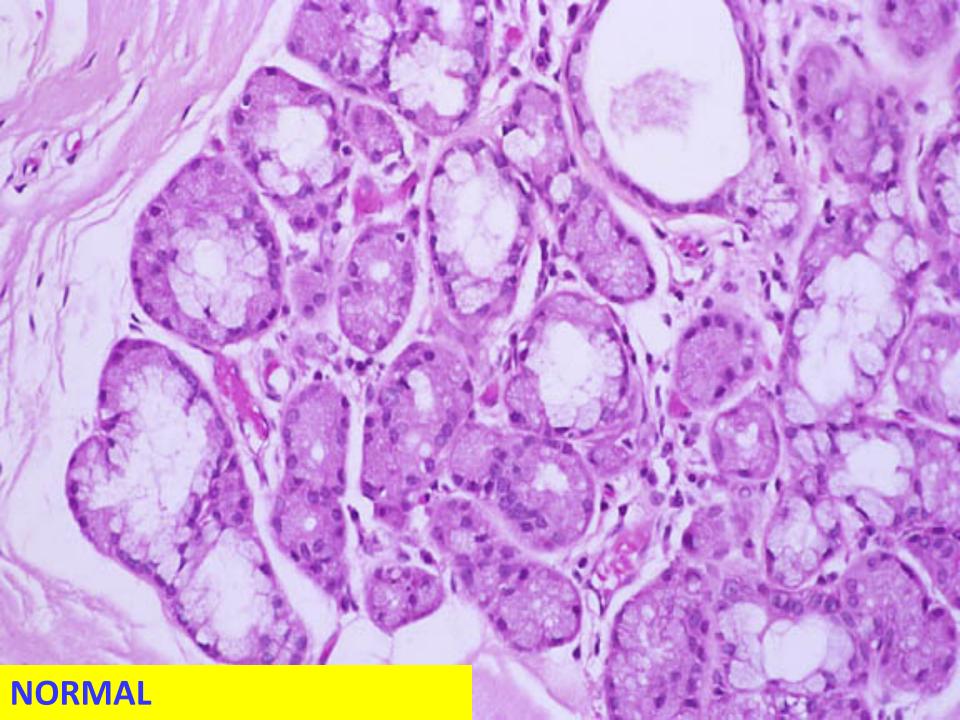
GNT Block Pathology Practical

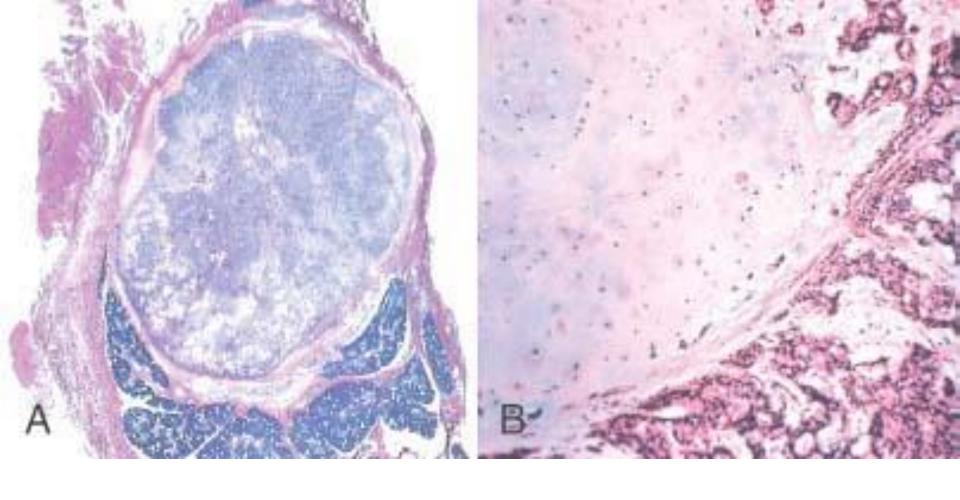
Salivary gland
Esophagus
Stomach
Small intestine
Large intestine

SALIVARY GLAND



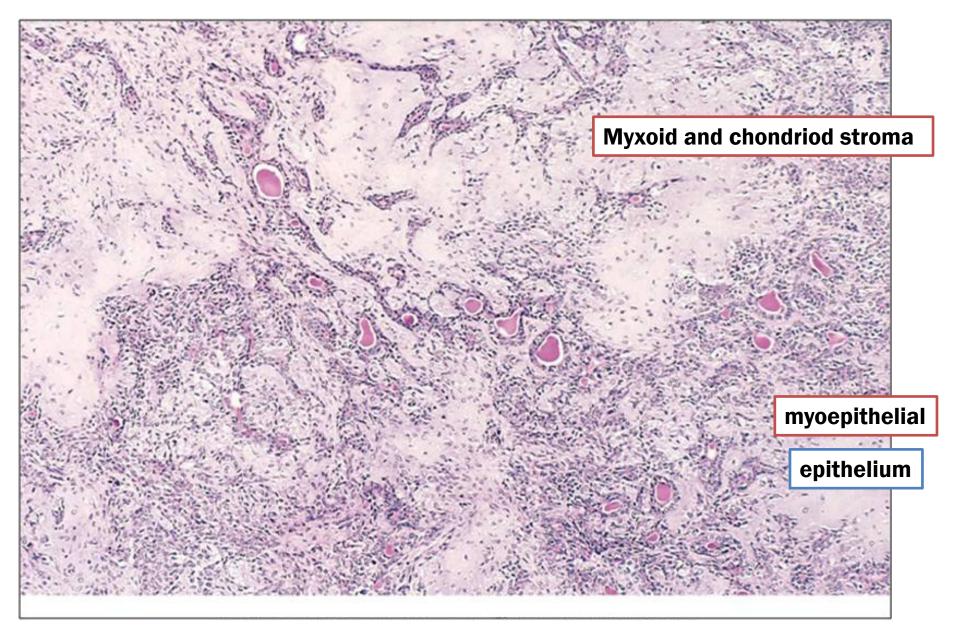


R



PLEOMORPHIC ADENOMA

i.e., MIXED TUMOR



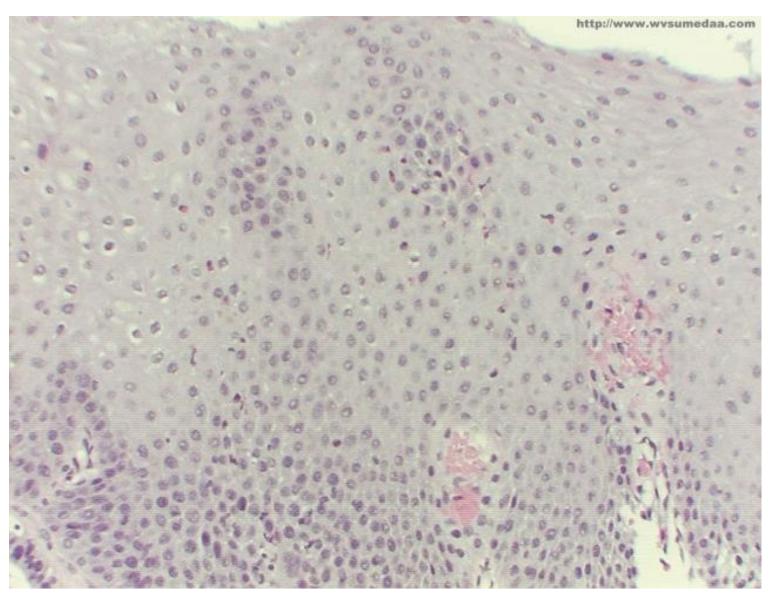
Mixed tumor of the parotid gland contains epithelial cells forming ducts and myxoid stroma that resembles cartilage.

Pleomorphic adenoma of the salivary gland: Section shows an incomplete fibrous capsule separating the tumour from normal salivary gland:

- Tumour shows mixed cellular components like epithelial, myoepithelial, chondriod and myxoid elements.
- Epithelial areas shows small ducts, acini and strands or sheets of cells.
- Myxoid areas are formed of loose myxomatous tissue and chondriod areas consists of pale blue matrix.

Esophagus

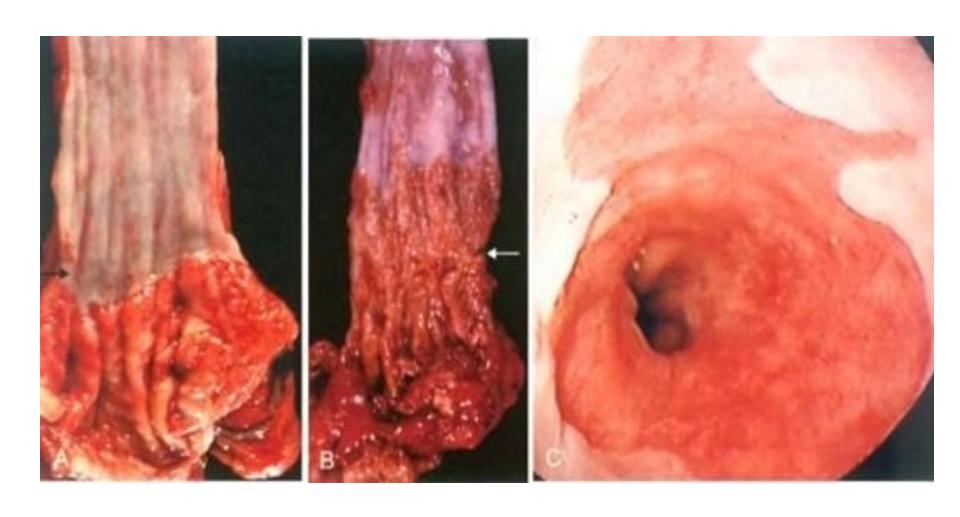
REFLUX/GERD

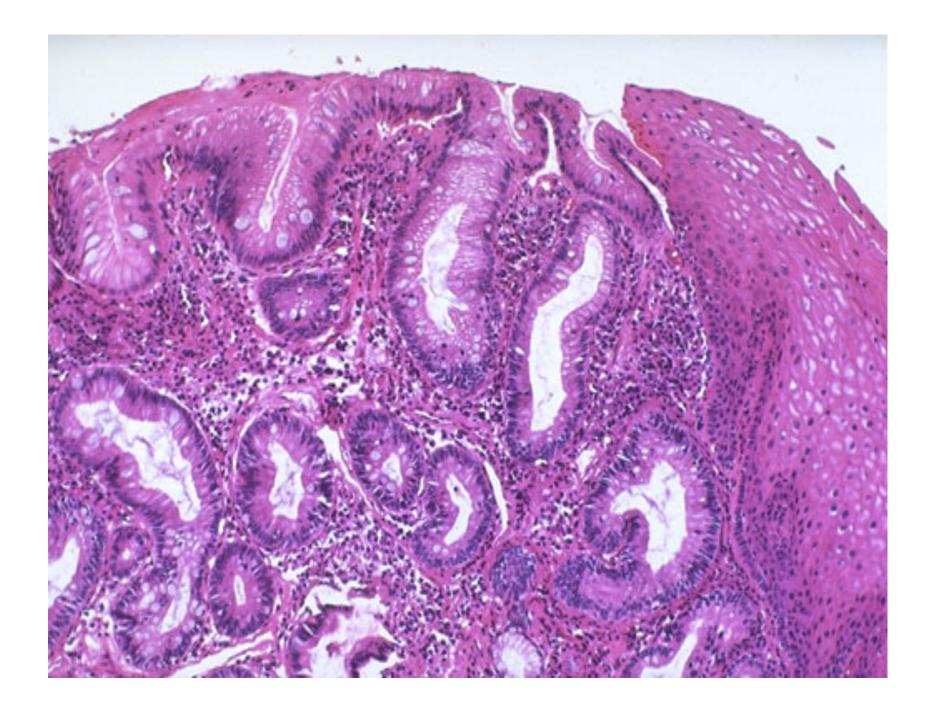


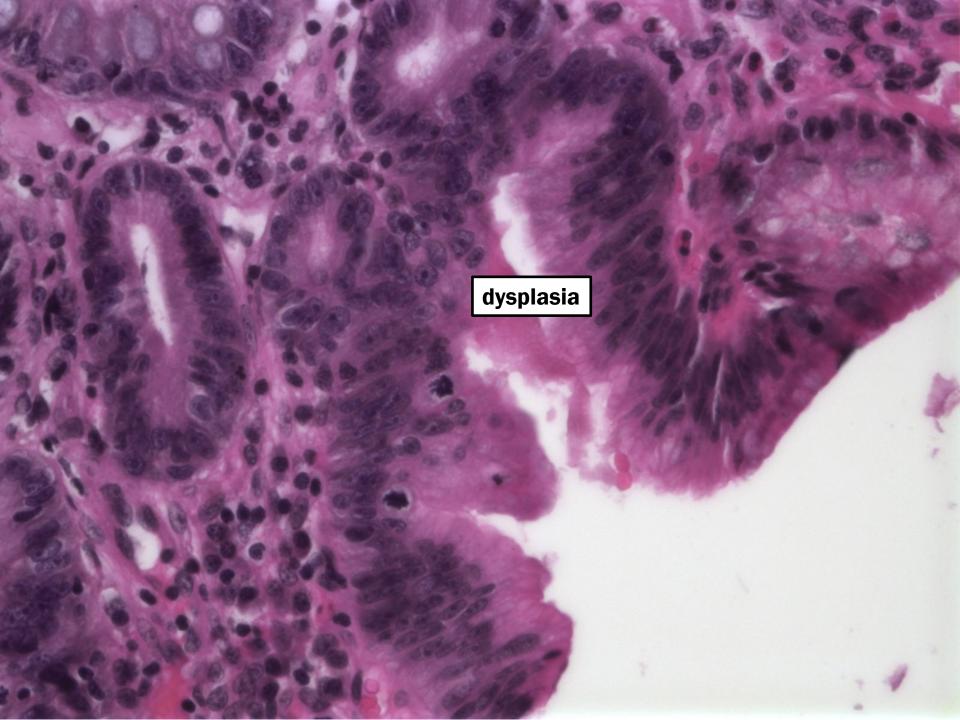
REFLUX/GERD

- Inflammatory Cells
 - -Eosinophils
 - -Neutrophils
 - **–Lymphocytes**
- Basal zone hyperplasia
- Lamina Propria papillae elongated and congested

BARRETT'S ESOPHAGUS



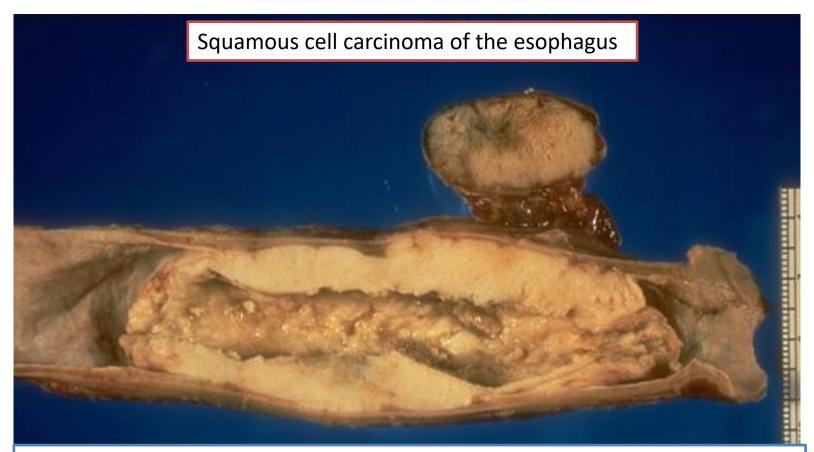




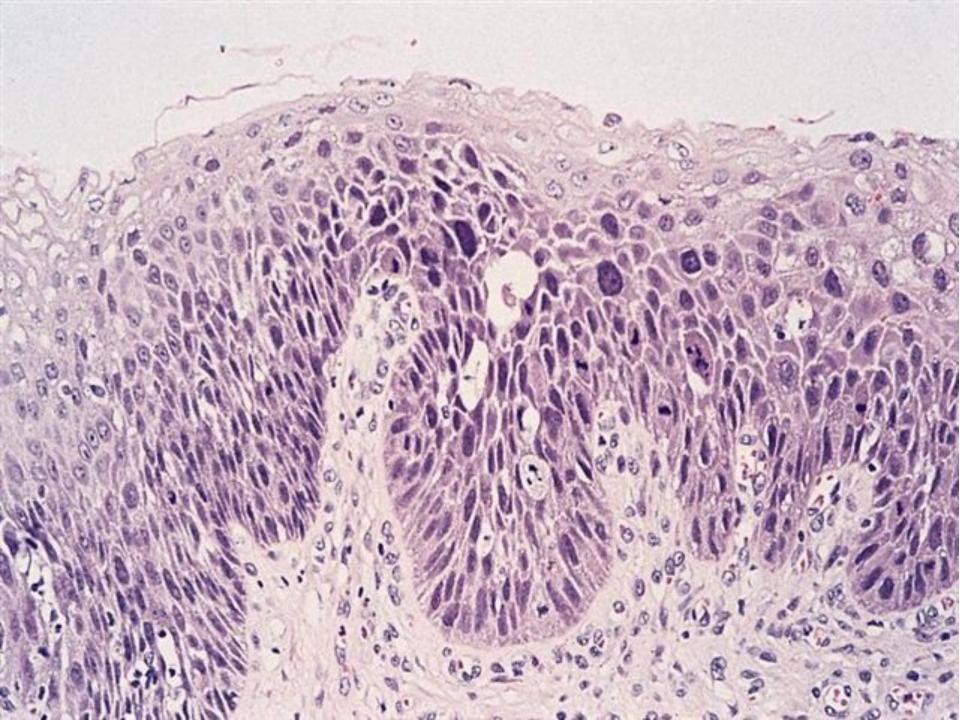
BARRETT'S ESOPHAGUS

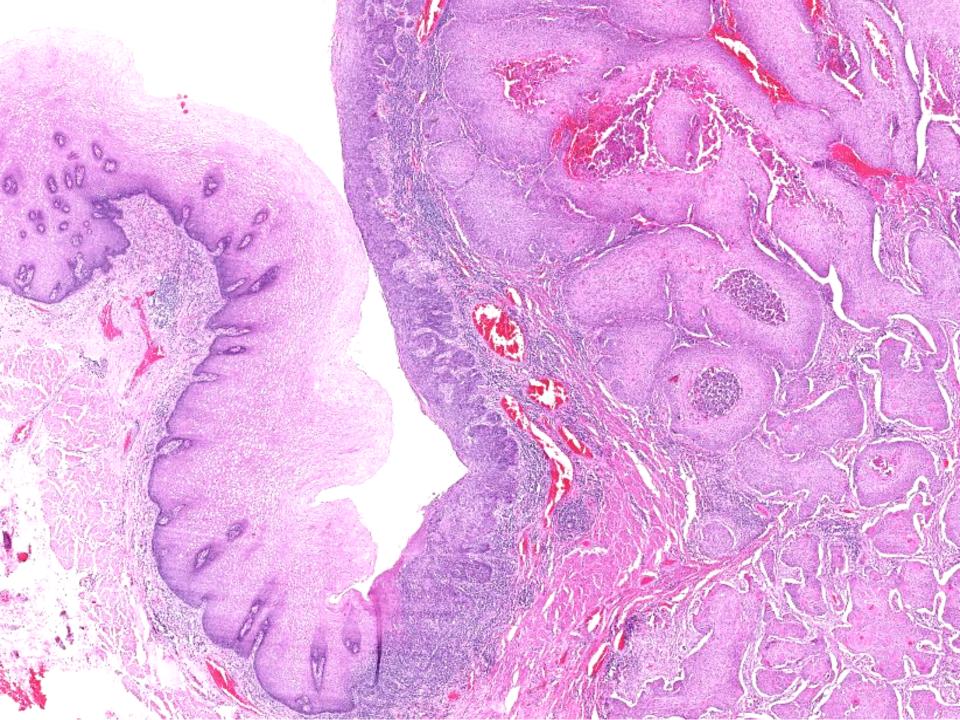
- INTESTINALIZED (GASTRICIZED) mucosa is AT RISK for glandular dysplasia.
- Searching for dysplasia when BARRETT's is present is of utmost importance
- MOST/ALL adenocarcinomas arising in the esophagus arise from previously existing BARRETT's

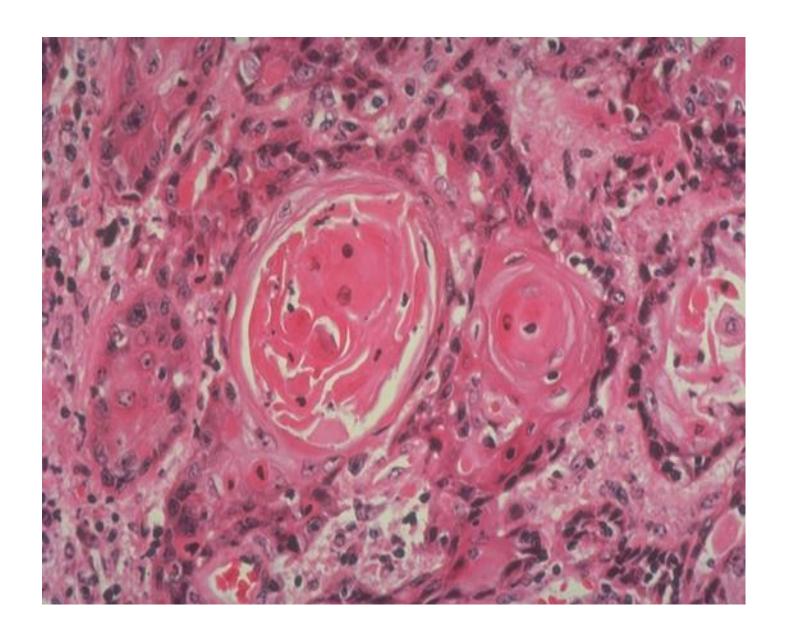
Carcinoma of the esophagus



Esophageal squamous cell carcinoma is associated with alcohol and tobacco use, poverty, caustic esophageal injury, achalasia, tylosis, and Plummer-Vinson syndrome.





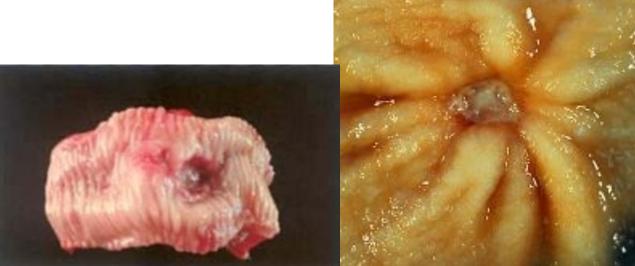


Stomach

Chronic gastric ulcer

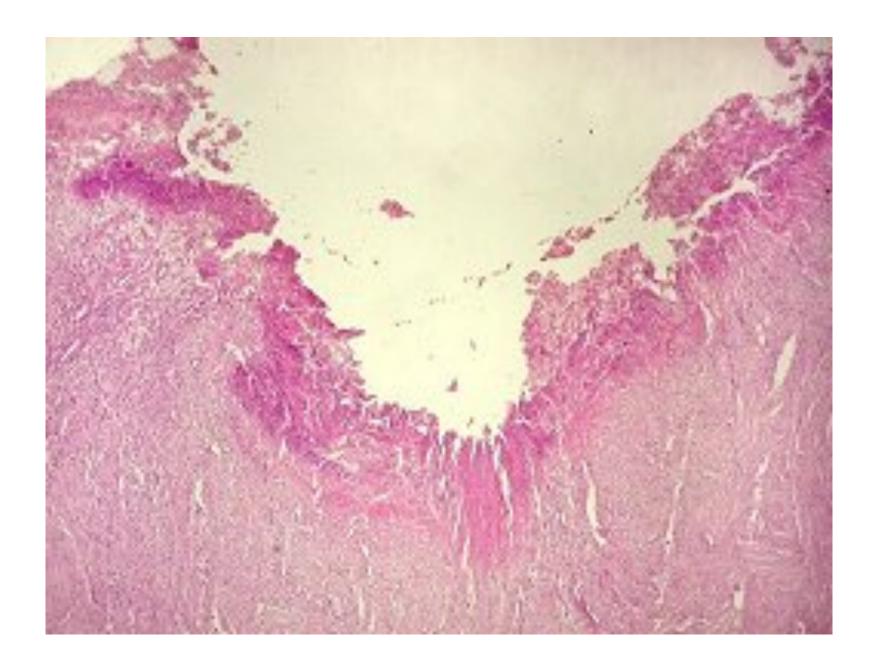
"PEPTIC" ULCERS

- "PEPTIC" implies acid cause/aggravation
- ULCER vs. EROSION (muscularis mucosa intact)
- MUC→SUBMUC→MUSCULARIS→SEROSA
- Chronic, solitary (usually), adults
- 80% caused by H. pylori in stomach
- 100% caused by H. pylori in duodenum
- NSAIDS "STRESS"





Chronic gastric ulcer



The Base of a Nonperforated Chronic Peptic Ulcer

Necrosis (N)

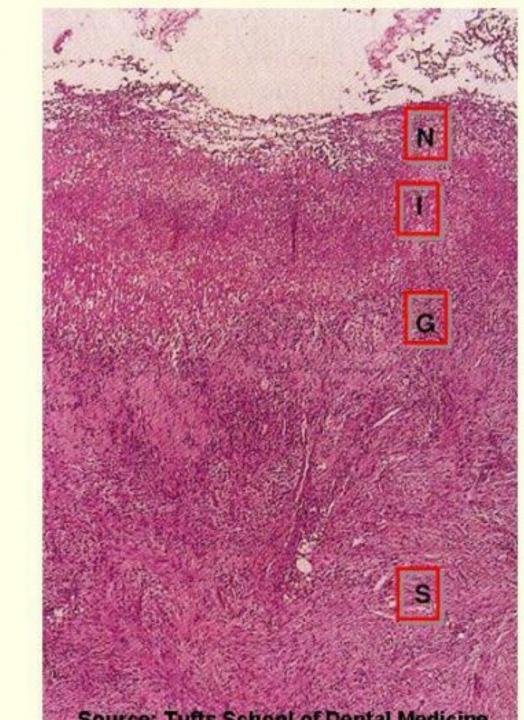
Inflammation (I)

Granulation tissue (G)

Scar (S)

(Top - luminal surface,

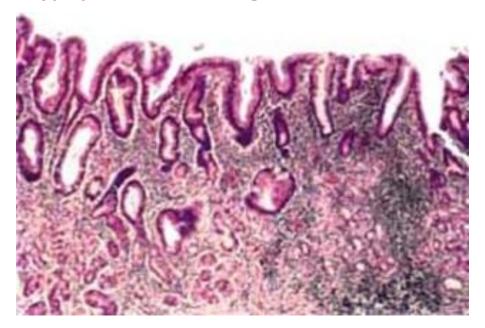
Bottom - muscular wall)

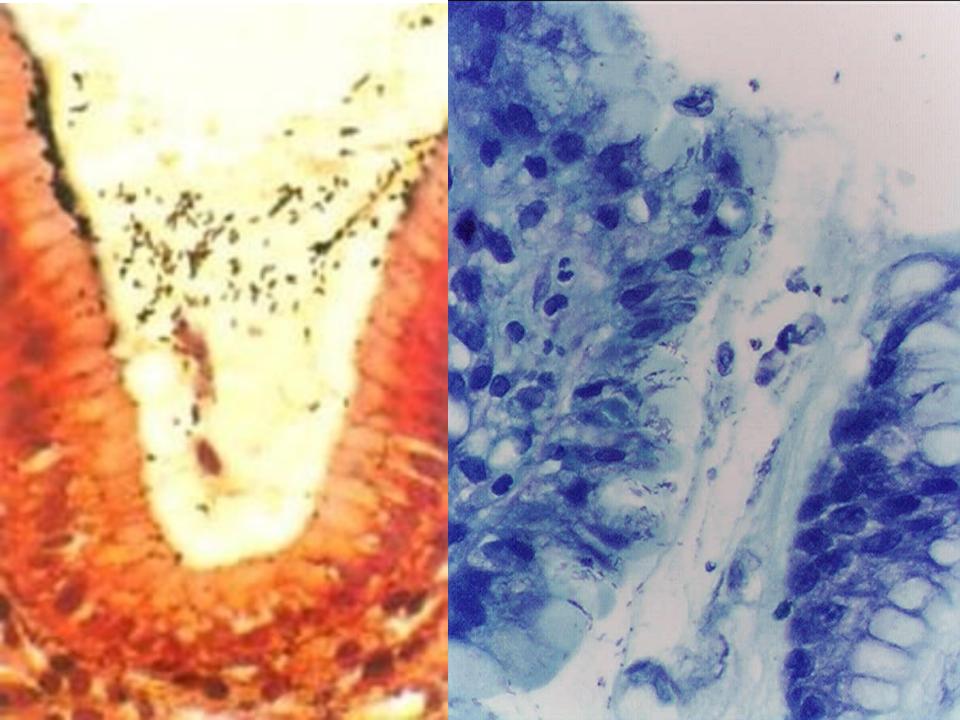


Gastritis Helicobacter induced

GASTRITIS

- CHRONIC, NO EROSIONS, NO HEMORRHAGE
- Perhaps some neutrophils
- Lymphocytes, lymphoid follicles
- REGENERATIVE CHANGES
 - METAPLASIA, intestinal
 - ATROPHY, mucosal hypoplasia, "thinning"
 - DYSPLASIA



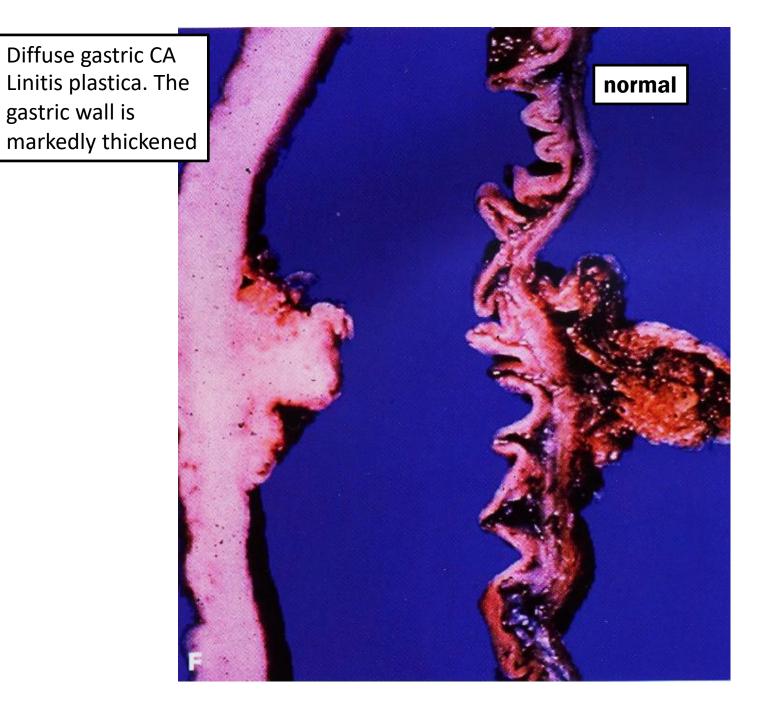


Carcinoma of the stomach

Two types:

1.Diffuse

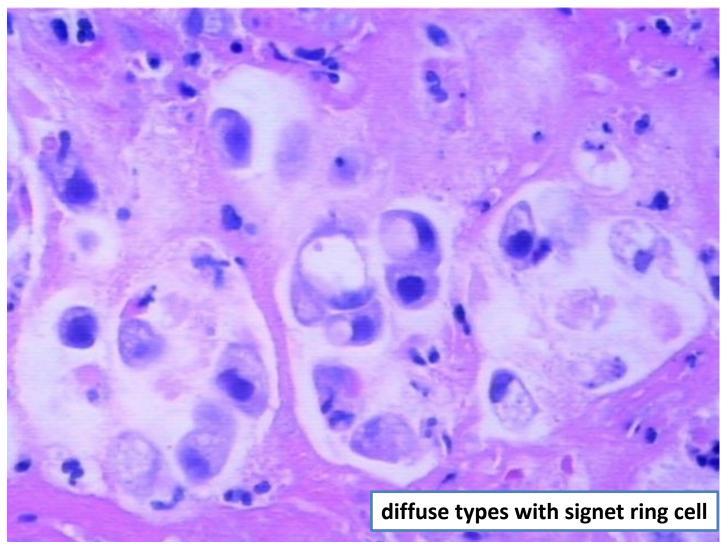
2. Intestinal



ADENOCARCINOMA DIFFUSE GROWTH PATTERNS

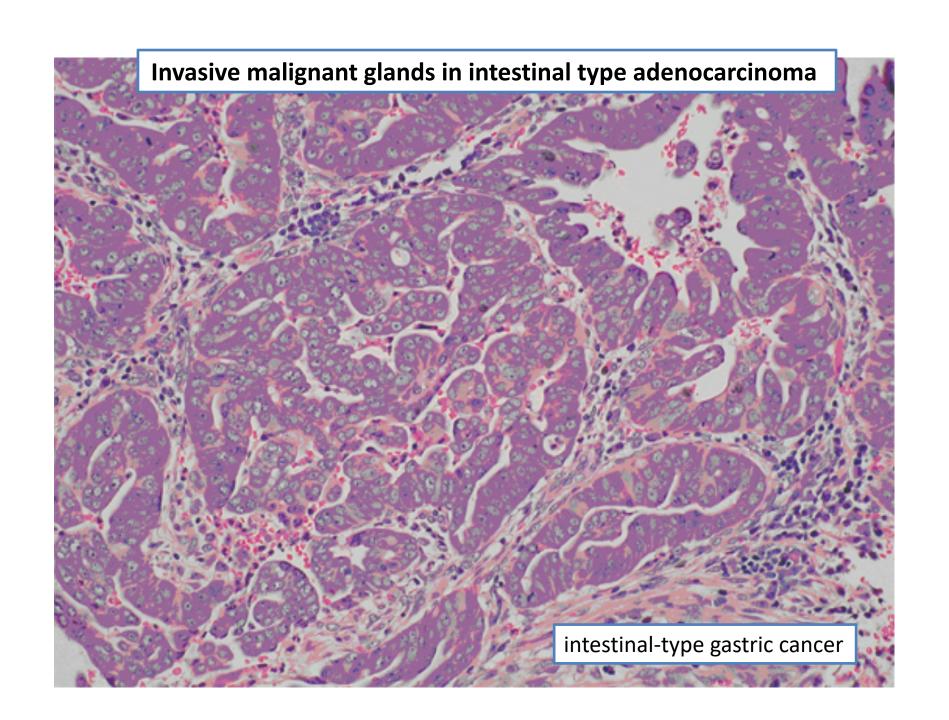


Gastric adenocarcinoma of the diffuse signet ring cell type



Intestinal-type adenocarcinoma of stomach consisting of an elevated mass with heaped-up borders and central ulceration

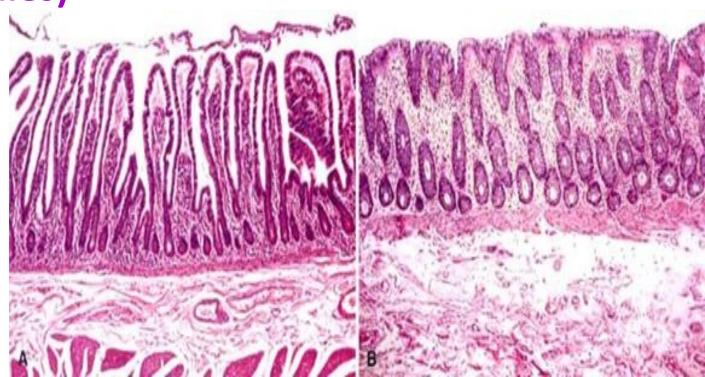




Small intestine

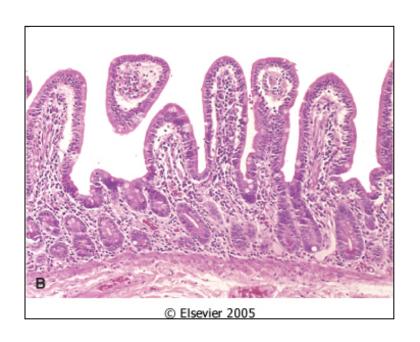
MUCOSA

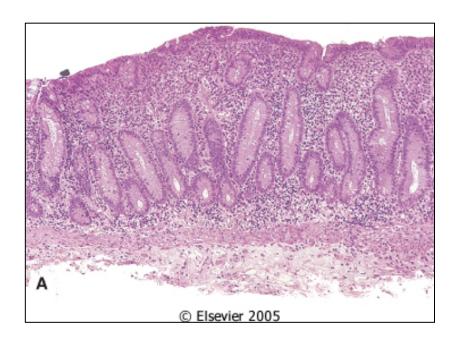
- SI: ABSORPTIVE, MUCUS, PANETH (apical granules)
 - VILLI
- LI: MUCUS, ABSORPTIVE, ENTEROENDOCRINE (basal granules)
 - CRYPTS



Gross and histopathology

Celiac disease



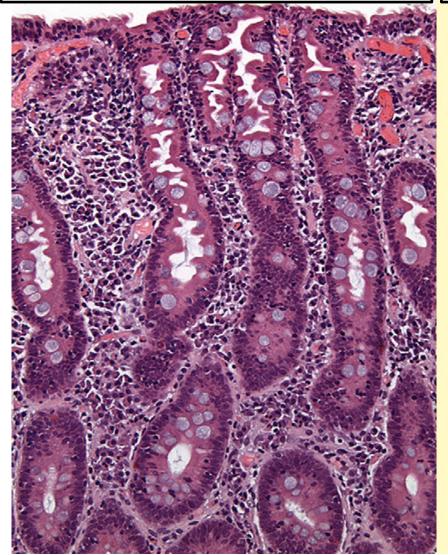


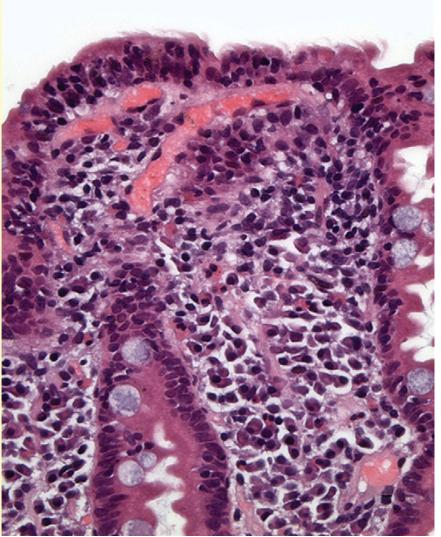
Villous length to crypt length 3/1

1/28/21 38

Low-power view of fully developed celiac disease. Note the elongated crypts with complete lack of villi.

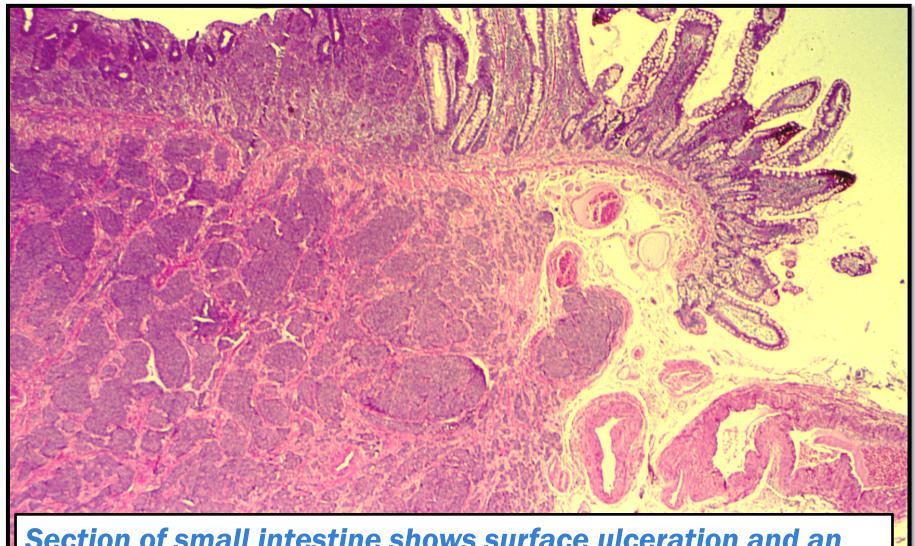
damaged surface epithelium with large numbers of intraepithelial lymphocytes.





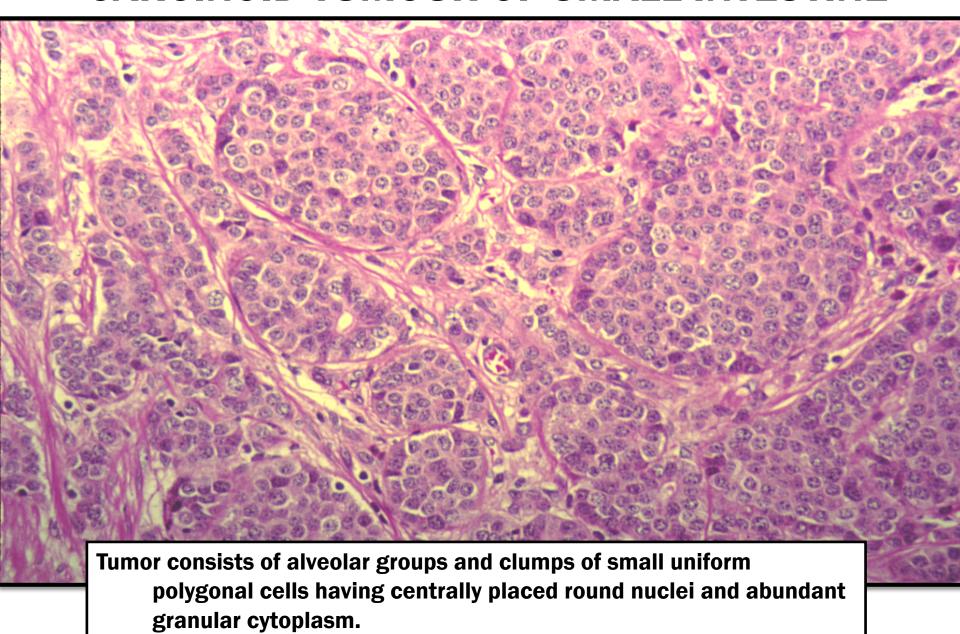
Carcinoid tumour

CARCINOID OF SMALL INTESTINE

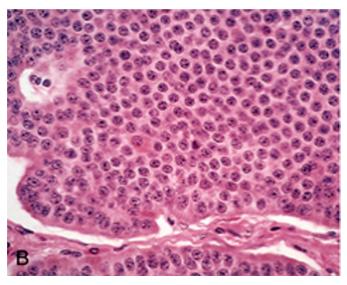


Section of small intestine shows surface ulceration and an infiltrating tumour mass in mucosa and submucosa

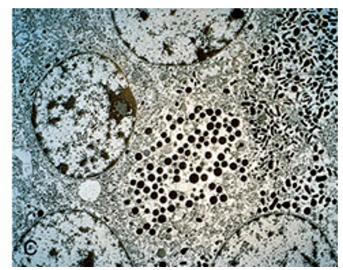
CARCINOID TUMOUR OF SMALL INTESTINE



CARCINOID TUMOUR



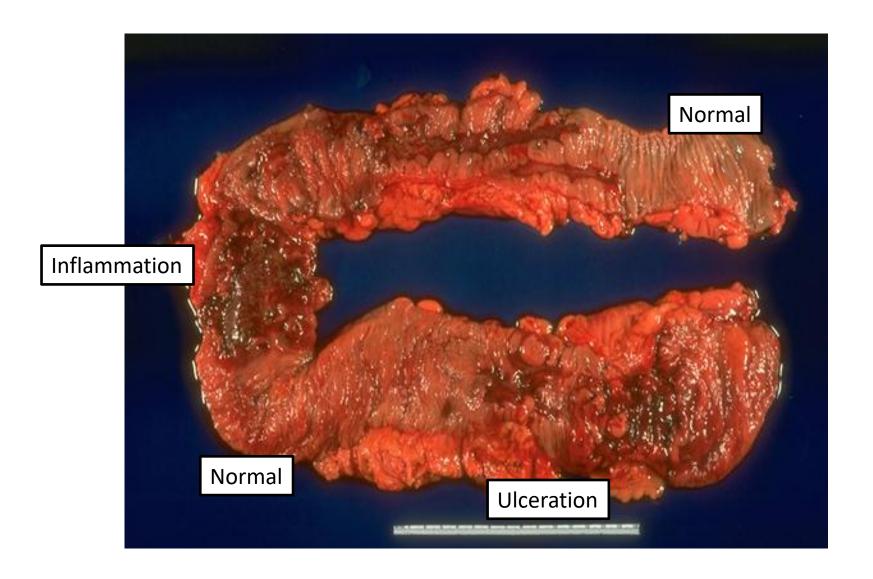
 Monotonously similar cells having a scant pink granular cytoplasm and a round-to-oval stippled nucleus



 Ultrastructral features: neurosecretory electron dense bodies in the cytoplasm

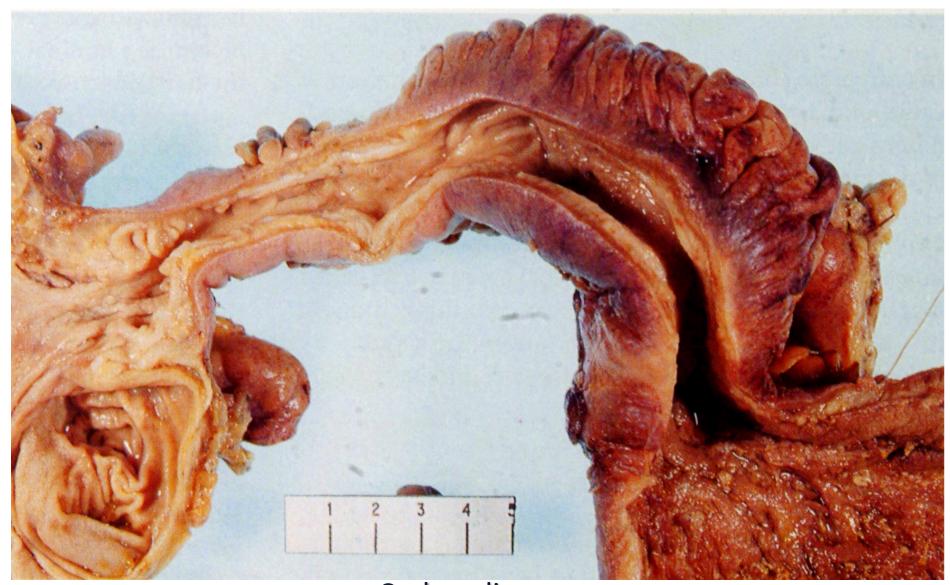
Large intestine

Crohn's disease



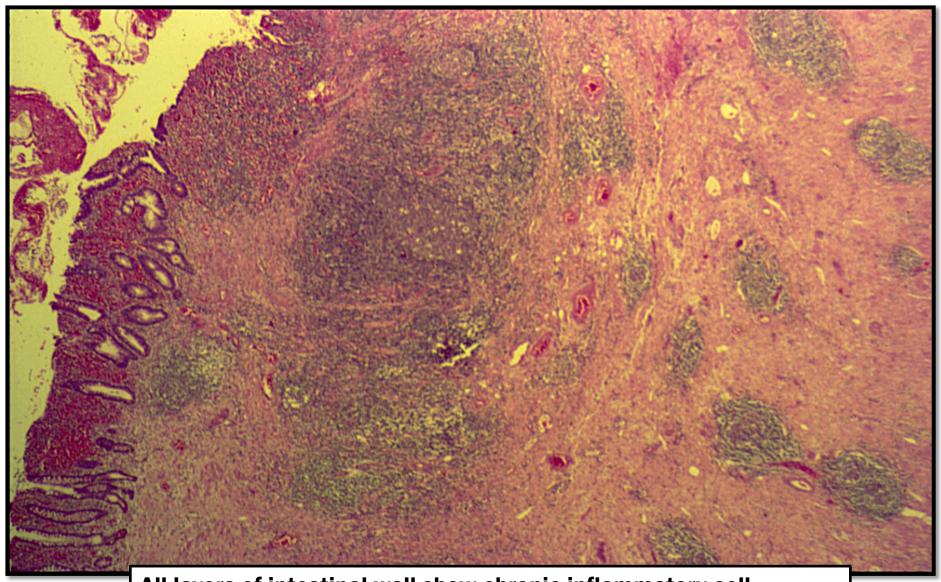


Crohn,'s disease
Linear mucosal ulcers and
thickened intestinal wall.



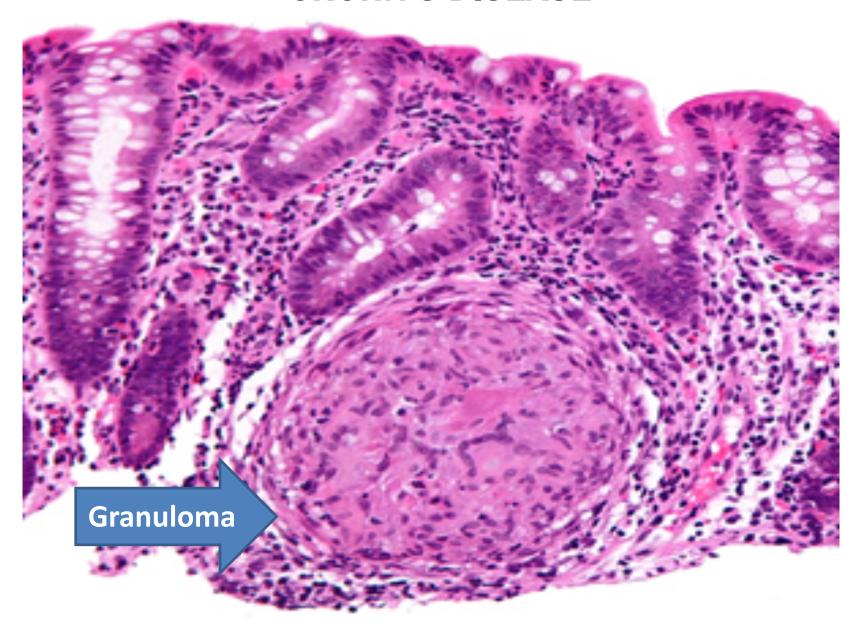
Crohns disease alternating normal and ulcerating mucosa

CROHN'S DISEASE (LARGE BOWEL)



All layers of intestinal wall show chronic inflammatory cell infiltrate (transmural), lymphoid aggregates and mild fibrosis

CROHN'S DISEASE

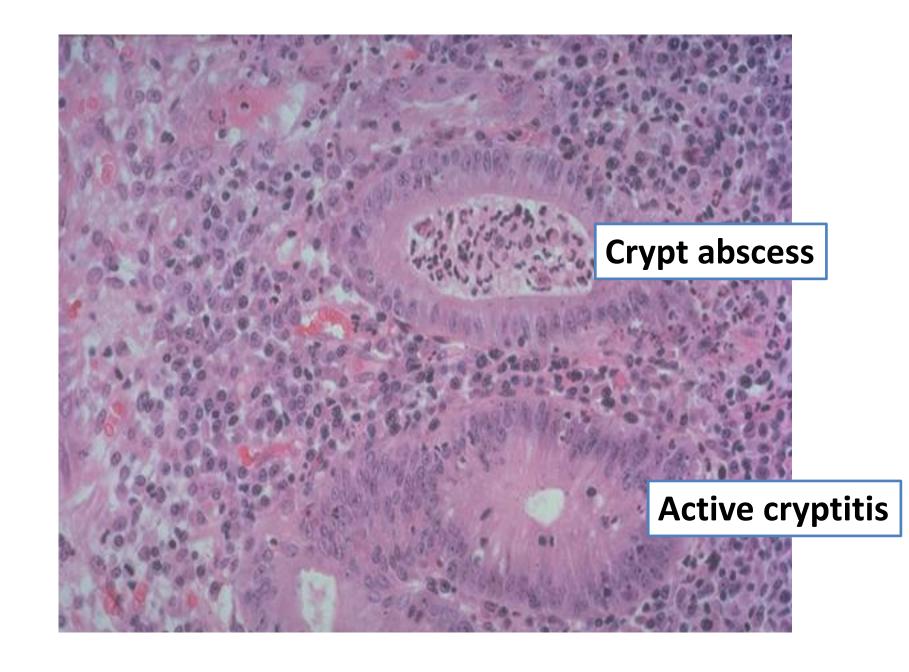


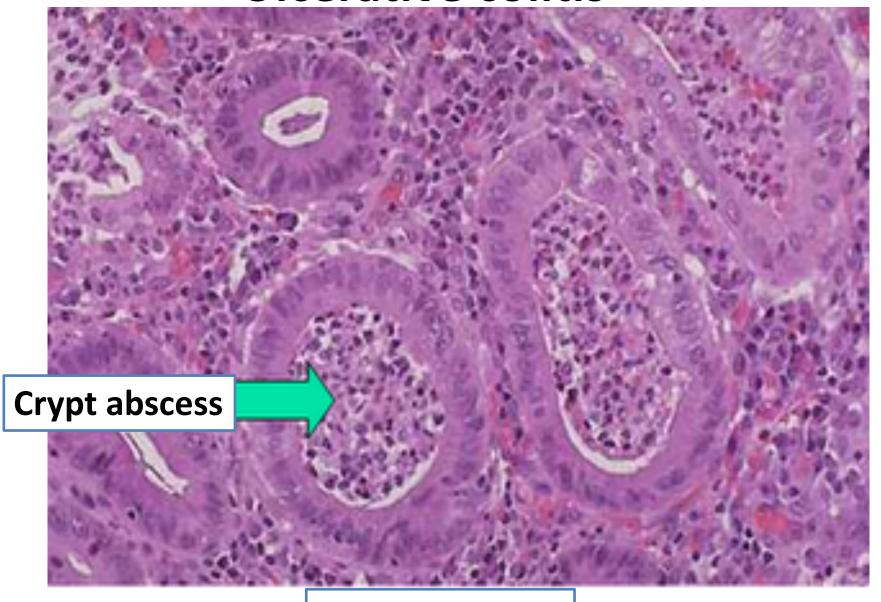


The entire colon is abnormal, and the usual transverse rugal folds have been almost completely effaced.

large bowel show relatively superficial ulcers lined by acute inflammatory exudate. Marked oedema and vascular congestion are seen in the intact mucosa

Inflammatory pseudopolyp





No granulomas

Polyps of rectum / colon

Non-neoplastic polyps 90%

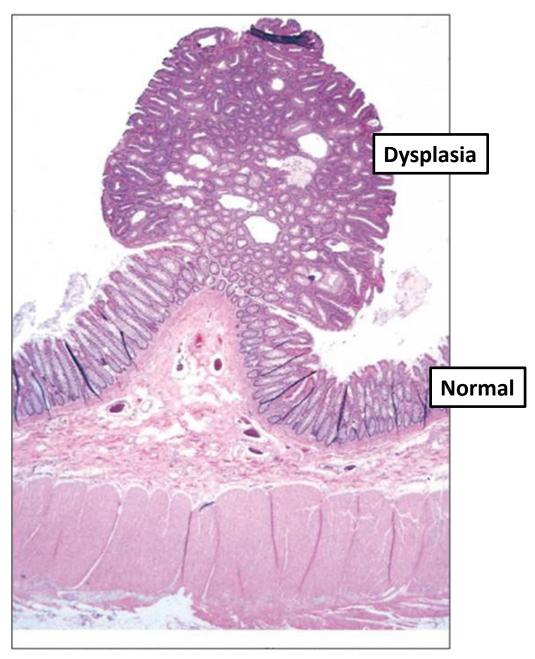
Hyperplastic polyps
Hamartomatous polyps (Juvenile & Peutz-Jeghers polyps)
Inflammatory polyps
Lymphoid polyps

Neoplastic polyps 10%
Adenoma

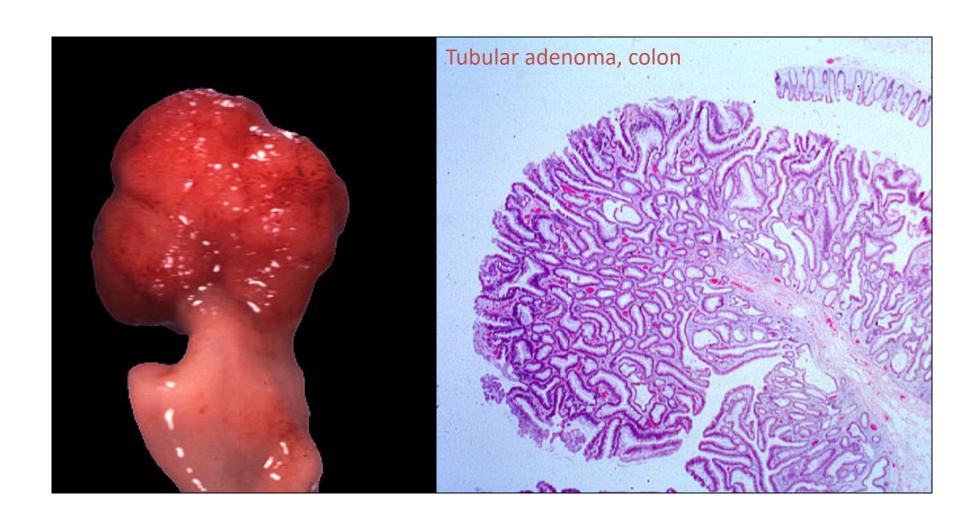
Adenomatous polyp of rectum / colon



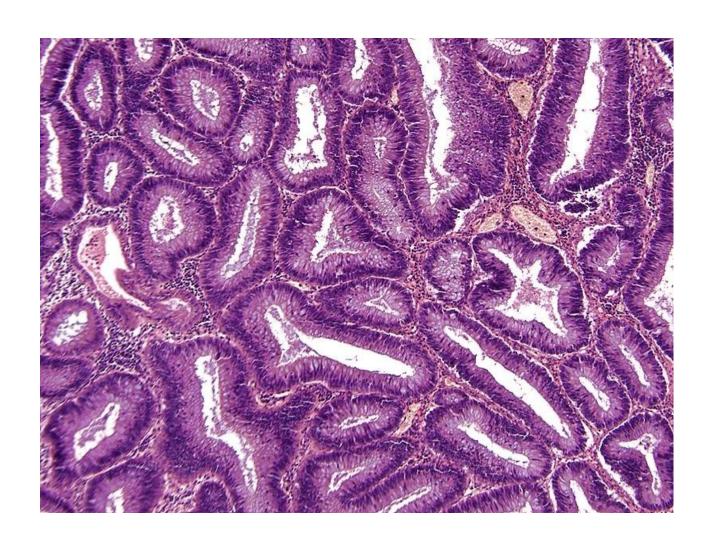
Organ: Colon Dx: Tubular adenoma



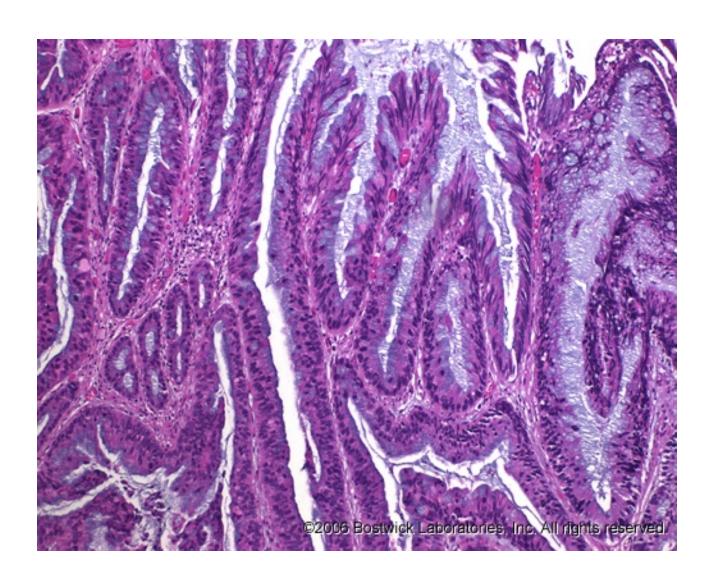
Colonic polyp: Dx: Tubular adenoma

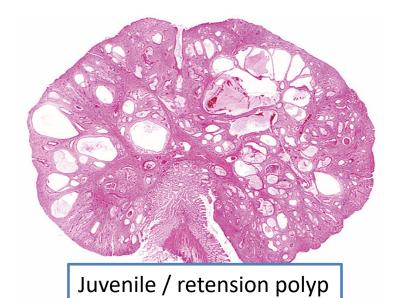


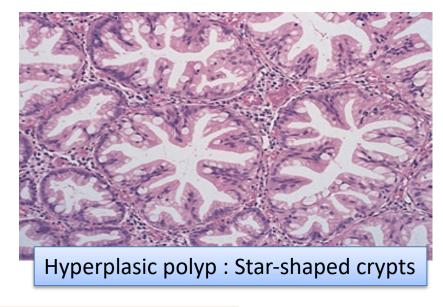
ADENOMATOUS POLYP (TUBULAR)

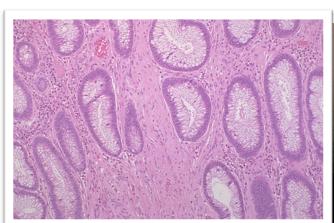


ADENOMATOUS POLYP (VILLOUS)











Hamartomatous polyps : Peutz-Jeghers syndrome

Familial adenomatous polyposis



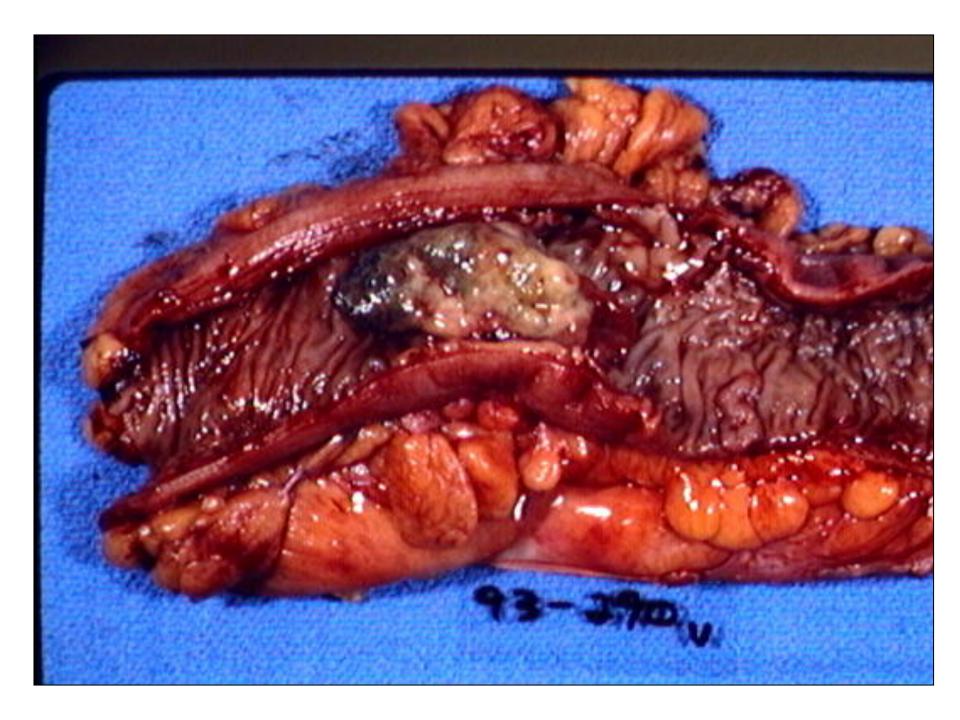
Familial adenomatous polyposis

Numerous neoplastic polyps
It is caused by mutations of the adenomatous polyposis coli , or APC gene

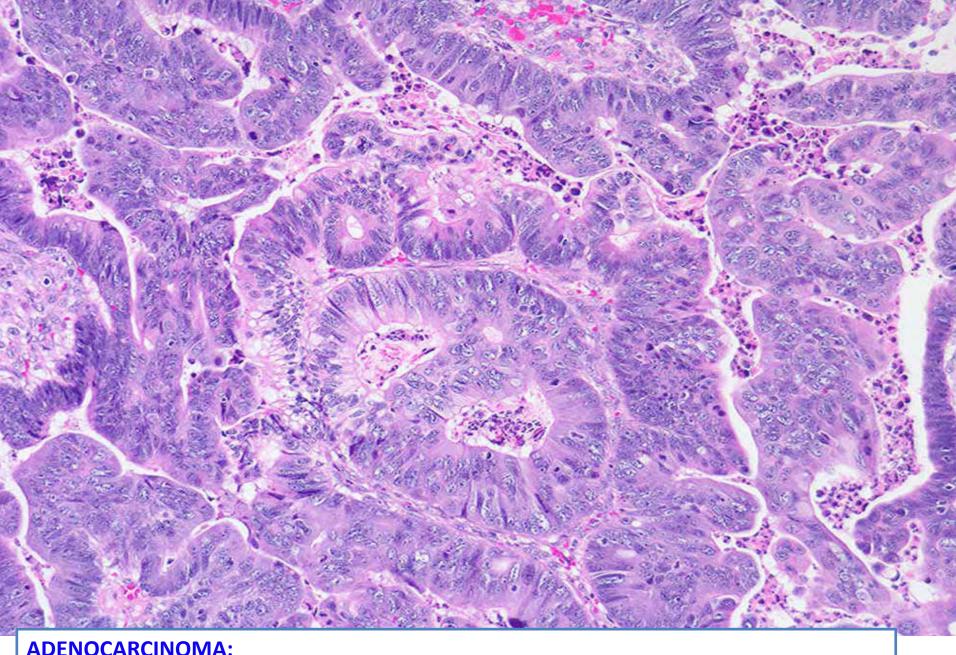
Colon carcinoma



Organ: Colon Dx: adenocarcinoma







ADENOCARCINOMA:

Invasive malignant glands showing pleomorphism, hyperchromatism and mitoses

