transmural inflammation in crohn's disease



Fissuring, linear ulcers in Crohn's Disease



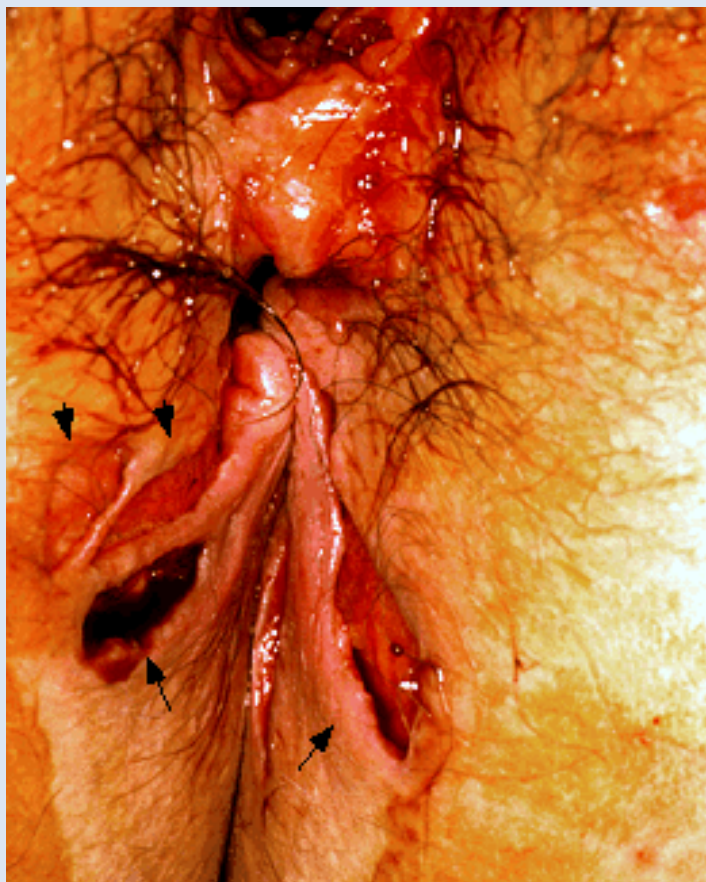
**Typical granuloma of Crohn's disease** Light micrographs showing granulomatous lesion that is diagnostic of Crohn's disease. Low and high power views show a central giant cell surrounded by epithelioid cells and rimmed by lymphocytes. Courtesy of the American Gastroenterological Association®. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.



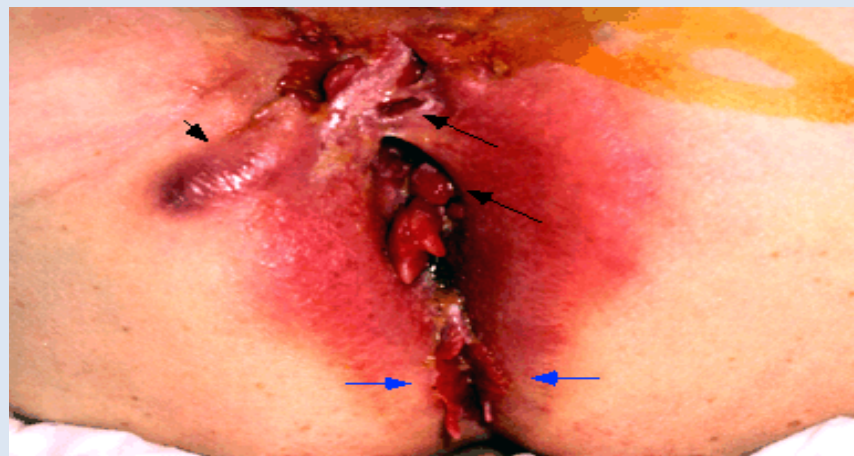
# Clinical Features

Depend mainly on anatomical location of the disease

- Symptoms
  - Abd. Pain
  - Diarrhea
  - Wt. loss
  - Bleeding per rectum
  - Anorexia, fever, N ,V
  - Recurrent oral aphthous ulceration
  - Hx. Of perianal disease, extraintestinal manifestation
  - In children growth retardation & failure of 2<sup>nd</sup> sex Chx.



**Perianal Crohn's disease** Perianal fistulas and ulcers in a patient with Crohn's disease. There are bilateral posterior perianal fistulas (arrows) and two right posterior ulcers (arrowheads). Courtesy of Alain Bitton, MD, FRCPC.



**Anal fistulas and abscess in Crohn's disease** This picture shows anterior perianal fistulas involving the vulva and anterior perineum (arrows) in a woman with Crohn's disease being prepared for surgery. The surrounding skin is erythematous and indurated. An abscess (arrowhead) appears as a localized swelling. Hypertrophic skin tags (blue arrows) in the anal canal are commonly observed in perianal Crohn's disease and may be confused with external hemorrhoids. Courtesy of Alain Bitton, MD, FRCPC.



**Perianal Crohn's disease** Perineum in a woman with Crohn's disease shows a large posterior ulcer (arrow) with a large, edematous fissure (arrowhead). Courtesy of Alain Bitton, MD, FRCPC.

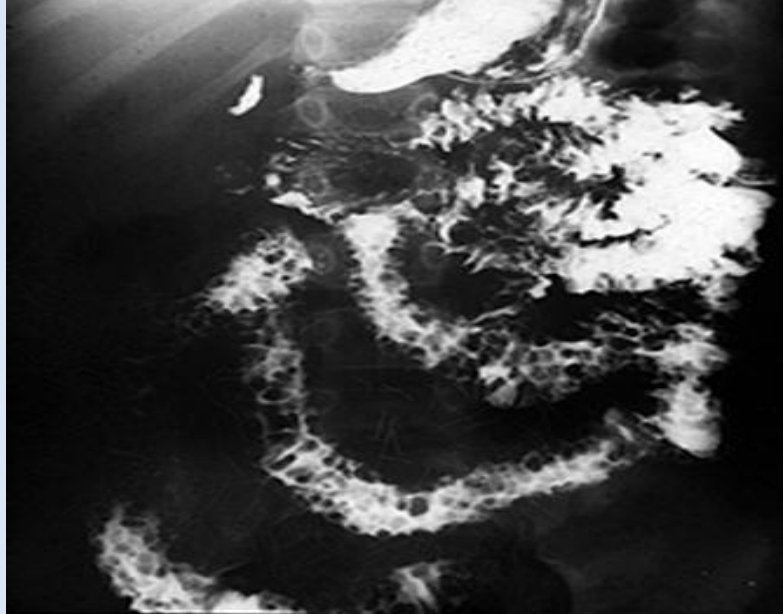
# Clinical Features-cont'd

- Signs
  - Pallor
  - Cachexia
  - Clubbing
  - Abdominal mass or tenderness
  - ? Evidence of obstruction
  - Anemia
  - Hypoproteinemia
- Exacerbating factors
  - Intercurrent infection (URTI or enteric)
  - Cigg. Smoking
  - NSAIDs.
  - ?Stress & psychological predisposition



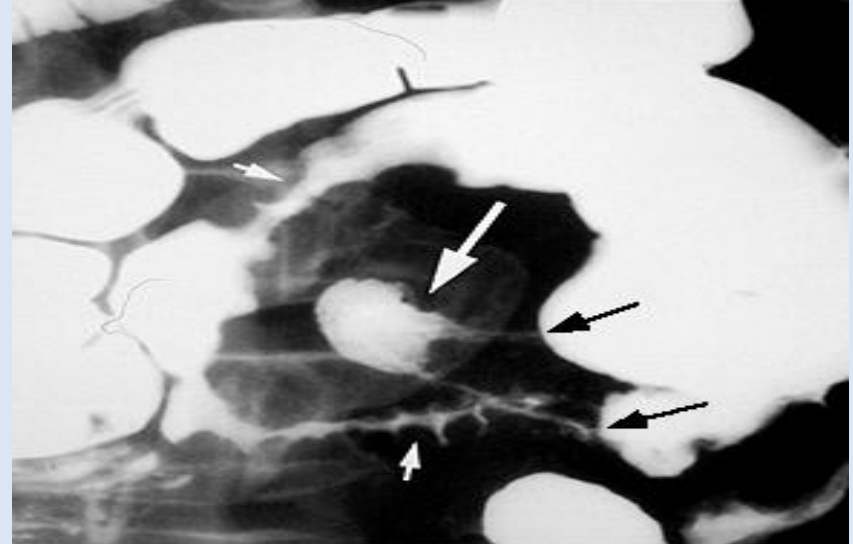
# Dx.

- The onset of C.D. is nearly always obscure & poorly defined thus, delayed diagnosis
- In only 30% of pts. Dx. is made within one year of onset of Sx.
- Dx. Is usually made during an acute exacerbation
- Hx. & ph.
- Radiology
  - Contrast radiography is essential for DDx. & to determine the severity of the disease
  - Indications include recurrence of Sx. After surgery & pts. With progressive disease requiring surgical Rx. or change in his medical plan.
  - Barium studies.
  - CT scan----- masses & abcesses.
  - ERCP ----- hepatobiliary involv.(sclerosing cholangitis).



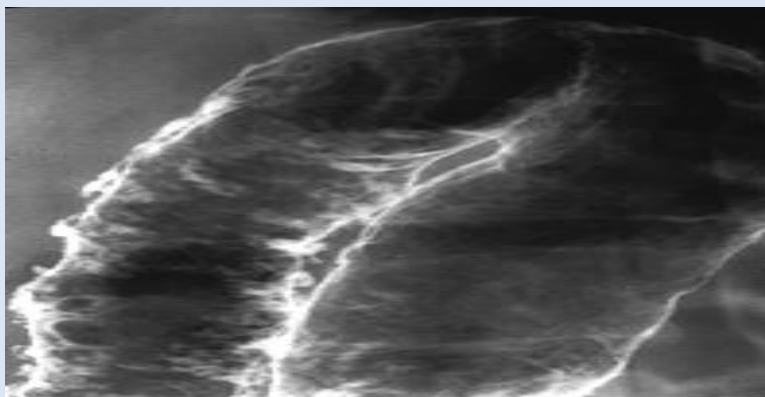
**Cobblestone appearance in Crohn's disease**

Small bowel follow through study demonstrates diffuse thickening of the small bowel mucosa in a patient with Crohn's disease. The cobblestone appearance is produced by barium being dispersed between the edematous inflamed mucosa. Courtesy of Norman Joffe, MD.

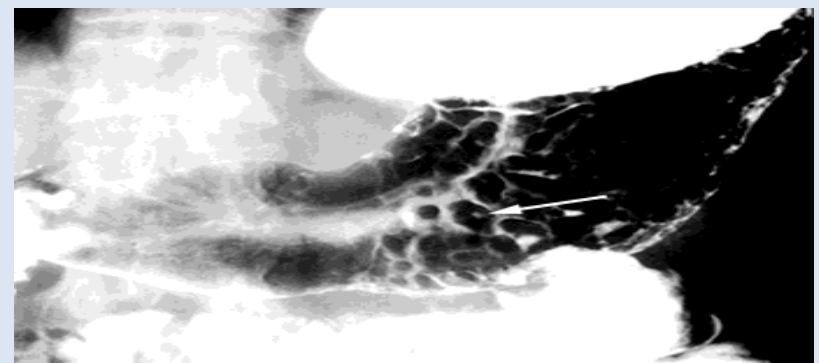


**Crohn's disease with abscess and fistulae**

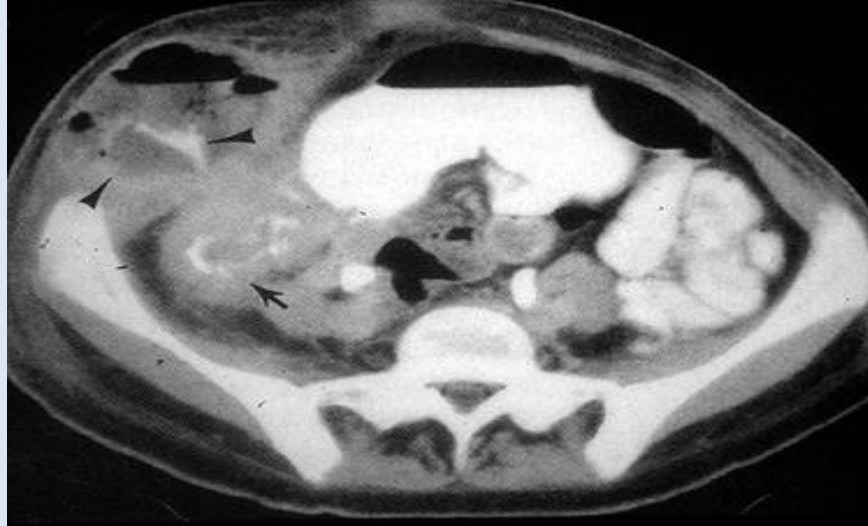
Small bowel follow through study demonstrates an abscess cavity (white arrow) with fistulae connecting the cavity to the adjacent small bowel (black arrows). Note the marked thickening of the inflamed mucosal folds (small arrows). Courtesy of Jonathan Kruskal, MD, PhD.



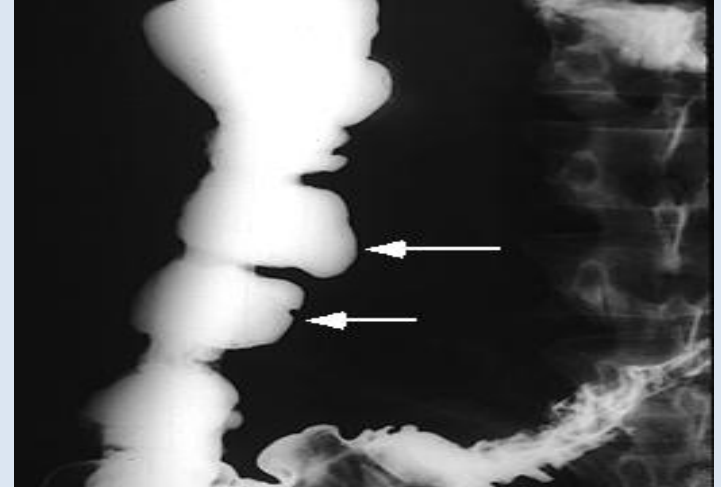
**Crohn's colitis** Double contrast barium enema in a patient with Crohn's disease shows extensive ulceration of the wall of the colon associated with mucosal thickening and inflammation. Courtesy of Jonathan Kruskal, MD, PhD.



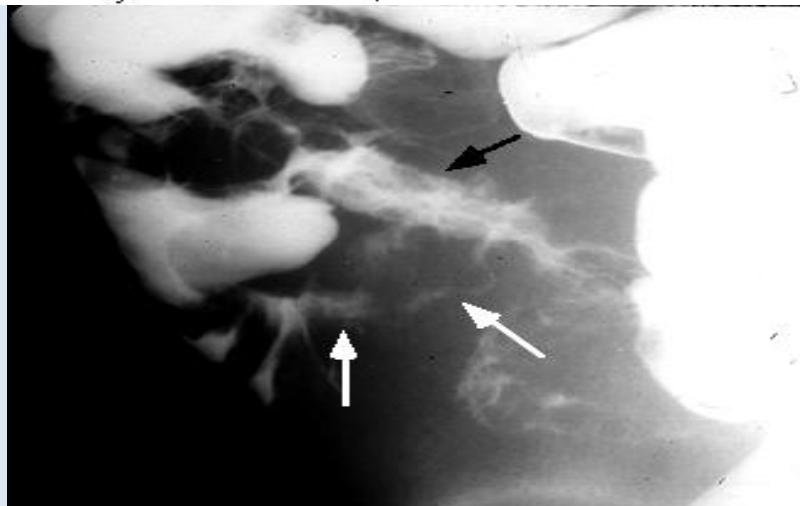
**Crohn's disease of the stomach** This upper GI series, performed in a young man with known Crohn's disease of the terminal ileum shows numerous rounded filling defects in the stomach produced by edematous mucosa. In some of these areas, small central collections of barium are demonstrated (arrow) resulting from superficial erosions. These features are suggestive of Crohn's disease, but may also be seen in patients with peptic ulcer disease, and in viral gastritis. Courtesy of Jonathan Kruskal, MD, PhD.



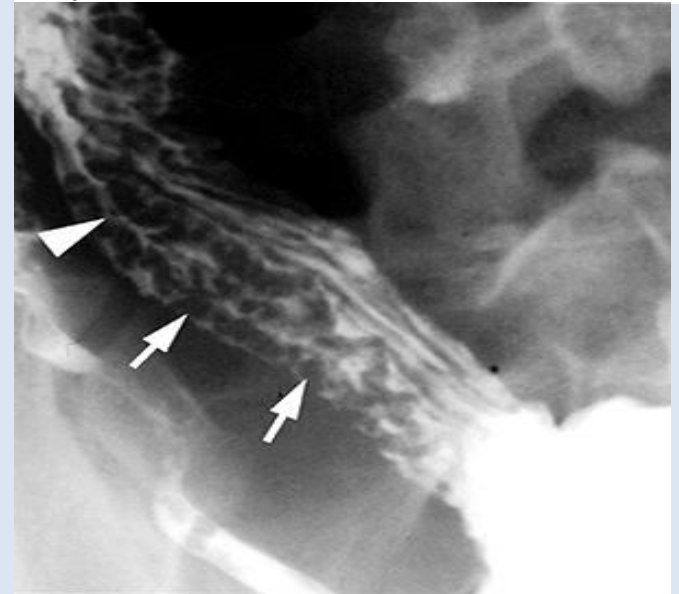
**Right lower quadrant abscess in Crohn's disease** Single axial CT scan of the lower abdomen demonstrates an abscess (arrowheads) extending from the markedly thickened and inflamed terminal ileum (arrow). The presence of contrast material within the abscess confirms a communication with the adjacent ileum. Courtesy of Norman Joffe, MD.



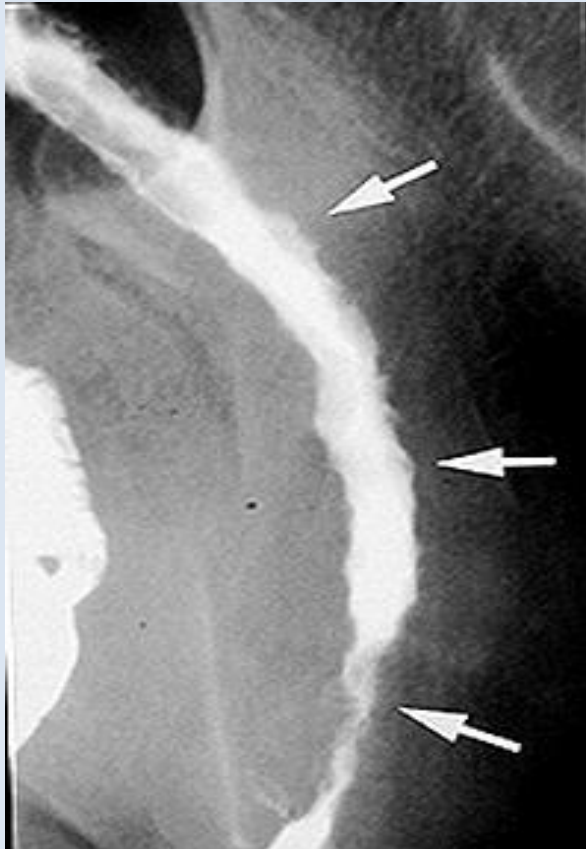
**Chronic Crohn's colitis** Barium enema demonstrates sacculations along the medial border of the ascending colon (arrows) produced by scarring and fibrosis in a patient with Crohn's disease. Courtesy of Jonathan Kruskal, MD, PhD.



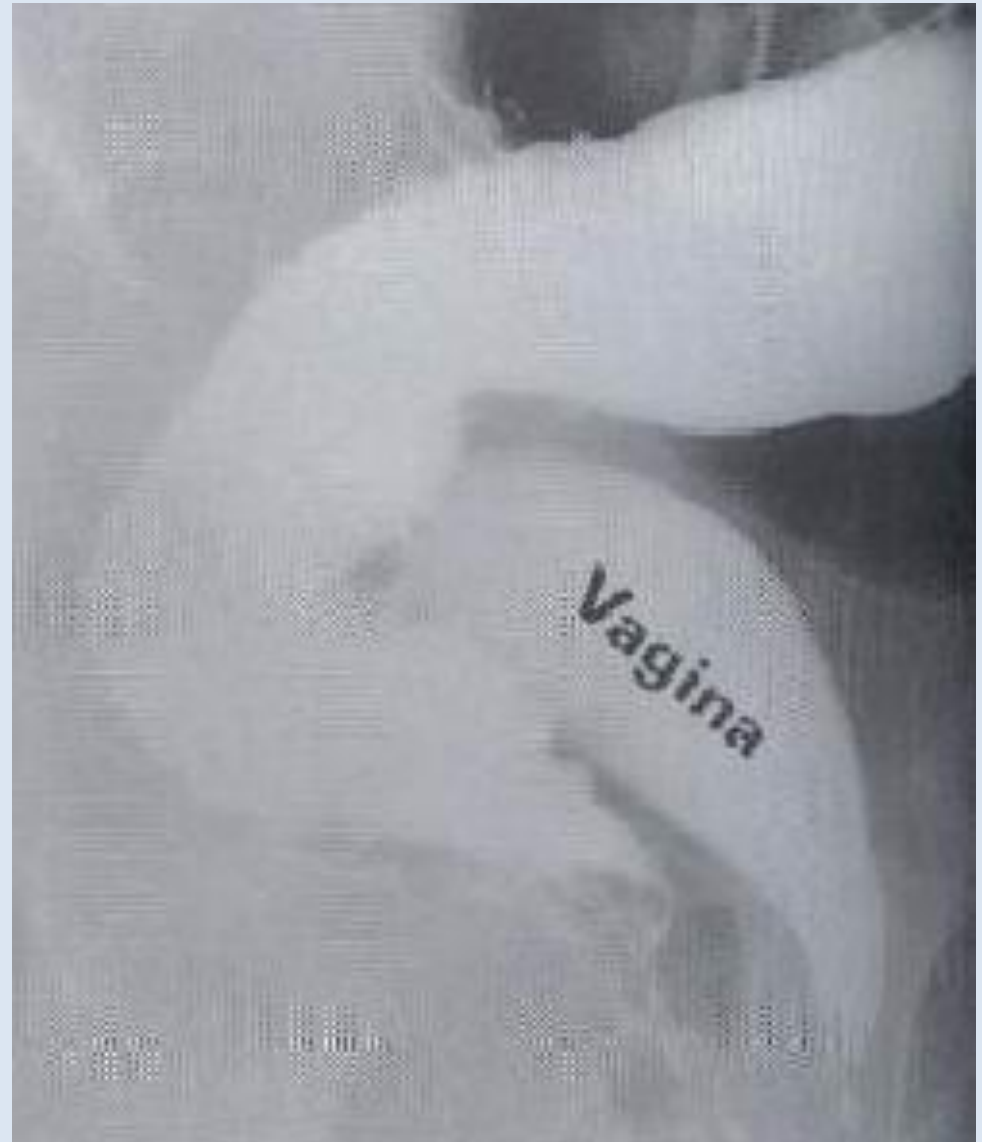
**Ileocecal fistulae in Crohn's disease** Small bowel follow through examination demonstrates nodular thickening of the terminal ileal mucosal folds in a patient with Crohn's disease (black arrow). Several fistulae extend from the terminal ileum to the adjacent cecum (white arrows). Courtesy of Jonathan Kruskal, MD, PhD.



**Crohn's disease** Small bowel follow through examination demonstrates nodular filling defects arising on thickened folds in the terminal ileum (arrows). These features are characteristic of Crohn's disease. Courtesy of Jonathan Kruskal, MD, PhD.



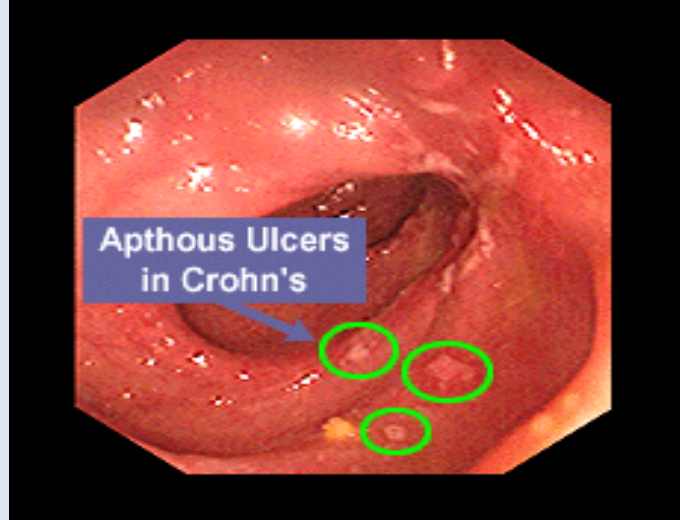
**String sign in Crohn's disease** Small bowel follow through study shows marked narrowing, irregularity and ulceration in the distal ileum (arrows) in a patient with Crohn's disease. Courtesy of Jonathan Kruskal, MD, PhD.



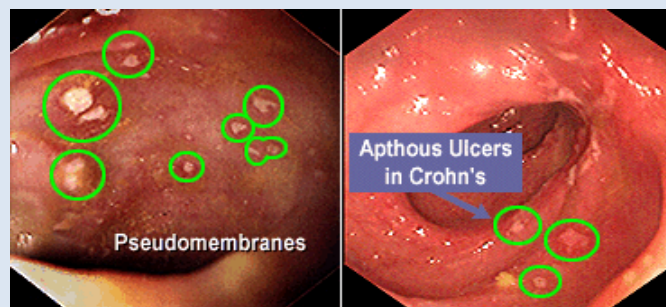
# Cont'd

- Endoscopy.
  - Colonoscopy
    - Extent of the dis.
    - Confirm radiographic abnormalities.
    - Bx.
    - Shows aphthus ulcers, linear ulcers, ulcers in otherwise normal appearing mucosa, cobblestoning & asymmetric discontinuous involvement.

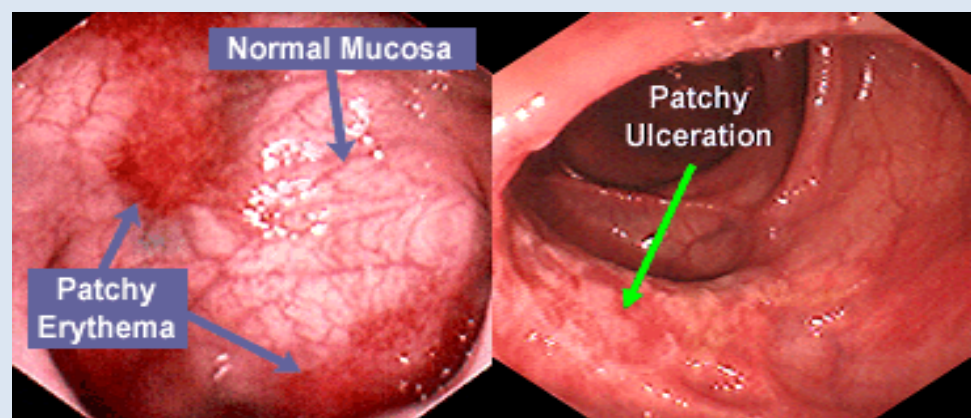




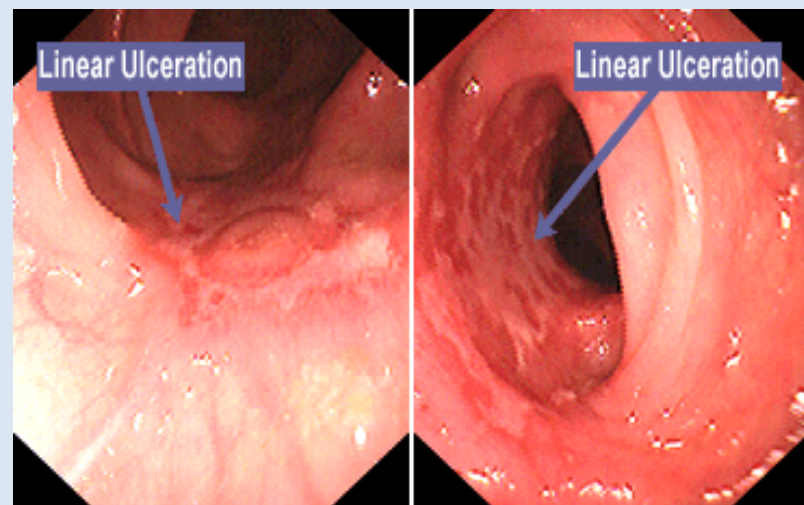
**Aphthous ulcers in Crohn's disease** Lower endoscopy demonstrates small discreet aphthous ulcers that are characteristic of early lesions in Crohn's disease. Courtesy of James B McGee, MD.



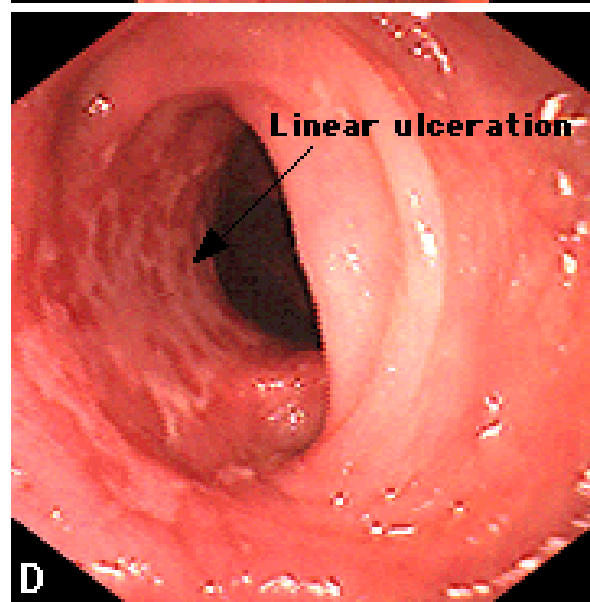
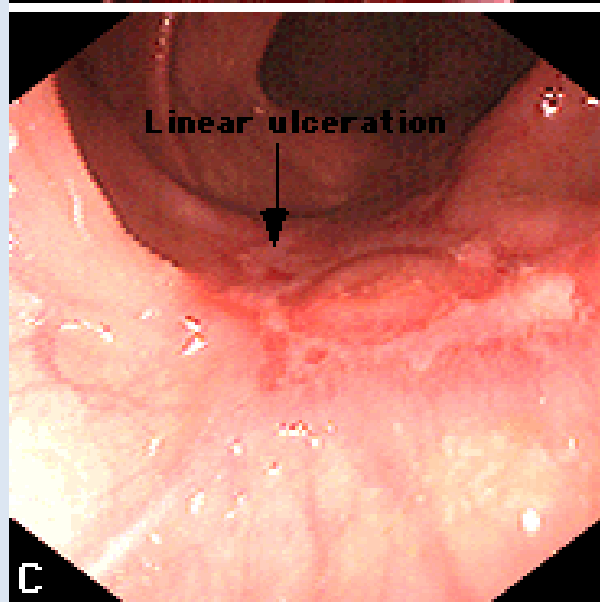
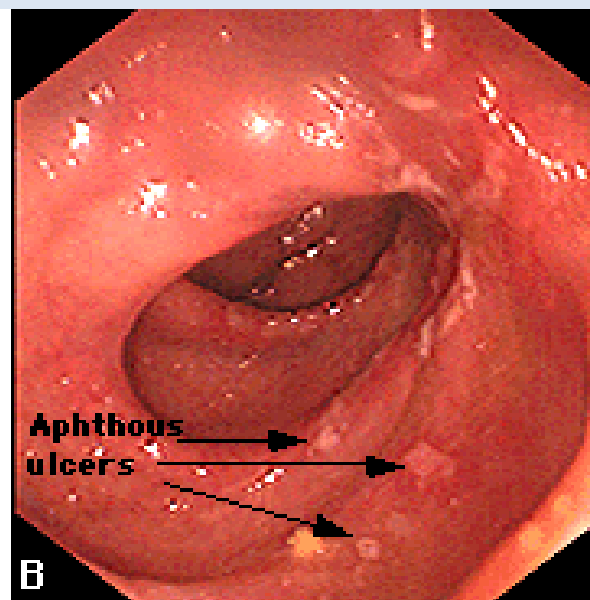
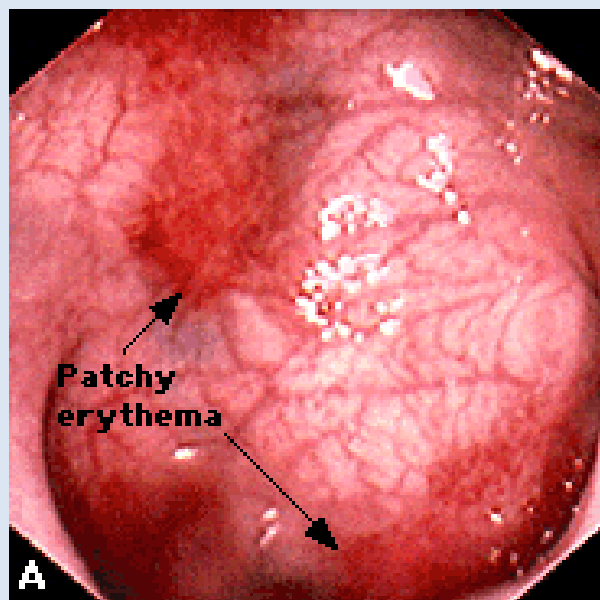
**Pseudomembranous colitis** Endoscopy of pseudomembranous colitis reveals small groups of pseudomembranes (left panel) that may be confused grossly with the aphthous ulcers of Crohn's disease (right panel). These lesions can be distinguished since pseudomembranes are present on top of the mucosa and do not result in ulceration of the underlying tissue. Courtesy of James B McGee, MD.



**Asymmetric distribution of lesions in Crohn's disease** Lower endoscopy in Crohn's disease demonstrates the characteristic patchy erythema (left panel) and ulceration (right panel) that occur next to areas of normal mucosa. Courtesy of James B McGee, MD.



**Linear ulcerations in Crohn's disease** Lower endoscopy shows linear ulcers that can course for several centimeters along the longitudinal axis of the colon in Crohn's disease. Courtesy of James B McGee, MD.



## Endoscopic progression of Crohn's disease

Ulcers are the dominant endoscopic feature in Crohn's disease. These tend to be linear and discontinuous, or "skip lesions". Early changes may be only patchy erythema (panel A) or aphthoid ulcers (panel B). Linear ulcers (panel C) are seen with more advanced disease, culminating in very deep and long serpiginous ulcers (panel D). Courtesy of James B McGee, MD.

# D.Dx.

- Chronic U.C.
  - The initial Dx. of U.C. Is confirmed in 80% of instances, is changed to crohn's colitis in 10% to15%, and remains indeterminant in 5% to 10%.
  - Acute appendicitis.
  - Other infections
    - Yersinia
    - Compylobacter
    - Shigella
    - Salmonella
    - E.coli
  - T.B.
  - Lymphoma.
  - Ch. Mesenteric ischemia.



# Extraintestinal manifestation

- Peptic ulcers.
- Arthritis.
- Skin lesions---(pyoderma gang.,Eryth.Nodosum)
- Cholelithiasis.
- Renal calculi.
- Eye lesions.

## Extraintestinal Manifestations of IBD†

### Common Extraintestinal Manifestations

#### Musculoskeletal

Arthritis – colitic type, ankylosing spondylitis, isolated joint involvement

Hypertrophic osteoarthropathy – clubbing, periostitis, metastatic Crohn's disease

Miscellaneous – osteoporosis, aseptic necrosis, polymyositis

#### Skin and mouth

Reactive lesions – erythema nodosum, pyoderma gangrenosum, aphthous ulcers, vesiculopustular eruption, necrotizing vasculitis

Specific lesions – fissures and fistulas, oral Crohn's disease, drug rashes

Nutritional deficiency – acrodermatitis enteropathica (Zn), purpura (vitamin C & K), glossitis (vitamin B), hair loss and brittle nail (protein)

Associated diseases – vitiligo, psoriasis, amyloidosis, epidermolysis bullosa acquisita

#### Hepatobiliary

Specific complications – primary sclerosing cholangitis and bile duct carcinoma

Associated inflammation – autoimmune chronic active hepatitis, pericholangitis, portal fibrosis and cirrhosis, granuloma in Crohn's disease

Metabolic – fatty liver, gallstones associated with ileal Crohn's disease

#### Ocular

Uveitis (Iritis), episcleritis, scleromalacia, corneal ulcers, retinal vascular disease

#### Metabolic

Growth retardation in children and adolescents, delayed sexual maturation

## **Less Common Extraintestinal Manifestations**

### **Blood and vascular**

Anemia due to iron, folate or B12 deficiency or autoimmune hemolytic anemia, thrombocytopenic purpura; leukocytosis and thrombocytosis; Thrombophlebitis and thromboembolism, arteritis and arterial occlusion.

### **Renal and genitourinary tract**

Urinary calculi (oxalate stones in ileal disease), local extension of Crohn's disease involving ureter or bladder, amyloidosis

Renal tubular damage with increased urinary excretion of various enzymes, eg, beta N-acetyl-D-glucosaminidase.

### **Neurological**

Up to 3 percent of patients may have non-iatrogenic various neurologic involvements including peripheral neuropathy, myelopathy, myasthenia gravis, and cerebro vascular disorders. Incidence equal in both UC & CD usually five to six years after the onset of IBD and frequently associated with other extraintestinal manifestations.

### **Broncho pulmonary**

Pulmonary fibrosis, vasculitis, bronchitis, acute laryngotracheitis. Abnormal pulmonary function tests, without clinical symptoms, are common in up to 50 percent of cases.

### **Cardiac**

Pericarditis, myocarditis and heart block – UC>CD.

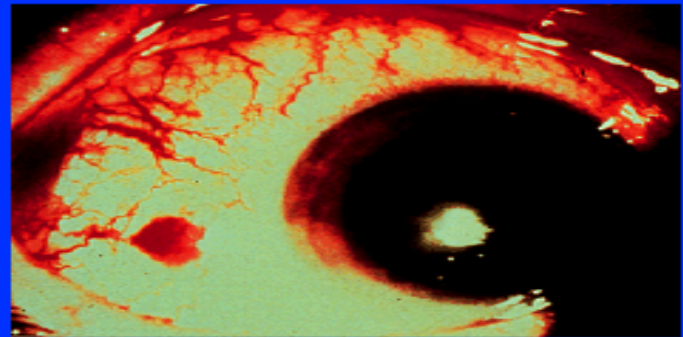
Pericarditis may also occur from sulfasalazine/5ASA

### **Pancreas**

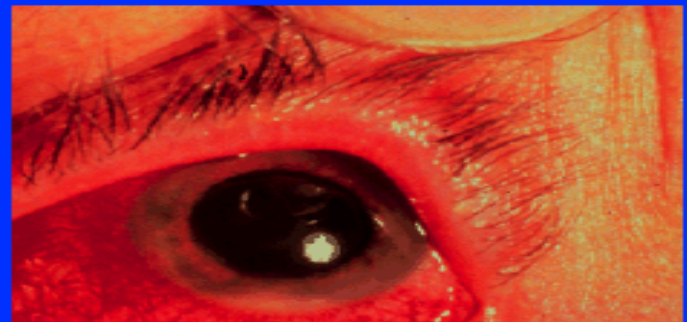
Acute pancreatitis – CD>UC: risk factors include 6 mercaptopurine and 5 amino salicylate therapy, duodenal Crohn's disease



**Erythema nodosum** Patient with inflammatory bowel disease with red nodular areas on the shins which are characteristic of erythema nodosum. (Courtesy of the American Gastroenterological Association®. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.)



**Episcleritis** Patient with episcleritis associated with inflammatory bowel disease showing the characteristic injection of the ciliary vessels. (Courtesy of the American Gastroenterological Association®. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.)



**Anterior uveitis** Anterior uveitis in a patient with inflammatory bowel disease is characterized by injection of the conjunctiva and opacity in the anterior chamber. Courtesy of the American Gastroenterological Association®. This slide cannot be downloaded but may be purchased as part of a set from the AGA through Milner-Fenwick, Inc at 1-800-432-8433.

# Comparison of Ulcerative Colitis and Crohn's Colitis

Manifestation	Ulcerative Colitis	Crohn's Colitis
<b>Clinical Features</b>		
Bleeding per rectum	3+	1+
Diarrhea	3+	3+
Abdominal pain	1+	3+
		Especially with involvement of ileum
Vomiting	R	3+
Fever	R	2+
Palpable abdominal mass	R	2+
Weight loss	+	3+
Clubbing	R	1+
Rectal involvement	4+	1+
Small bowel involvement	0	4+
Anal and perianal involvement	R	4+
Risk of carcinoma	1+	1+
Clinical course	Relapses/remission	Slowly progressive
<b>Radiologic</b>		
Thumb printing sign on barium enema	R	1+

R=rare, 0=not found,  
1+=maybe present,  
2+=common,  
3+=usual finding,  
4+=characteristic

# Comparison of Ulcerative Colitis and Crohn's Colitis-cont'd

Manifestation	Ulcerative Colitis	Crohn's Colitis
<b>Endoscopic</b>		
Distribution	Symmetric	Asymmetric
Continuous involvement	4+	1+
Rectal	4+	1+
Vascular architecture	Absent	1+
Friability	4+	1+
Erythema	3+	1+
Spontaneous petechiae	2+	R
Profuse bleeding	1+	R
Aphthous ulcer	0	4+
Serpiginous ulcer	R	4+
Deep longitudinal ulcer	0	4+
Cobblestoning	0	4+
Mucosa surrounding ulcer	Abnormal	+normal
Pseudopolyps	2+	2+
Bridging	R	1+
<b>Gross Appearance</b>		
Thickened bowel wall	0	4+
Shortening of bowel	2+	R
Fat creeping onto serosa	0	4+
Segmental involvement	0	4+
Aphthous ulcer	R	4+
Linear ulcer	0	4+

# Comparison of Ulcerative Colitis and Crohn's Colitis-cont'd

Manifestation	Ulcerative Colitis	Crohn's Colitis
<b>Microscopic Pictures</b>		
Depth of involvement	Mucosa and submucosa	Full thickness
Lymphoid aggregation	0	4+
Sarcoid-type granuloma	0	4+
Fissuring	0	2+
Goblet cell mucin depletion	4+	1+
Intramural sinuses	0	1+
<b>Operative Treatment</b>		
Total proctectomy	Excellent option in selected patients	Indicated in total large bowel involvement
Segmental resection	R	Frequent
Ileal pouch procedure	"Gold standard"	Contraindicated
<b>Prognosis</b>		
Recurrence after total proctocolectomy	0	3+
<b>Complications</b>		
Internal fistula	R	4+
Intestinal obstruction (stricture of infection)	0	4+
Hemorrhage	1+	1+
Sclerosing cholangitis	1+	R
Cholelithiasis	0	2+
Nephrolithiasis	0	2+





"Mr. Osborne, may I be excused?  
My brain is full."



# **Common small and large intestinal surgical diseases**

## **Part II**

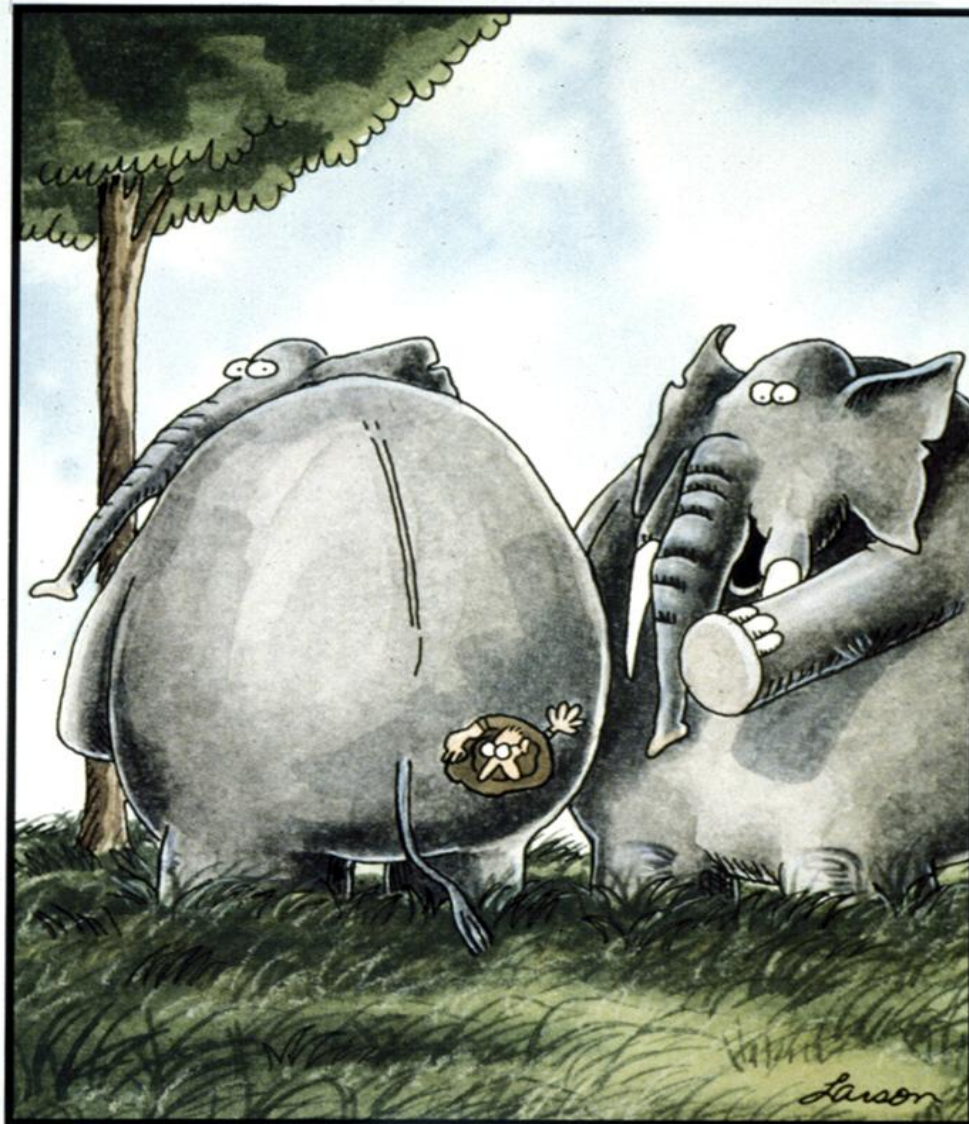
Khayal AlKhayal, MD, FRCSC  
Assistant Professor of Surgery  
Consultant Colorectal Surgeon  
2010

# Colorectal cancer

# Outline

- Definitions
- Polyps
- Basics of colorectal cancer
- Surgery
- Staging

# Perspective



"Whoa, Frank ...  
guess what youuuuuuuuu sat in!"

# Definitions

- Colon = large bowel = large intestine
- Rectum - terminal portion of the colon
- Polyp - benign growth; not invasive
- Adenoma - type of polyp
- Cancer - malignant growth; invasive
- Stage - where the cancer is growing
- Primary - the original tumour, where it started
- Metastases - where the tumour has spread to

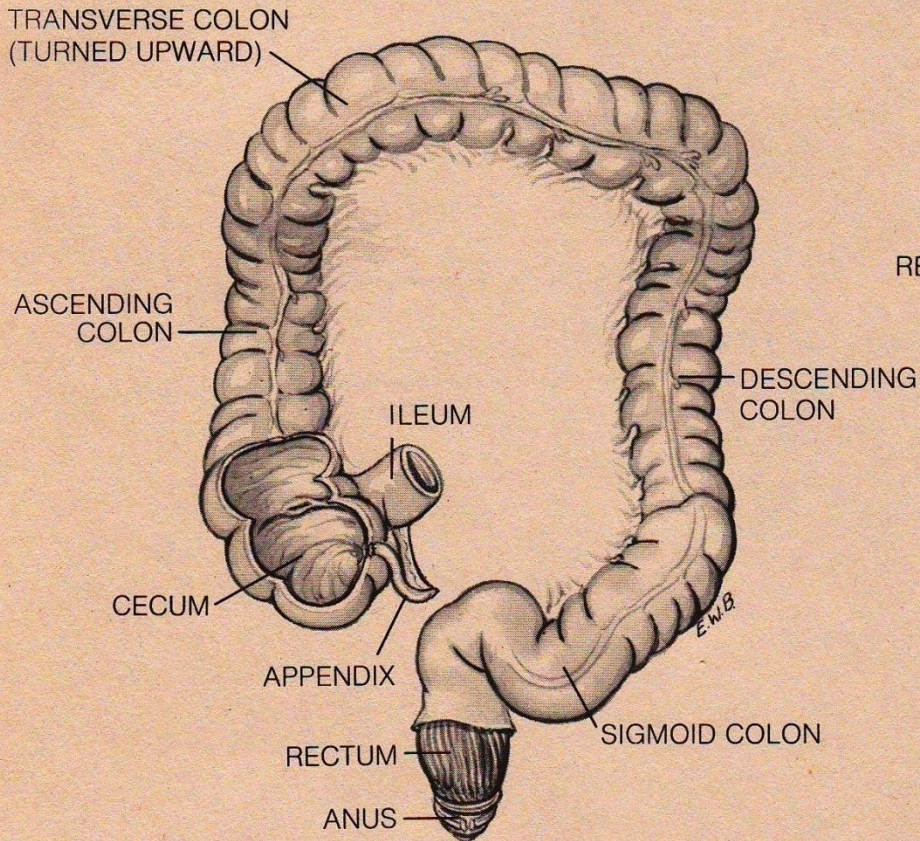
# Cancer

A cancer cell :

- is immortal ( lives forever)
- multiplies uncontrollably
- can live on its own without neighbors
- can live in other parts of the body

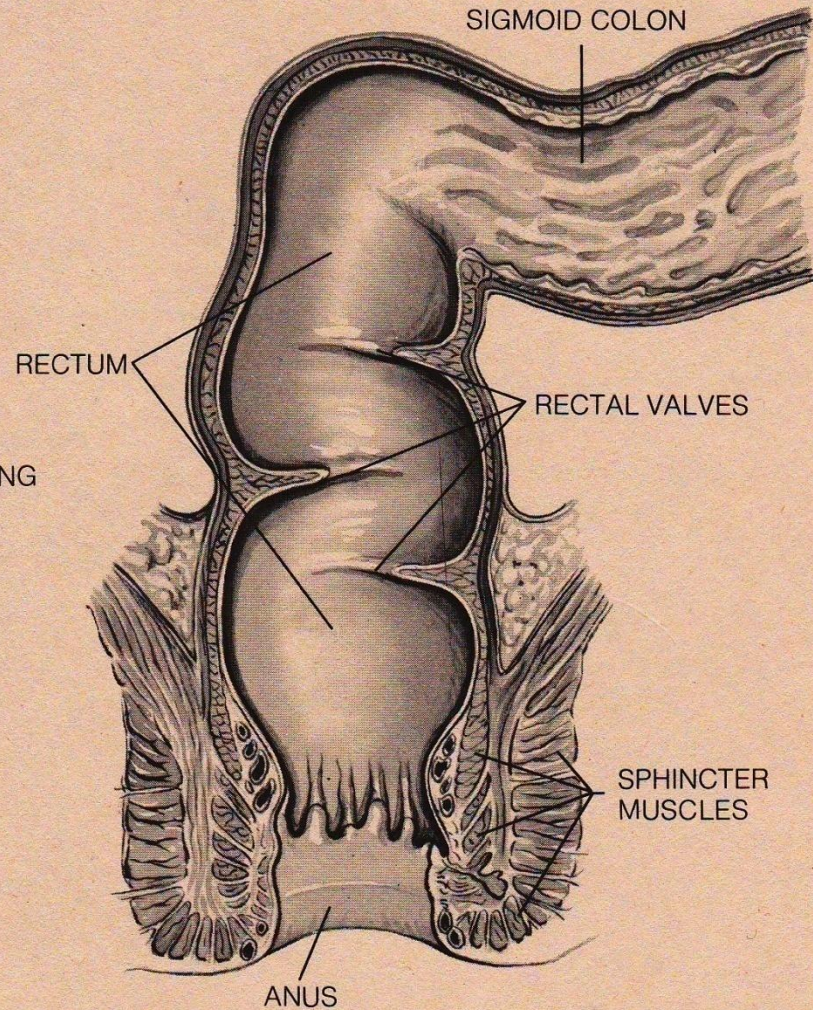


# Colon and Rectum



THE LARGE INTESTINE

8/16/2018



RECTUM AND ANUS

M.A. REED



# Colorectal Cancer

- Most cancers are acquired some are inherited
- Almost all cancers begin as a benign polyp or adenoma
- Only a tiny percentage of adenomas become cancers



# What is a polyp?



Fig. 22-7 Pedunculated polyp.



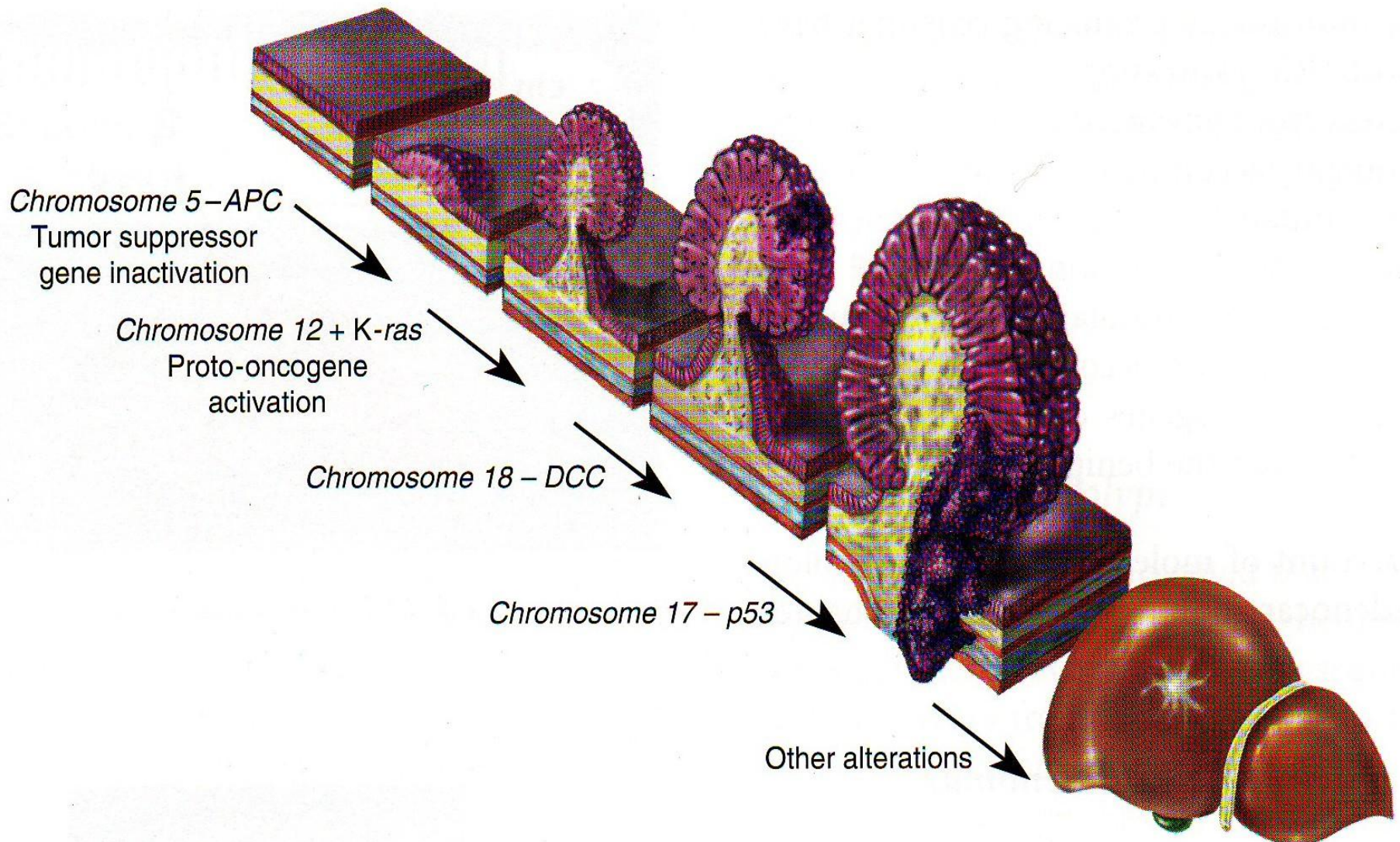
Fig. 22-8 Sessile polyp.

# Polyp - Cancer Sequence

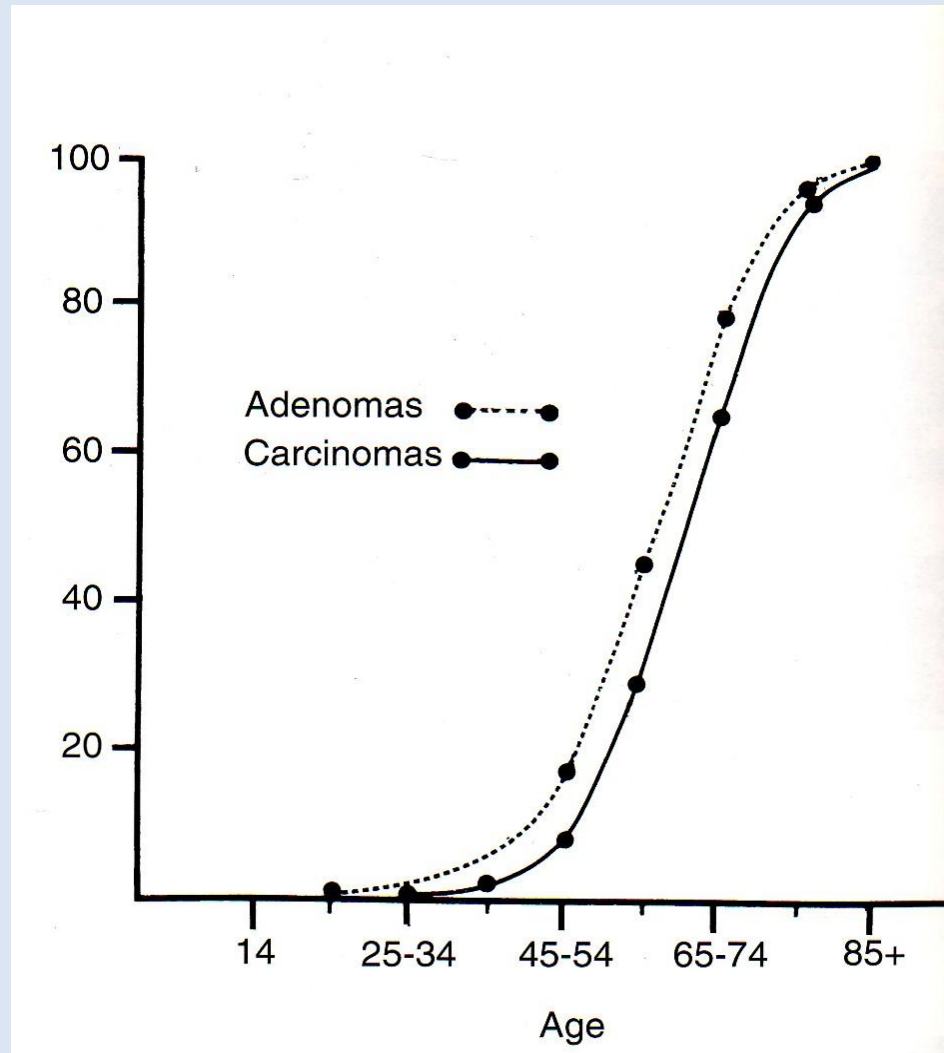
- The process from benign polyp to cancer takes from 7 - 10 years
- The transformation into cancer is based on
  - the type of polyp
  - Size of polyp
- Multiple polyps = greater risk of cancer







## The Effect of Age on the Incidence of Colorectal Cancer and Colorectal Polyps



# **Removing polyps prevents cancer**

## Colonoscopy

# Colorectal Carcinoma

## Classification

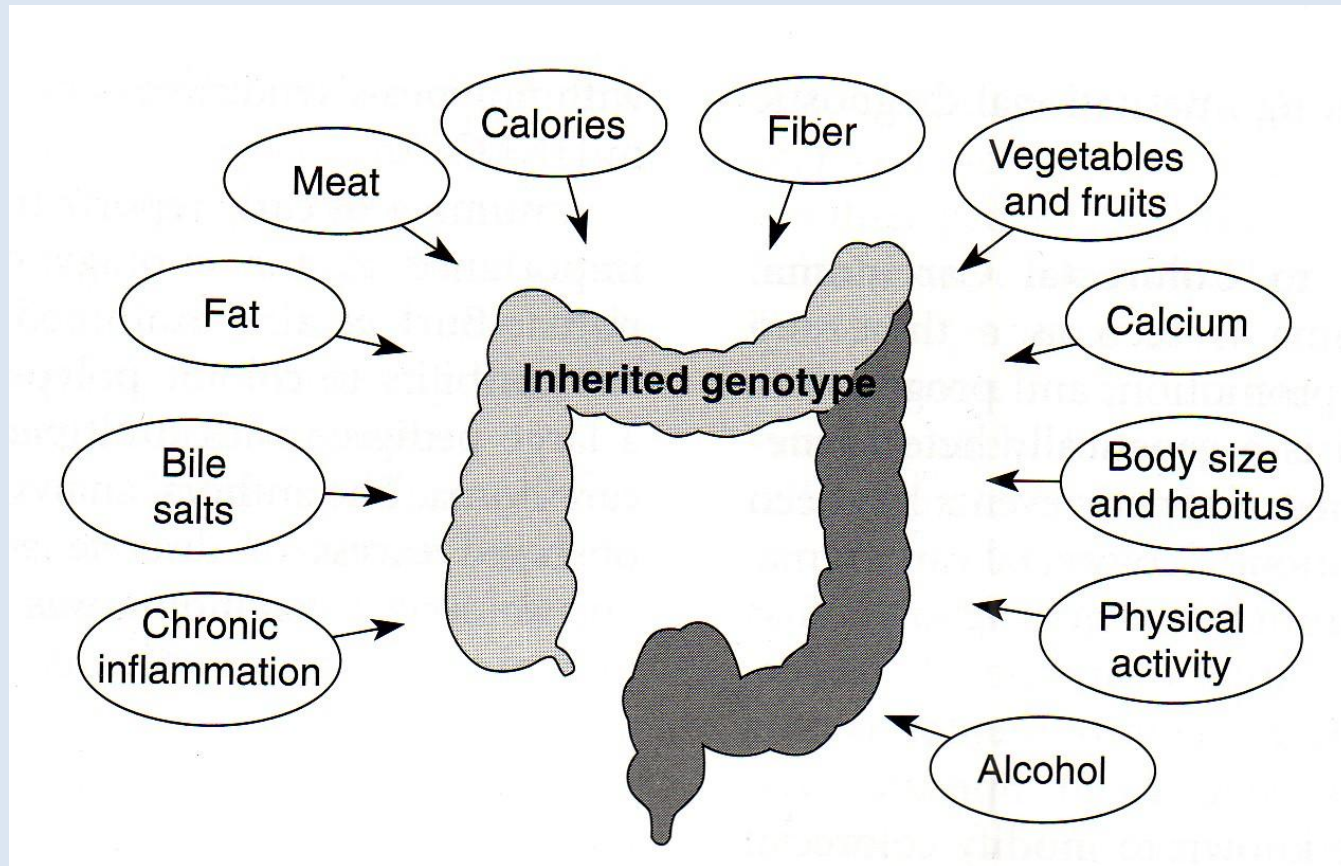
Adenocarcinoma 95%  
Carcinoid  
Lymphoma  
Sarcoma  
Squamous cell carcinoma



# Epidemiology

- 3<sup>th</sup> most common malignancy worldwide.
- 1<sup>st</sup> most common in Saudi males.
- second to lung cancer as a cause of cancer death
- 21,500 new cases, 8900 will die (2008)
- risk of CRC – women 1/16 , men 1/14
- peak incidence in 7<sup>th</sup> decade but it can occur at any age

# Etiology of Colorectal Cancer



# Risk Factors

## 1. Genetics, Family history

- Personal history
- One first degree family member doubles risk
- Hereditary colorectal cancer syndromes

## 2. Polyps

## 3. Inflammatory bowel disease

## 4. Other

- Diet, nutrients, smoking, ETOH

# Colorectal Cancer Risk Based on Family History

- General population 6%
- One 1st degree CRC 2-3X\* (12-18%)
- Two 1st degree CRC 3-4X\*
- One 1st degree CRC < 50 y 3-4\*
- One 2nd or 3rd CRC 1.5X
- 2 2nd degree CRC 2-3X\*
- 1 first degree with polyp 2X\*

# Clinical presentation

1. Bleeding - gross, occult, anemia (37%)
2. Change in bowel habit – pain, diarrhea, constipation, alternating pattern
3. Obstruction – more common with left sided lesions most common cause of bowel obstruction in the elderly
4. Vague abdominal pains
5. Change in caliber of the stools
6. Weight loss
7. Abdominal mass
8. Asymptomatic

# Investigations

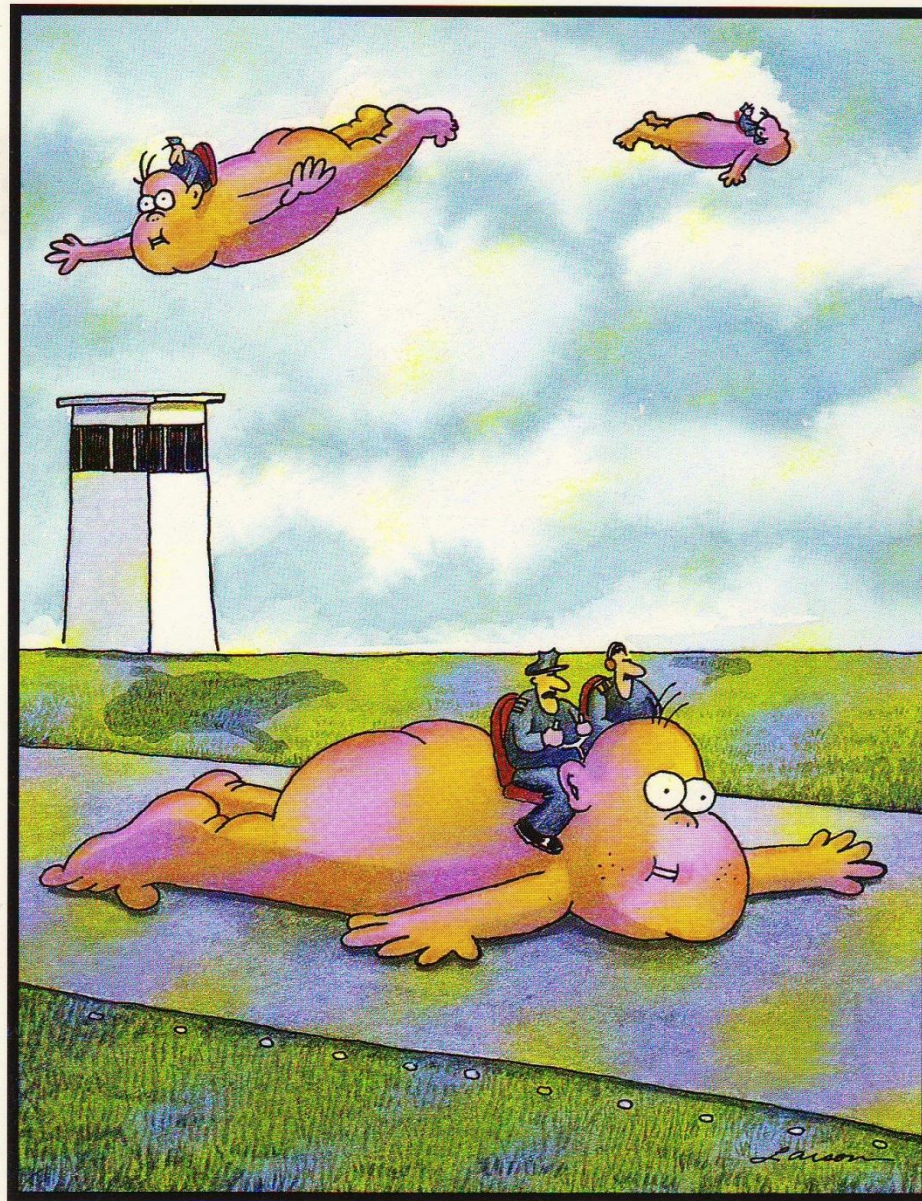
- General:
  - Complete history and physical (DRE)
- Endoscopic (identify primary, synchronous lesions)
  - Flexible sigmoidoscopy
  - Colonoscopy
- Staging
  - Endorectal ultrasound (rectal cancer)
  - Chest x-ray (metastases)
  - Liver ultrasound (metastases)
  - Abdominal CT scan (metastases)
- Bloodwork
  - CBC electrolytes, CEA (tumour marker)





# Surgical therapy

- Surgery is the most important variable in the treatment of colorectal cancer
- Radiation and chemotherapy alone cannot cure any stage of colorectal cancer
- The site of tumour dictates the basic procedure



"Fuel ... check. Lights ... check. Oil pressure ... check. We've got clearance. OK, Jack — let's get this baby off the ground."

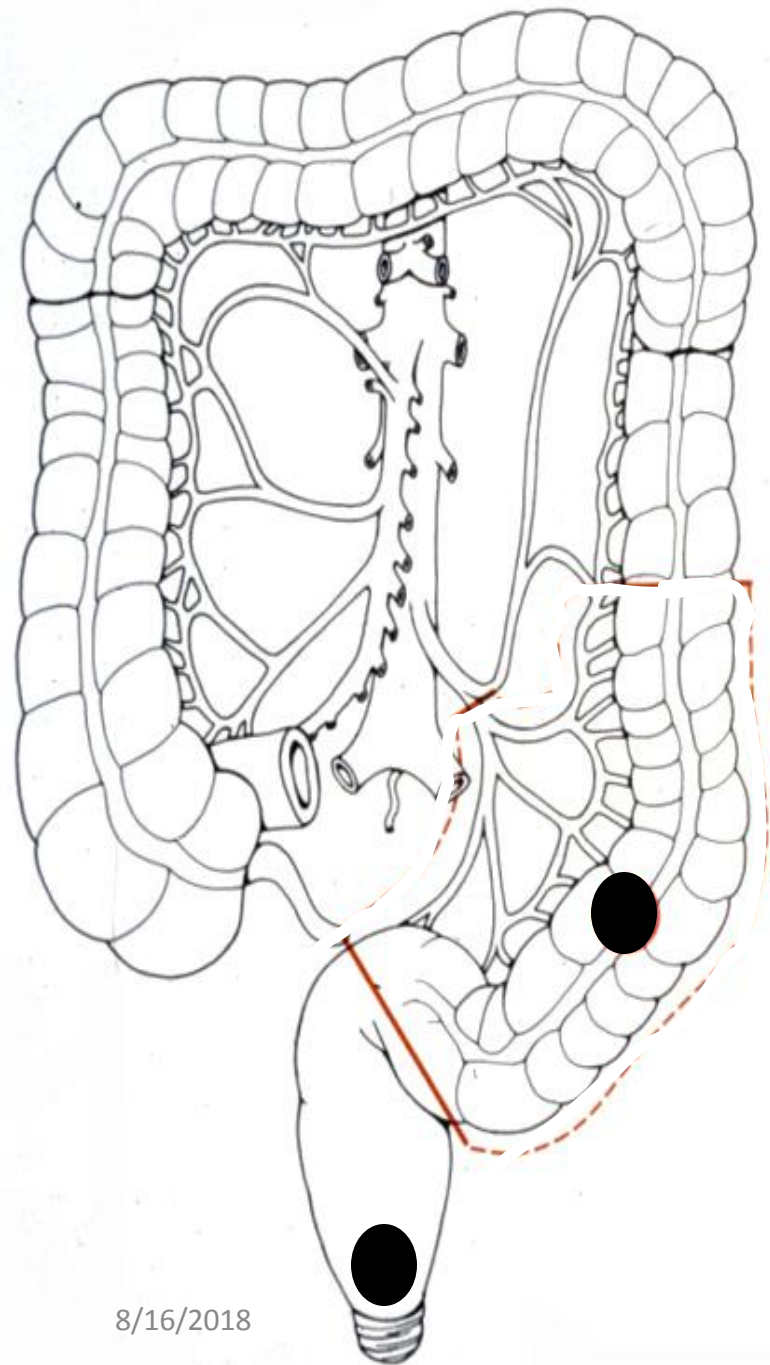
# Preoperative preparation

- Evaluation of medical problems
- Mechanical bowel preparation
  - Colyte , Oral fleet
- IV antibiotics
- DVT prevention ( blood clots in the legs)
  - Heparin shots
  - Compression stockings
- Foley catheter
- Epidural catheter for pain

# Principles of Surgery

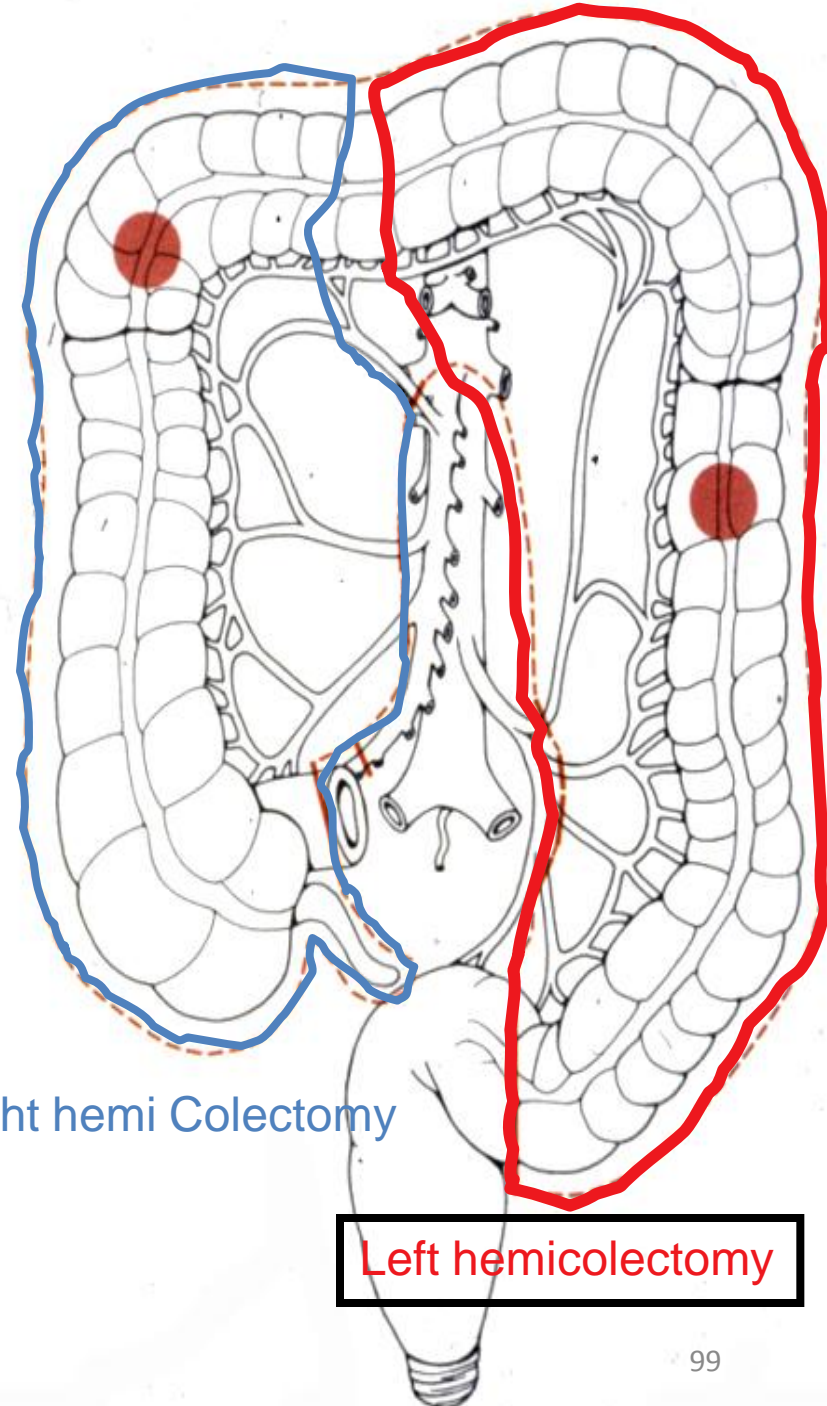
- Examine the entire abdomen
- Remove the appropriate segment of the colon with adequate margins
- Remove the corresponding lymph nodes
- Open vs laparoscopic approach





8/16/2018

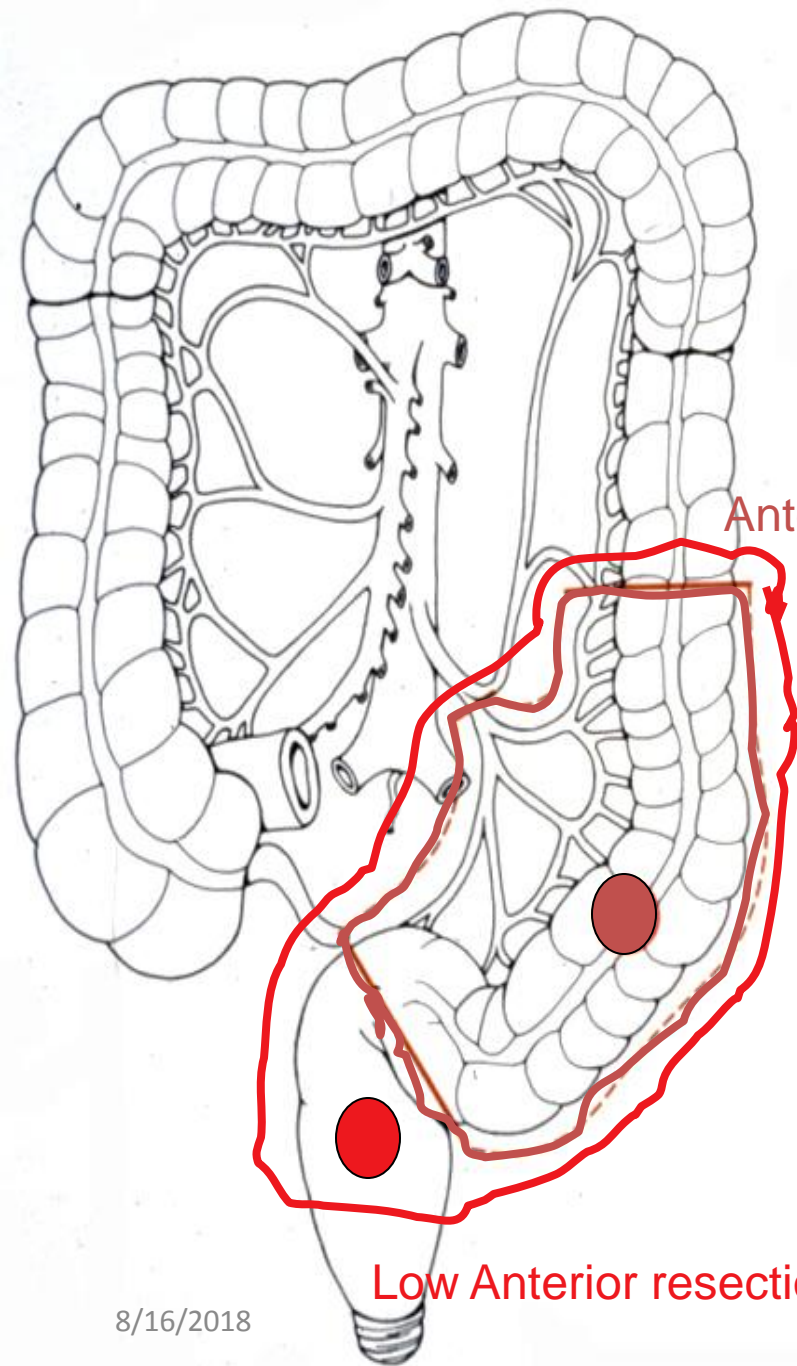
M.A.Kubtan



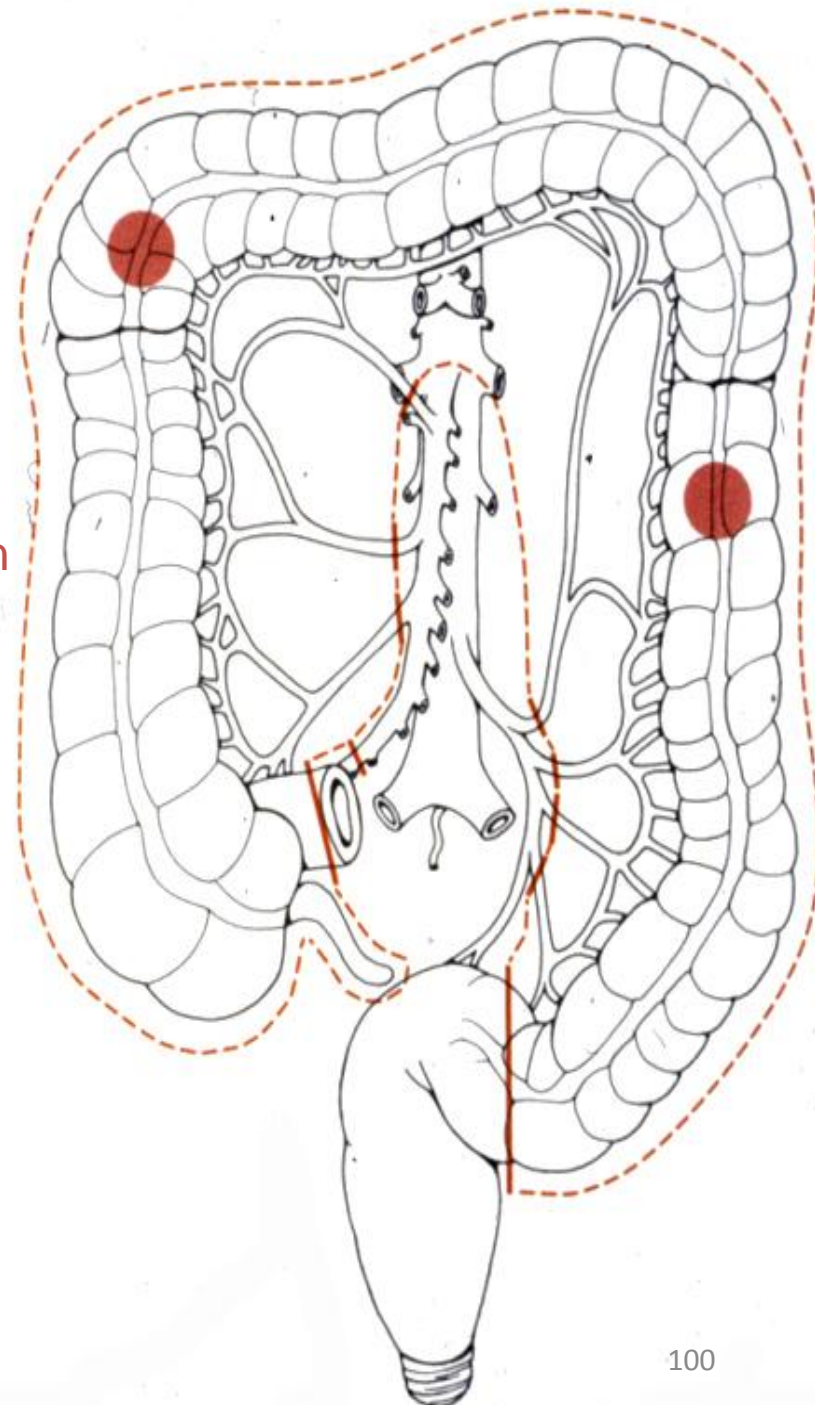
Right hemi Colectomy

Left hemicolectomy

99



Anterior resection



# Ostomy

- The intestine is brought out through a hole in the abdominal wall

## Colostomy ( colon on the skin)

- Permanent when the rectum is removed
- Temporary when it is unsafe to make a join

## Ileostomy ( ileum on the skin)

- Temporary when the join needs time to heal

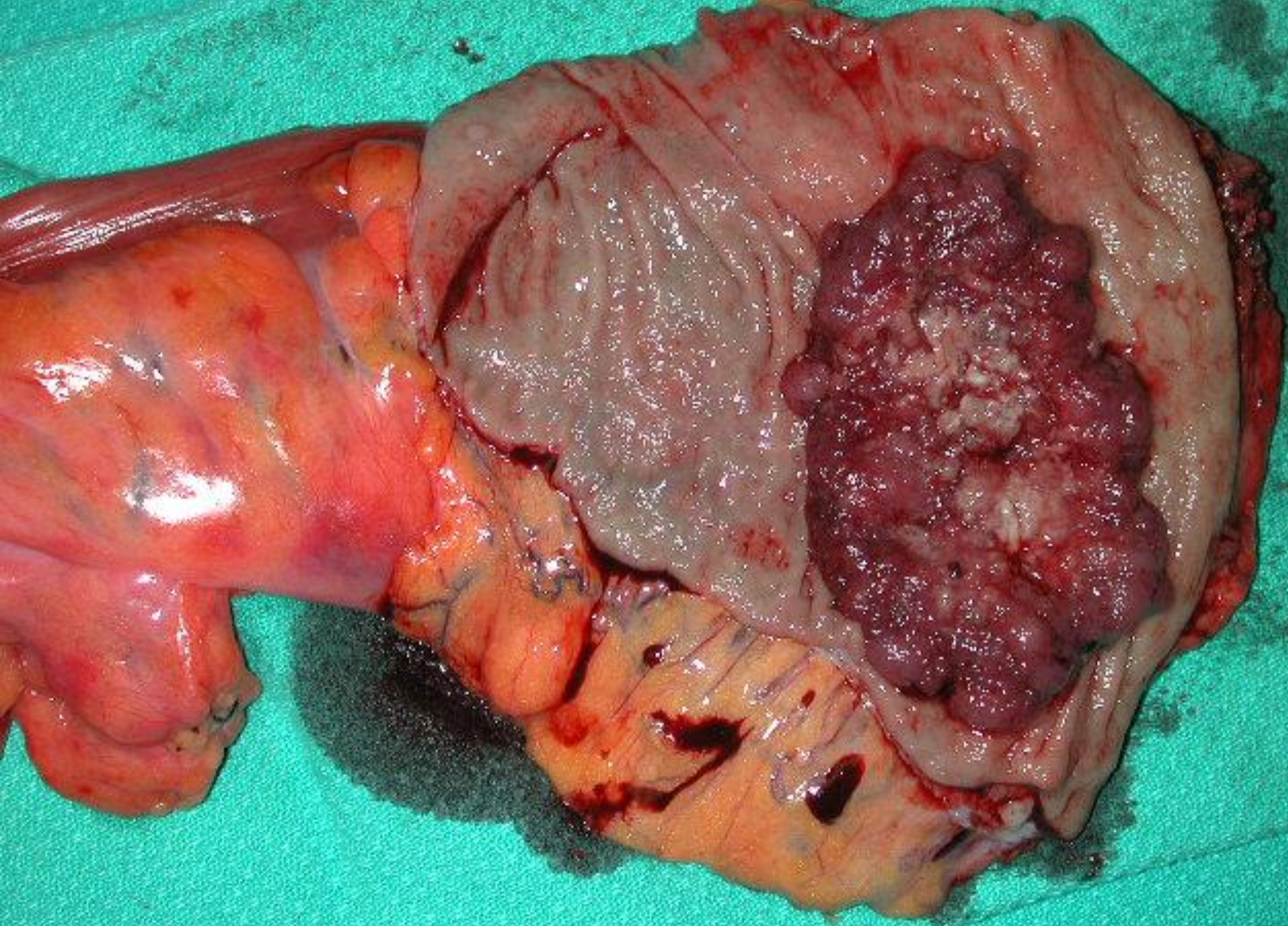




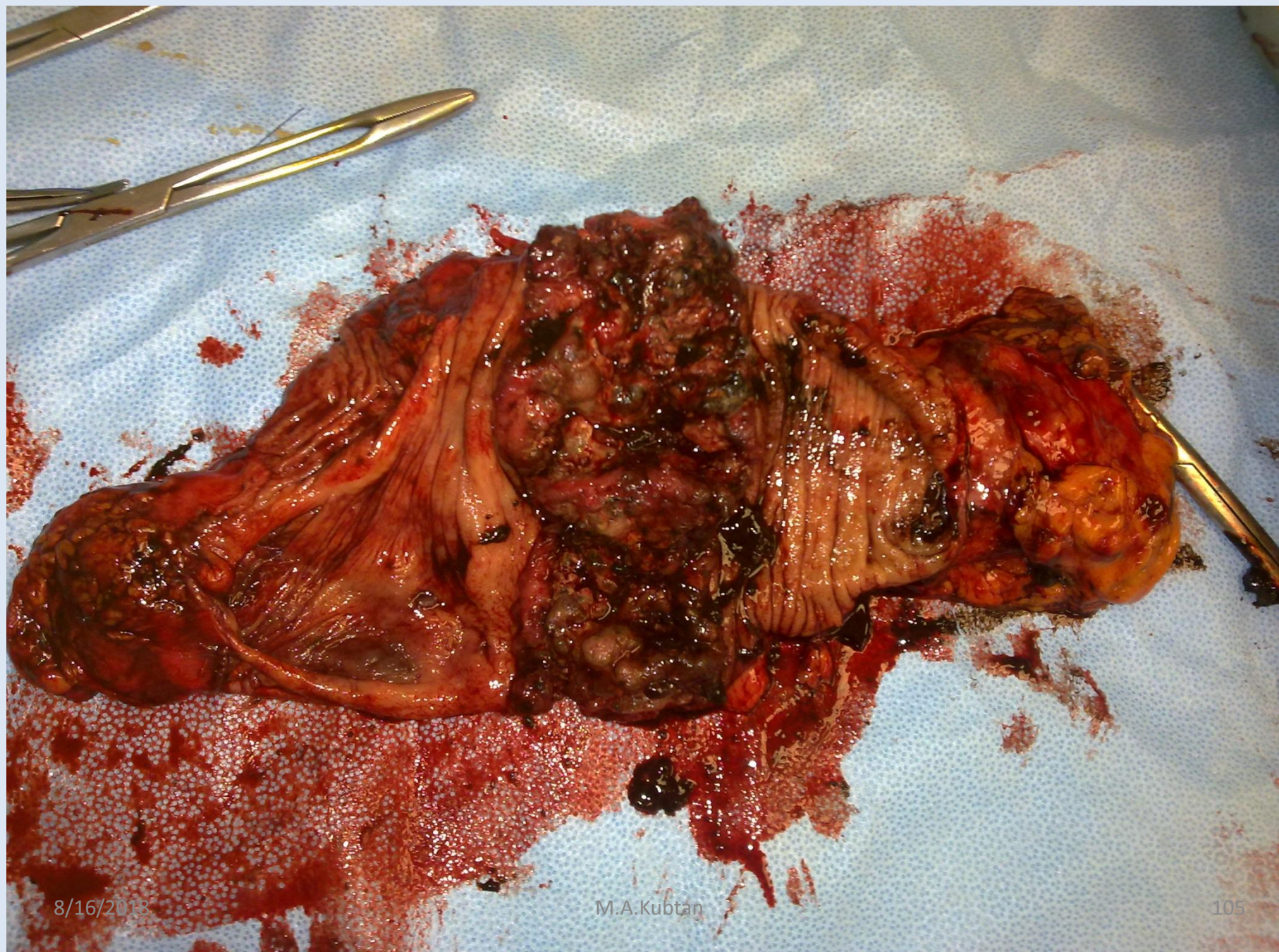












# Recovery

- Surgery 2 to 4 hours
- Hospital stay 4 to 10 days
  - IV, urine catheter, compression stockings, intravenous pain killers, blood thinner
  - Discharge when ambulating, eating, bowel function, good pain control
- Recovery 4 weeks

# Follow up

- Office visit every 3 months for two years then every 6 months for 3 years
- Regular blood work (CEA)
- Colonoscopy at year 1 and 4 and every 5 years
- CT scan yearly



# Pathology of Colorectal Cancer

- Macroscopic:
- Microscopic (differentiation):
  - Well
  - Moderately
  - Poorly
- Lymph node involvement



# Staging ( Where is it Growing?)

## 1. How far into the wall has it grown? T stage

- Tis - invasion of mucosa only
- T1 - Invasion of submucosa
- T2 - Invasion of muscularis propria
- T3 - Full thickness/perirectal fat
- T4 - Invasion into adjacent organs

# Staging ( Where is it Growing?)

2. Is it growing in other places?    N  
stage, M stage

- N1 - 1-3 lymph nodes
- N2 - >4 lymph nodes
- N3 - distant lymph nodes
- M1 - Distant organ ( liver, lung)

# TNM Staging

- Stage 0 - Tis tumors
- Stage 1 - T1 and T2 tumors
- Stage 2 - T3 and T4 tumors
- Stage 3 - Any lymph node involvement
- Stage 4 - Distant metastases

# Who Gets Additional Treatment?

- COLON

- All stage 3 patients (positive nodes) - chemotherapy
- ?High risk stage 2 patients

- RECTUM

- All stage 2 and stage 3 patients should get radiation and chemo

# Survival and TNM Stage

- | <u>STAGE</u> | <u>5-Year Survival</u> |
|--------------|------------------------|
| 1            | 90%                    |
| 2            | 80%^                   |
| 3            | 27-69%*                |
| 4            | 8%                     |

^for T3N0 tumors

\*depends on # of nodes involved

# Summary

1. Common Cancer
2. Can be prevented through screening and resection of polyps
3. Surgery is the primary treatment
4. Slow but steady improvement in survival



"Mr. Osborne, may I be excused?  
My brain is full."