

**Pathology of tonsils,
esophagus, stomach.**

Pathology of tonsils, esophagus, stomach.

I. Microspecimens:

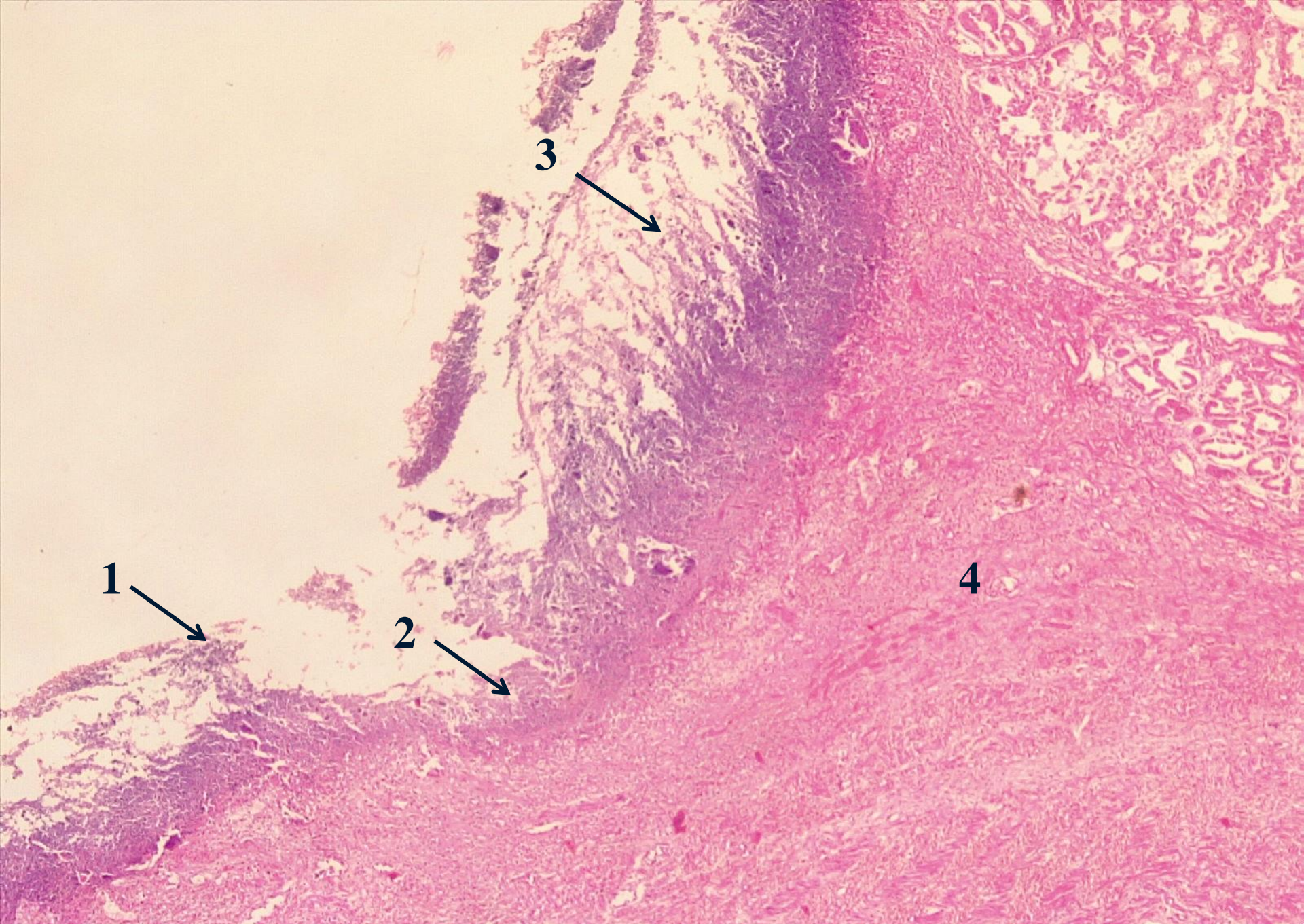
№ 176. Acute gastric ulcer. (H.E. stain).

Indications:

1. The superficial layer of the ulcer, consisting of leukocytes and erythrocytes.
2. Necrotic masses and tissue debris in the area of the ulcer.
3. Foci of necrosis in the muscular layer of the gastric wall.
4. Leukocyte infiltration in the edges and bottom of the ulcer.

A lesion in the gastric wall is observed, which involves the mucosa, the muscularis mucosa and the submucosa; the bottom of the ulcer is presented by necrotic masses with diffuse leukocyte infiltration, which extends into the muscular layer of the gastric wall; blood vessels are dilated, hyperemic; there is no granulation tissue or mature fibrocollagenous tissue.

Acute gastric ulcers are located more frequently on the lesser curvature, in the antral and pyloric region, they can also be on the greater curvature. They are usually multiple, more often round in shape, diameter up to 1.0-1.5 cm, the bottom is dark brown due to the accumulation of the hemoglobinogen pigment, hydrochloric hematin. The edges and bottom have a flaccid consistency, they are not hardened, the arrangement of the folds of the adjacent mucosa is not changed. In some cases, the acute ulcer can progress with involvement of the muscular layer of the gastric wall and even the serosa, which can lead to perforation and peritonitis. Deep acute ulcers often look like a funnel, with the base facing the gastric mucosa and the tip facing the serous membrane. Another complication is gastric bleeding. Acute ulcers in the region of the small curve often heal poorly and turn into a chronic gastric ulcer.



№ 176. Acute gastric ulcer. (*H.E. stain*).

№ 87. Active chronic gastric ulcer. (H.E. stain).

Indications:

1. The distal edge of the ulcer.
2. The proximal edge of the ulcer.
3. The ulcer base:
 - a. zone of necrotic fibrinoid debris;
 - b. zone of infiltration with neutrophils;
 - c. zone of granulation tissue;
 - d. zone of fibrous, collagenous scar.

A defect in the gastric wall can be observed, in which the mucosa, submucosa and partially the muscular layer are missing, in the bottom region there are 4 layers from inside to outside: 1) exudate containing fibrin and leukocytes, 2) fibrinoid necrosis, 3) granulation tissue, 4) mature connective tissue; edema, lympho-histiocytic infiltration are revealed in the edges of the ulcer, the blood vessels are dilated, hyperemic, some with fibrinoid necrosis and thrombosis. These changes are characteristic of the period of exacerbation of chronic gastric ulcer.

Chronic gastric ulcer has an undulating evolution, the periods of exacerbation alternate with periods of remission. The remission ulcer is morphologically characterized by attenuation of the inflammatory process, resorption of necrotic masses, gradual transformation of granulation tissue into mature fibrocollagenous tissue, finally the bottom of the ulcer is represented by scar tissue, and in some cases ulcer epithelialization occurs; the blood vessels are thickened, sclerosed, the lumen stenotic or obliterated.



№ 87. Active chronic gastric ulcer. (*H.E. stain*).

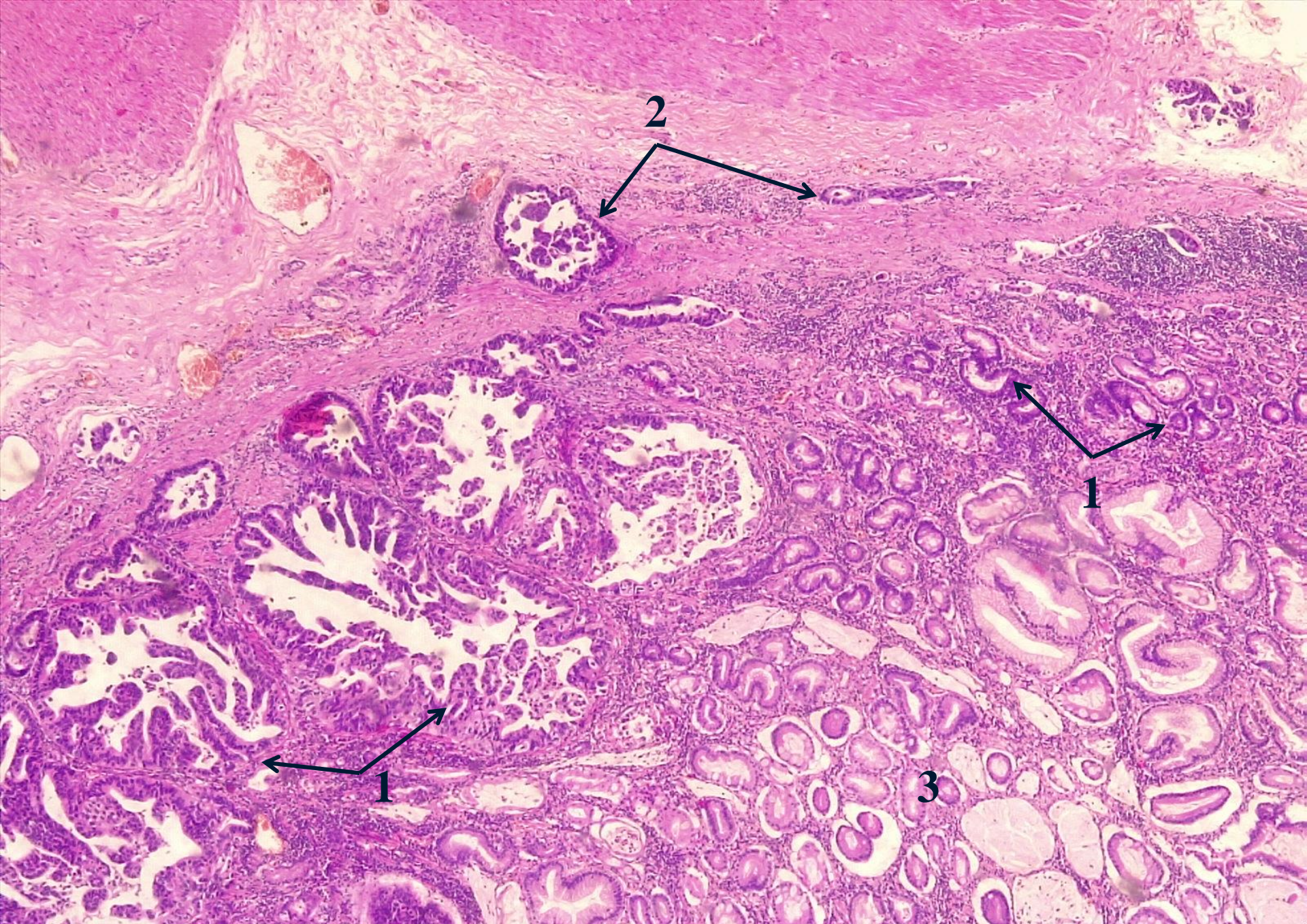
№ 192. Gastric tubular adenocarcinoma – intestinal type. (*H.E. stain*).

Indications:

1. Agglomerations of cancer cells in the gastric mucosa.
2. Cancerous glandular structures in the thickness of the muscular layer.
3. Unmodified mucosal areas.

In the gastric wall is proliferation of atypical glandular structures can be noticed, arranged disorderly, which infiltrate the wall, including the muscular layer, until the serous layer, cancerous cells are polymorphic, with hyperchromic nuclei, sometimes form compact cell nests, in the lumen of glands isolated or small cell groups can be noticed that are floating in mucus, in the stroma lymphocyte infiltration is observed, there is also dilation and hyperemia of blood vessels, hemorrhage; in the adjacent normal mucosa signs of intestinal metaplasia of the gastric epithelium can be present .

[macrospecimen № 60]



№ 192. Gastric tubular adenocarcinoma – intestinal type. (*H.E. stain*).

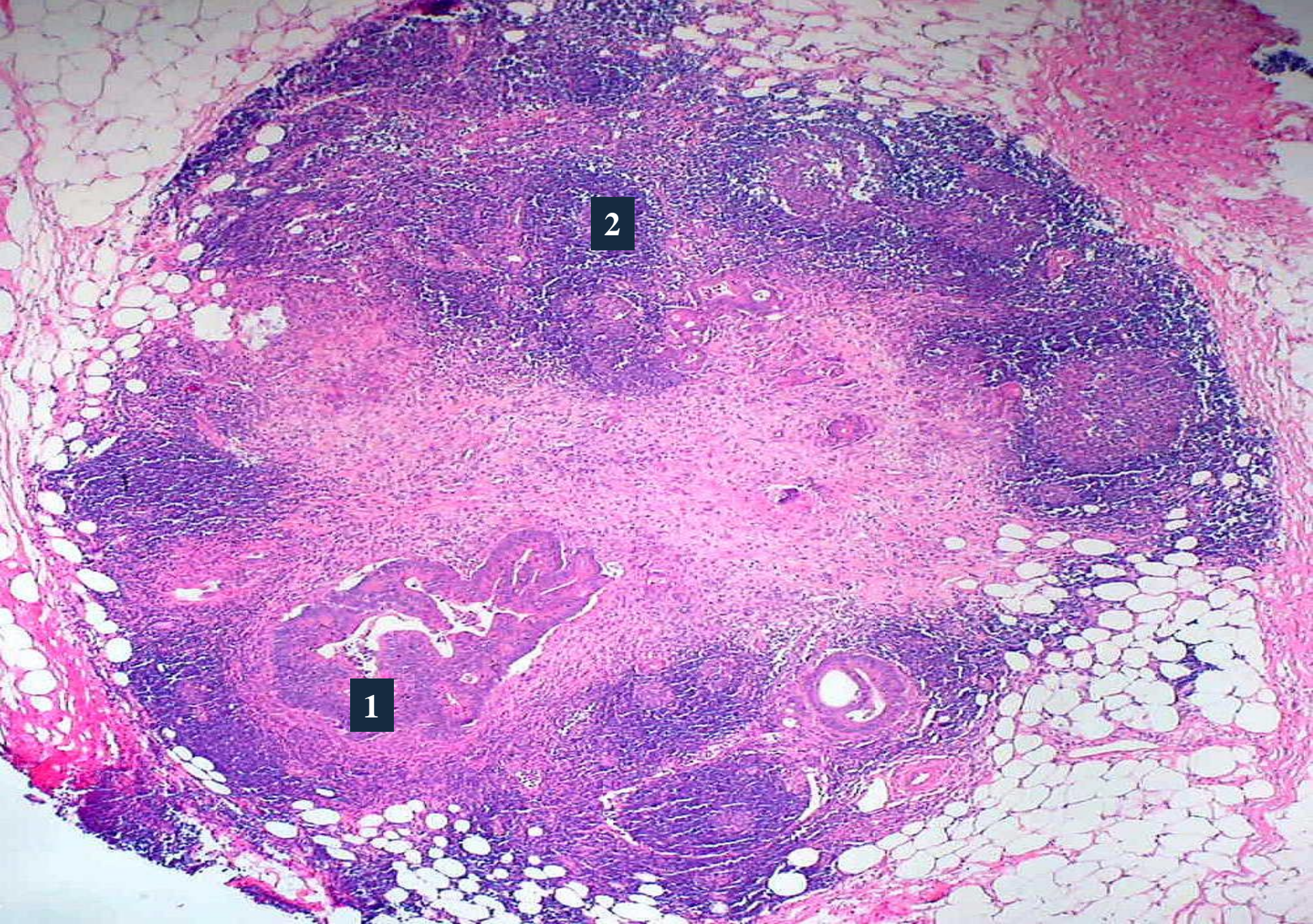
№ 192a. Metastasis of gastric carcinoma into lymph node. (H.E. stain).

Indications:

1. Agglomerations of cancer cells in the marginal and medullary sinuses of lymph node.
2. Unmodified lymphoid follicles.

On the cut-section of the lymph node, foci of atypical glandular proliferation are present, polymorphic, with different shapes and sizes, located mainly in the marginal and medullary sinuses.

Lymphatic metastases have a major importance in the clinical course of gastric cancer. They appear orthogradically (in the direction of the lymphatic flow), firstly can be located in the regional lymph nodes along the lesser and greater curvatures of the stomach. The presence of metastases in regional lymph nodes, change the volume and extent of the surgery, making it larger. Metastases can also occur in a retrograde direction (against the lymphatic flow), for example, in the supraclavicular lymph nodes, usually on the left, which are called - metastasis or Virchow's gland, being in some cases the first symptom of gastric cancer. Retrograde metastases are also observed in pararectal lymph nodes (Schnitzler metastases).



№ 192a. Metastasis of gastric carcinoma into lymph node. (*H.E. stain*).

II. Macrospecimens:

№ 59. Esophageal carcinoma.

The esophagus is sectioned longitudinally, in the middle third a tumor is revealed, which grows circularly, protruding and stenosing the lumen, with an irregular, ulcerated surface, covered by necrotic masses.

Most esophageal cancers are located in the 1/3 middle portion. Histologically the most common histological type - 90% of the total number is keratinized or non-keratinized squamous cell cancer. Complications: infiltration into the stomach, hypopharynx, trachea with the formation of esophageal-tracheal fistula, larynx, mediastinum, lungs, pleura, aorta. Lymphatic metastases - into the cervical, para-esophageal, tracheobronchial, subdiaphragmatic nodules. Hematogenous metastases are rare.



№ 59. Esophageal carcinoma.

№ 51. Gastric polyp.

On the surface of the gastric mucosa there are multiple prominent formations, with thin base (peduncled polyps) or wide (sessile polyps), dimensions from a few mm to 1-1.5 cm, oval shape, smooth surface, flaccid consistency, in some cases hemorrhagic foci can be seen.

Gastric polyps are more frequently located in the anthro-pyloric region, they can be solitary or multiple. The absolute majority of gastric polyps ($\approx 90\%$) are of inflammatory origin, non-neoplastic (hyperplastic polyps). Microscopically they consist of hyperplastic glands, irregularly arranged, some cystically dilated and elongated; are covered with superficial gastric epithelium, but can also parietal and main cells can be noted, the stroma is edematous, hyperemic, with moderate lympho-histiocytic infiltration. No cellular atypia is observed and, as a rule, it has no malignant potential. However, in polyps larger than 1.5 cm there is a risk of gastric epithelial dysplasia, which is a premalignant lesion. Hyperplastic polyps can be complicated by superficial erosions and gastric hemorrhage.



№ 51. Gastric polyp.

№ 52. Chronic gastric ulcer.

A defect of the gastric wall in the region of the lesser curvature can be noted with elongated, oval shape, dimensions 3-4 cm x 1.5-2 cm, with dense consistency edges, the folds of the mucosa converge towards this defect, directly in the edges of the ulcer the folds are atrophied, the bottom is gray-brown due to the presence of necrotic masses and blood clots. On the perpendicular section the edge from the cardia is slightly "dug", steep, hangs over the defect, and the edge from the pylorus is elongated, "in terrace", the steps of which are formed by the layers of the wall - mucosa, submucosa and muscle layer (this aspect is due to displacement of the layers in the peristalsis of the gastric wall).

Peptic gastric ulcer is usually solitary (80%), located more frequently in the region of lesser curvature and anthro-pyloric region. The development of chronic ulcer is often preceded by gastric erosion and acute ulcer. In 10-20% of cases gastric ulcer coexists with duodenal ulcer. During the acute period, the bottom of the ulcer is covered with necrotic masses, fibrin-purulent exudate, there may be blood clots, the mucosa at the edges of the ulcer is edematous and hyperemic. During the remission period, the bottom is presented by scar tissue, it is smooth, clean, dense, the edges of the same firm consistency. Complications of chronic gastric ulcer can be classified into 5 groups: 1) destructive - a) hemorrhage by erosion of blood vessels, which can be manifested by vomiting with "coffee grounds" and melena, b) perforation with peritonitis and c) penetration, which can produce in the pancreas, the small omentum, the hepato-duodenal ligament, much less often in the liver, the transverse colon, the gallbladder; 2) inflammatory complications - periulcerous gastritis and perigastritis, which can lead to adhesions with neighboring organs; 3) scar complications - stenosis and stomach deformity, more frequently pyloric stenosis, which can be manifested by food retention and frequent vomiting; 4) malignancy, transformation into gastric carcinoma, which is observed in about 1% of cases; 5) mixed complications, e.g. perforation and hemorrhage, penetration and hemorrhage.



№ 52. Chronic gastric ulcer.

№ 53. Chronic gastric ulcer with perforation.

In the gastric wall a chronic ulcer can be noted, in which a perforative defect can be observed, through which the gastric contents are eliminated in the peritoneal cavity and peritonitis develops.

Perforation occurs during the period of exacerbation of chronic gastric ulcer and leads to peritonitis. More often, the pyloric ulcers or ulcers of the anterior wall of the duodenal bulb are perforating. Perforation of duodenal ulcer is more common than gastric ulcer. At first, fibrinous peritonitis develops, located around the perforation defect, and later the inflammation spreads throughout the peritoneum, becoming generalized and, as a rule, fibrino-purulent. If there are adhesions in the bottom region of the ulcer they can delimit the inflammatory process and the peritonitis becomes localized.

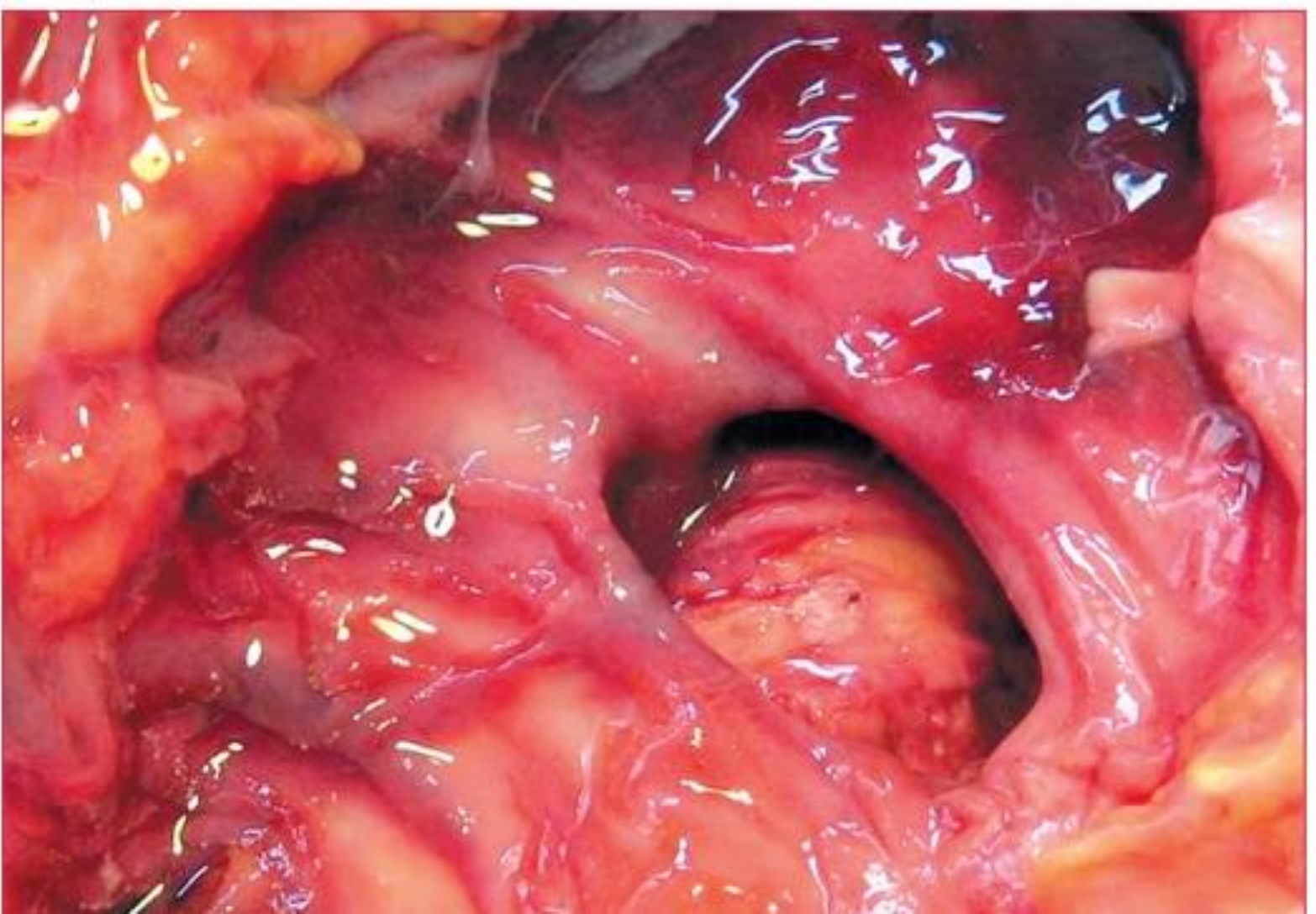
№ 54. Chronic duodenal ulcer.

In the duodenal wall an ulcer defect can be observed with dense edges, dimensions of 1.5-2 cm, irregular shape.

Duodenal ulcer is more frequent compared to gastric ulcer, it is located immediately in the postpyloric region, in the first few cm after the pyloric valve, usually in the anterior wall of the duodenal bulb, but can also be in the posterior wall (bulbar ulcer) and much less frequently - in the post-bulbar portion. Sometimes the ulcers are multiple and can be placed face to face on the anterior and posterior walls – mirror ulcers. Complications: a) hemorrhage, usually from the ulcer of the posterior wall, b) perforation of the ulcer of the anterior wall, c) penetration of the ulcer of the posterior wall, usually in the head and body of the pancreas, which can lead to pancreonecrosis, d) inflammatory processes - periulcerous duodenitis and periduodenitis with the formation of adhesions with adjacent organs, e) scar complications - stenosis and deformation of the duodenal bulb, which is found only in the ulcer of the posterior wall. Malignancy is never observed.



No 53. Chronic gastric ulcer with perforation.



№ 54. Chronic duodenal ulcer.

№ 60. Gastric adenocarcinoma.

In the stomach is a voluminous tumor with exophytic growth, irregular surface, hemorrhagic foci, dense-elastic consistency, white-gray color, fungus appearance (fungoid). It is located more frequently in the region of the lesser curvature and the pyloric canal.

Gastric cancer is most often preceded by precancerous conditions such as chronic gastric ulcer (ulcer-cancer), chronic atrophic gastritis with intestinal metaplasia of the epithelium, epithelial dysplasia, adenomatous polyps, Helicobacter pylori infection. The most common location is in the region of lesser curvature, pylorus, pyloric antrum. The most common histological variant is adenocarcinoma with different degrees of differentiation. Gastric cancer can spread by continuity into the esophagus, peritoneum (peritoneal carcinomatosis), large omentum, pancreas, liver, transverse colon, and by implantation - in mono- or bilateral ovaries - Krukenberg tumor. Locally it can be complicated by hemorrhage, perforation, inflammation of the gastric wall (phlegmon). It metastasizes primarily in the regional lymph nodes in the region of small curvature, cardiac, suprapancreatic. A pathognomonic sign is metastasis to the left supraclavicular lymph nodes - the Virchow or Troisier sign. Hematogenous metastases occur first in the liver, later - in the lungs, brain, bones, kidneys.

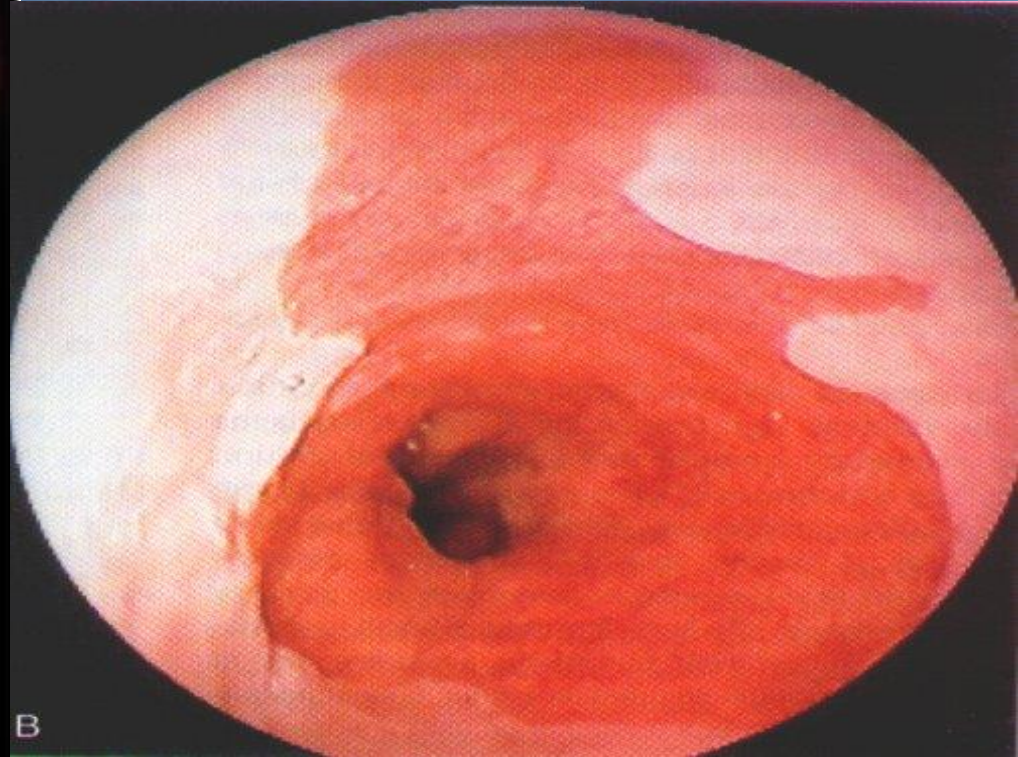


№ 60. Gastric adenocarcinoma.

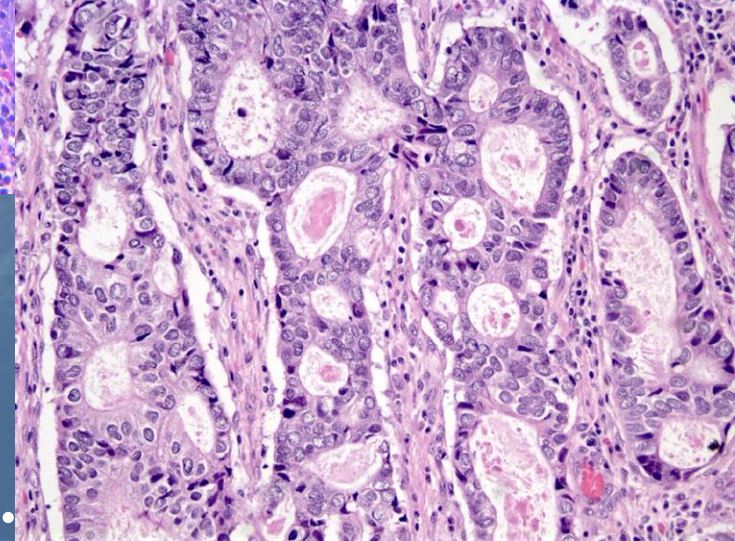
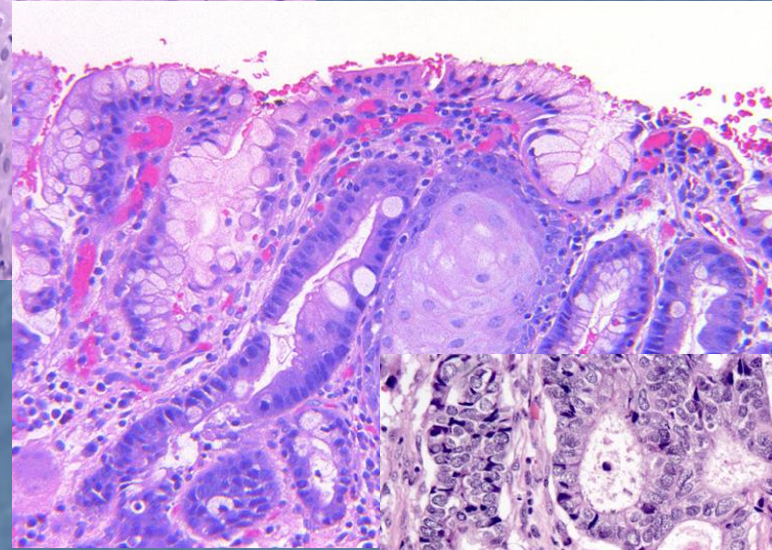
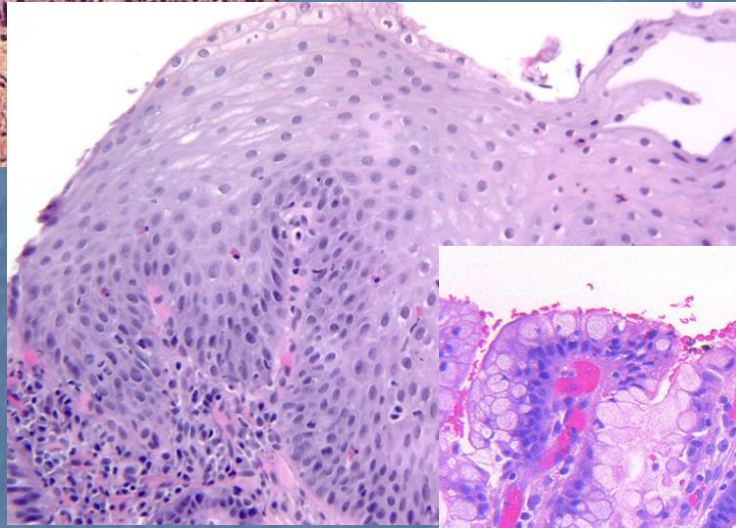
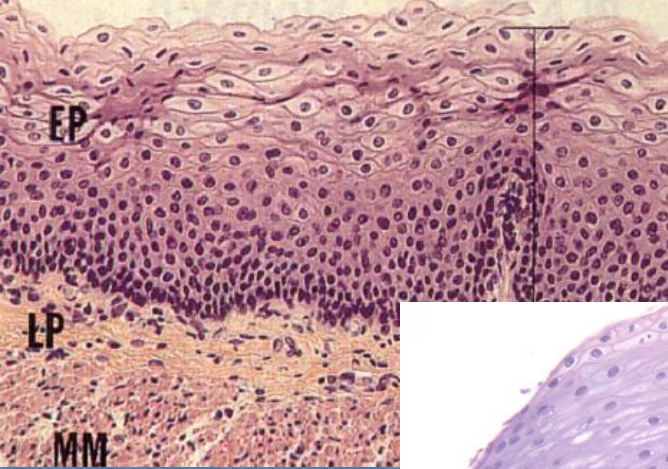


Esophageal development abnormalities (esophageal atresia, esophagotracheal fistulas).

**Reflux esophagitis:
macroscopic and endoscopic
pattern.**



Morphopathogenesis of esophageal adenocarcinoma.



Reflux
esophagitis.



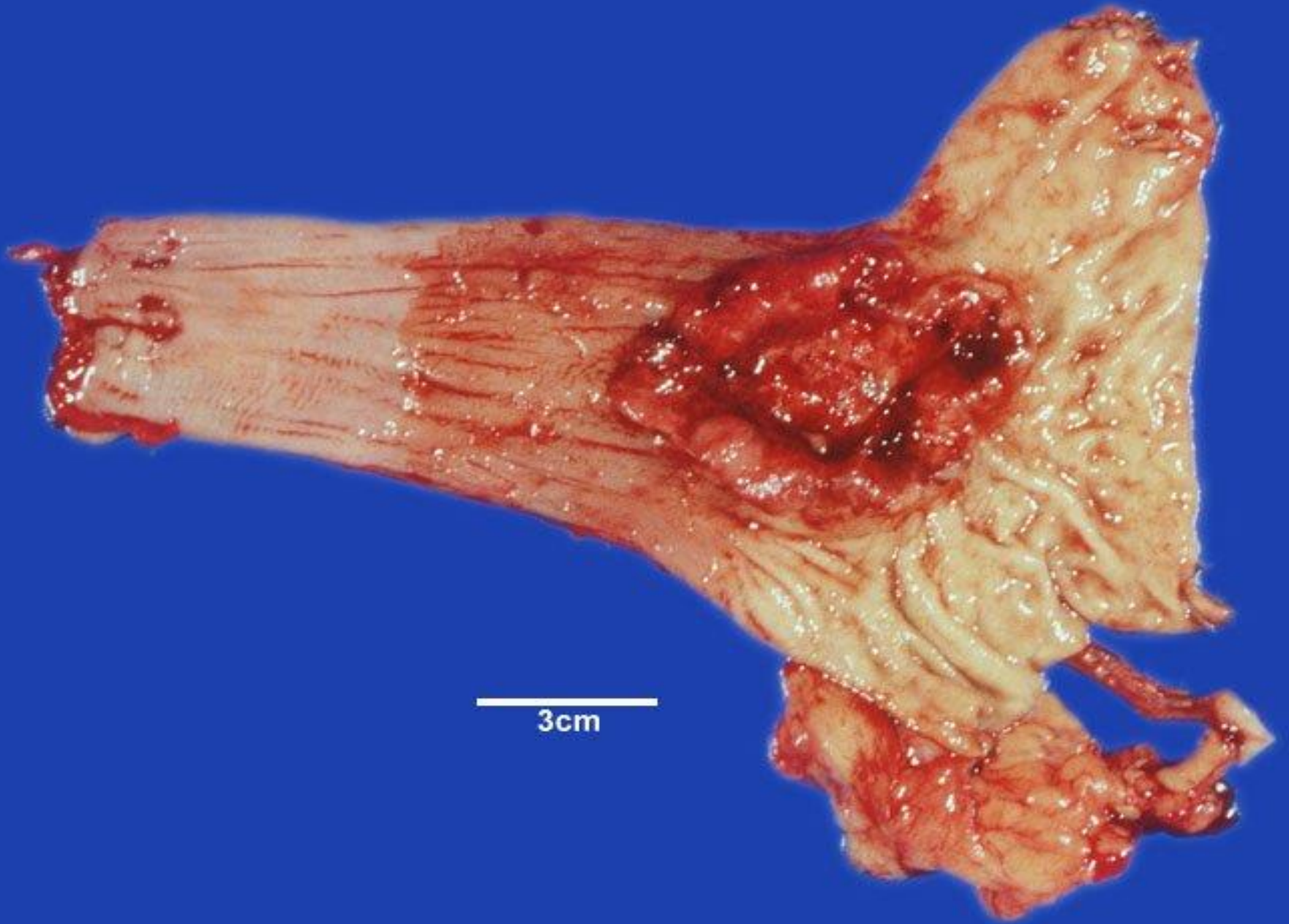
Barrett
esophagus
(intestinal type).



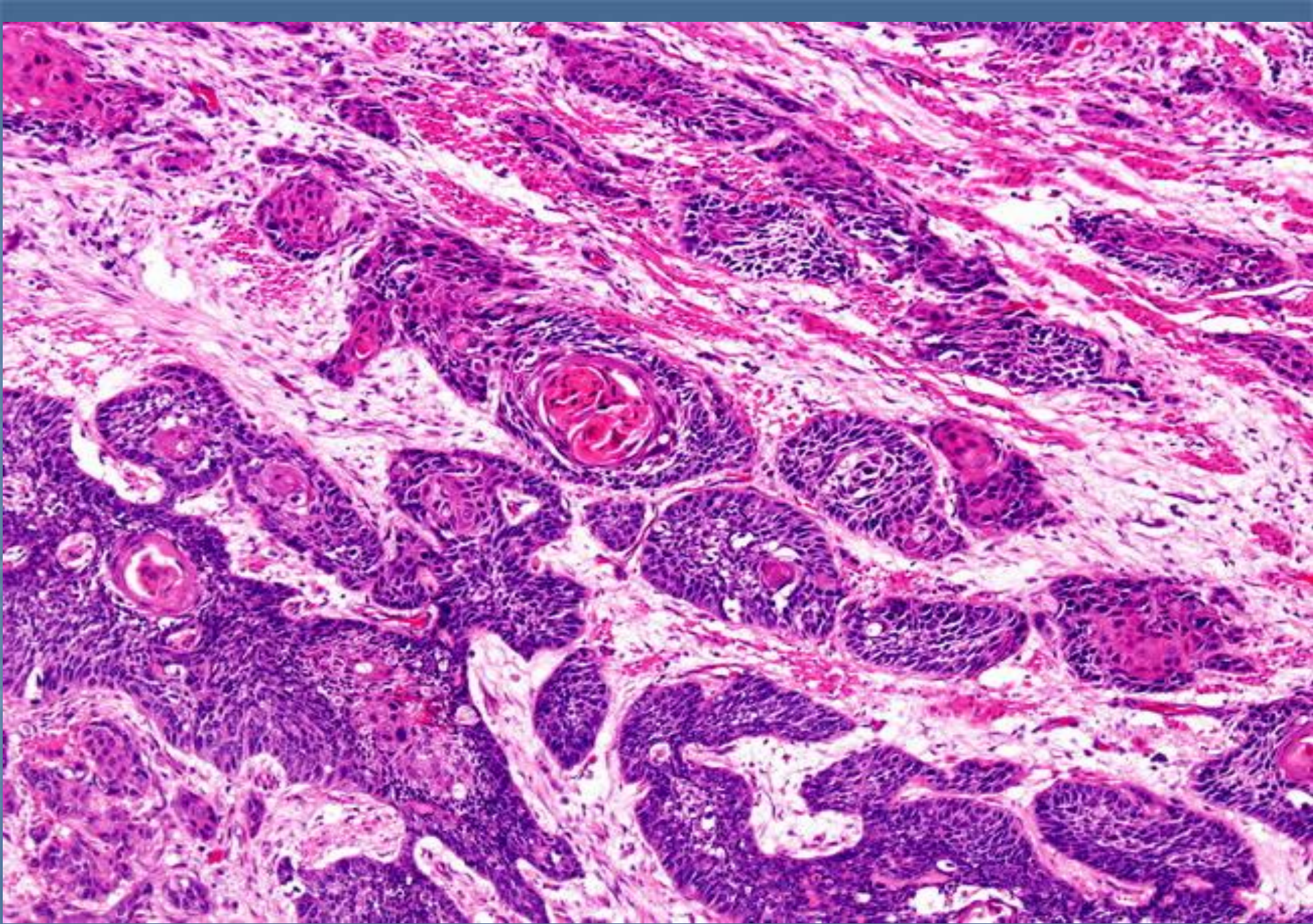
Barrett with
dysplasia
(carcinoma in-situ).



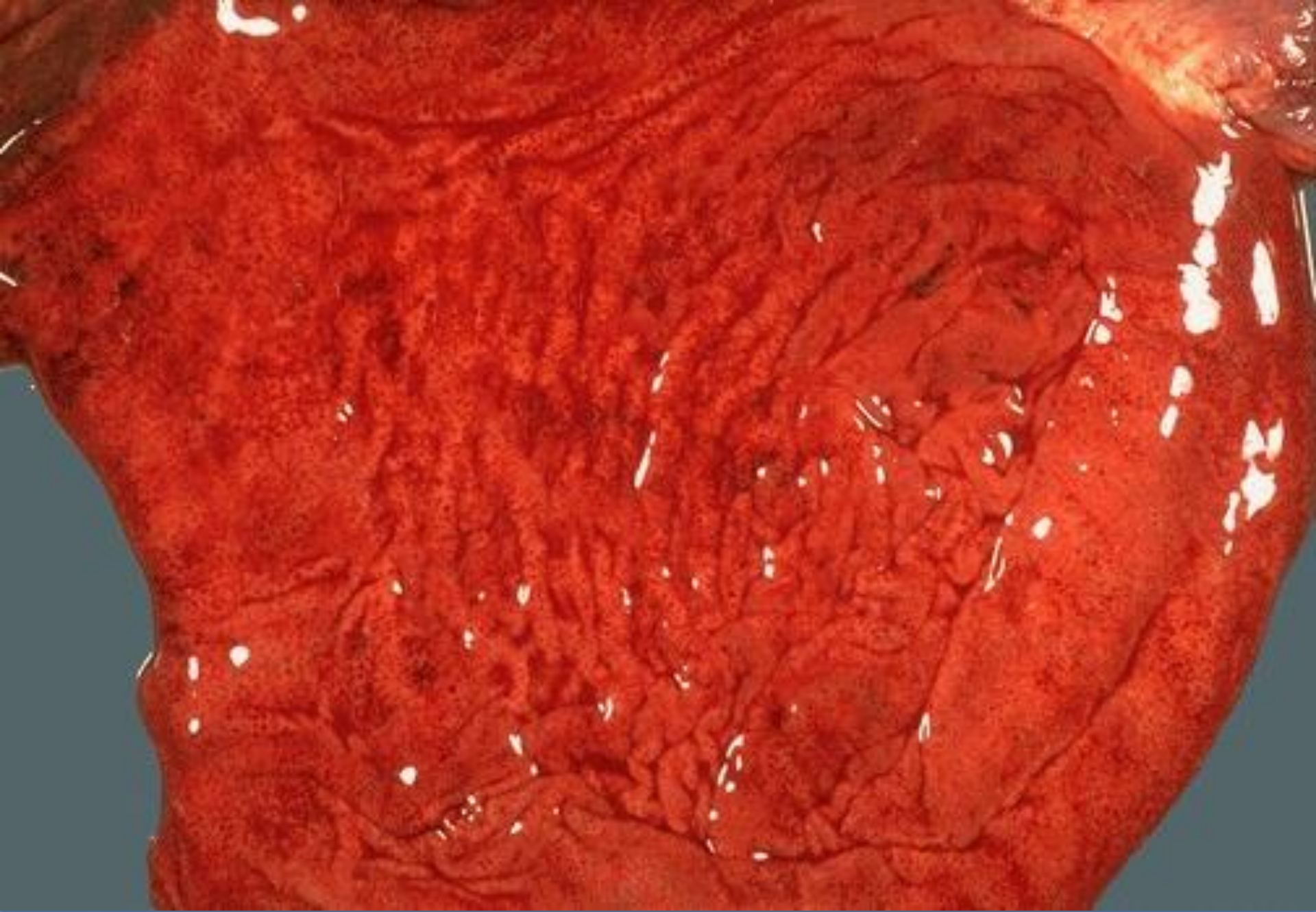
Invasive adenocarcinoma.



Esophageal adenocarcinoma.

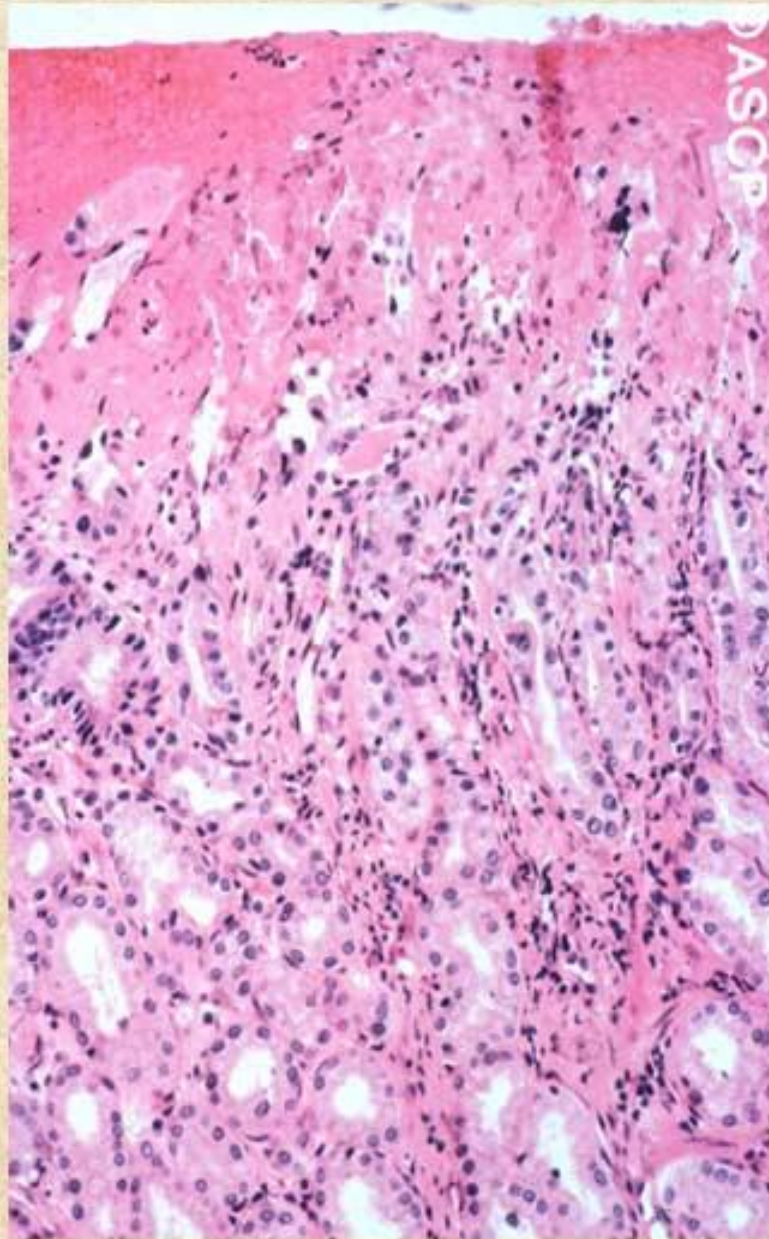


Esophageal keratinizing squamous cell carcinoma.

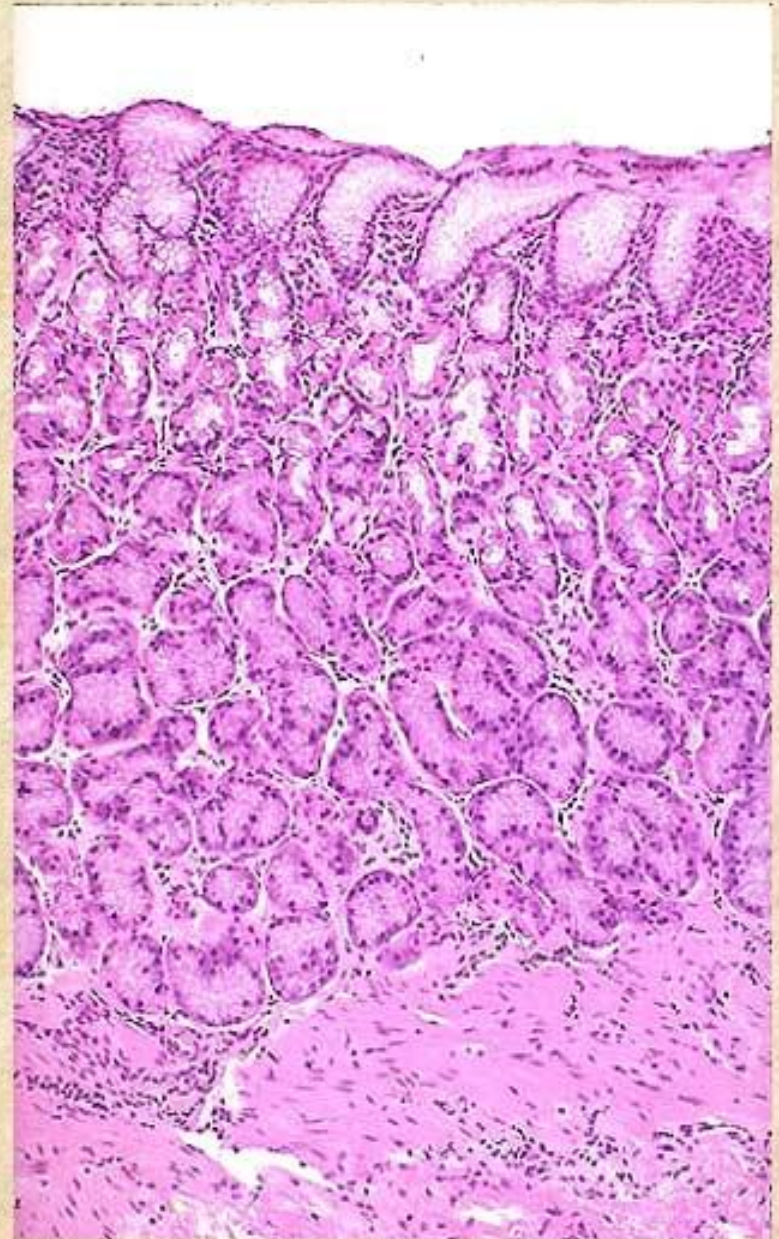


Acute gastritis.

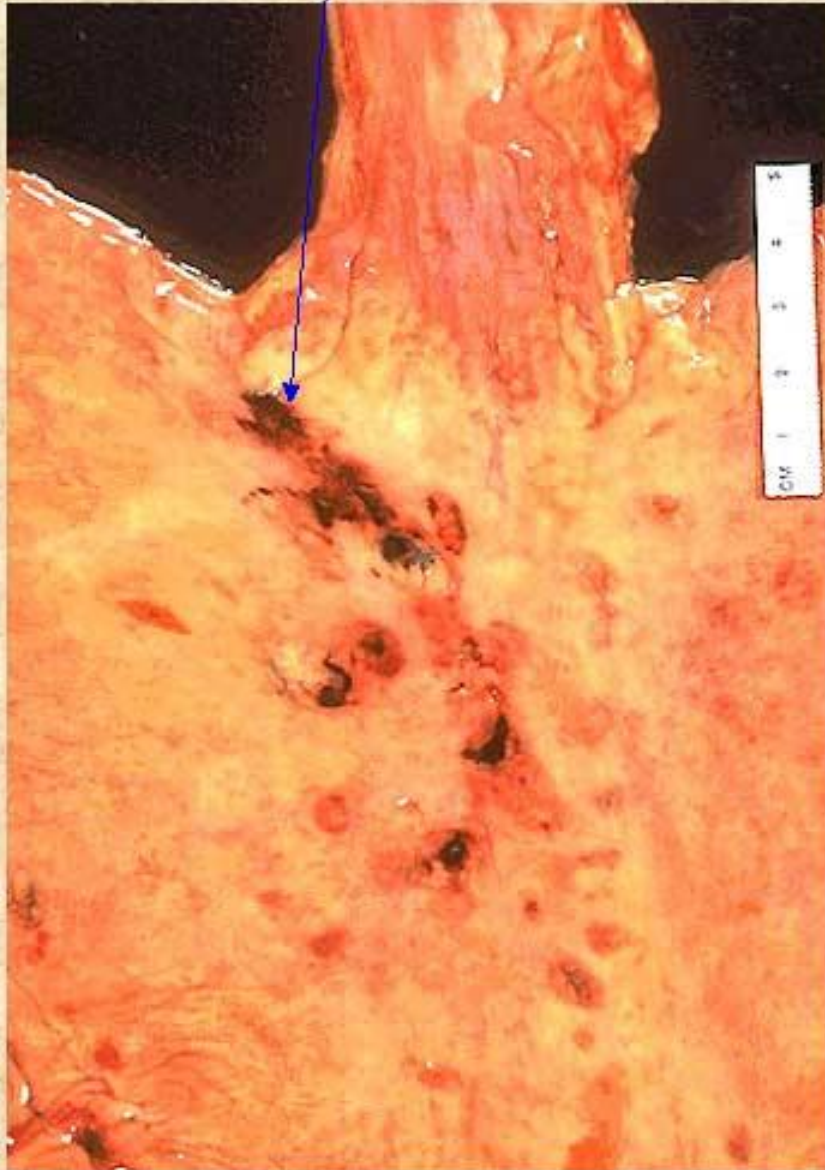
Acute erosive gastritis



Normal fundus



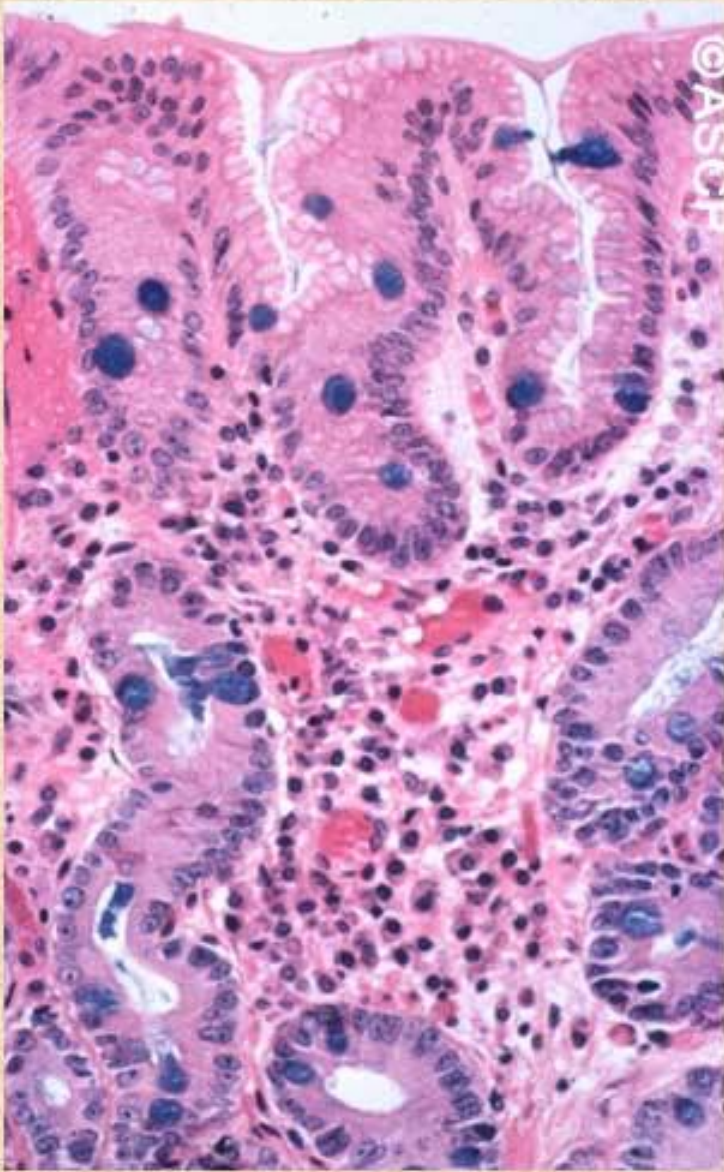
Acute erosion



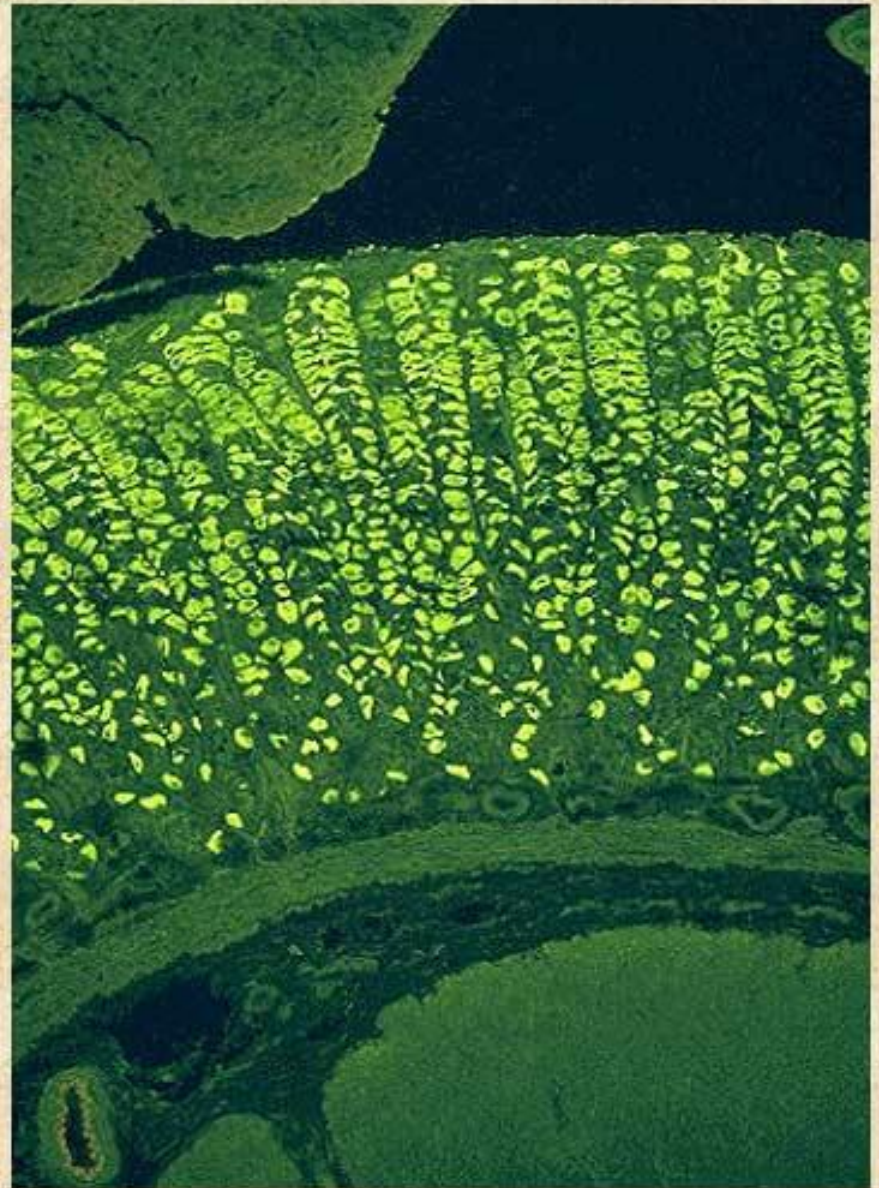
Acute ulcer



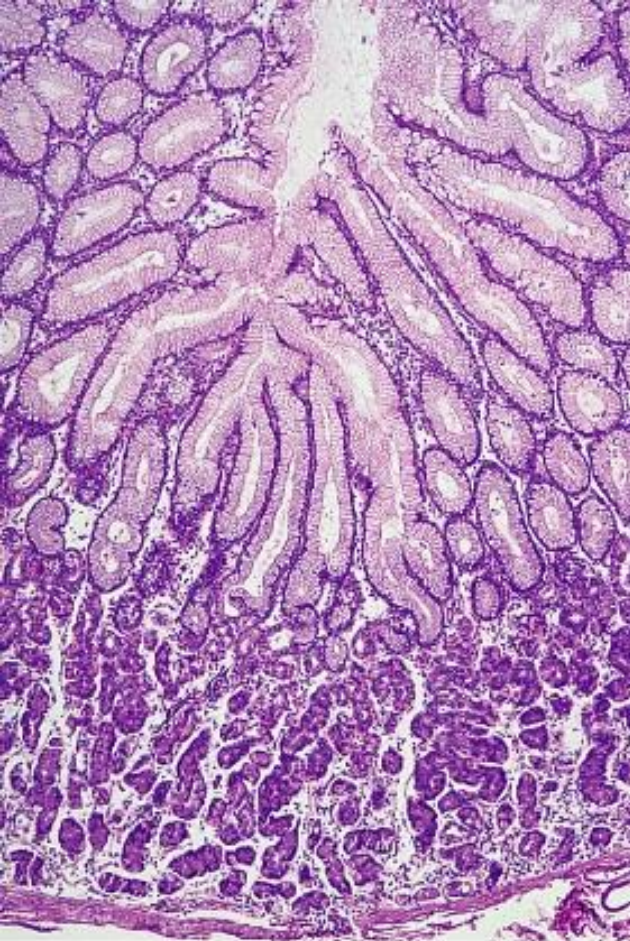
Autoimmune chronic gastritis



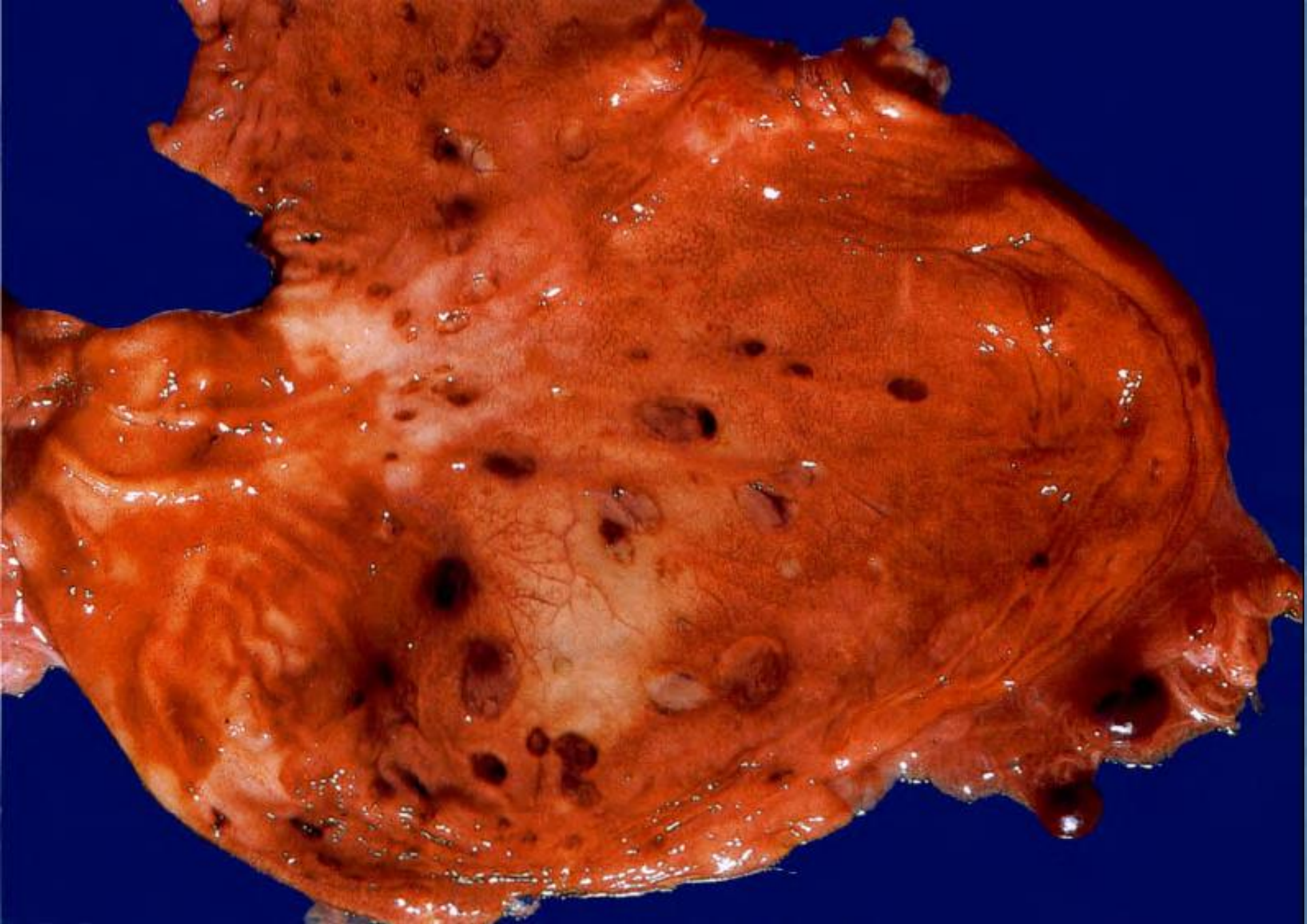
H & E



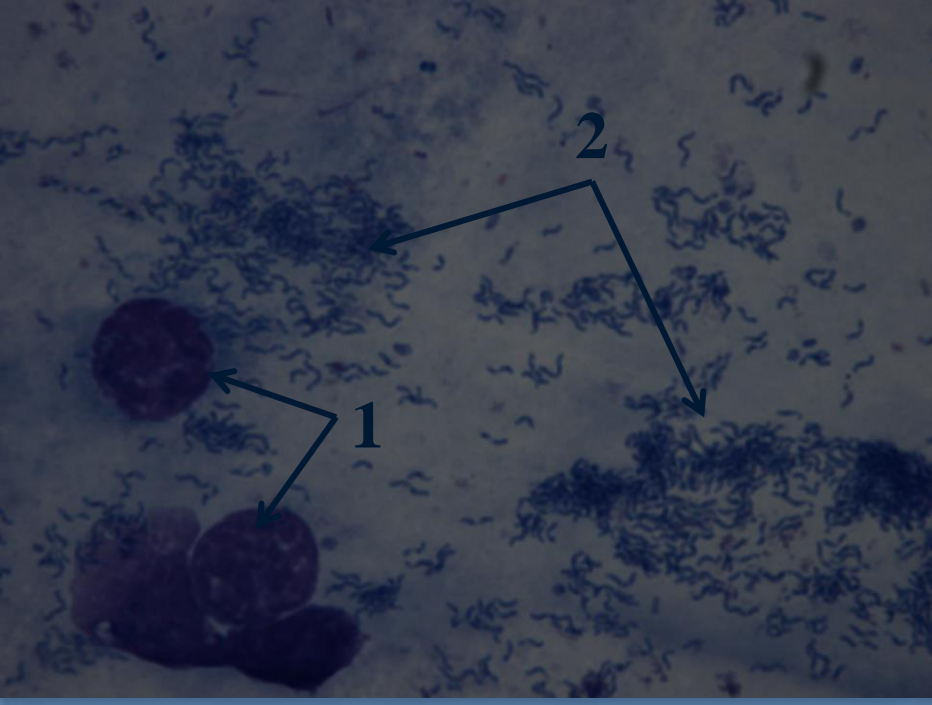
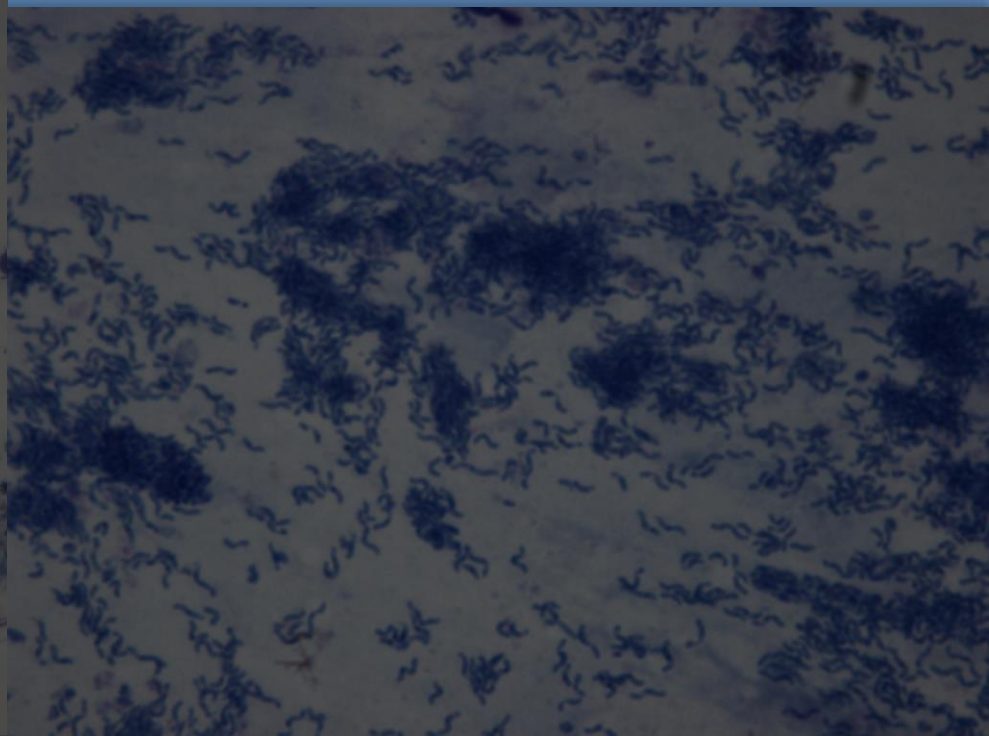
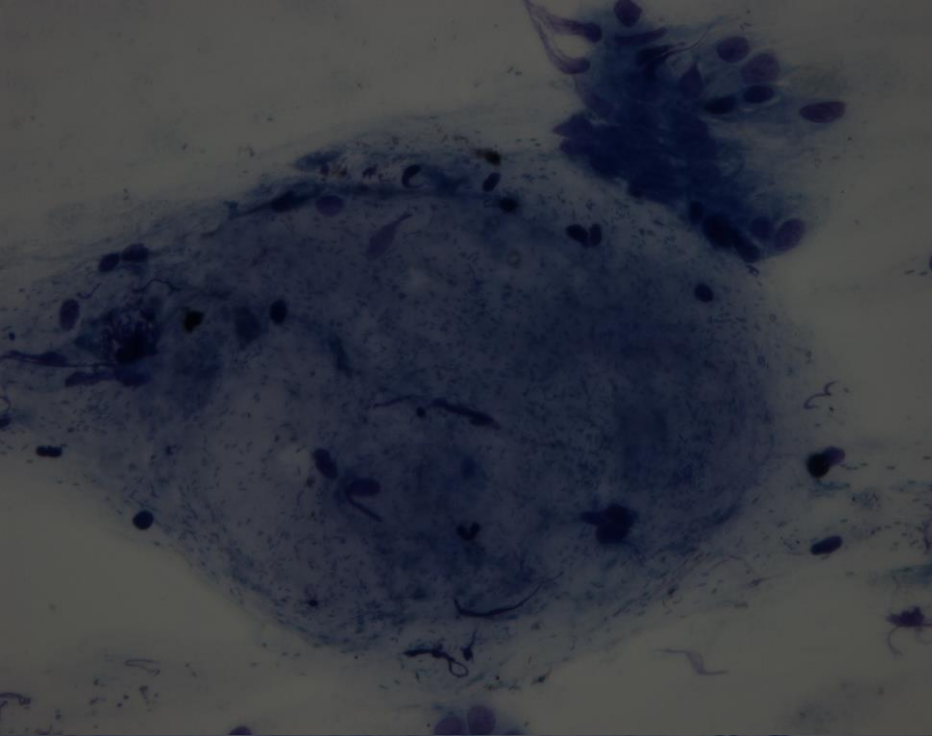
IF Anti-parietal cell Ab



**Menetrier
gastritis.**



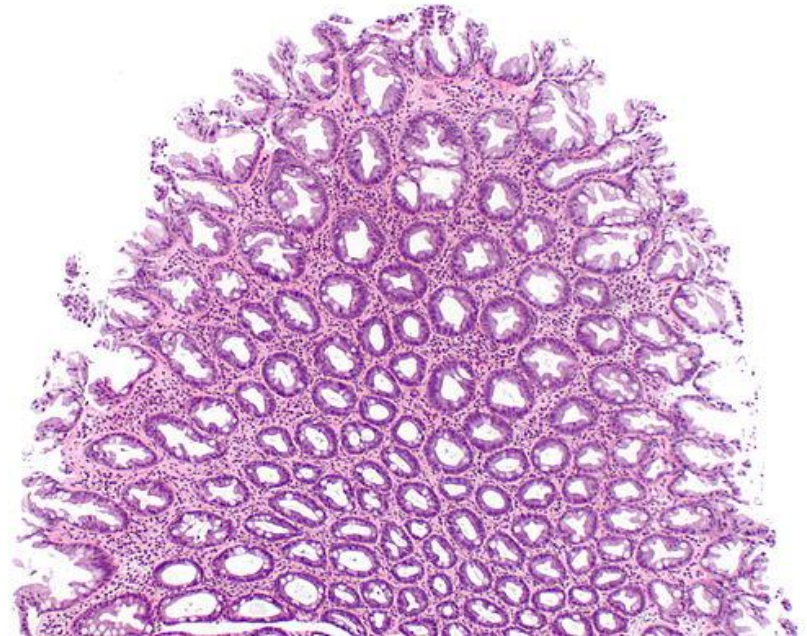
Acute gastric erosions.



Cytology of the gastric mucosa (smear).
1. Chief cells
2. Helicobacter pylori.

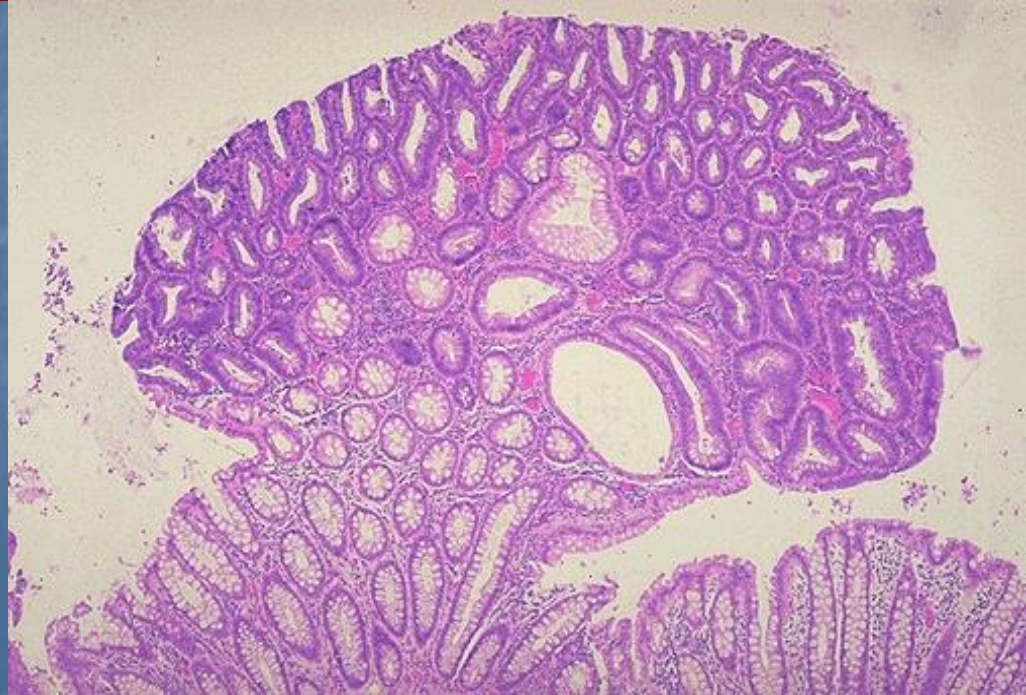


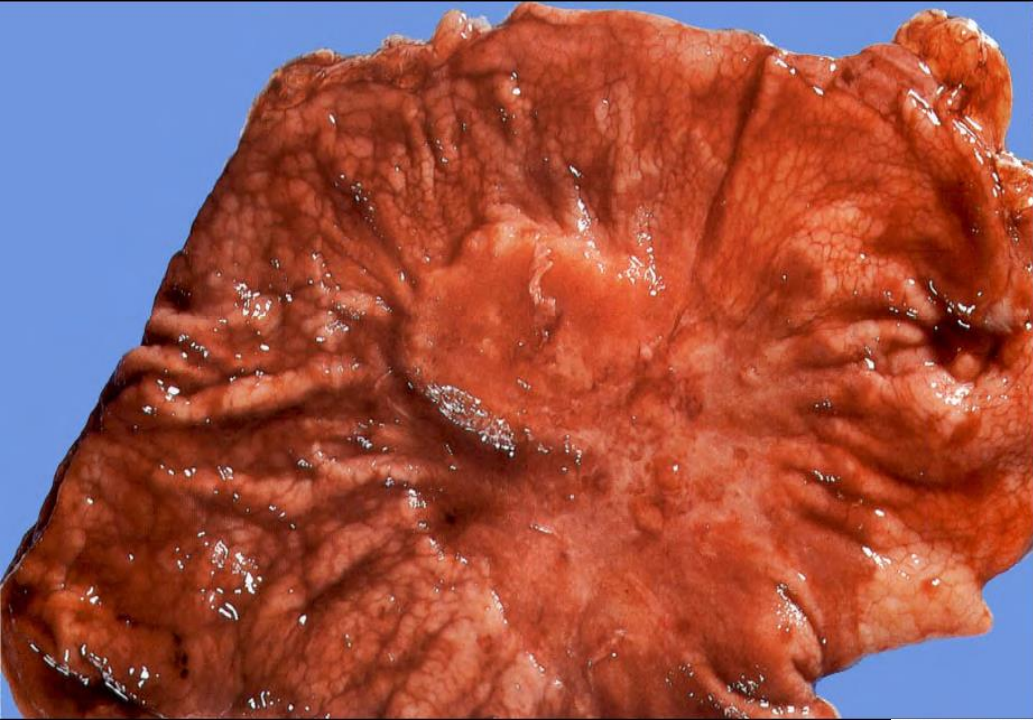
Gastric hyperplastic polyps.





Gastric adenoma.

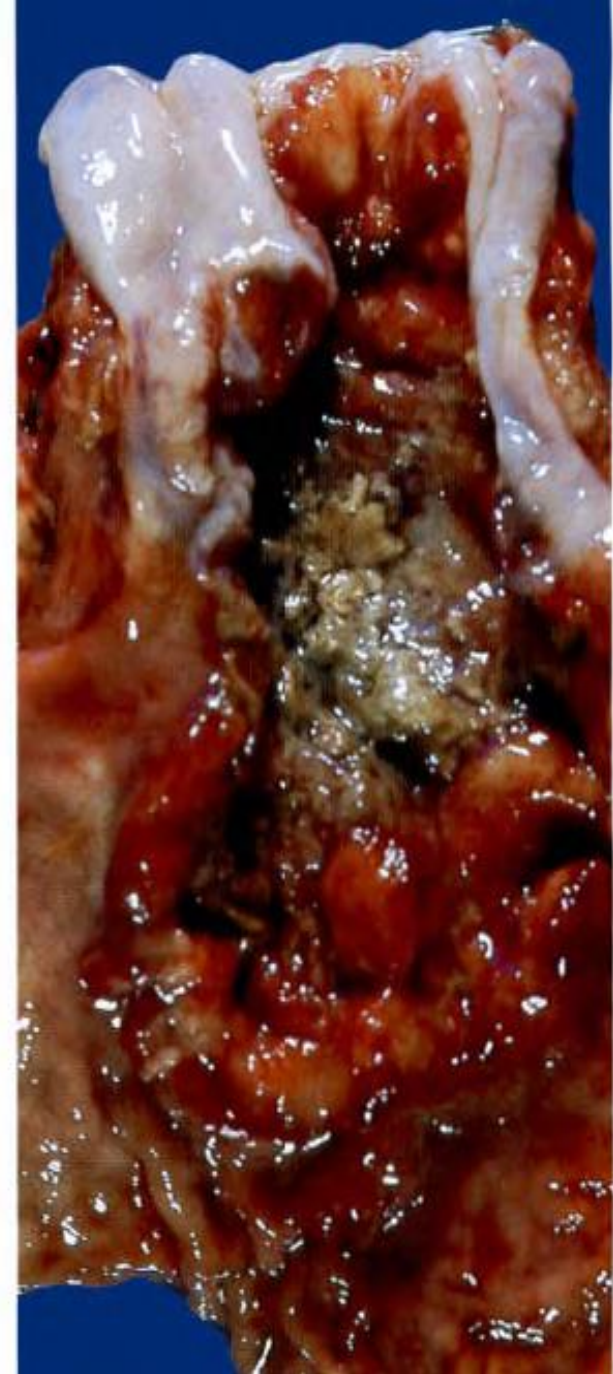




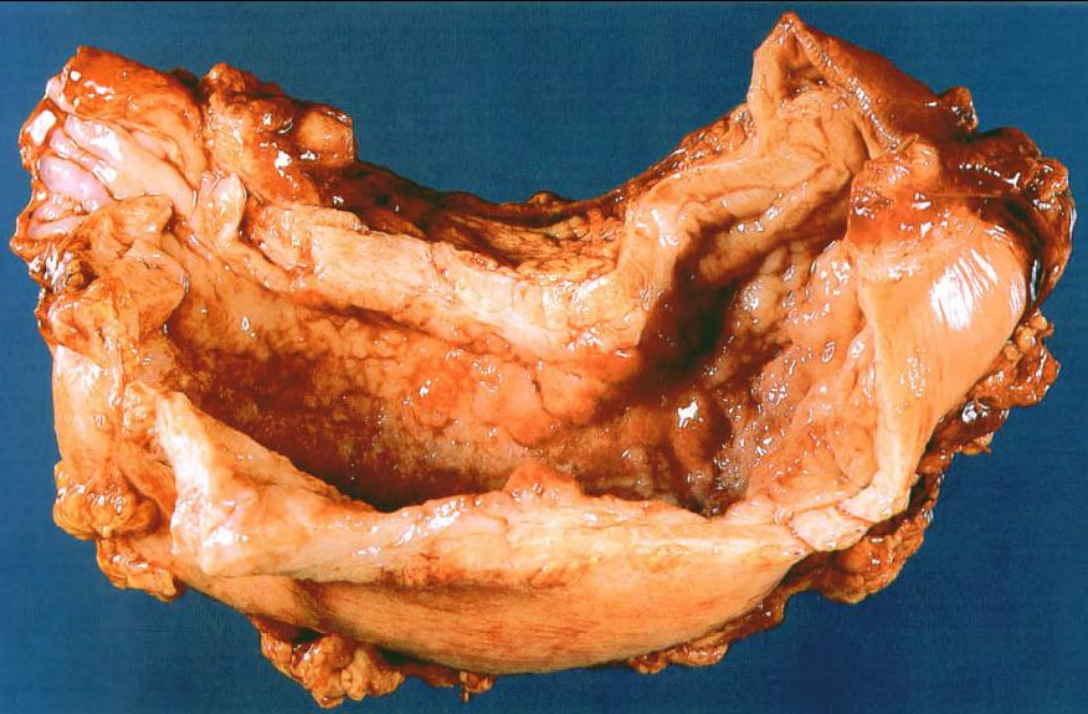
Polypoid gastric carcinoma

**Gastric carcinoma
plaque-like lesion**

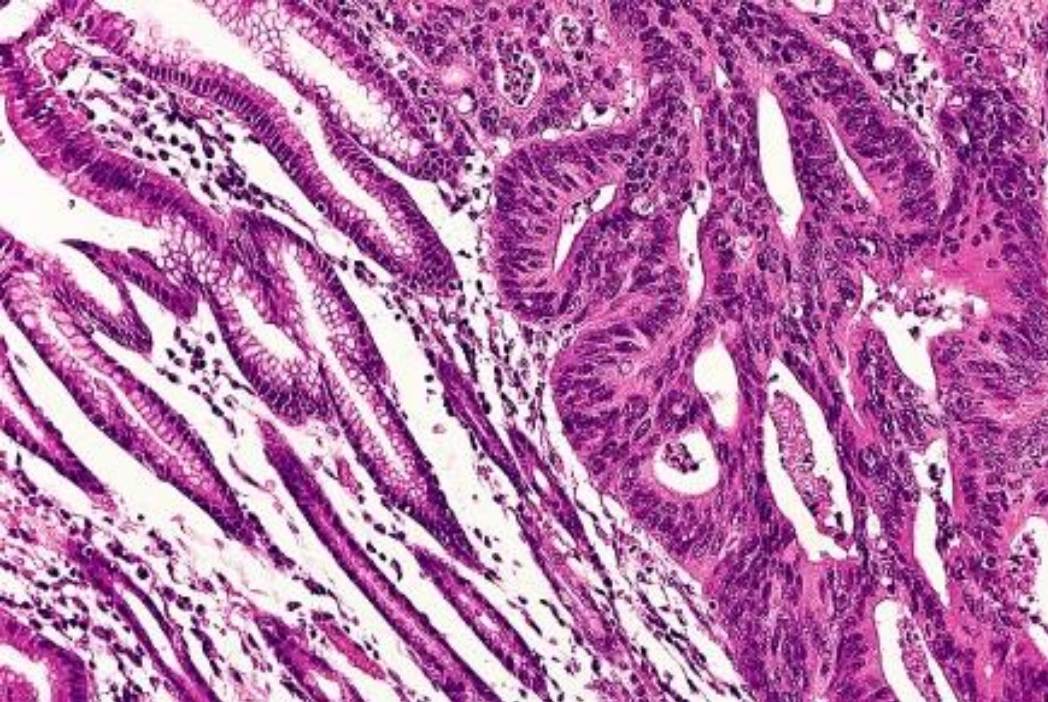




Exulcerated gastric carcinoma.

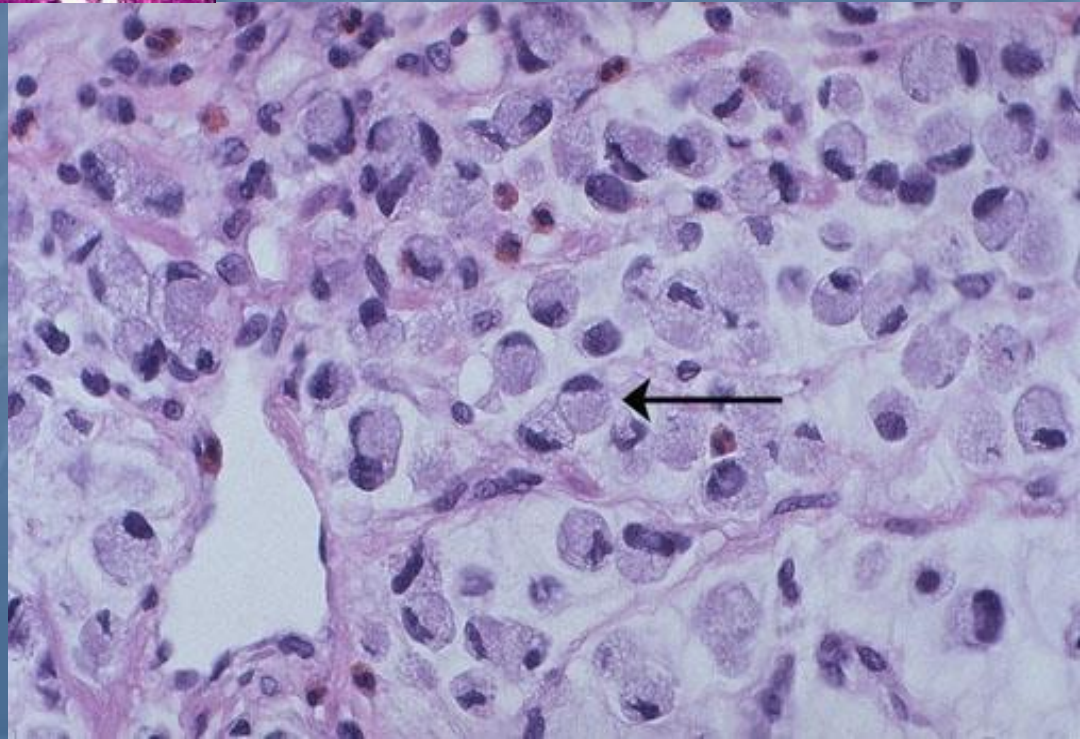


Infiltrative gastric carcinoma.



Gastric signet ring cell carcinoma

Gastric papillary adenocarcinoma.



ESOPHAGUS

- **Congenital Anomalies**
- **Achalasia**
- **Hiatal Hernia**
- **Diverticula**
- **Laceration**
- **Varices**
- **Reflux**
- **Barretts**
- **Esophagitis**
- **Neoplasm: Benign, Sq. Cell Ca., Adenoca.**

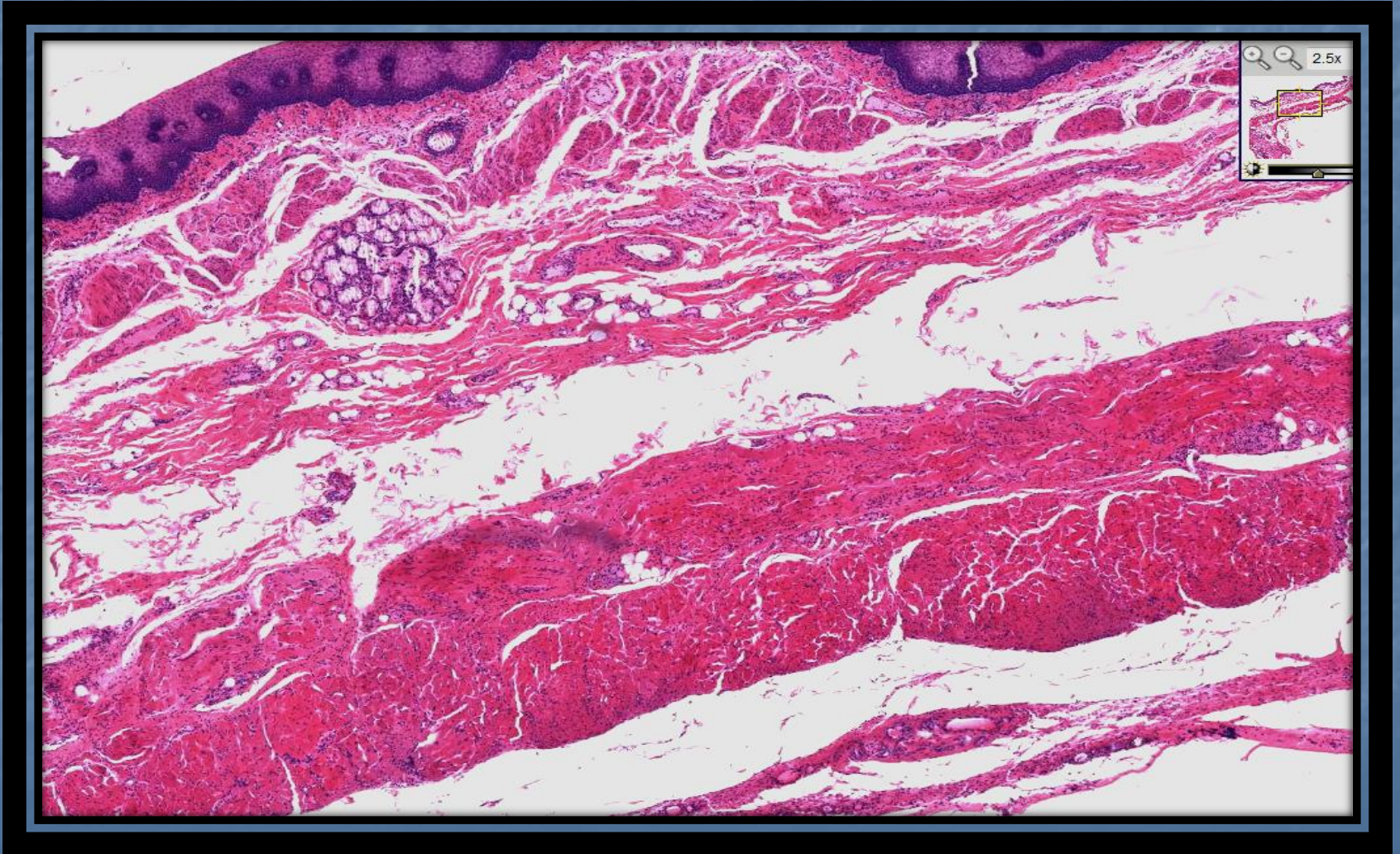
ANATOMY

25 cm.

UES/LES

Mucosa/Submucosa/Muscularis/Adventitia*

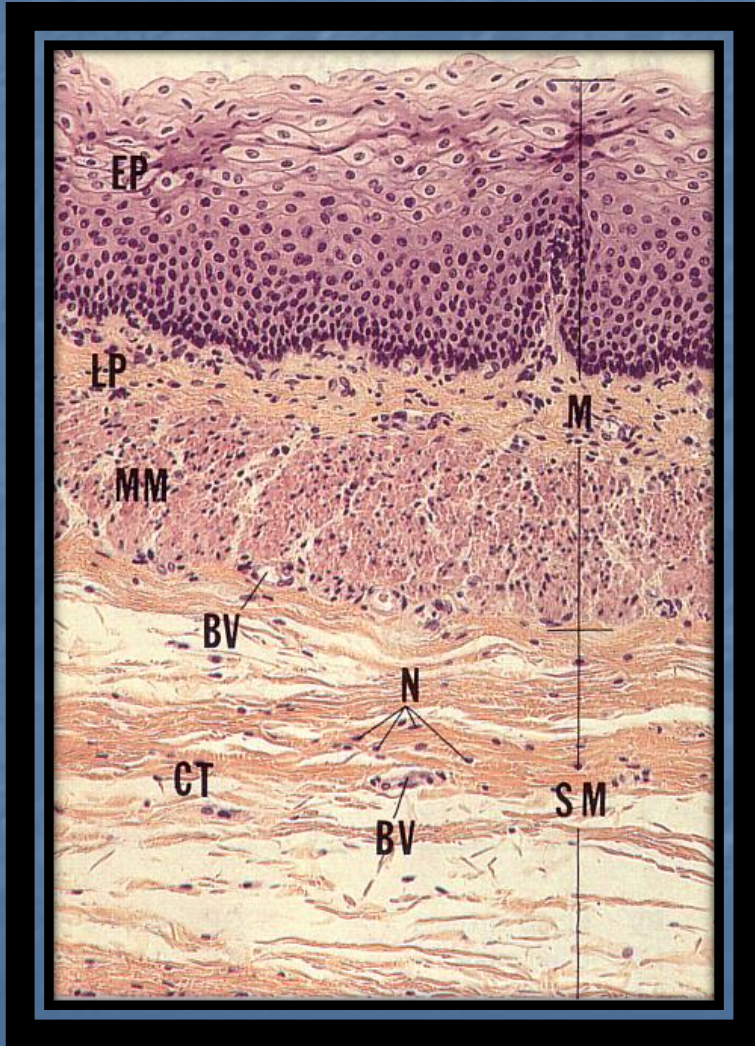
Histologic structure of esophagus



DEFINITIONS

- Heartburn (GERD/Reflux)
- Dysphagia
- Hematemesis
- Esophagospasm (Achalasia)

Esophagus Pathology

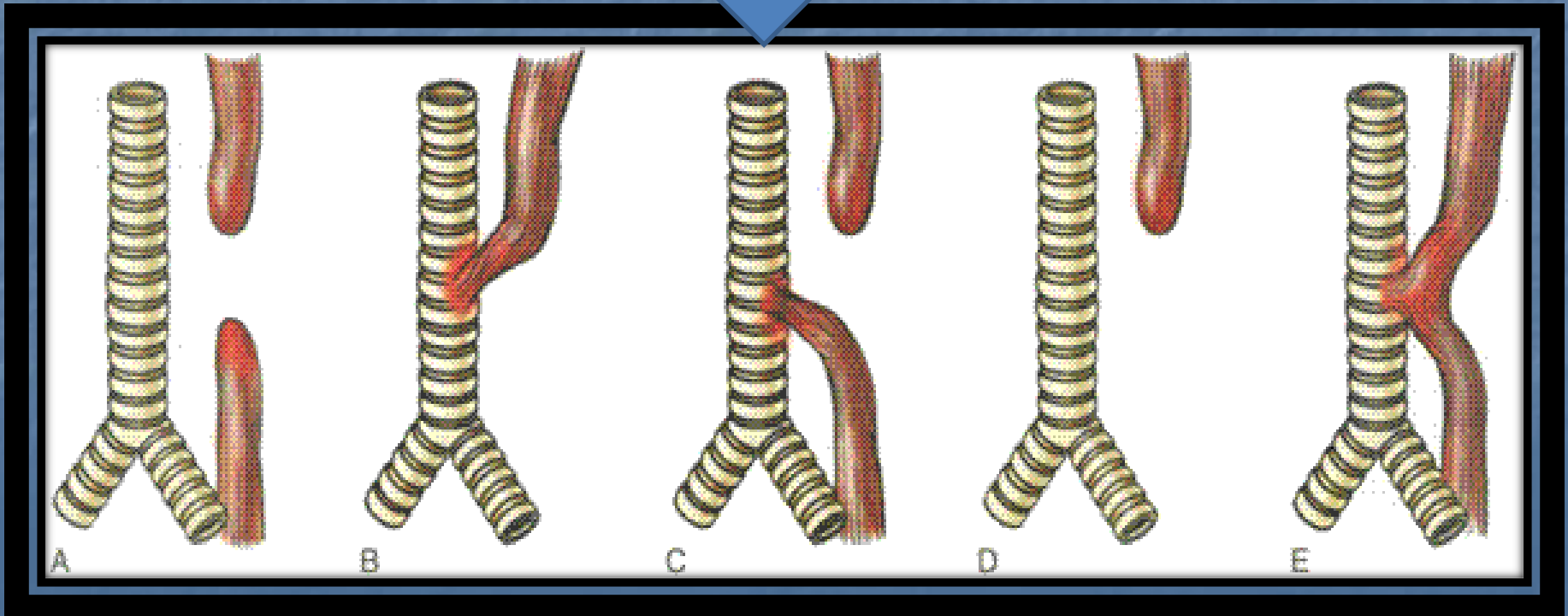


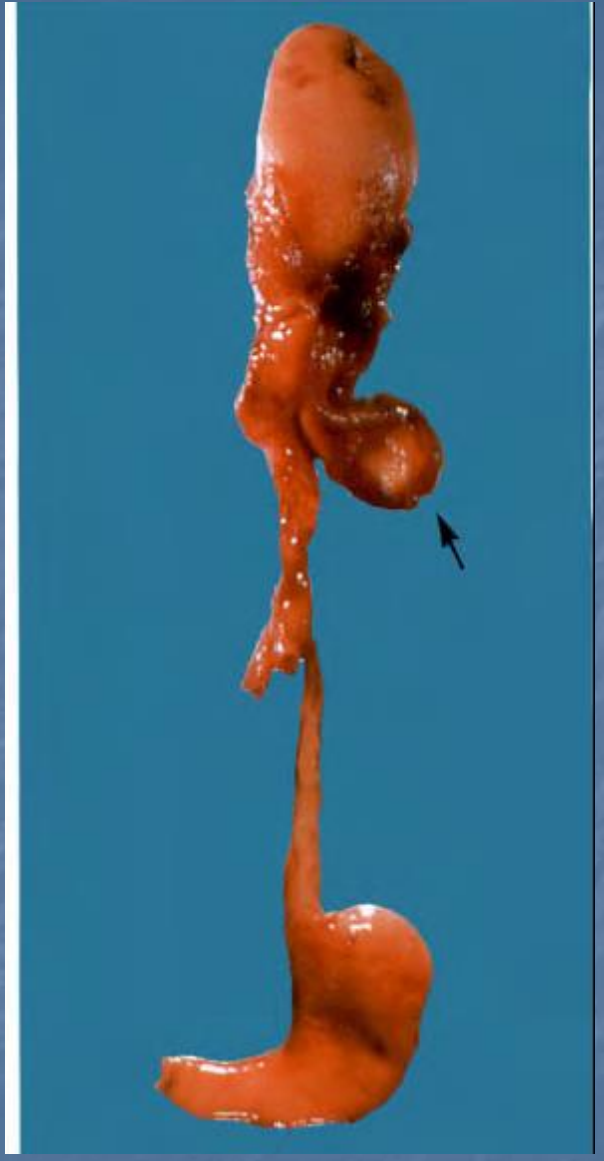
- **Esophageal motor disorders**
 - Achalasia
 - Hiatal hernia
 - Mallory-Weiss syndrome
- **Esophagitis**
 - Reflux esophagitis
 - Infectious esophagitis
 - Chemical esophagitis
- **Esophageal varices**
- **Tumors**
 - B – leiomyoma, papiloma, fibroma, lipoma, angioma
 - **Malignant**
 - Squamous cell carcinoma
 - Adenocarcinoma

CONGENITAL ANOMALIES

- ECTOPIC TISSUE (gastric, sebaceous, pancreatic)
- Atresia/Fistula/Stenosis/

MOST
COMMON





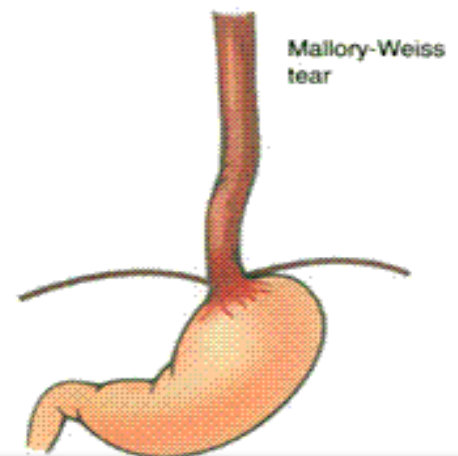
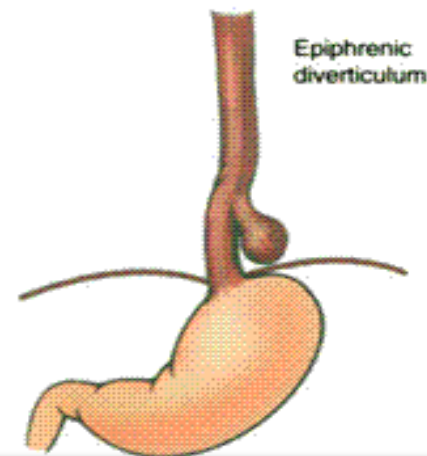
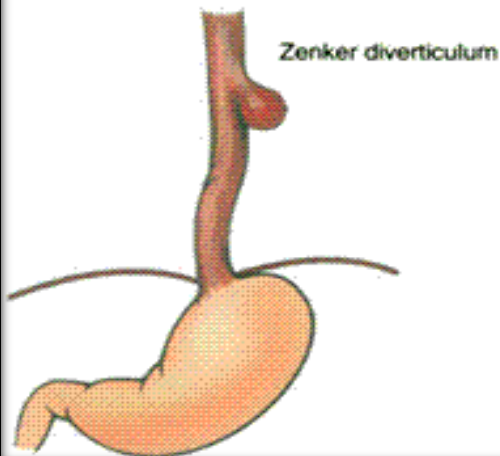
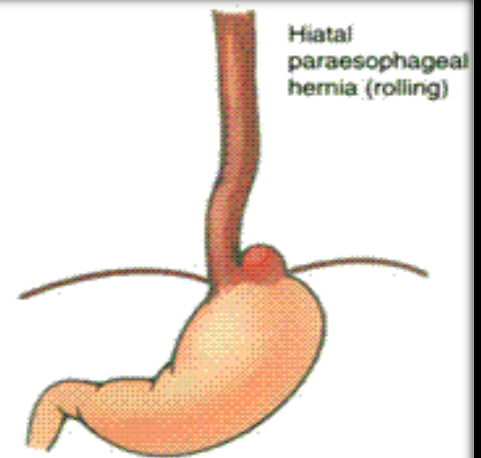
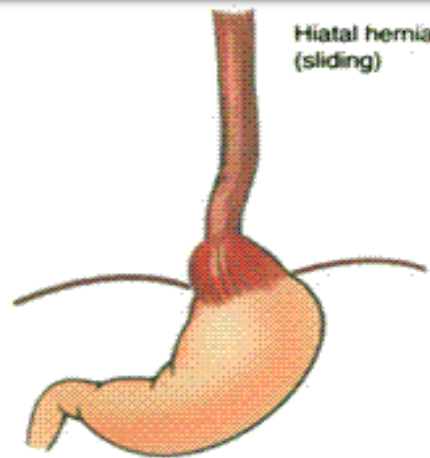
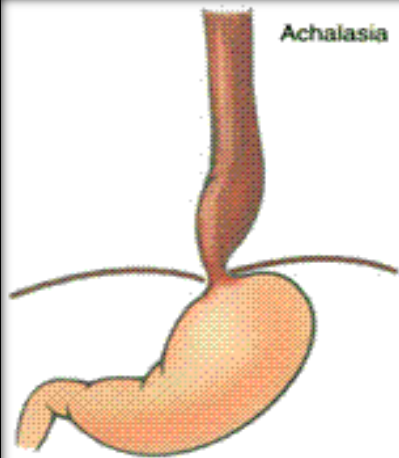
MOTOR DISORDERS

- **Achalasia**
- **Hiatal Hernia (sliding [95%], paraesophageal)**
- **“ZENKER” diverticulum**
- **Esophagophrenic diverticulum**
- **Mallory-Weiss tear**

Achalasia

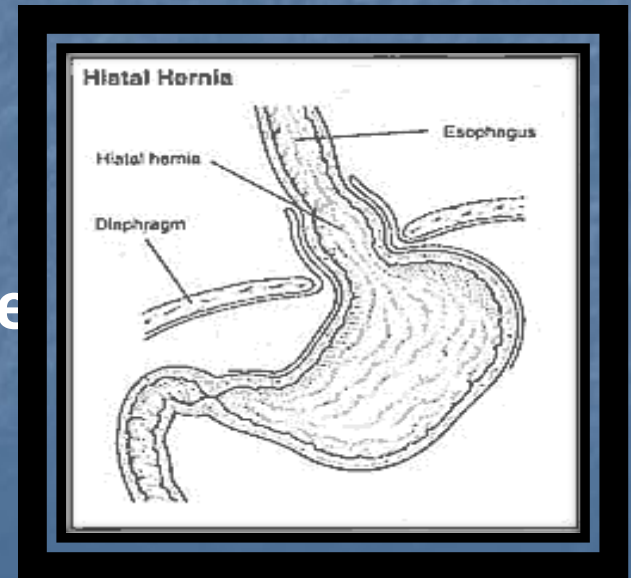
- “Failure to relax”
 - Aperistalsis
 - Incomplete relaxation of the LES
 - Increased LES tone
 - INCREASE: Gastrin, serotonin, acetylcholine, Prostaglandin F₂α, motilin, Substance P, histamine, pancreatic polypeptide
 - Progressive dysphagia starting in teens
 - Mostly UNCERTAIN etiology

MOTOR DISORDERS



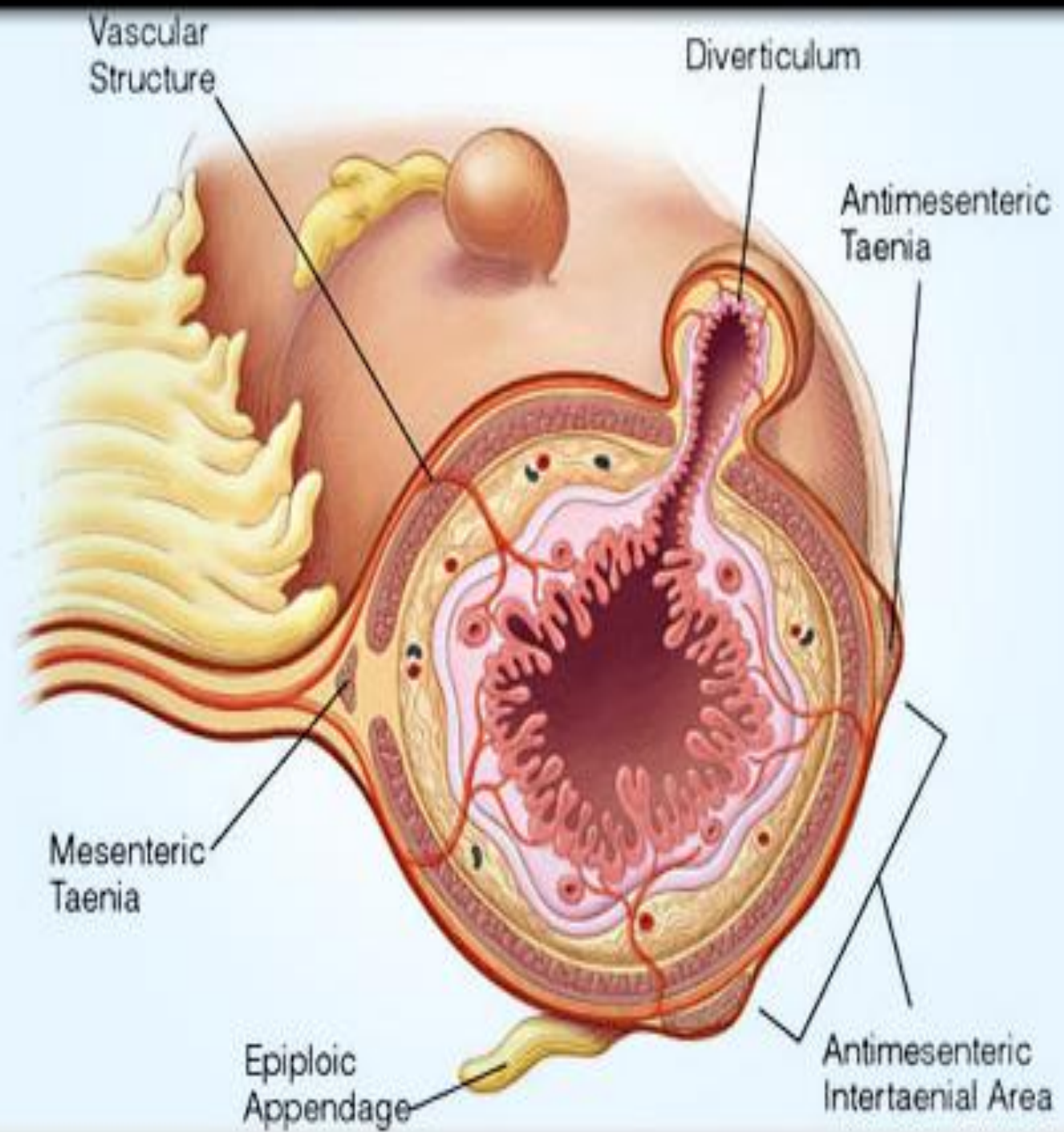
HIATAL HERNIA

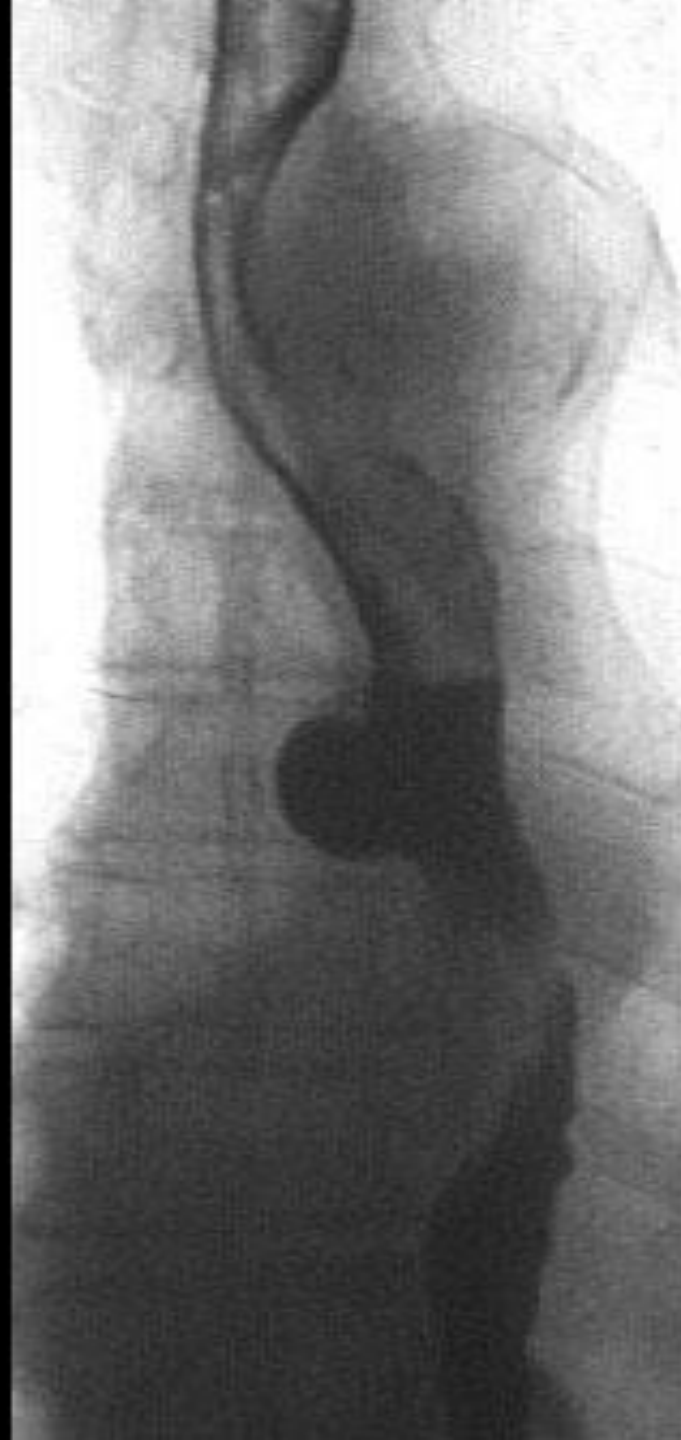
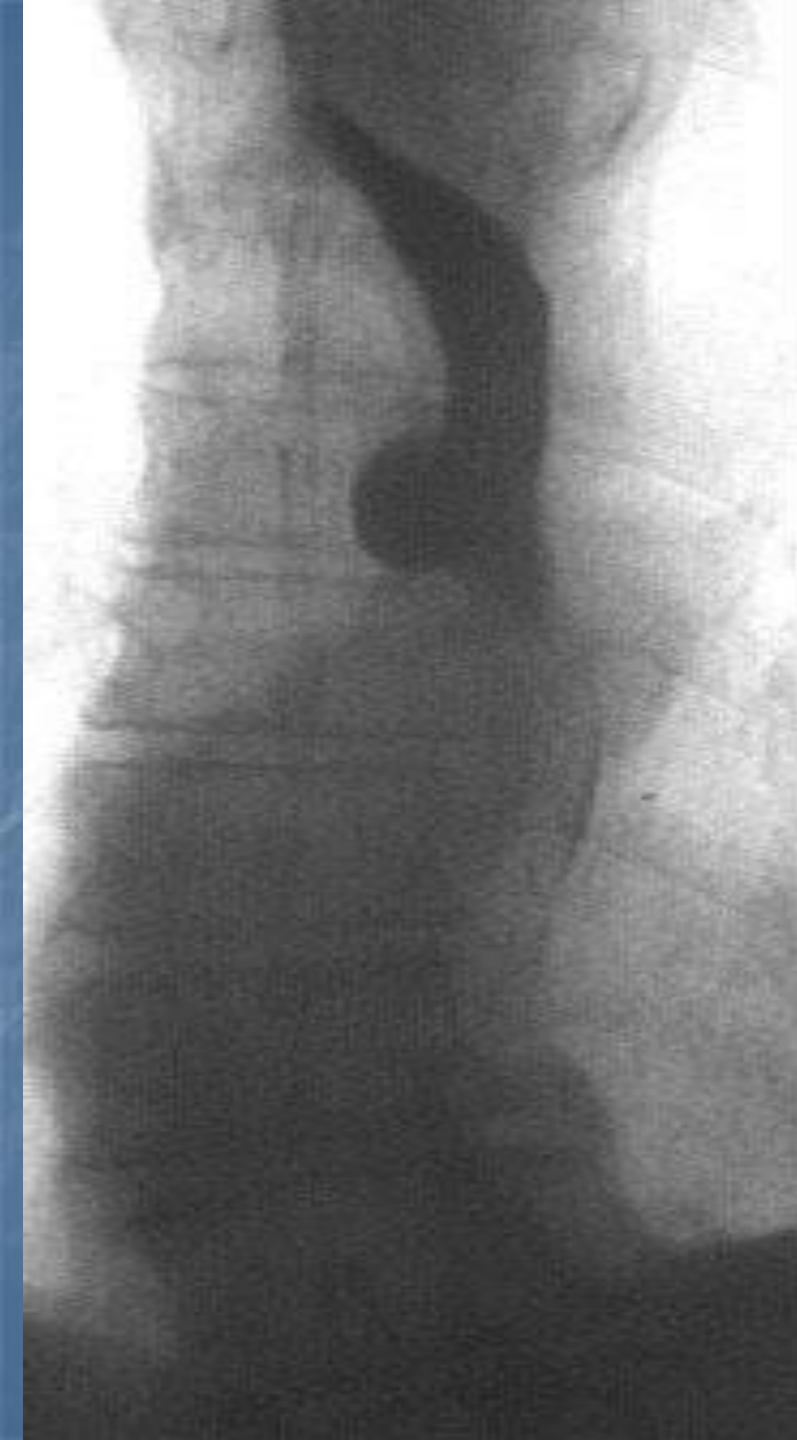
- Diaphragmatic muscular defect
- WIDENING of the space which the lower esophagus passes through
- IN ALL cases, STOMACH above diaphragm
- Usually associated with reflux
- Very common → Increases with age
- Ulceration, bleeding, perforation, strangulation



DIVERTICULA

- ZENKER (HIGH)
- TRACTION (MID)
- EPIPHRENIC (LOW)
- TRUE vs. FALSE?

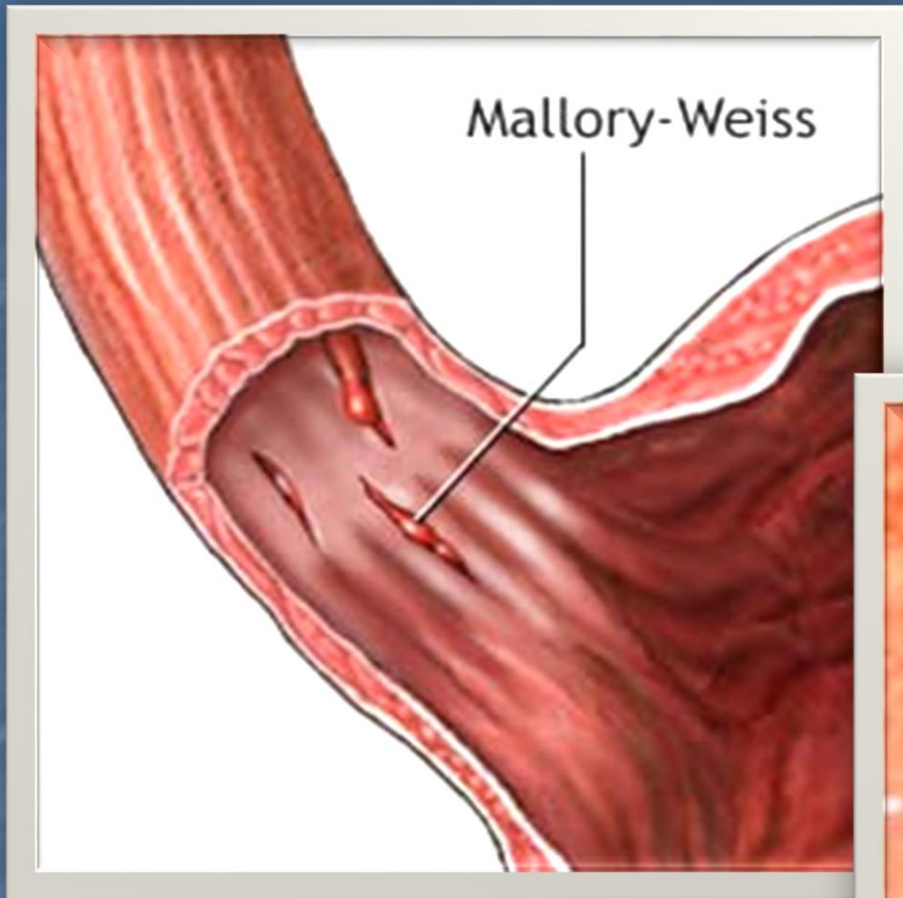




LACERATION

- Tears are **LONGITUDINAL** (lower esophagus)
- Usually secondary to severe **VOMITING**
- Usually in **ALCOHOLICS**
- Usually **MUCOSAL** tears
- By convention, they are all called:

MALLORY-WEISS



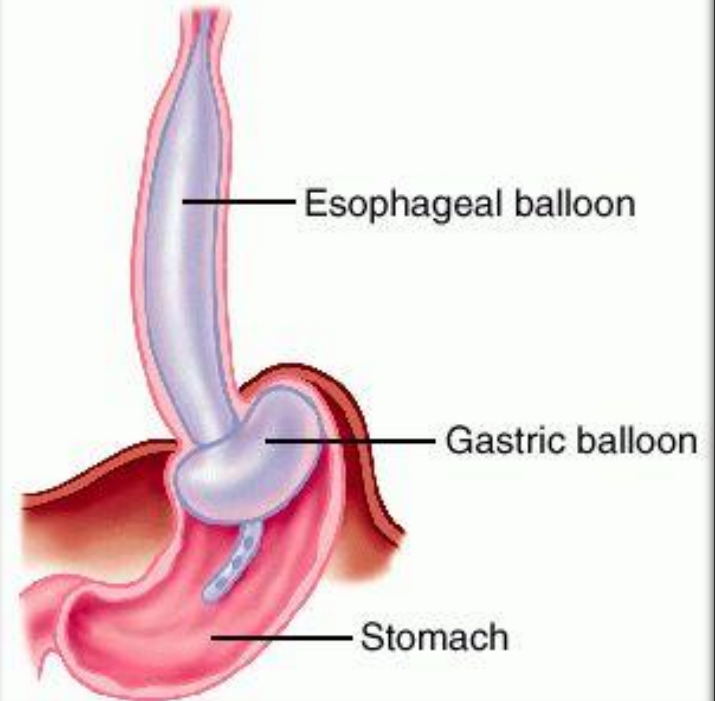
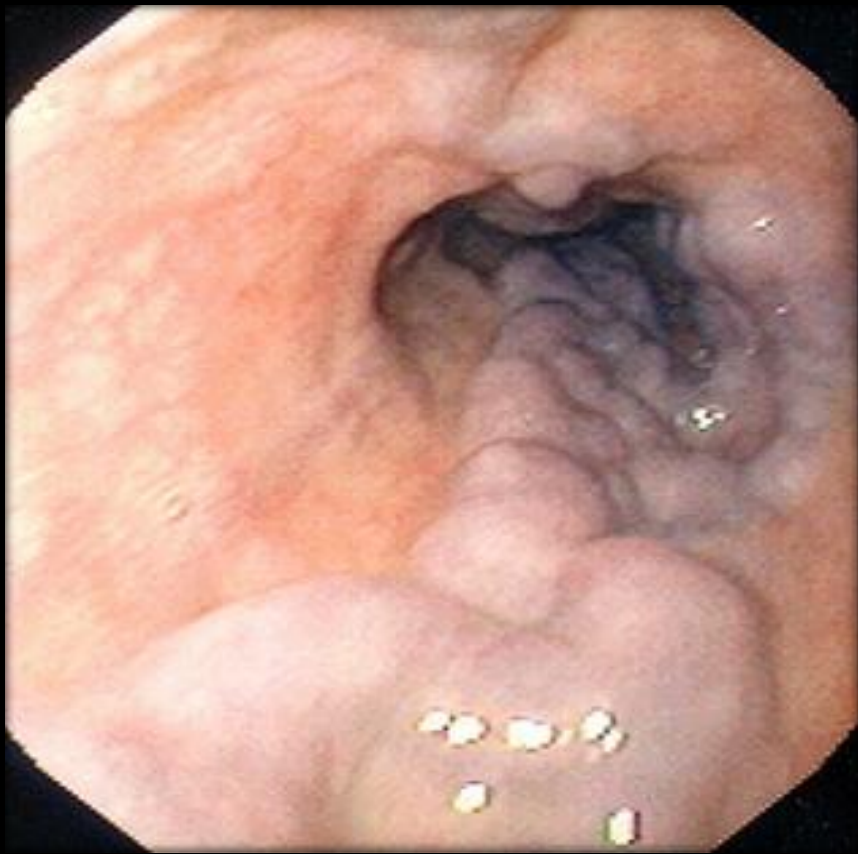
VARICES

- **THREE common areas of portal/caval anastomoses**
 - **Esophageal**
 - **Umbilical**
 - **Hemorrhoidal**
- **100% related to portal hypertension**
- **Found in 90% of cirrhotics**
- **MASSIVE, SUDDEN, FATAL hemorrhage is the most feared consequence**

VARIICES

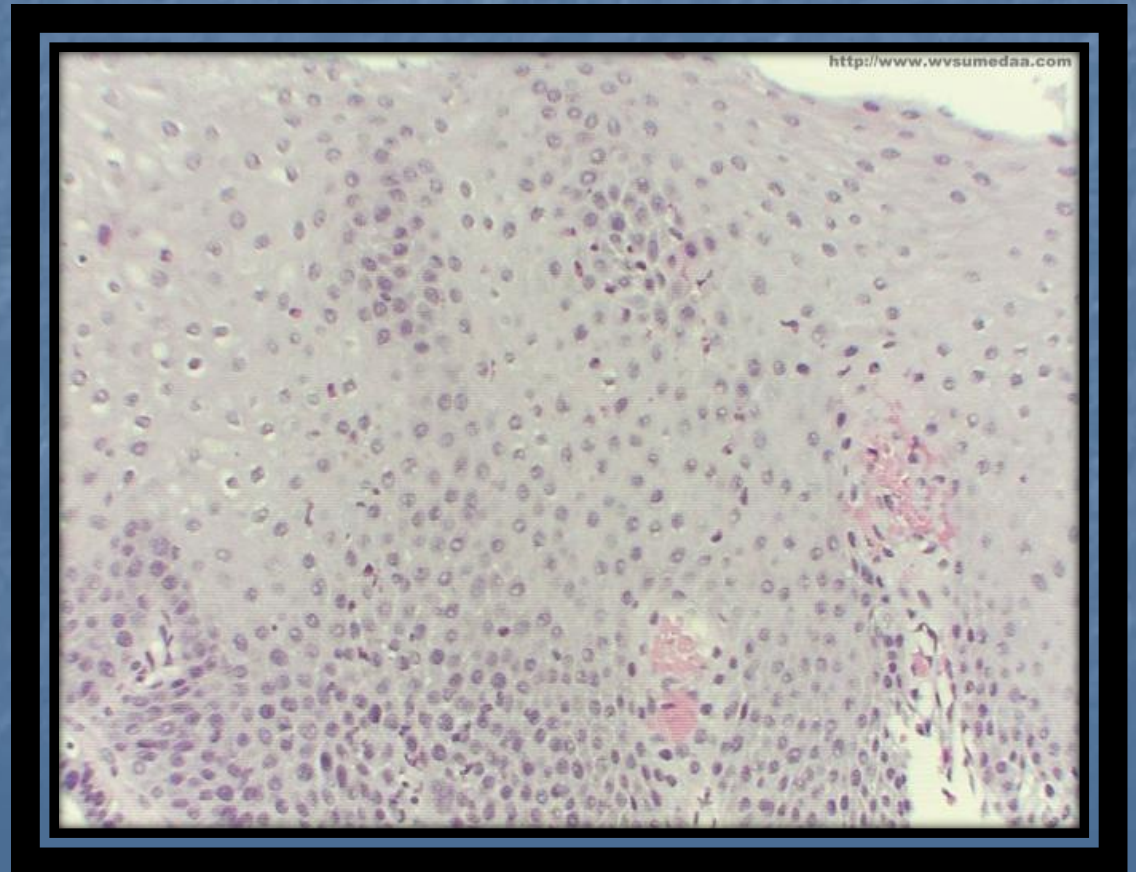


VARIICES



ESOPHAGITIS

- GERD/Reflux →
- Barrett's
- Chemical
- Infectious



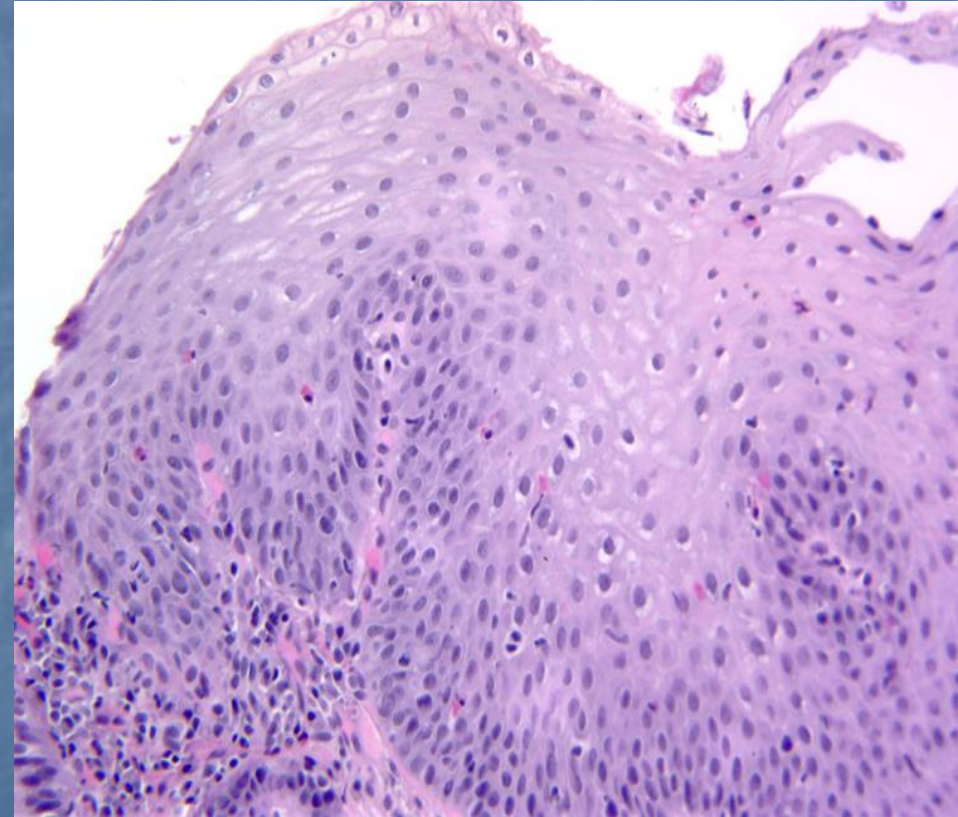
REFLUX/GERD

- **DECREASED LES tone**
- **Hiatal Hernia**
- **Slowed reflux clearing**
- **Delayed gastric emptying**

REFLUX/GERD

- Inflammatory Cells
 - Eosinophils
 - Neutrophils
 - Lymphocytes
- Basal zone hyperplasia
- Lamina Propria papillae elongated and congested, due to regeneration

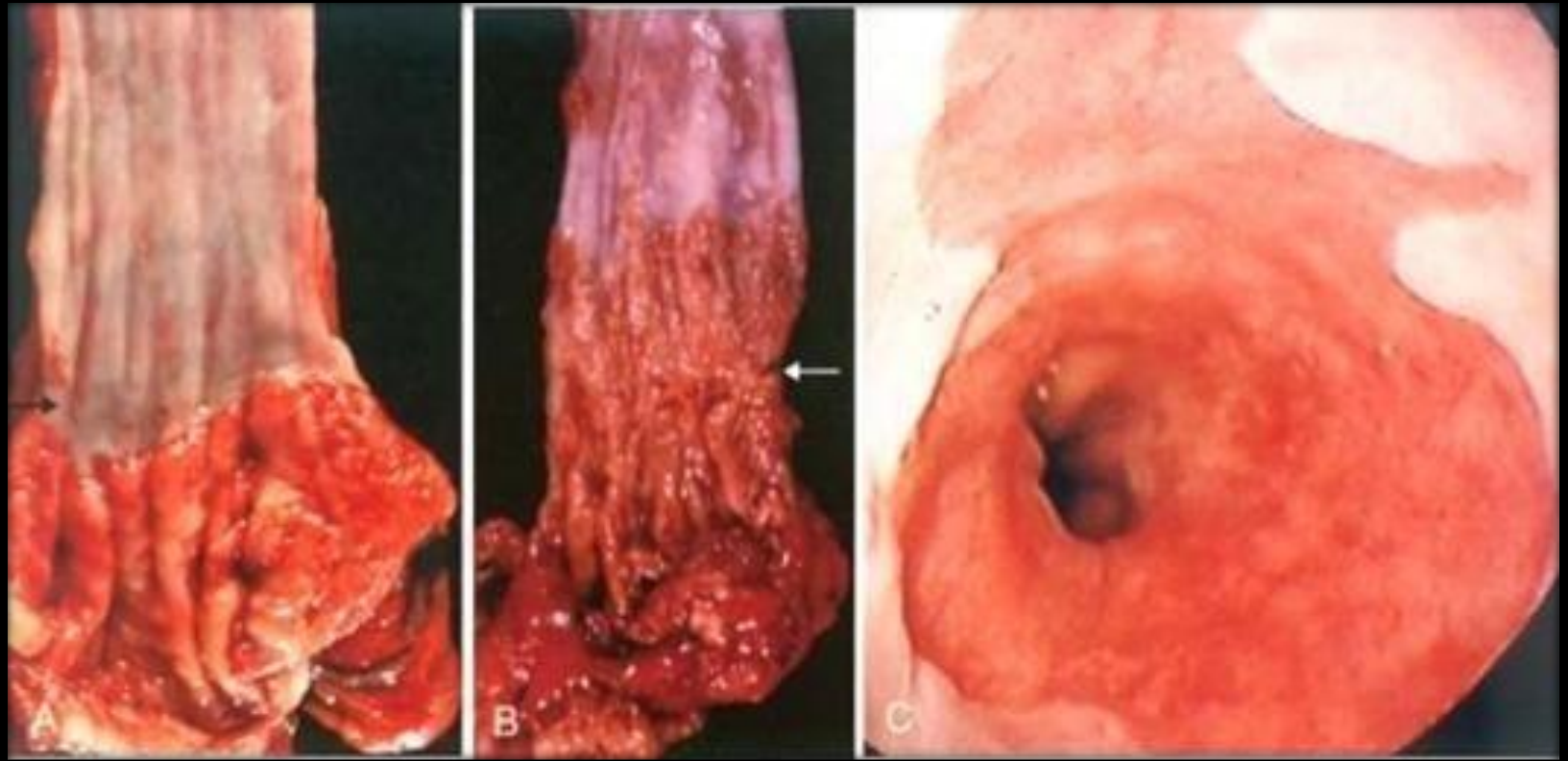
REFLUX/GERD

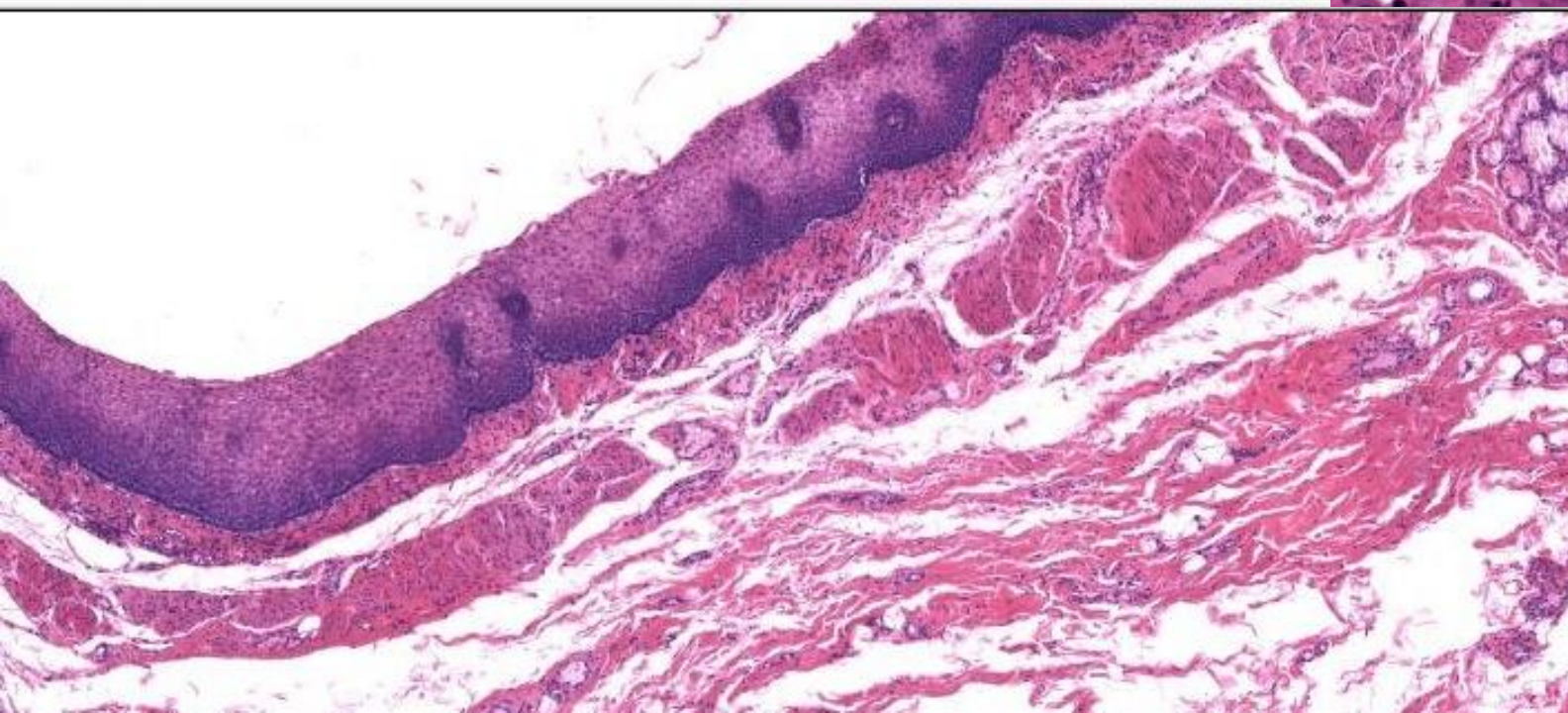
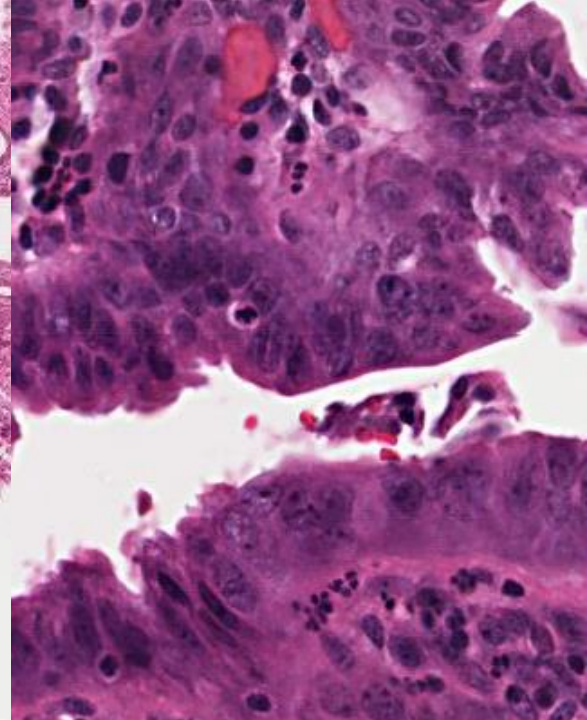
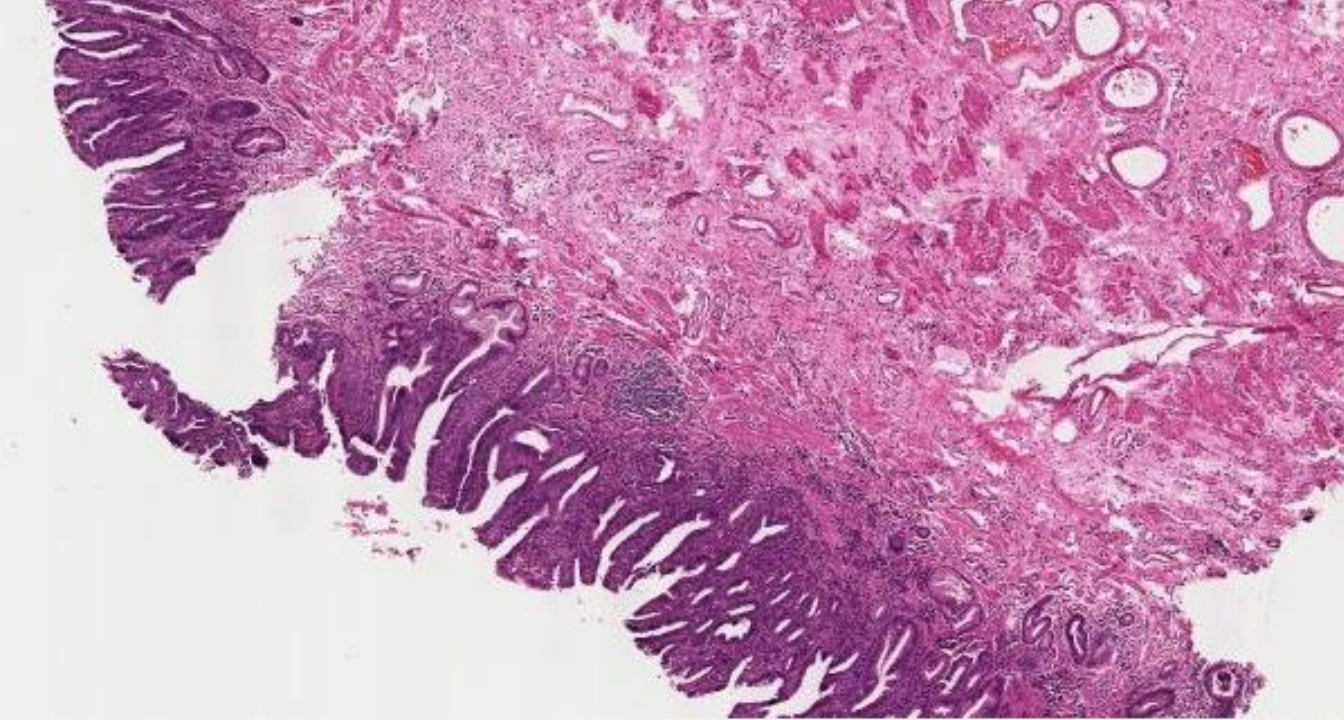


BARRETT'S ESOPHAGUS

- Can be defined as intestinal metaplasia of a normally SQUAMOUS esophageal mucosa. The presence of GOBLET CELLS in the esophageal mucosa is DIAGNOSTIC.
- SINGLE most common RISK FACTOR for esophageal adenocarcinoma
- 10% of GERD patients get it
- “BREACHED” G-E junction

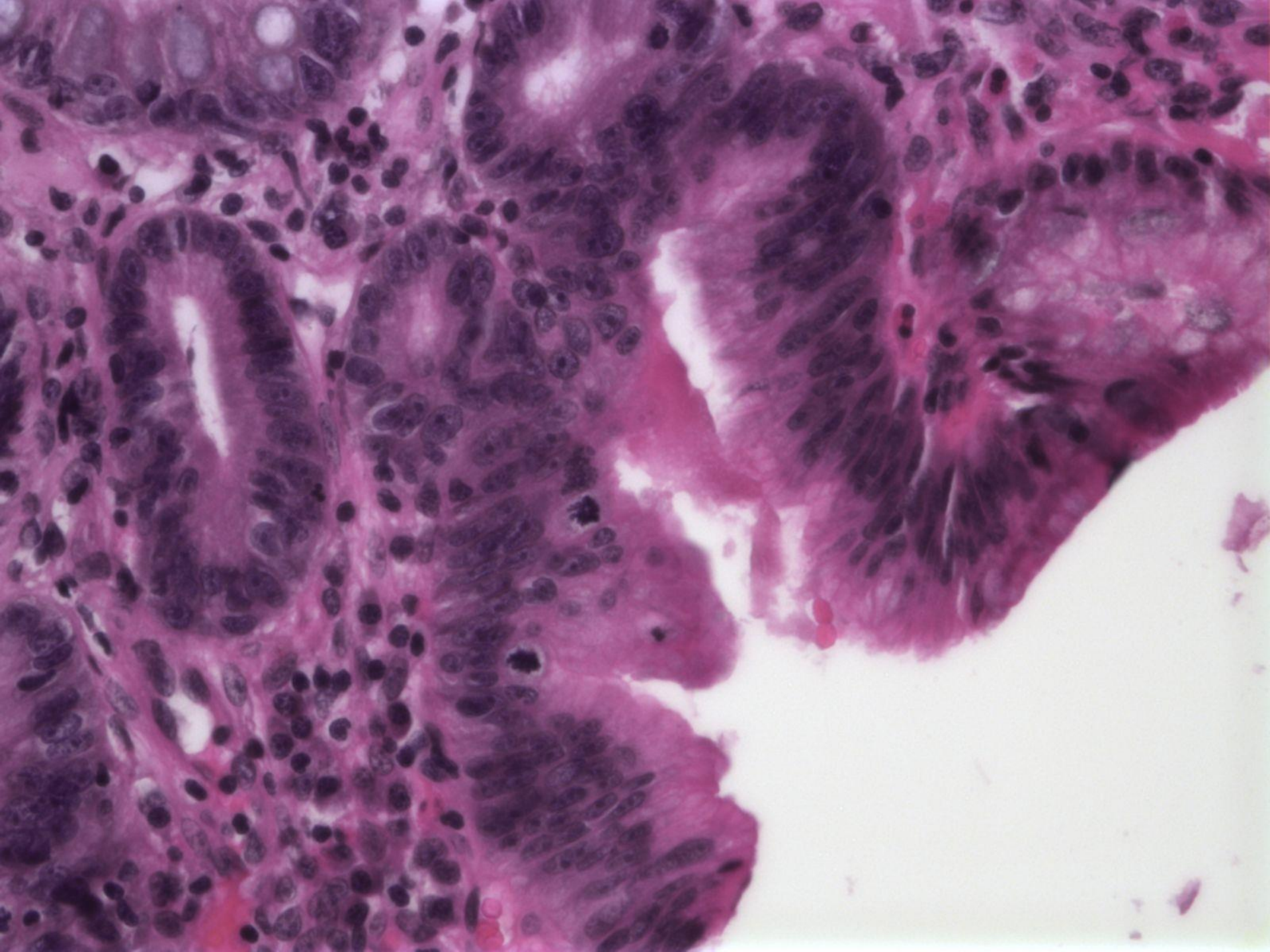
BARRETT'S ESOPHAGUS





BARRETT'S ESOPHAGUS

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



ESOPHAGITIS

- **CHEMICAL**

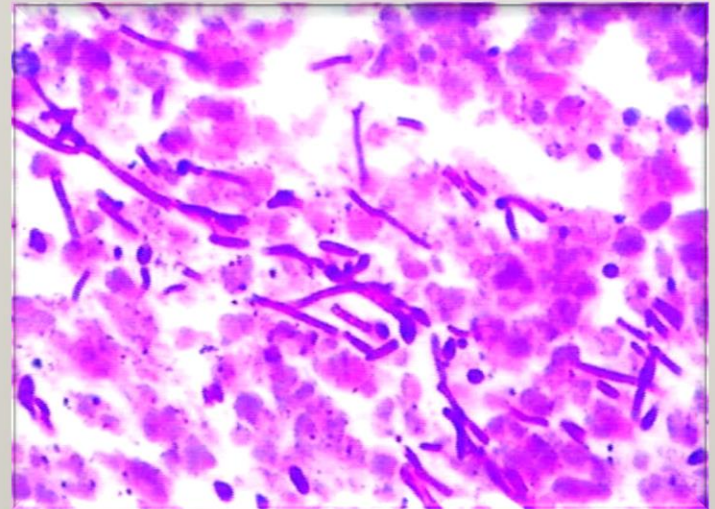
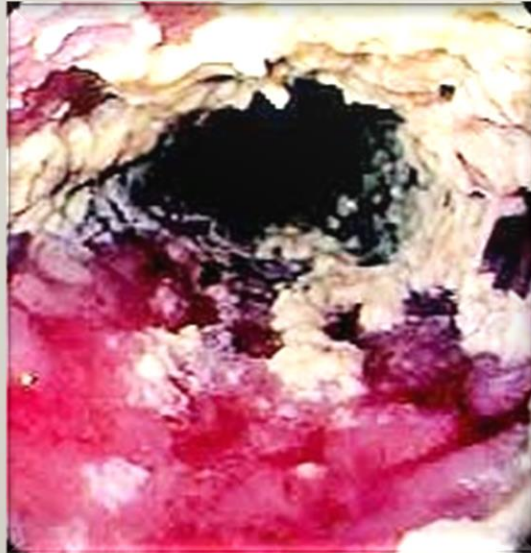
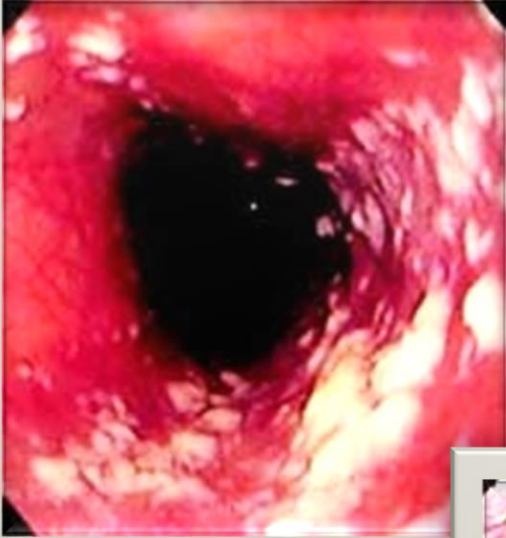
- Suicide attempts with strictures
- Alcohol
- Extremely HOT drinks
- CHEMO (often harmful to ALL high turnover mucosas)

- **INFECTIOUS**

- HSV, CMV, Fungal (especially CANDIDA)

ESOPHAGITIS

Candida, candida esophagitis in a HIV positive patient often is indicative of “full blown” AIDS.



TUMORS

- BENIGN
- MALIGNANT
 - Squamous cell carcinoma
 - Adenocarcinoma

BENIGN TUMORS

- LEIOMYOMAS
- FIBROVASCULAR POLYPS
- CONDYLOMAS (HPV)
- LIPOMAS
- “GRANULATION” TISSUE (PSEUDOTUMOR)

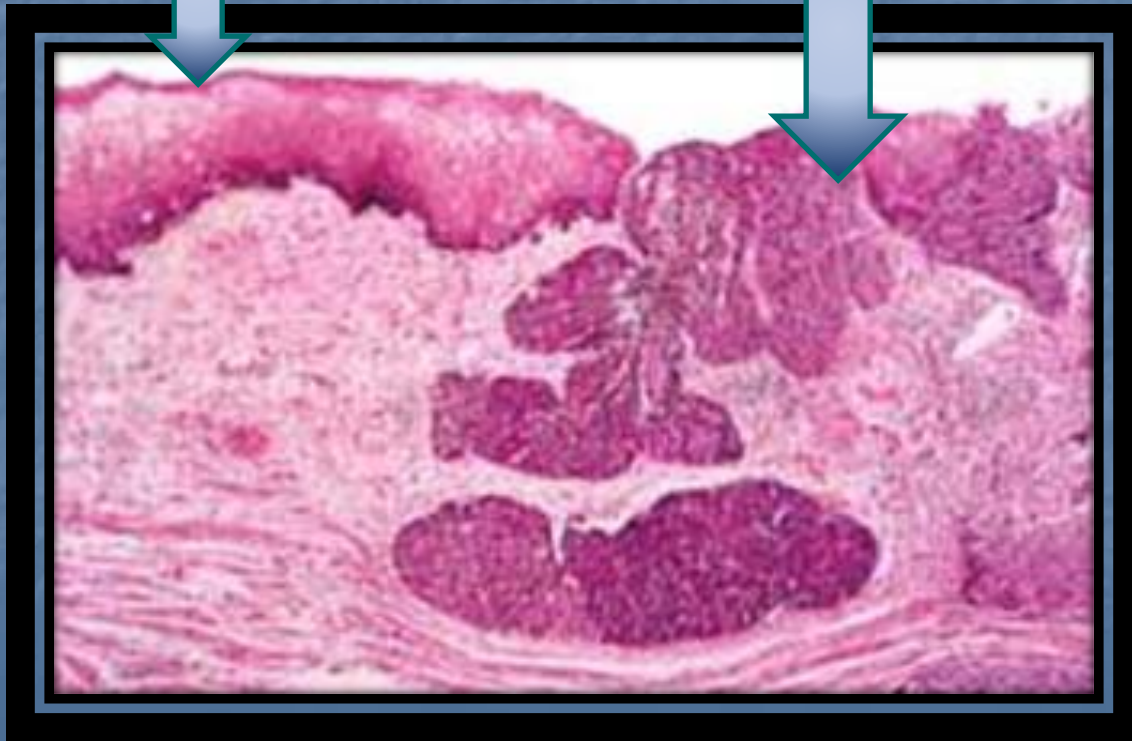


SQUAMOUS CARCINOMA

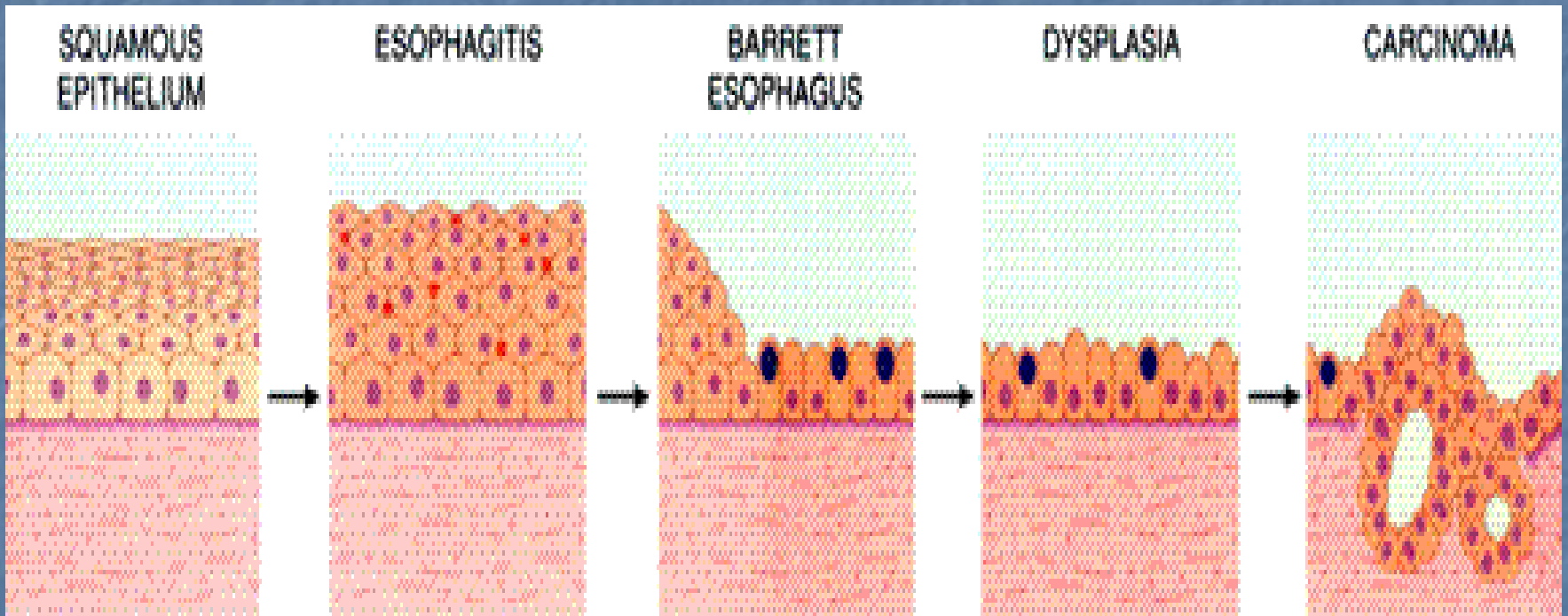
- Nitrites/Nitrosamines
- Fungi in food
- Tobacco
- Alcohol
- Esophagitis

SQUAMOUS CARCINOMA

- DYSPLASIA → IN-SITU → INFILTRATION

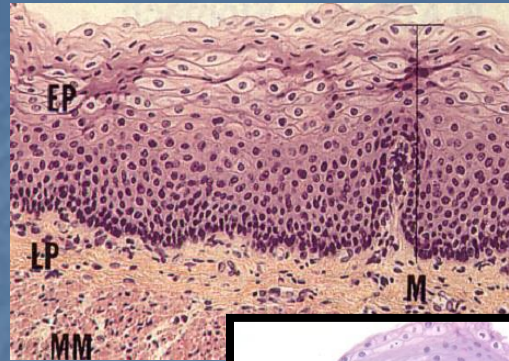


ADENOCARCINOMA

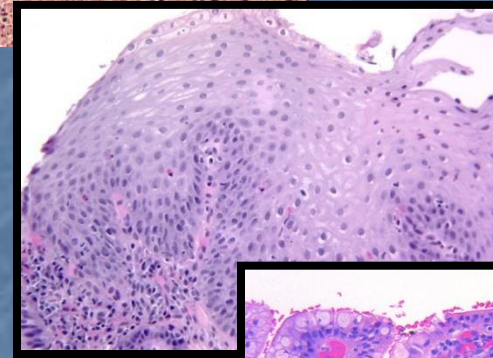


Pathogenesis of Adenocarcinoma

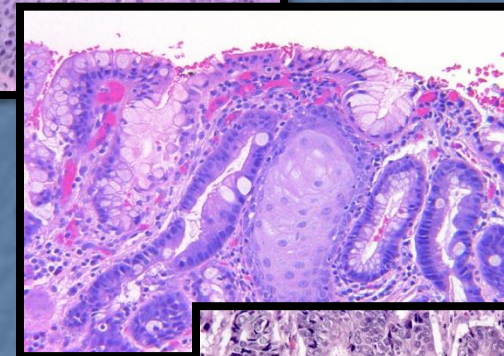
- Reflux Esophagitis



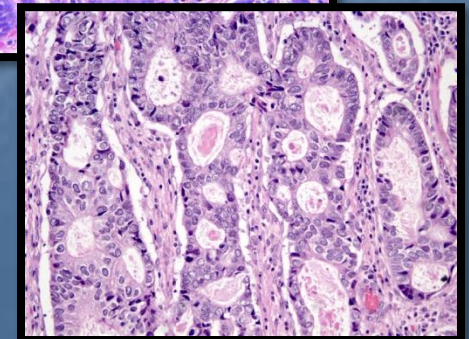
↓
Barrett's esophagus
(intestinal type)



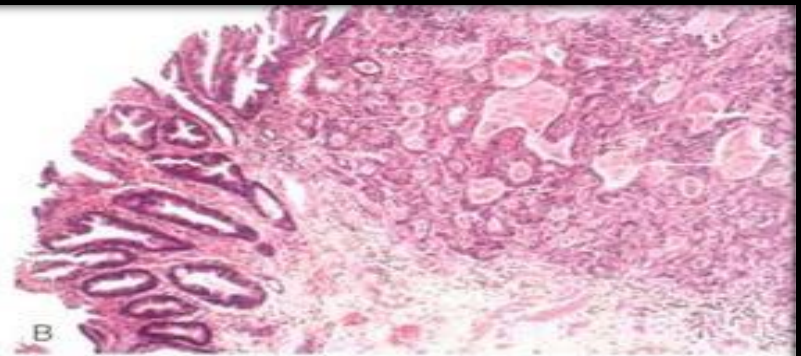
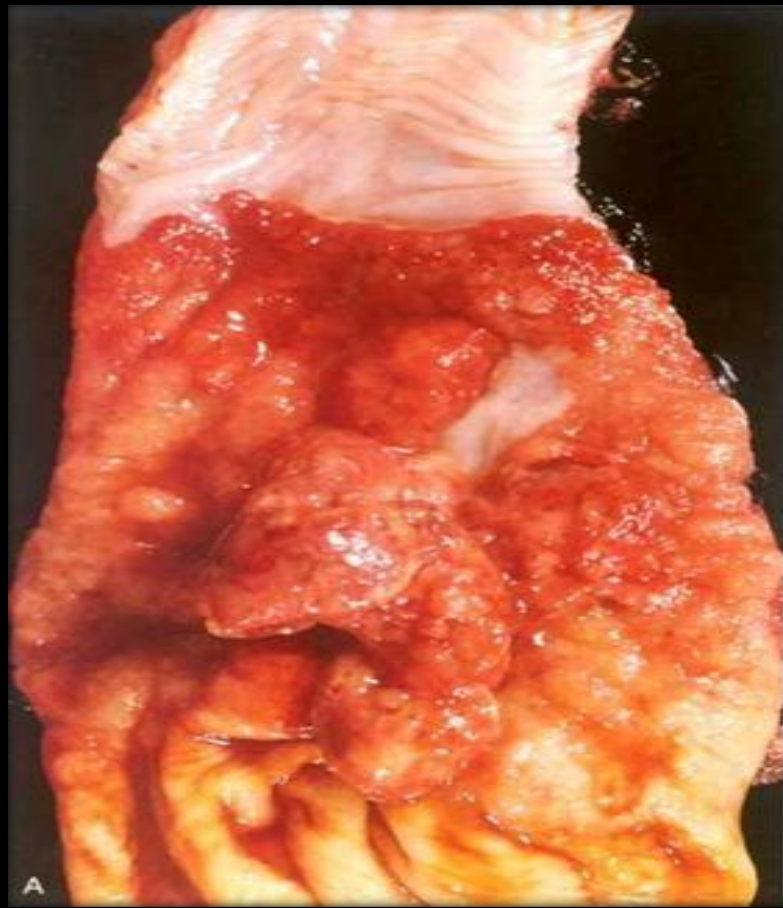
↓
Barrett's with dysplasia
(Carcinoma in-situ)



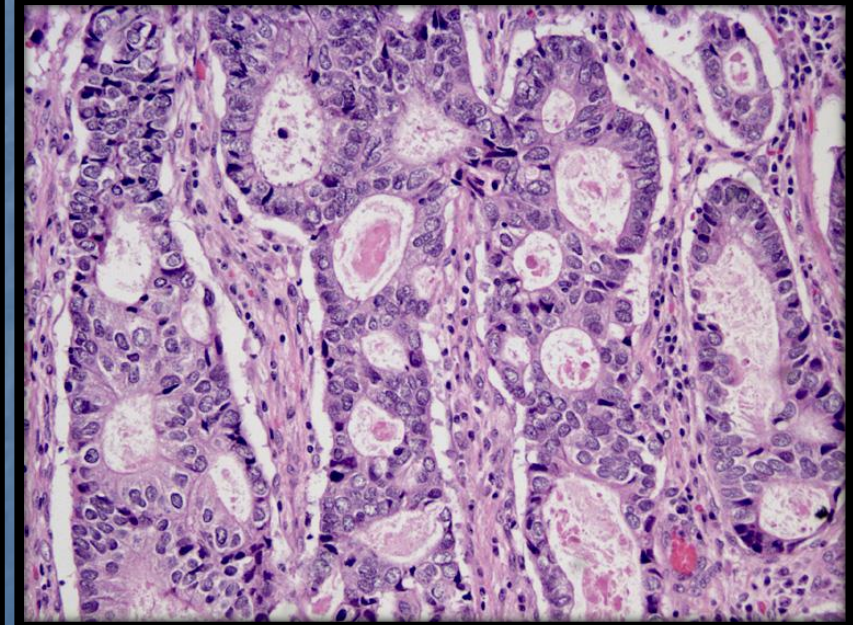
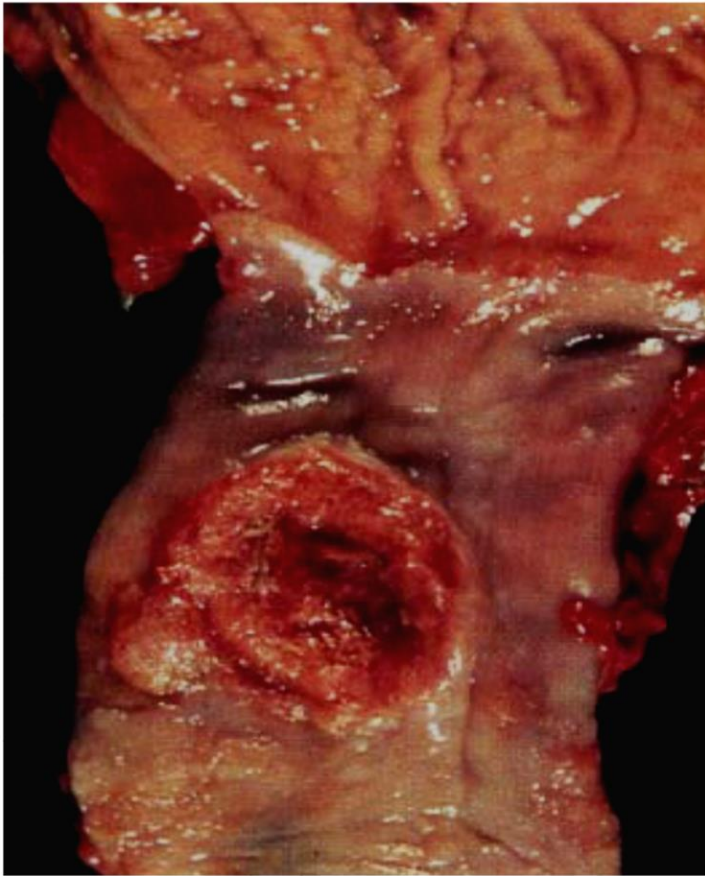
↓
Invasive
adenocarcinoma

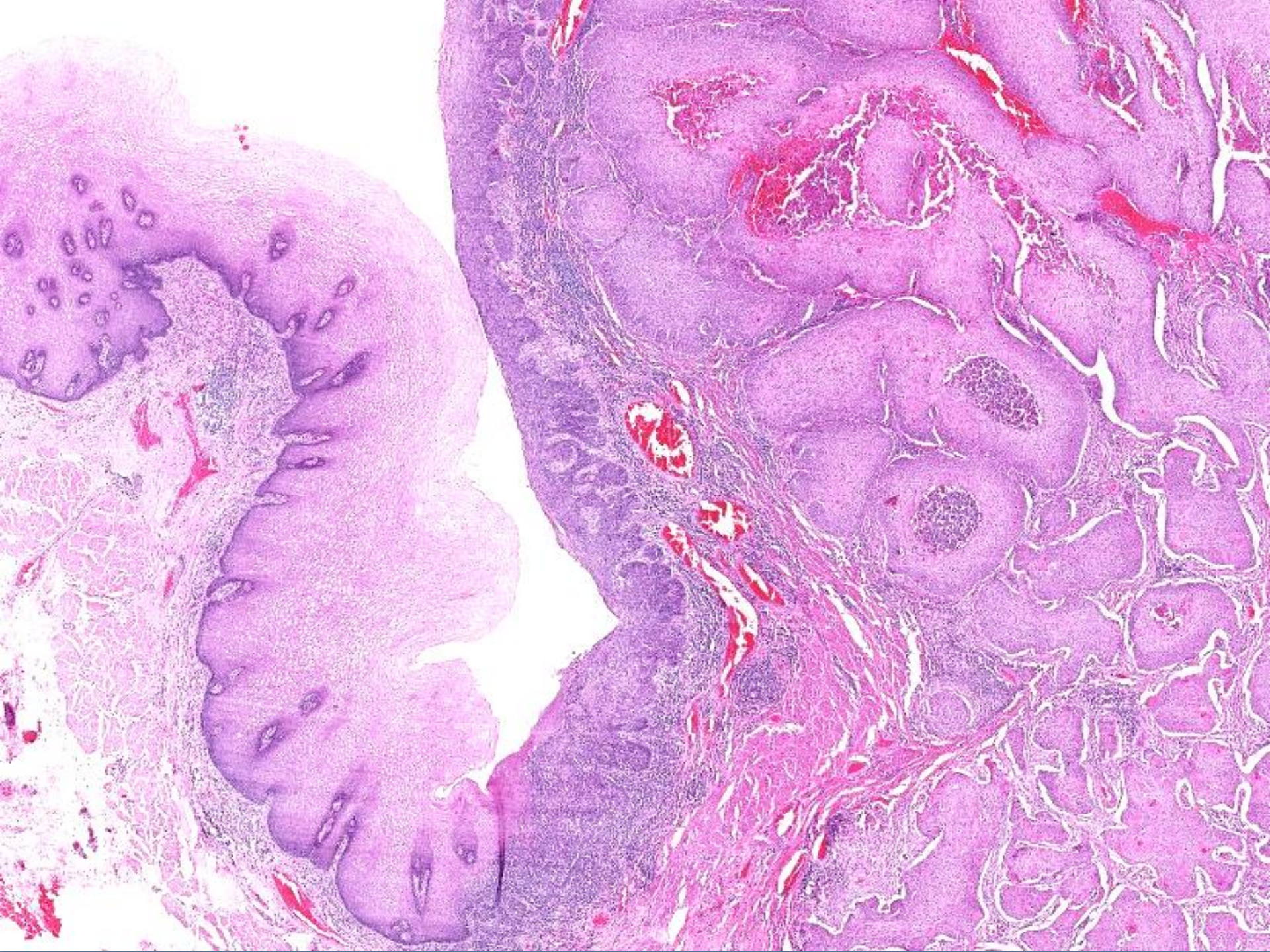


ADENOCARCINOMA

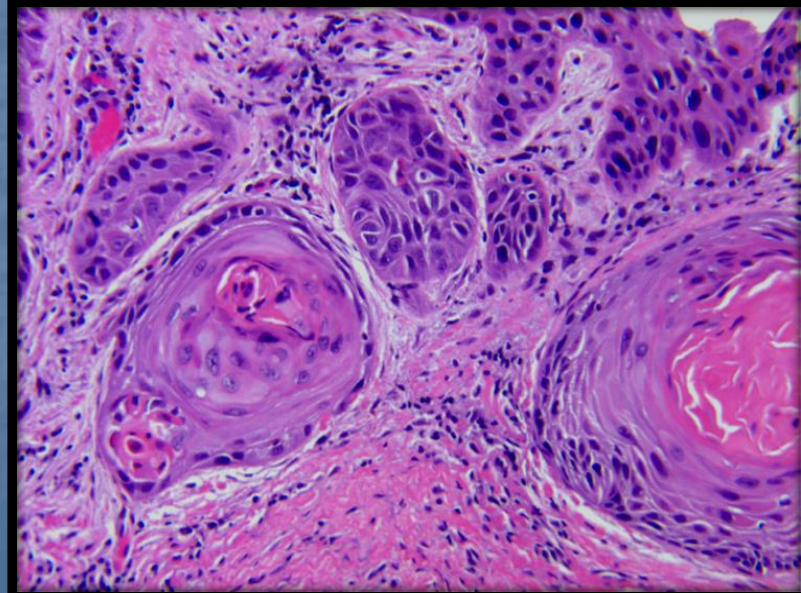


Esophageal Adenocarcinoma

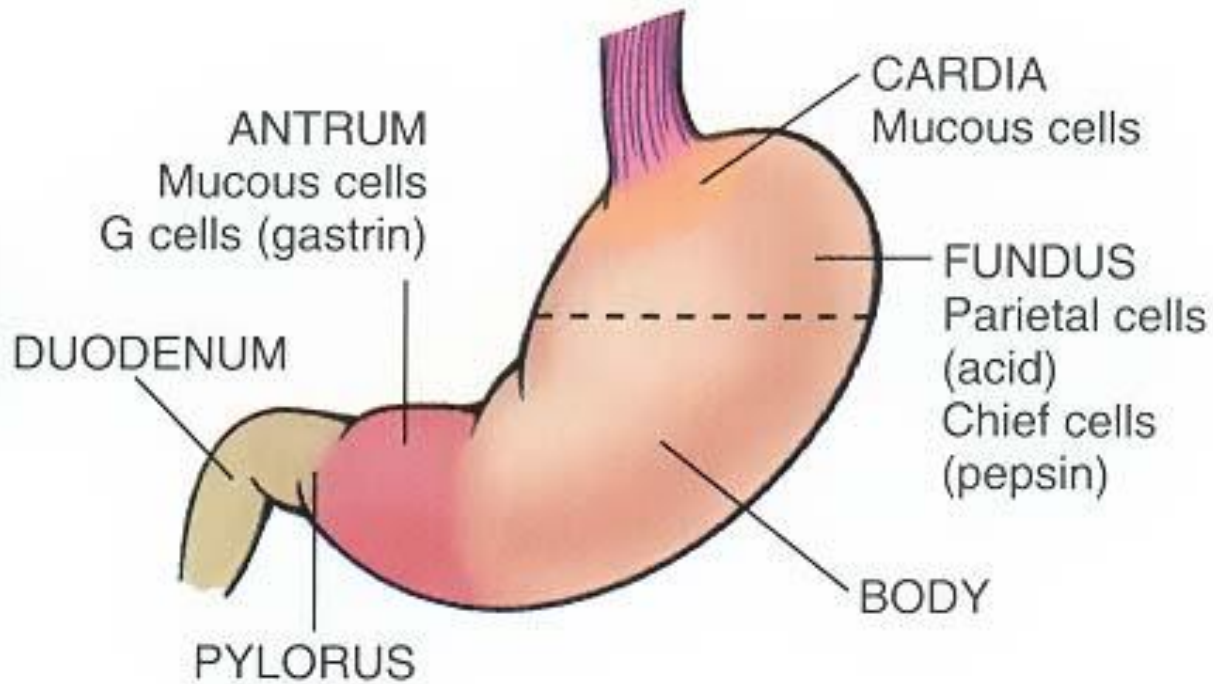




Esophageal Squamous Cell Carcinoma



Stomach



ANATOMY

**Cardia (esoph), Fundus (diaph),
Body (acid), Antrum, Pylorus**

Greater/Lesser Curvatures

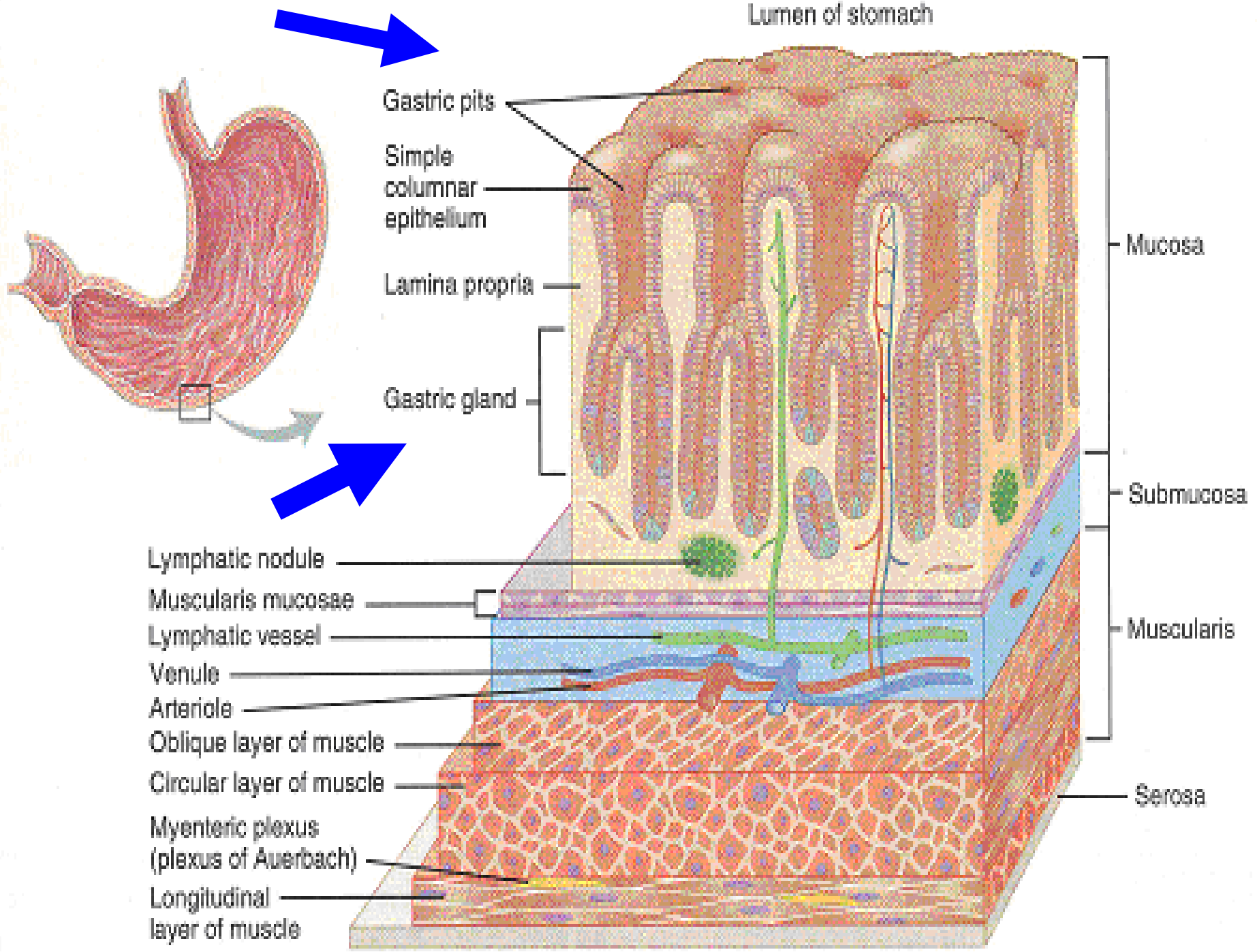
1500-3000 ml

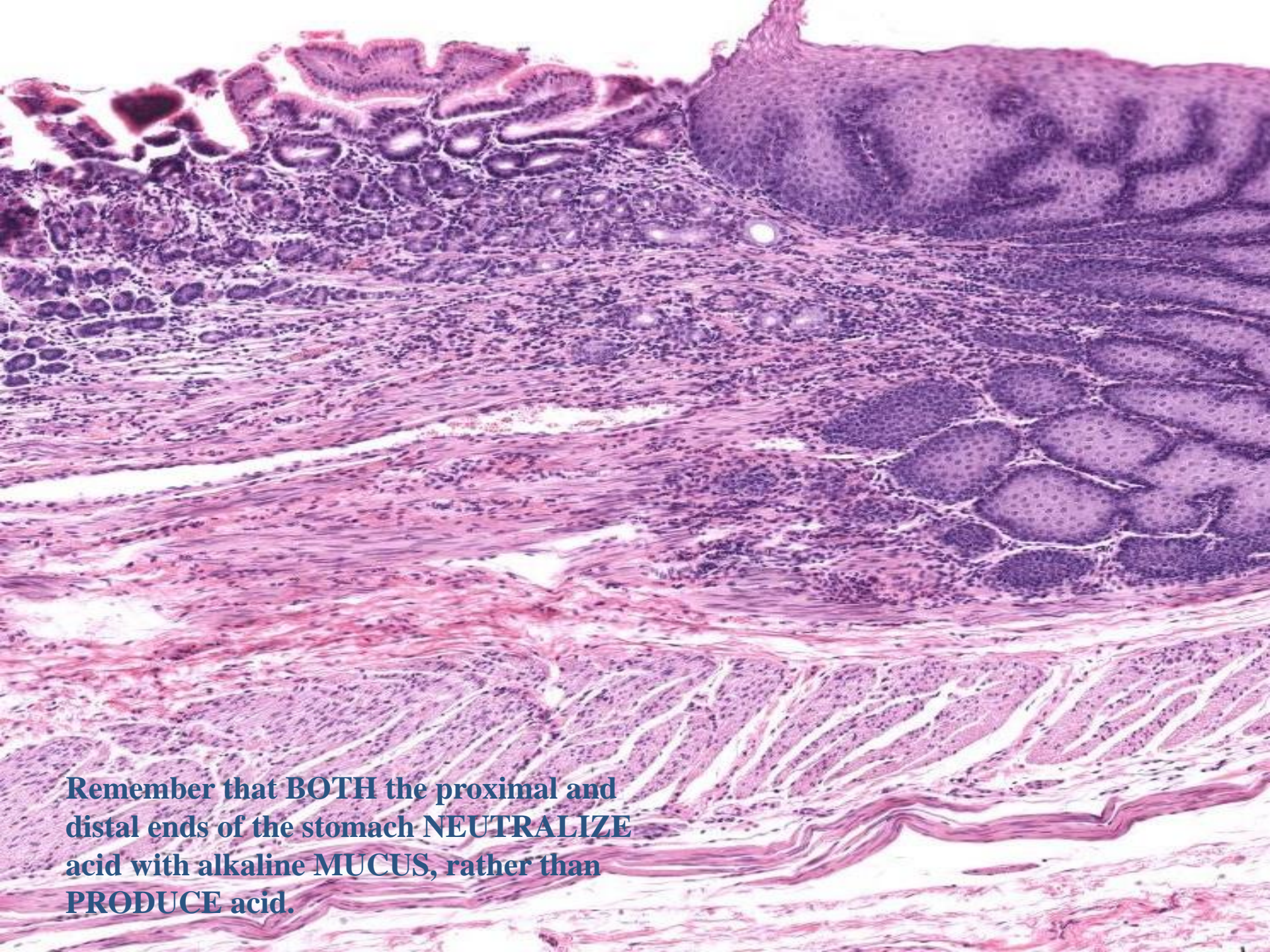
Rugae

INNERVATION: VAGUS, Sympathetic

VEINS: Portal

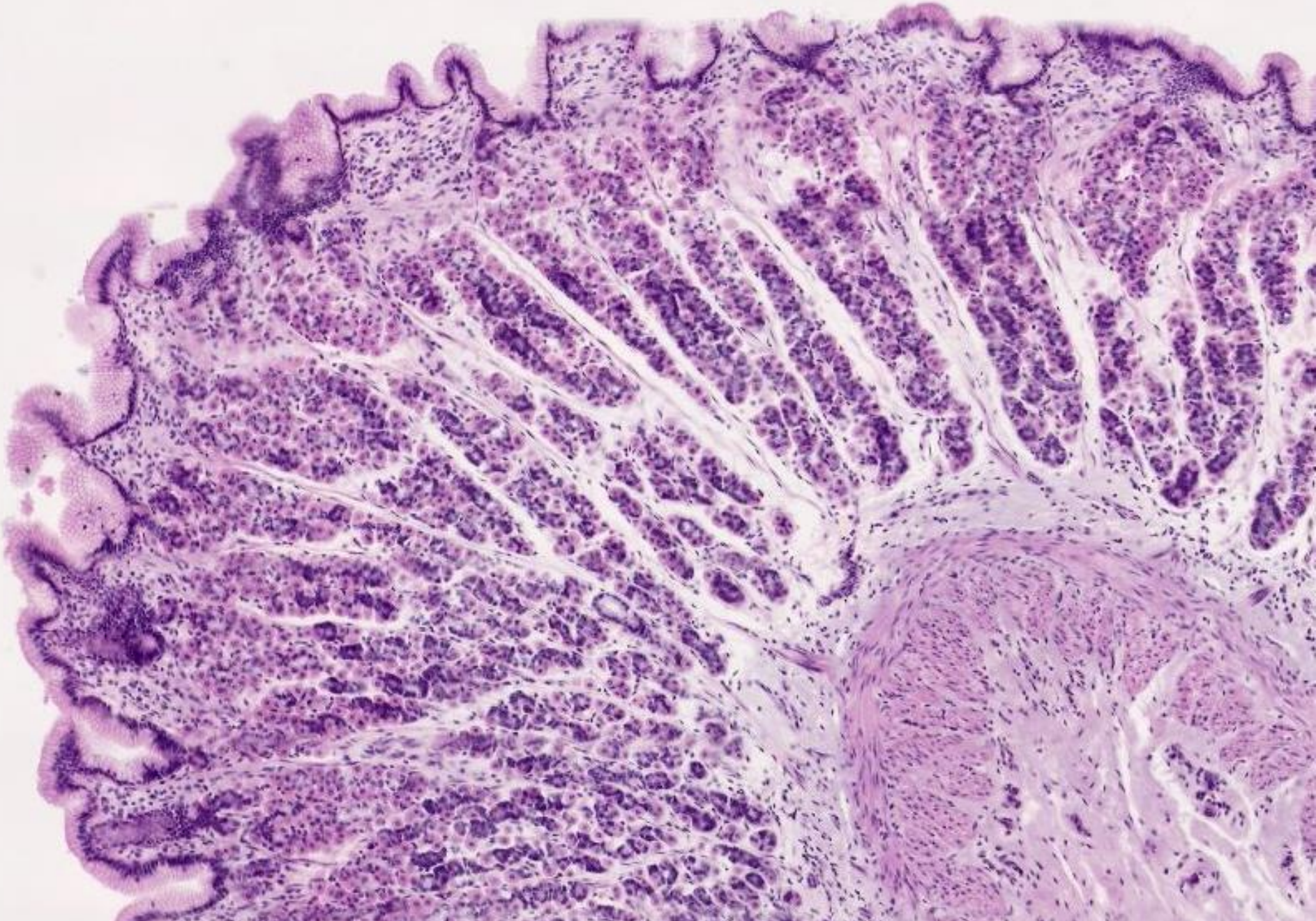
**Blood Supply: RG, LG, RGE(O), LGE(O),
SG, ALL 3 branches of the celiac, no
matter what the variations may be.**





Remember that BOTH the proximal and distal ends of the stomach NEUTRALIZE acid with alkaline MUCUS, rather than PRODUCE acid.

Body with numerous chief and parietal (acid producing) cells.





Pyloric sphincter, note a few submucosal glands seen a little BEFORE the sphincter itself.

CELLS

MUCOUS: MUCUS, PEPSINOGEN II

CHIEF: PEPSINOGEN I, II

PARIETAL: ACID

ENTEROENDOCRINE: HISTAMINE,
SOMATOSTATIN, ENDOTHELIN

ACID PROTECTION

MUCUS

HCO₃⁻

EPITHELIAL BARRIERS

BLOOD FLOW

PROSTAGLANDIN E, I

CONGENITAL

- **ECTOPIC PANCREAS** (ectopic pancreas tissue → stomach), very common
- **ECTOPIC GASTRIC** (ectopic gastric tissue → pancreas), not rare
- **Diaphragmatic HERNIA** → Failure of diaphragm to close, not rare

PYLORIC STENOSIS

- **CONGENITAL: (1/500),**
Neonatal obstruction
symptoms, pyloric splitting
curative
- **ACQUIRED: Secondary to**
extensive scarring such as
advanced peptic ulcer disease

GASTRITIS

- ACUTE
- CHRONIC
- AUTOIMMUNE
- OTHER
 - EOSINOPHILIC
 - ALLERGIC
 - LYMPHOCYTIC
 - GRANULOMATOUS

Acute Gastritis Causes

Heavy use of nonsteroidal anti-inflammatory drugs (NSAIDs), particularly aspirin

Excessive alcohol consumption

Heavy smoking

Treatment with cancer chemotherapeutic drugs

Uremia

Systemic infections (e.g., salmonellosis)

Severe stress (e.g., trauma, burns, surgery)

Ischemia and shock

Suicide attempts with acids and alkali

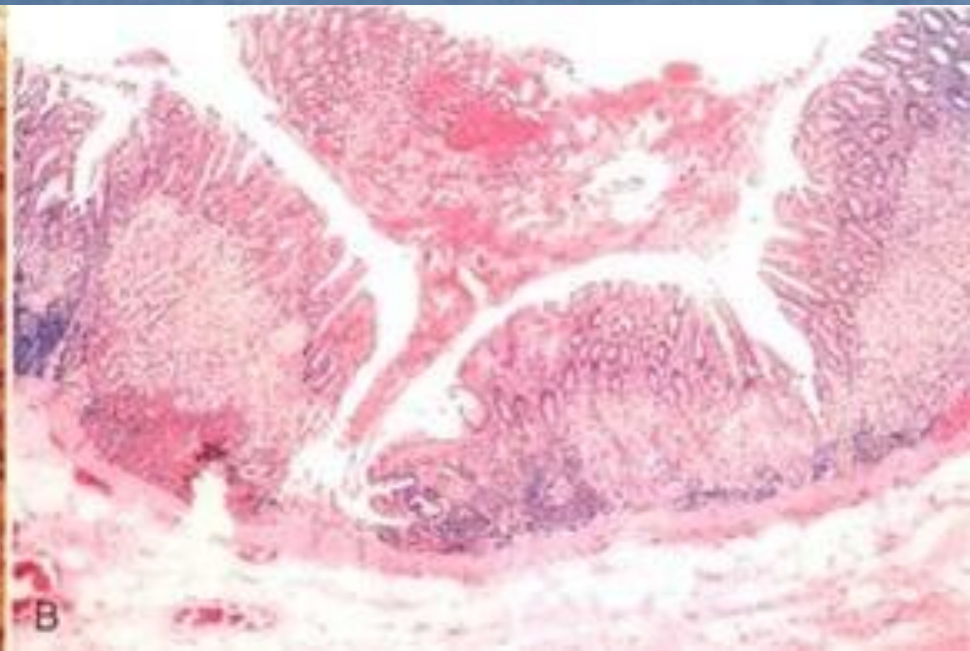
Mechanical trauma (e.g., nasogastric intubation)

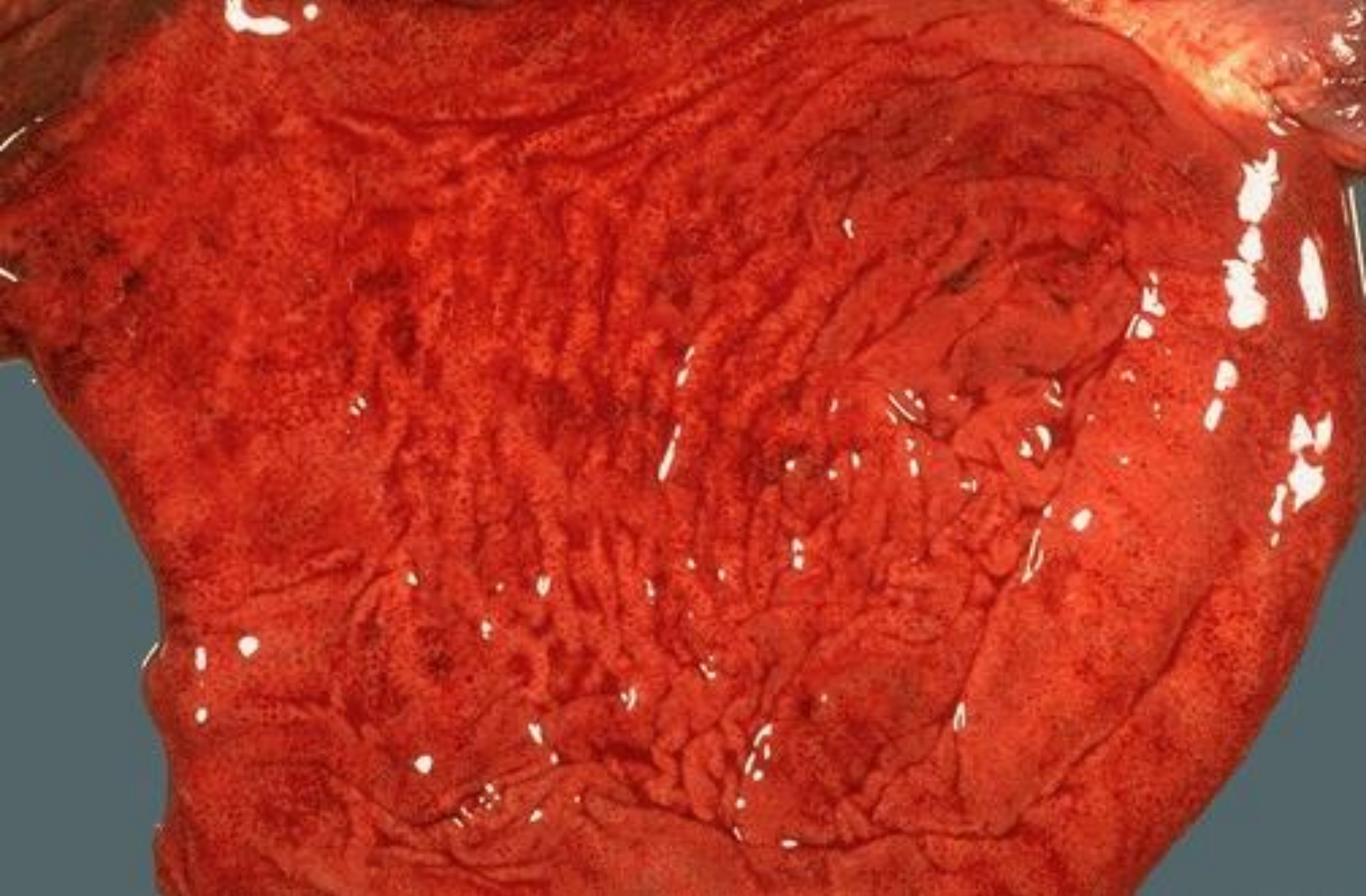
After distal gastrectomy with reflux of bilious material

The main CLINICAL differentiation between acute and chronic gastritis would be the presence or absence of blood, respectively.

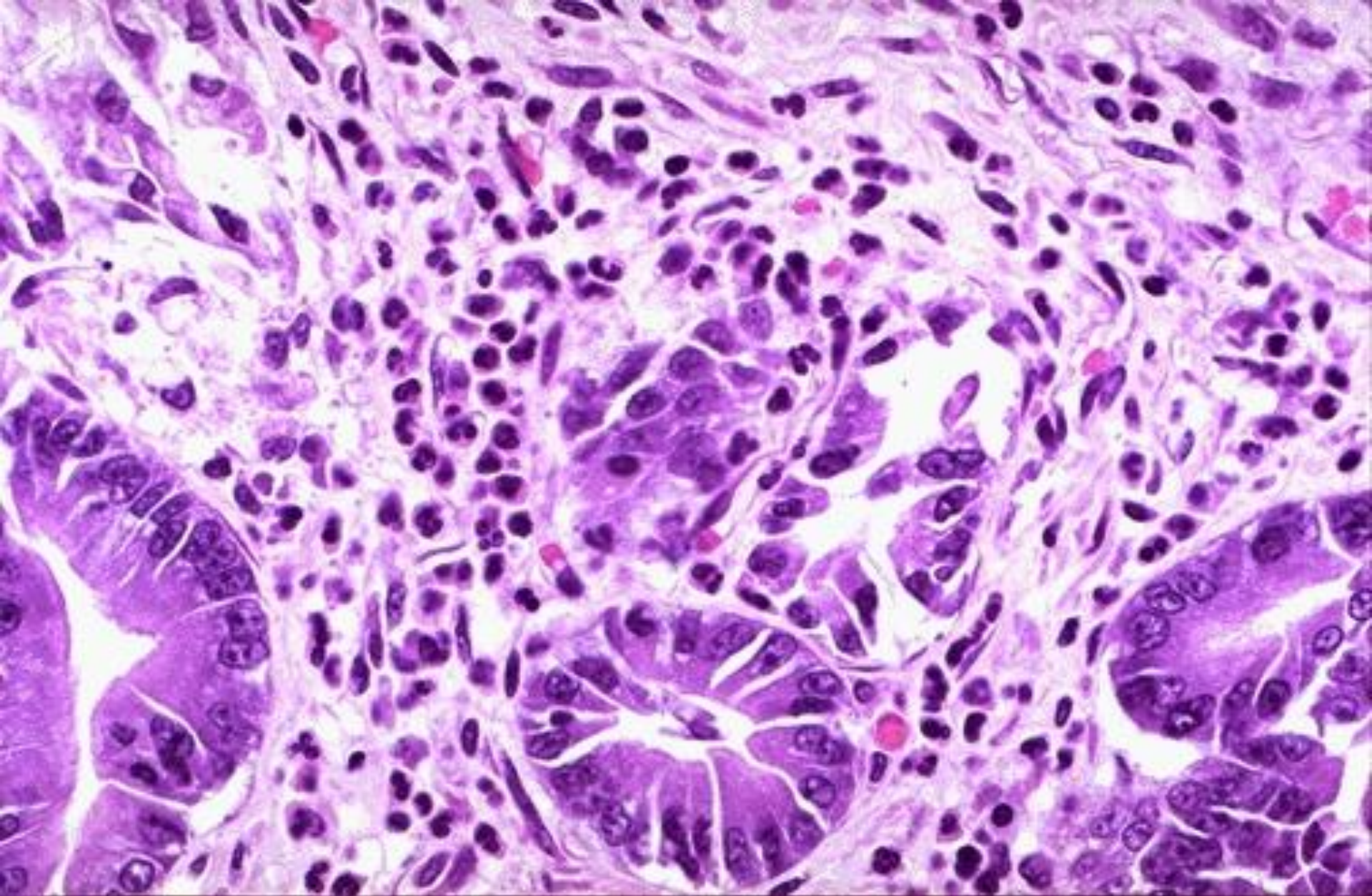
GASTRITIS

- **ACUTE, HEMORRHAGIC**
- **HISTOLOGY: Erosion, Hemorrhage, NEUTROPHILS**

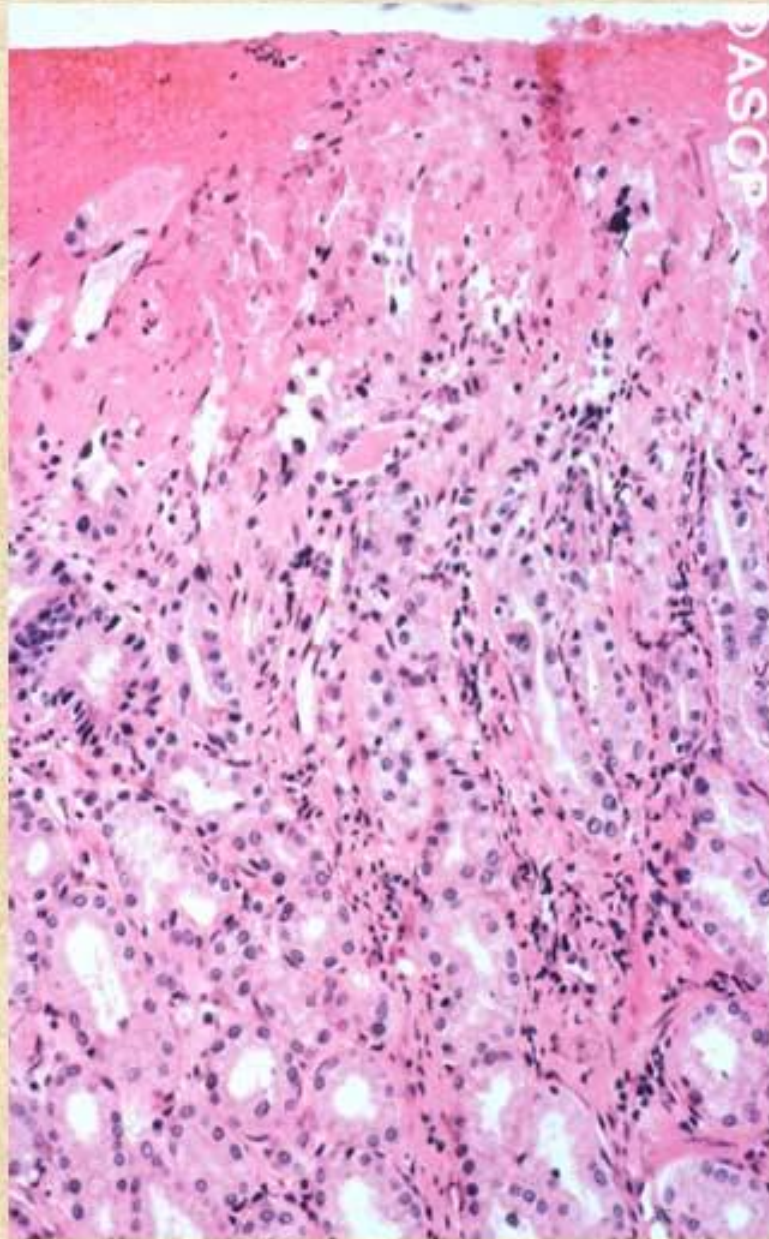




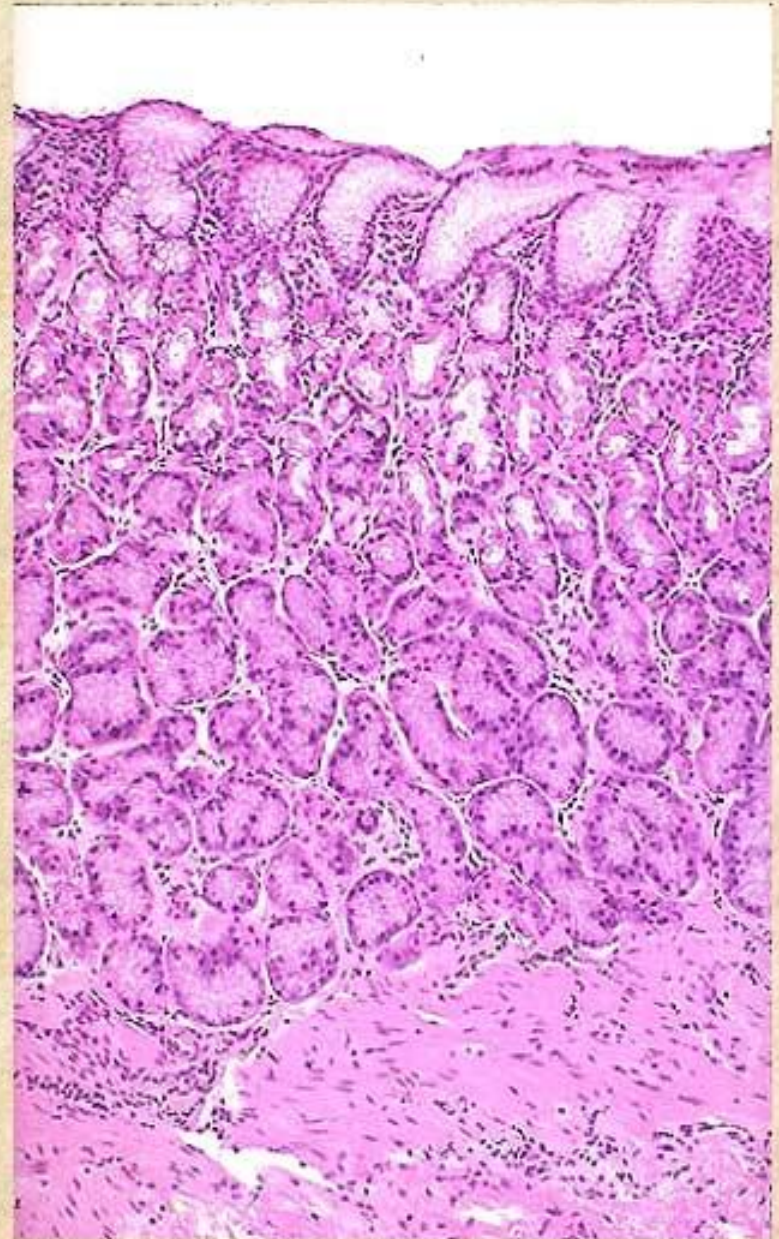
Acute gastritis



Acute erosive gastritis



Normal fundus



GASTRITIS

- CHRONIC, NO EROSIONS, NO HEMORRHAGE

- Chronic infection by *H. pylori*
- Immunologic (*autoimmune*),
- Toxic, as with alcohol and cigarette smoking
- Postsurgical, reflux of bile
- Motor and mechanical, including obstruction, bezoars (luminal concretions), and gastric atony
- Radiation
- Granulomatous conditions (e.g., Crohn disease)
- Uremia

GASTRITIS

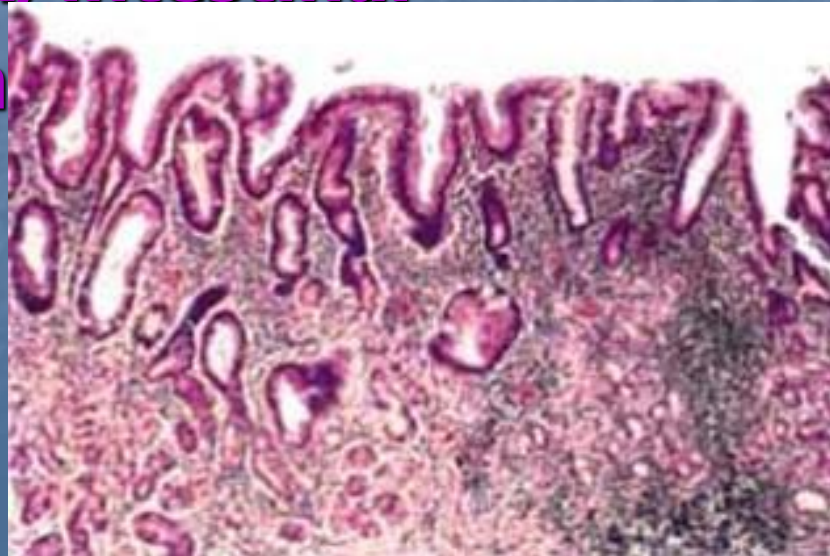
- **CHRONIC, NO EROSIONS, NO HEMORRHAGE**

- Perhaps some neutrophils
- Lymphocytes, lymphoid follicles
- **REGENERATIVE CHANGES**

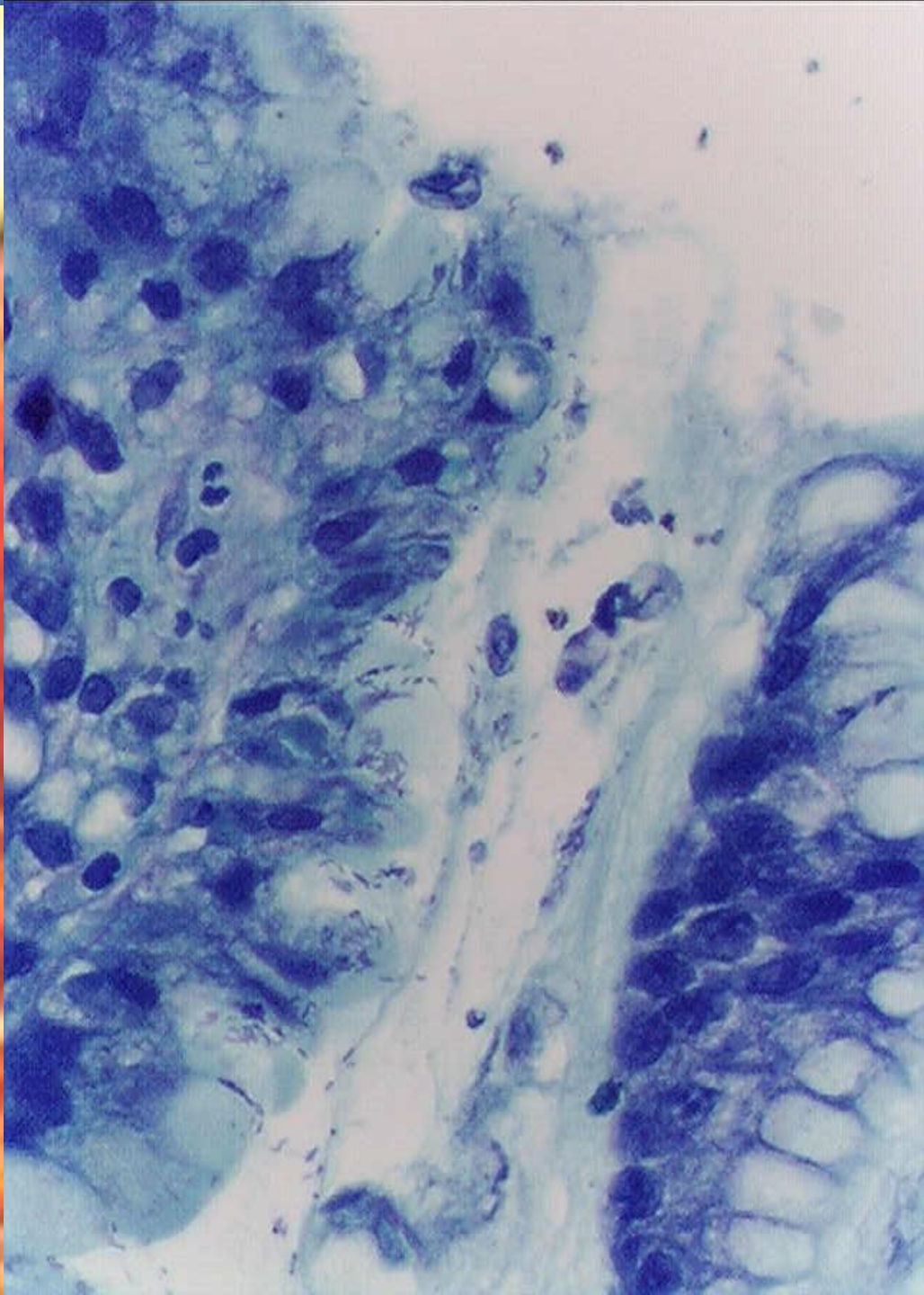
- **METAPLASIA, intestinal**

- **ATROPHY, m**

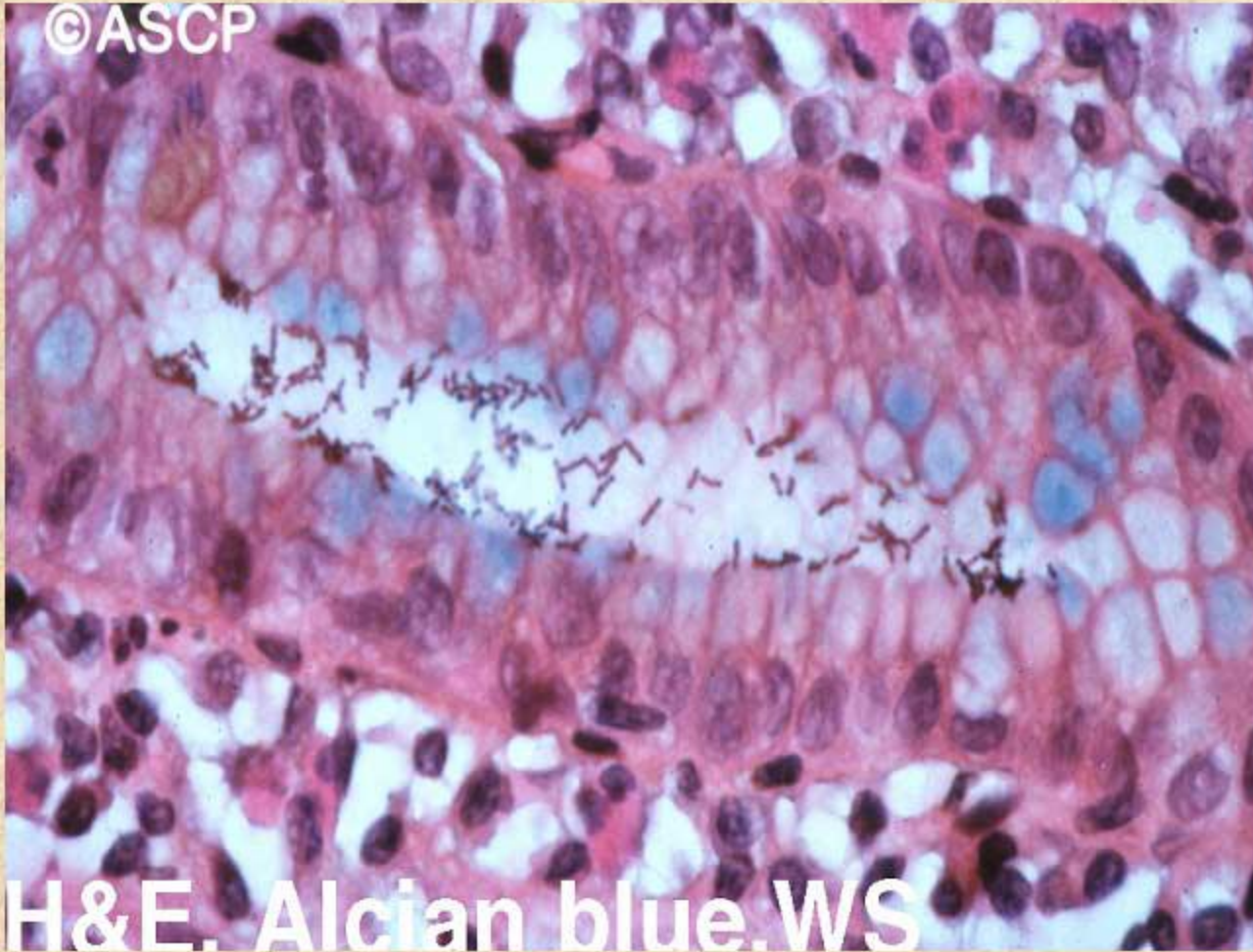
- **DYSPLASIA**

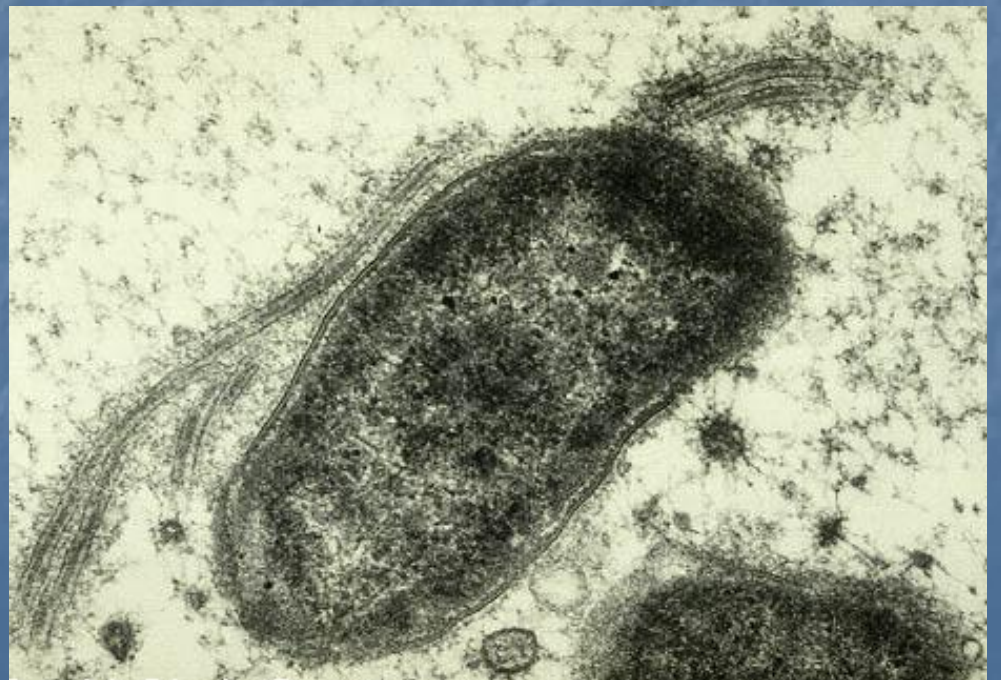
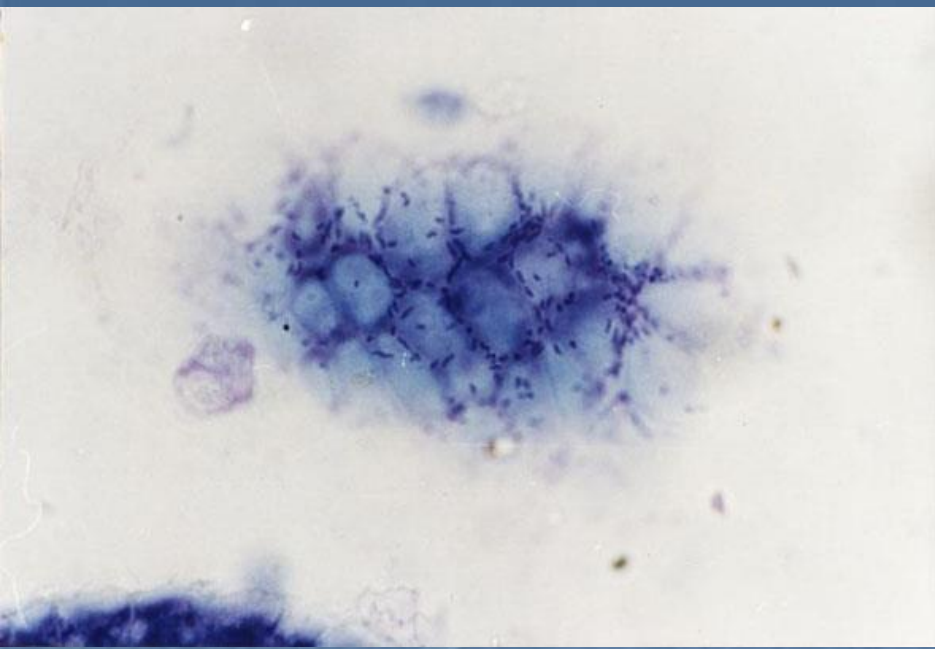


“inching”



H. pylori Gastritis

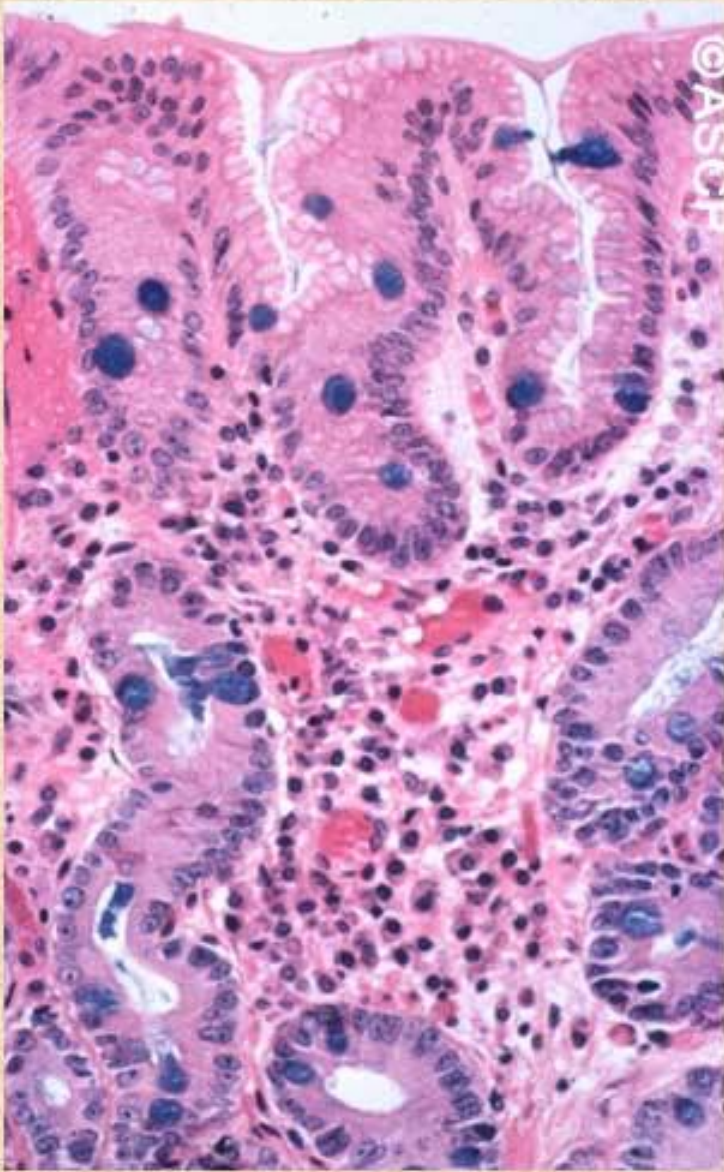




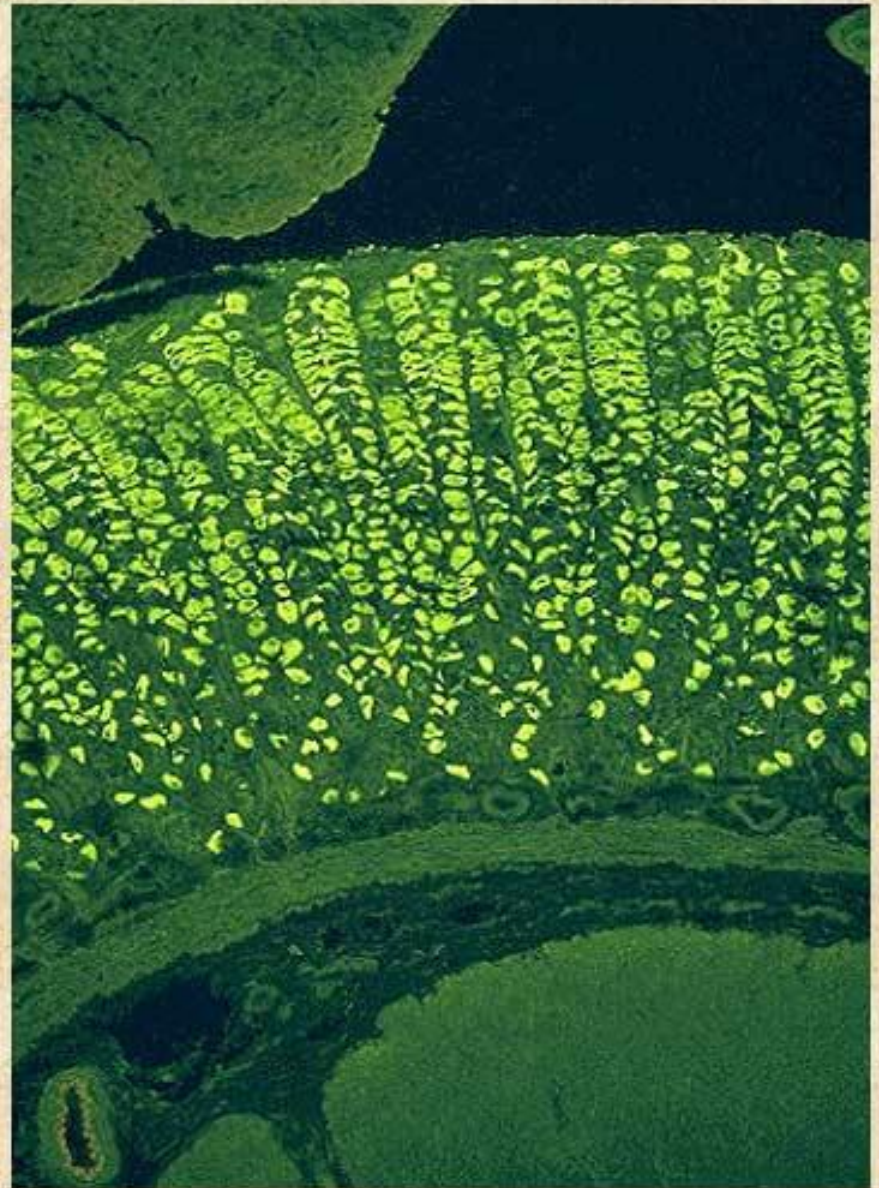
GASTRITIS

- **AUTOIMMUNE**
(10%)
- **ANTIBODIES AGAINST** →
 - **acid producing enzyme H^+**
 - **K^+ -ATPase**
 - **gastrin receptor**
 - **and intrinsic factor**

Autoimmune chronic gastritis



H & E



IF Anti-parietal cell Ab

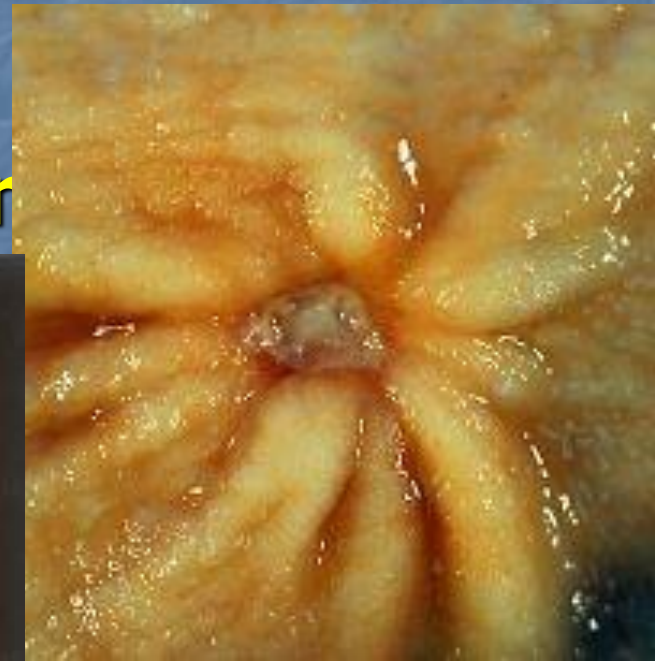
GASTRITIS

■ OTHER

- EOSINOPHILIC, middle aged women
- ALLERGIC, children (also eosinophils)
- LYMPHOCYTIC, T-Cells, body, DIFFUSE
- GRANULOMATOUS, Crohn's, other granulomas

"PEPTIC" ULCERS

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



Helicobacter pylori

- Causes 80% of gastric peptic ulcers
- Causes 100% of duodenal peptic ulcers
- Causes chronic gastritis
- Causes gastric carcinomas
- Causes lymphomas

"PEPTIC" ULCERS

- Burning, aching epigastric pain,
- Fe deficiency anemia
- Acute hemorrhage
- Penetration, perforation:
 - Pain in BACK
 - Pain in CHEST
 - Pain in LUQ
- **NOT** felt to develop into malignancy

"PEPTIC" ULCERS

■ Bleeding

- Occurs in 15% to 20% of patients
- Most frequent complication
- May be life-threatening
- Accounts for 25% of ulcer deaths
- May be the first indication of an ulcer

■ Perforation

- Occurs in about 5% of patients
- Accounts for two thirds of ulcer deaths
- Rarely, is the first indication of an ulcer

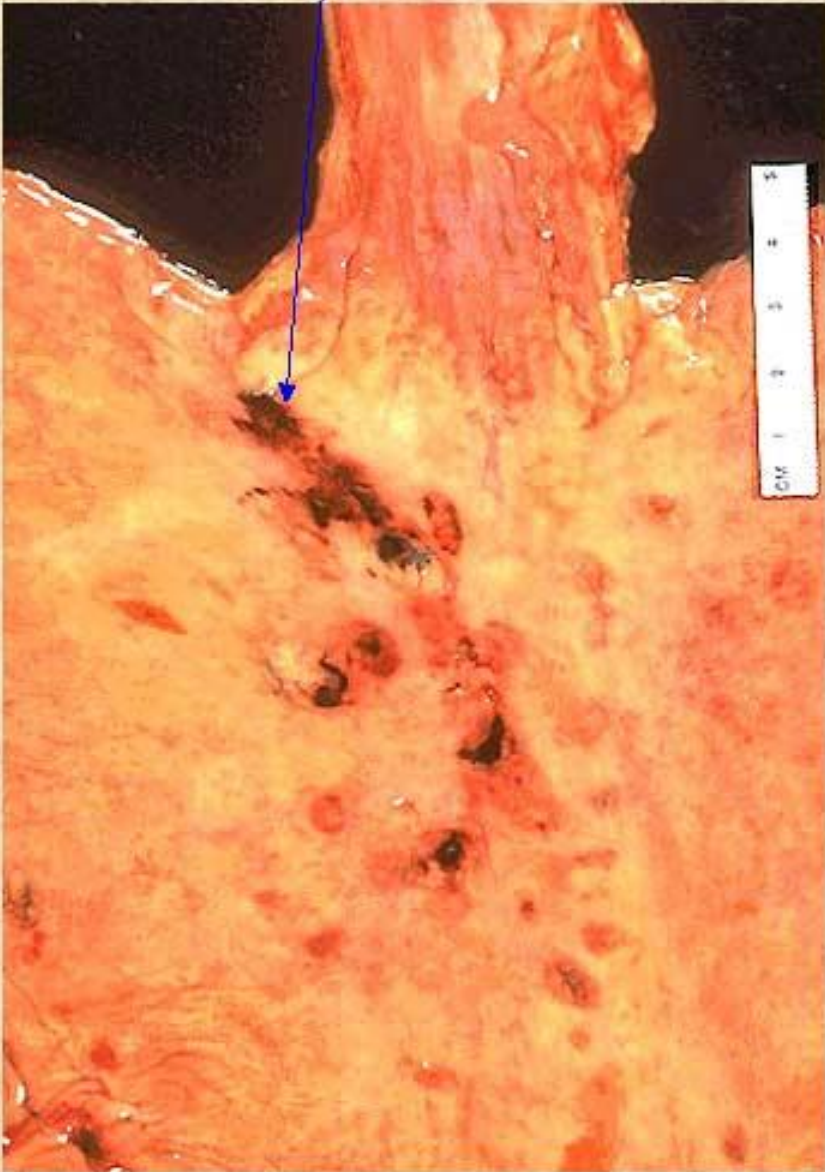
■ Obstruction from edema or scarring

- Occurs in about 2% of patients
- Most often due to pyloric channel ulcers
- May also occur with duodenal ulcers
- Causes incapacitating, crampy abdominal pain
- Rarely, may lead to total obstruction with intractable vomiting

"ACUTE" ULCERS

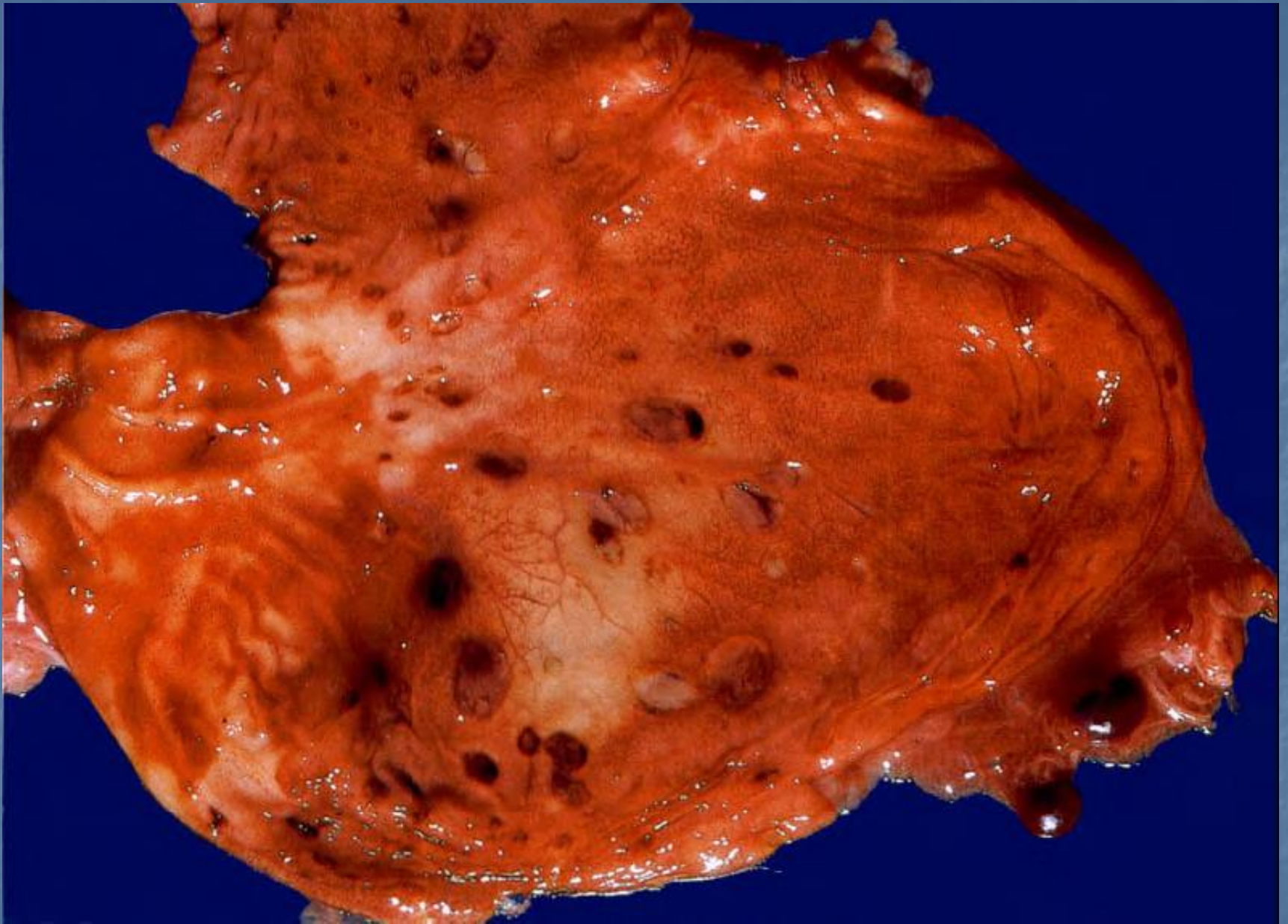
- **NSAIDS**
- **"STRESS" ULCERS**
 - **ENDOGENOUS STEROIDS**
 - **SHOCK**
 - **BURNS**
 - **MASSIVE TRAUMA**
 - **Intracranial trauma, Intracranial surgery**
 - **SEPSIS**
 - **EXOGENOUS STEROIDS**
 - **CUSHING ULCER**

Acute erosion



Acute ulcer

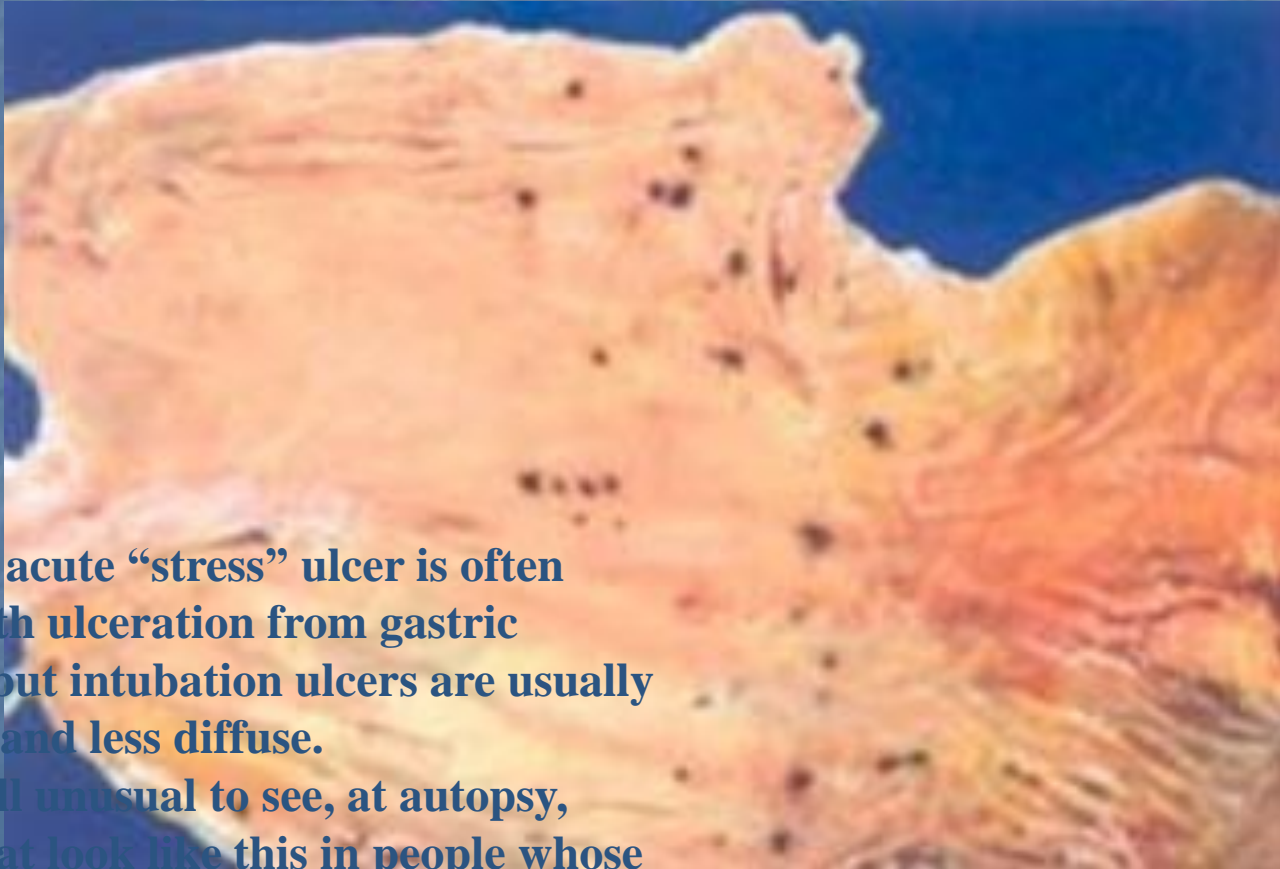




Acute gastric erosions

"ACUTE" ULCERS

- Usually small (<1cm), superficial, **MULTIPLE**



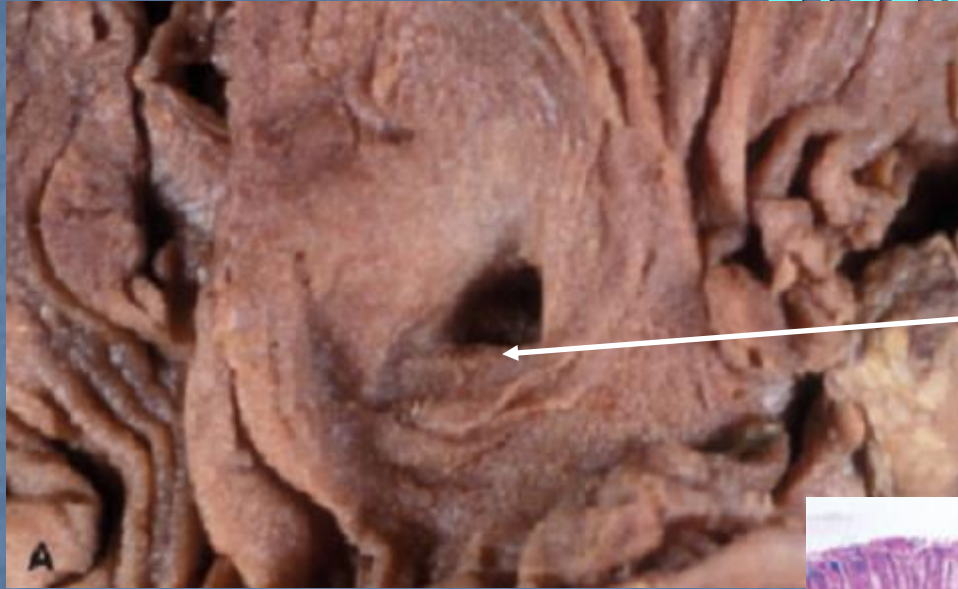
This type of acute “stress” ulcer is often confused with ulceration from gastric intubation, but intubation ulcers are usually more linear and less diffuse.

It is not at all unusual to see, at autopsy, stomachs that look like this in people whose death was preceded by extreme physiologic stress.

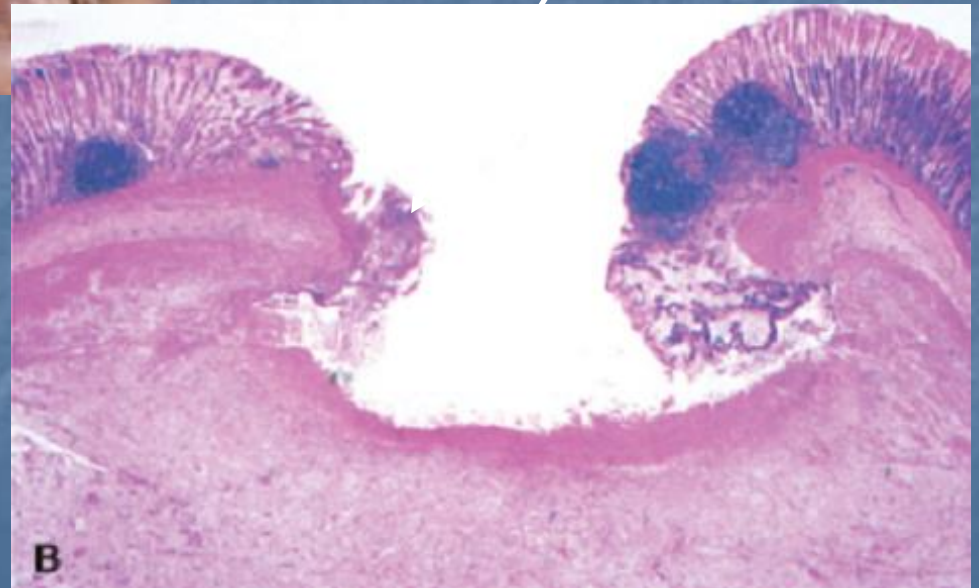


Active chronic ulcer of stomach

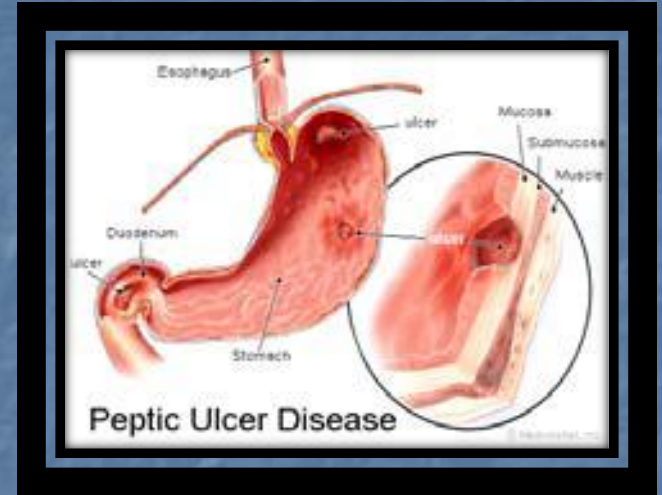
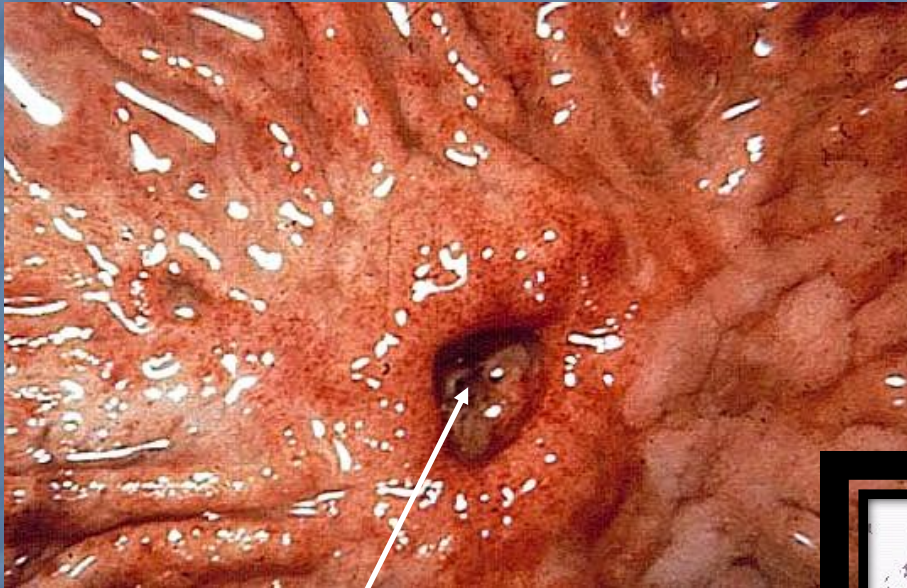
Duodenal Ulcer - Peptic Ulcer



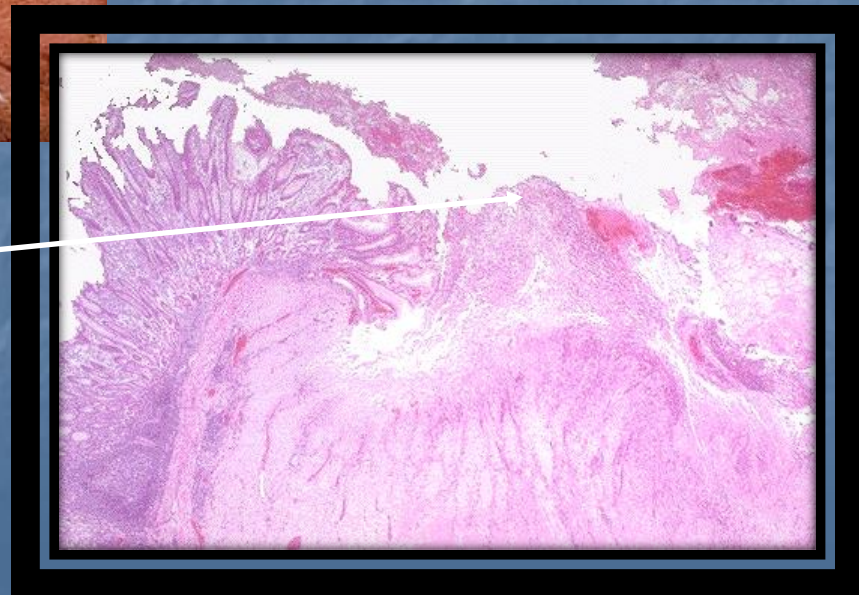
Loss of mucosal layer

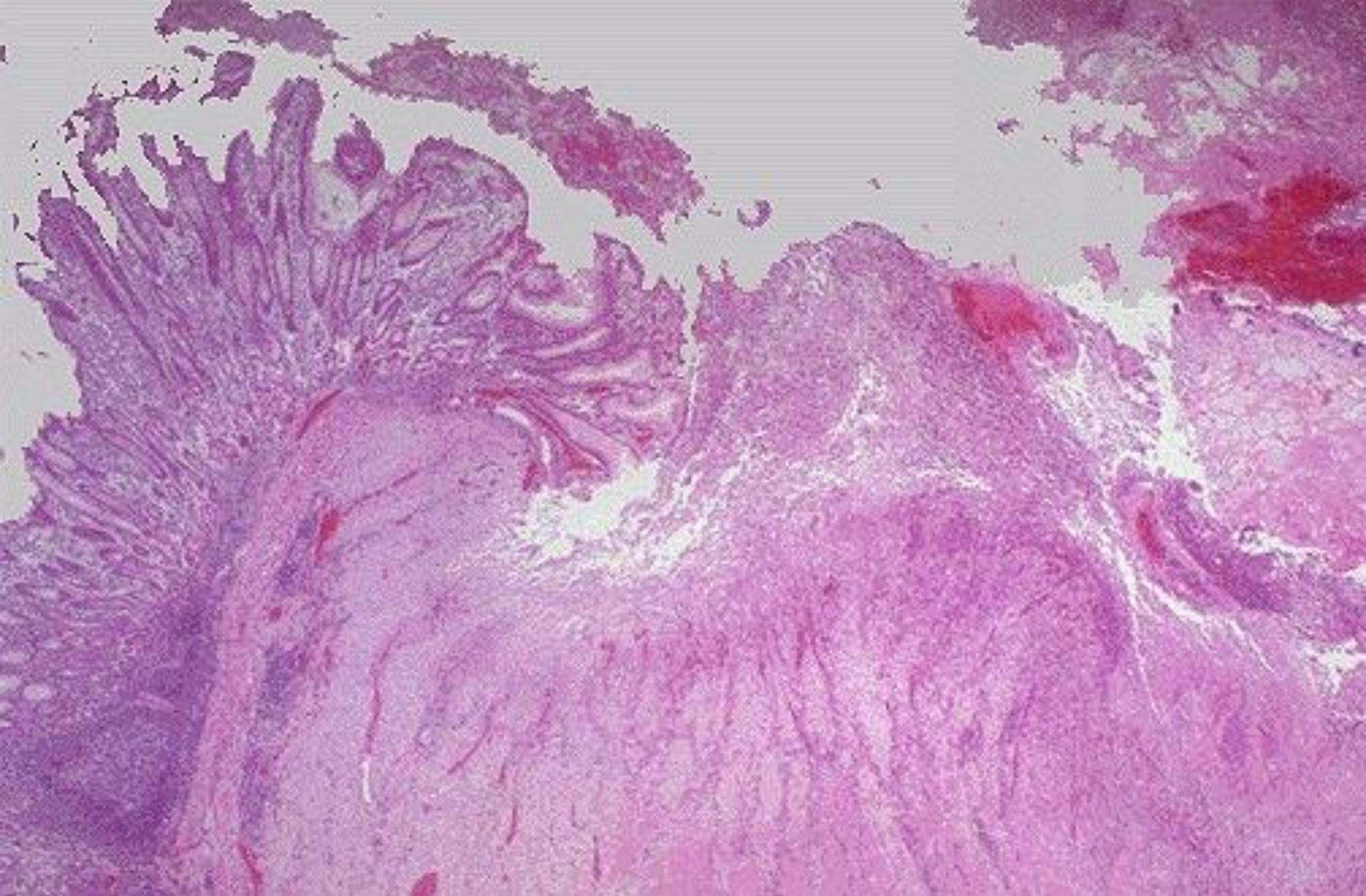


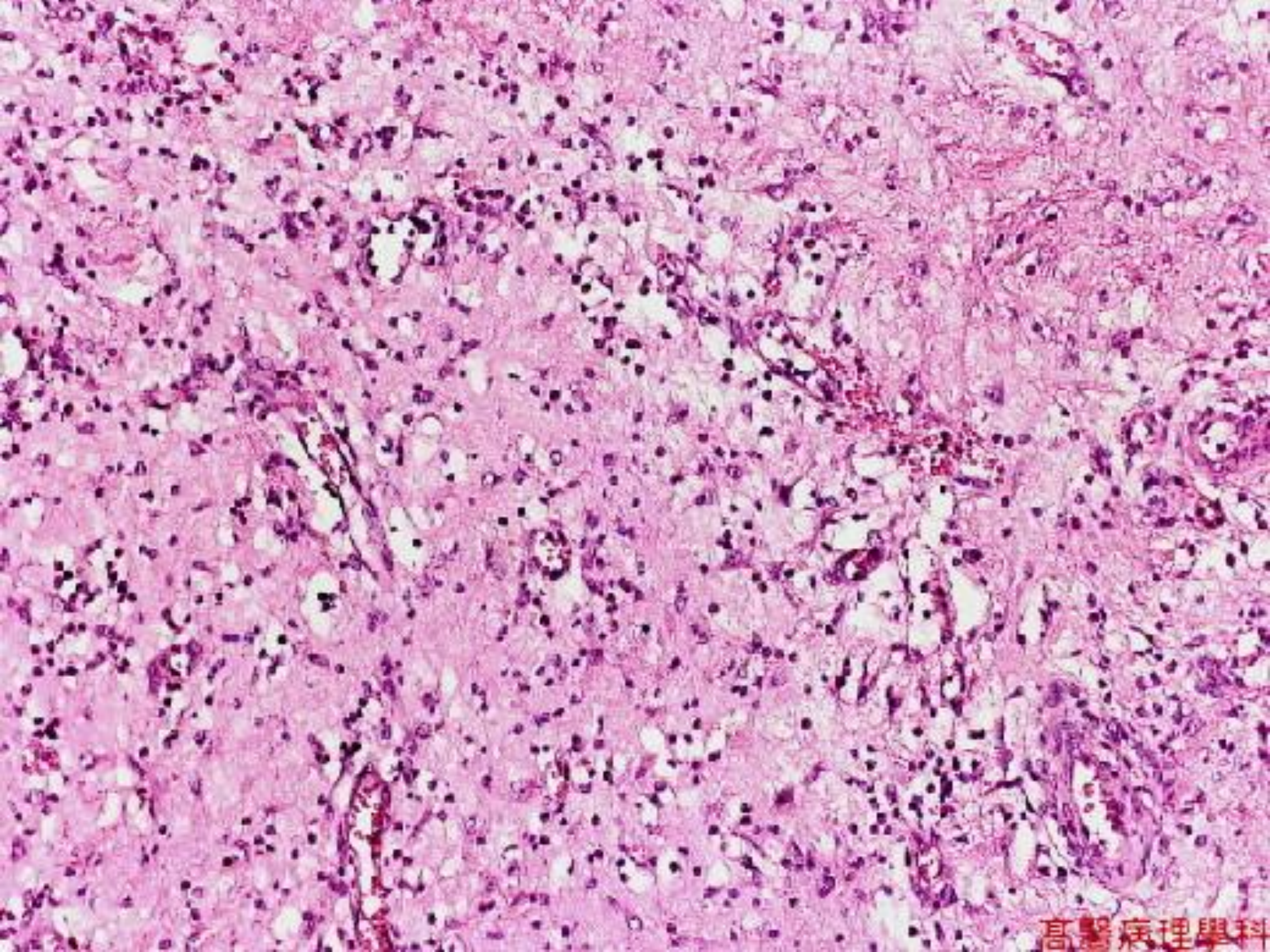
Gastric Ulcer

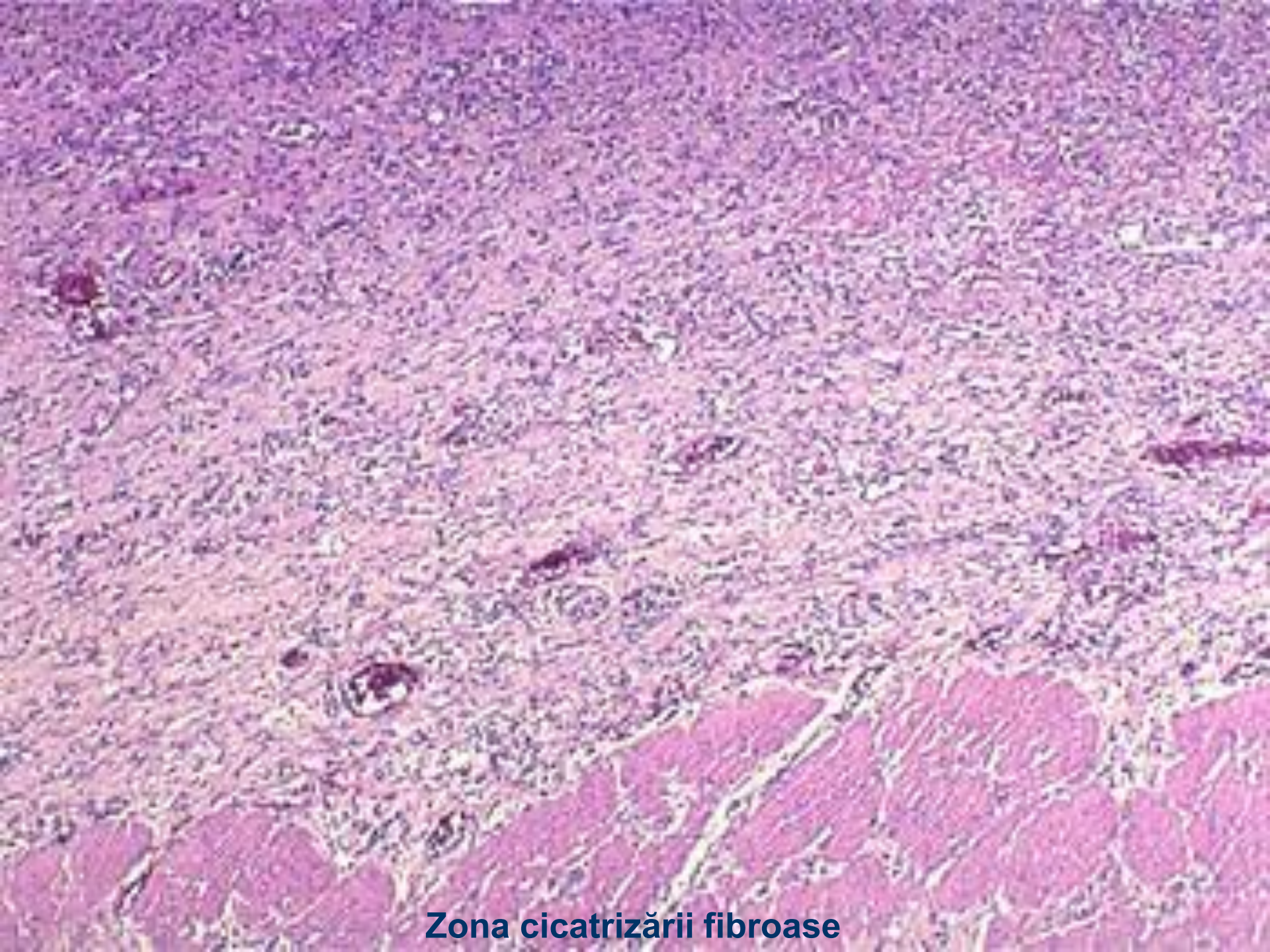


Loss of mucosa









Zona cicatrizării fibroase

GASTRIC DILATATION

- **PYLORIC STENOSIS**
- **PERITONITIS (→ pyloric stenosis)**
- **1.5-3.0 liters NORMAL**
- **10 liters can be present**
- **ACUTE RUPTURE is associated with a HIGH immediate mortality rate**

"HYPERTROPHIC"*
GASTROPATHY

RUGAL PROMINENCE
(cerebriform)

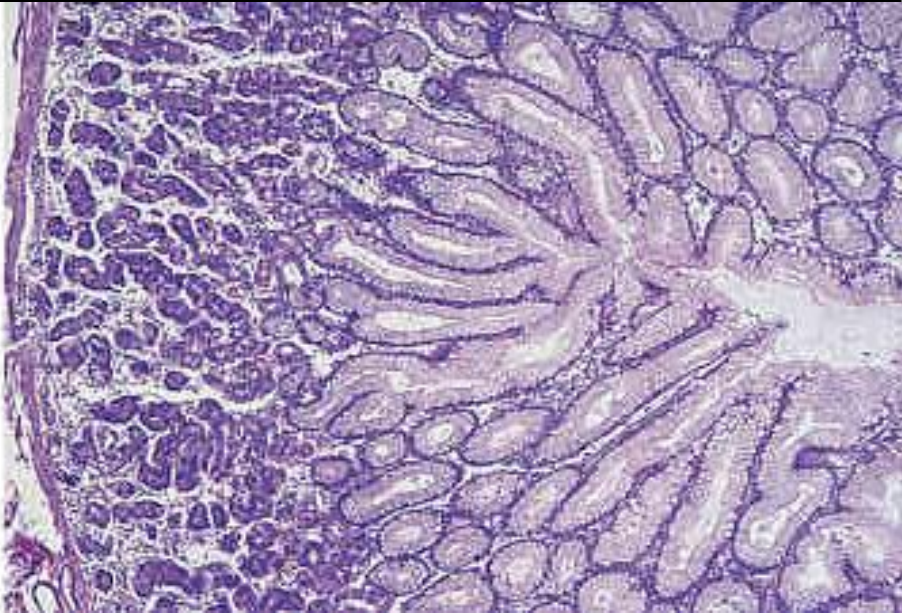
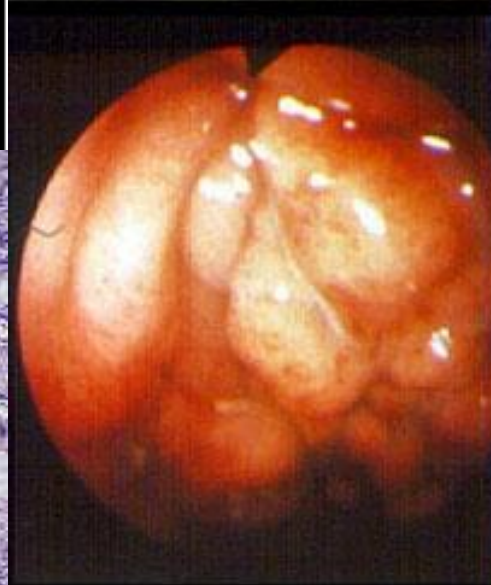
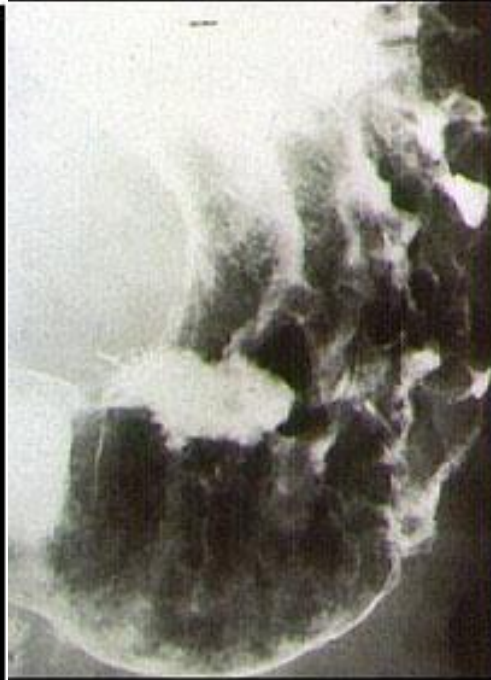
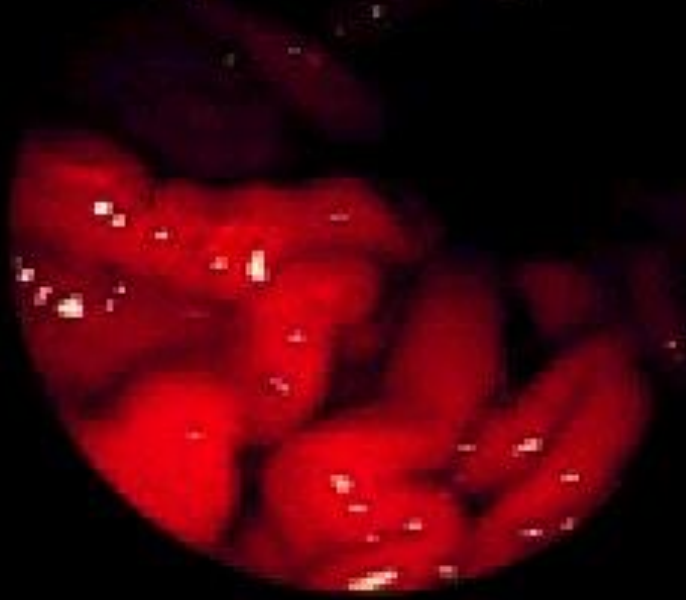
NO INFLAMMATION

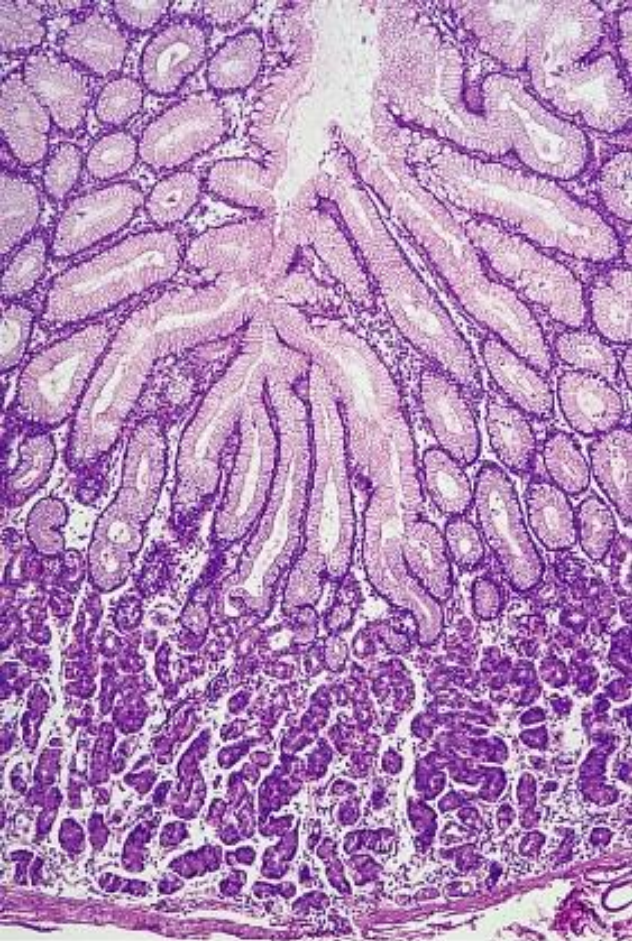
HYPERPLASIA of MUCOSA

"HYPERTROPHIC" GASTROPATHY

- Inaccurate name "hypertrophic gastritis"
- *Ménétrier disease*, resulting from profound hyperplasia of the surface mucous cells with accompanying glandular atrophy, ass. w. CMV, H. Pylori, ↑TGF- α
- *Hypertrophic-hypersecretory gastropathy*, associated with hyperplasia of the parietal and chief cells within gastric glands (normal gastrin)
- *Gastric gland hyperplasia secondary to excessive gastrin secretion*, in the setting of a gastrinoma (*Zollinger-Ellison syndrome*)

A





GASTRIC "VARICES"

- **SAME SETTING AND ETIOLOGY AS ESOPHAGEAL VARICES, i.e., PORTAL HYPERTENSION**
- **NOT AS COMMON AS ESOPHAGEAL VARICES**
- **MAY LOOK LIKE PROMINENT RUGAE**
- **IF A PATIENT HAS GASTRIC VARICES, HE ALSO PROBABLY HAS ESOPHAGEAL, (but probably not vice versa)**



GASTRIC TUMORS

■ BENIGN:

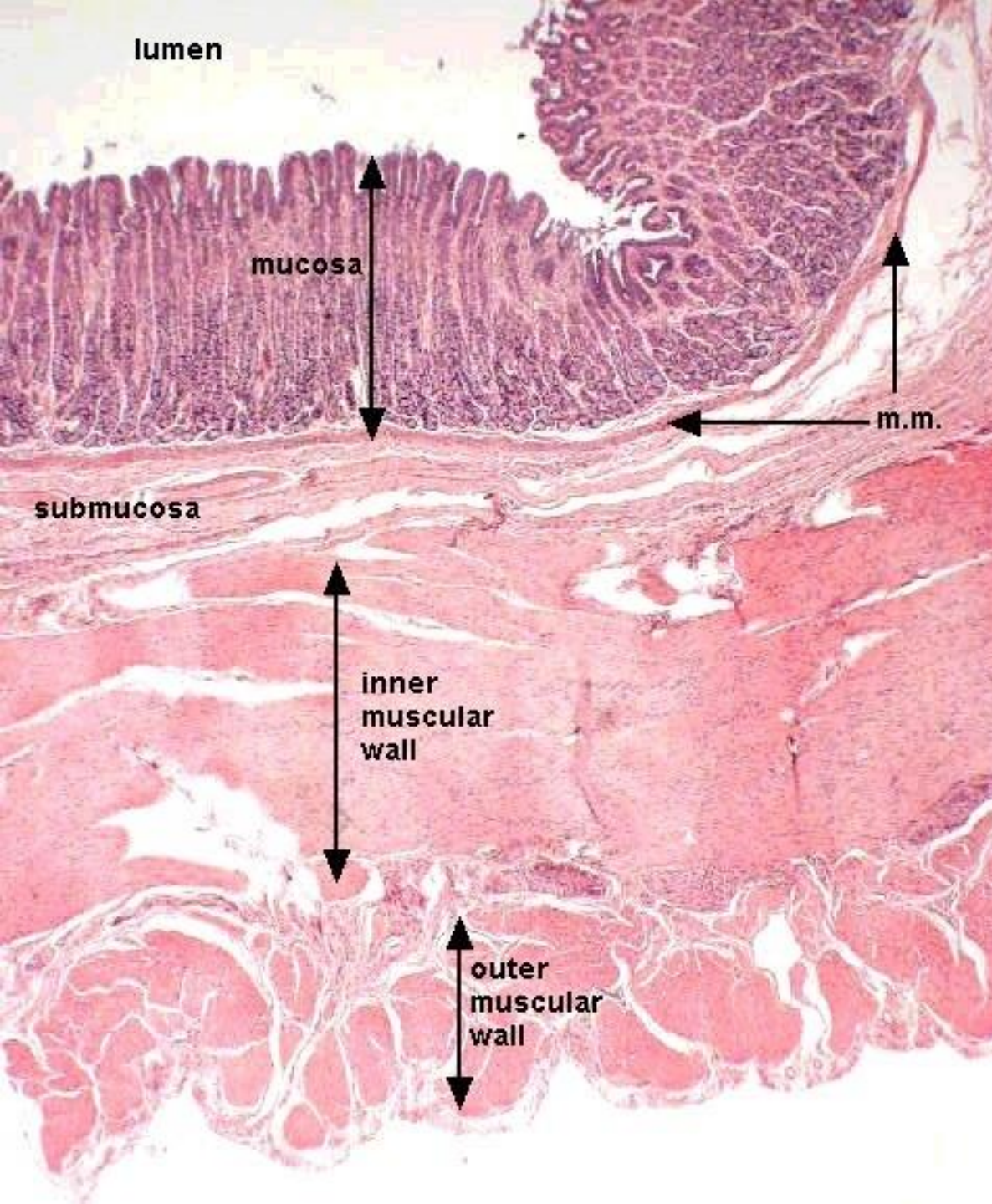
- "POLYPS*" (HYPERPLASTIC vs. ADENOMATOUS)
- LEIOMYOMAS (Same gross and micro as smooth muscle)
- LIPOMAS (Same gross and micro as adipose tissue)

■ MALIGNANT

- (ADENO)-Carcinoma
- LYMPHOMA

■ POTENTIALLY MALIGNANT

- G.I.S.T. (Gastro-Intestinal "Stromal" Tumor)



BENIGN TUMORS

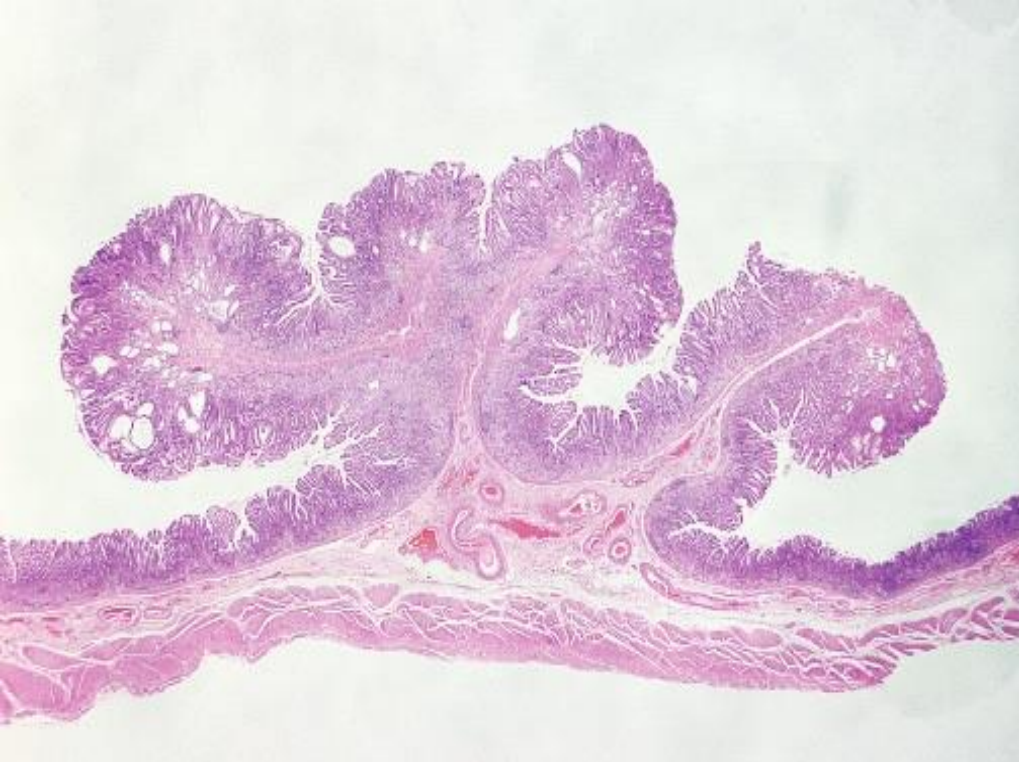
BEBNIBGNB

MUCOSA (POLYPS)

---HYPERPLASTIC

---Fundic

---ADENOMATOUS





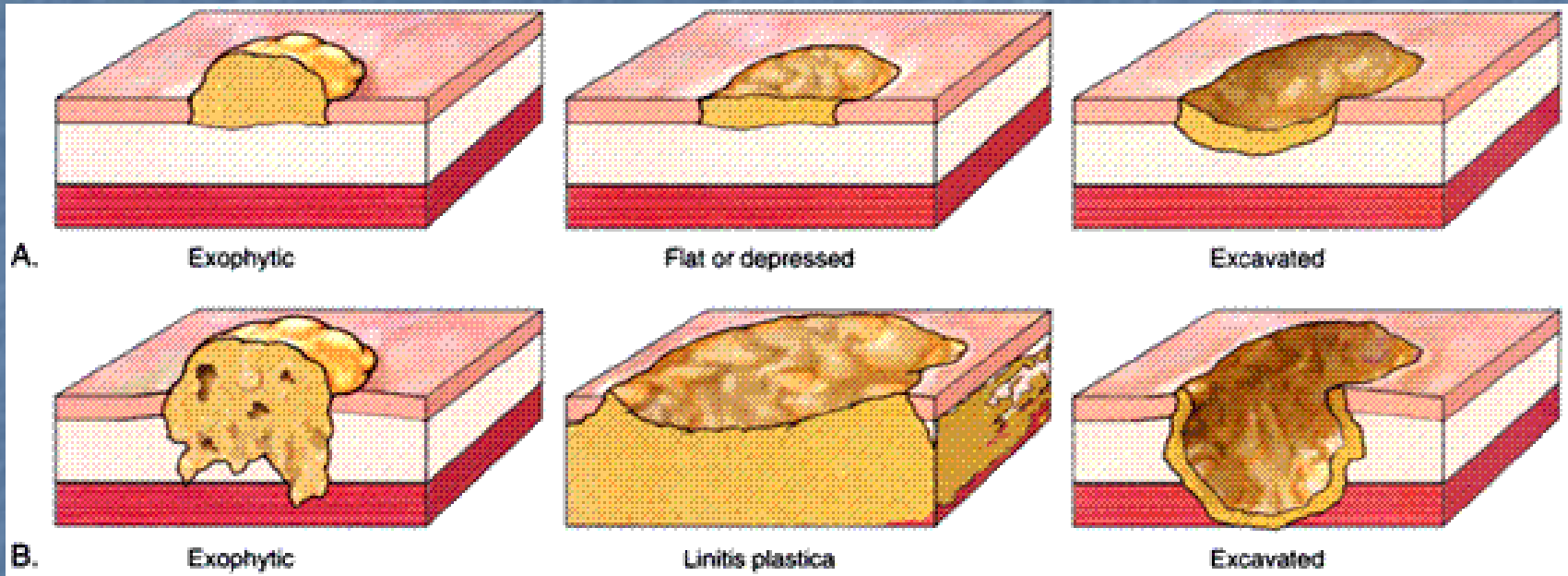
WHO GASTRIC NEOPLASMS

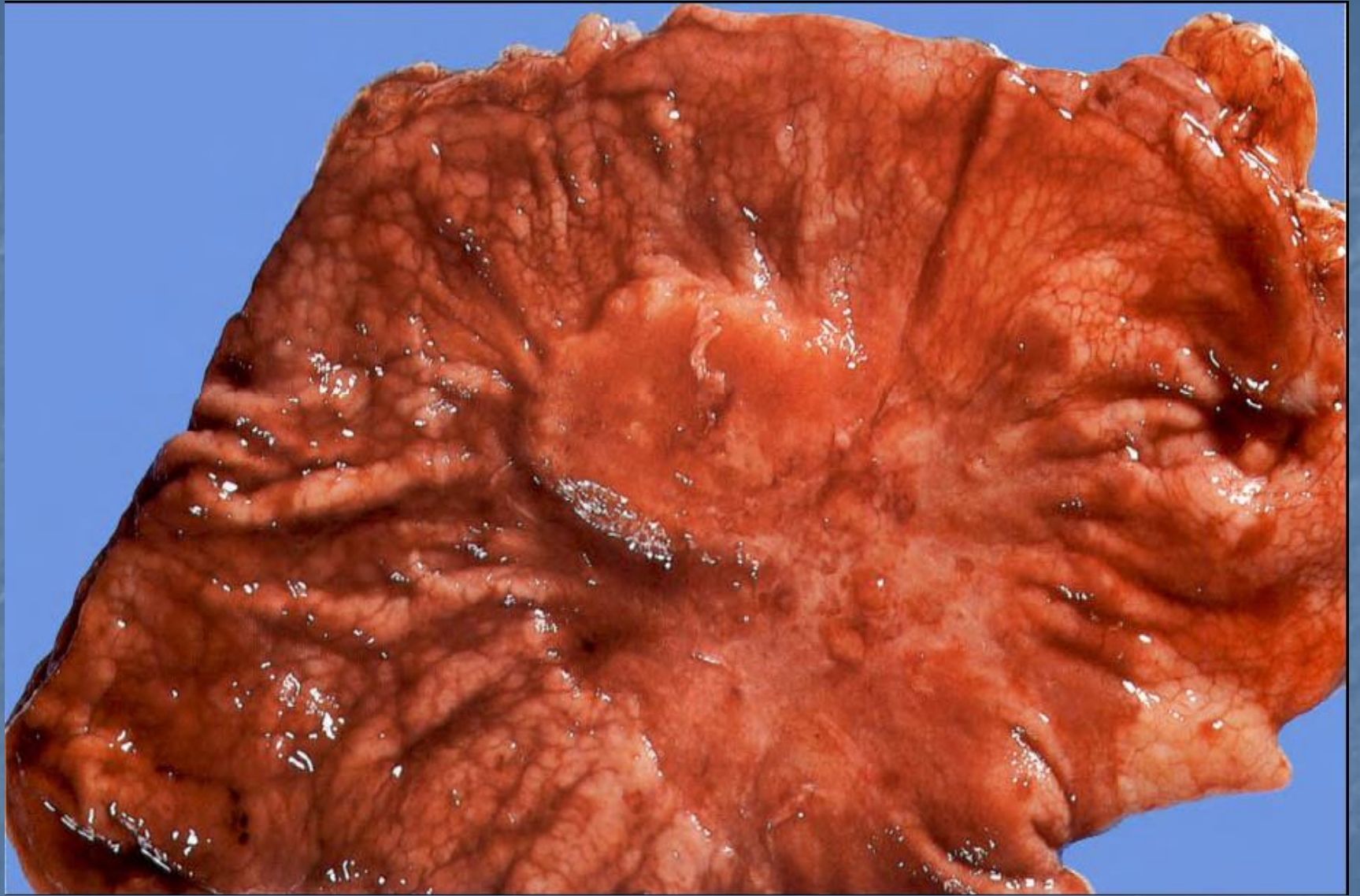
- *Epithelial Tumors: Adenomatous polyps, Adenocarcinoma (papillary, tubular, mucinous, signet ring, adenosquamous, unclassified), Small cell, Carcinoid (neuroendocrine)*
- *Nonepithelial Tumors: Leiomyo(sarc)oma, Schwannoma, GIST, Granular Cell Tumor, Kaposi sarcoma*
- *Malignant Lymphomas:*

ADENOCARCINOMA RISK FACTORS

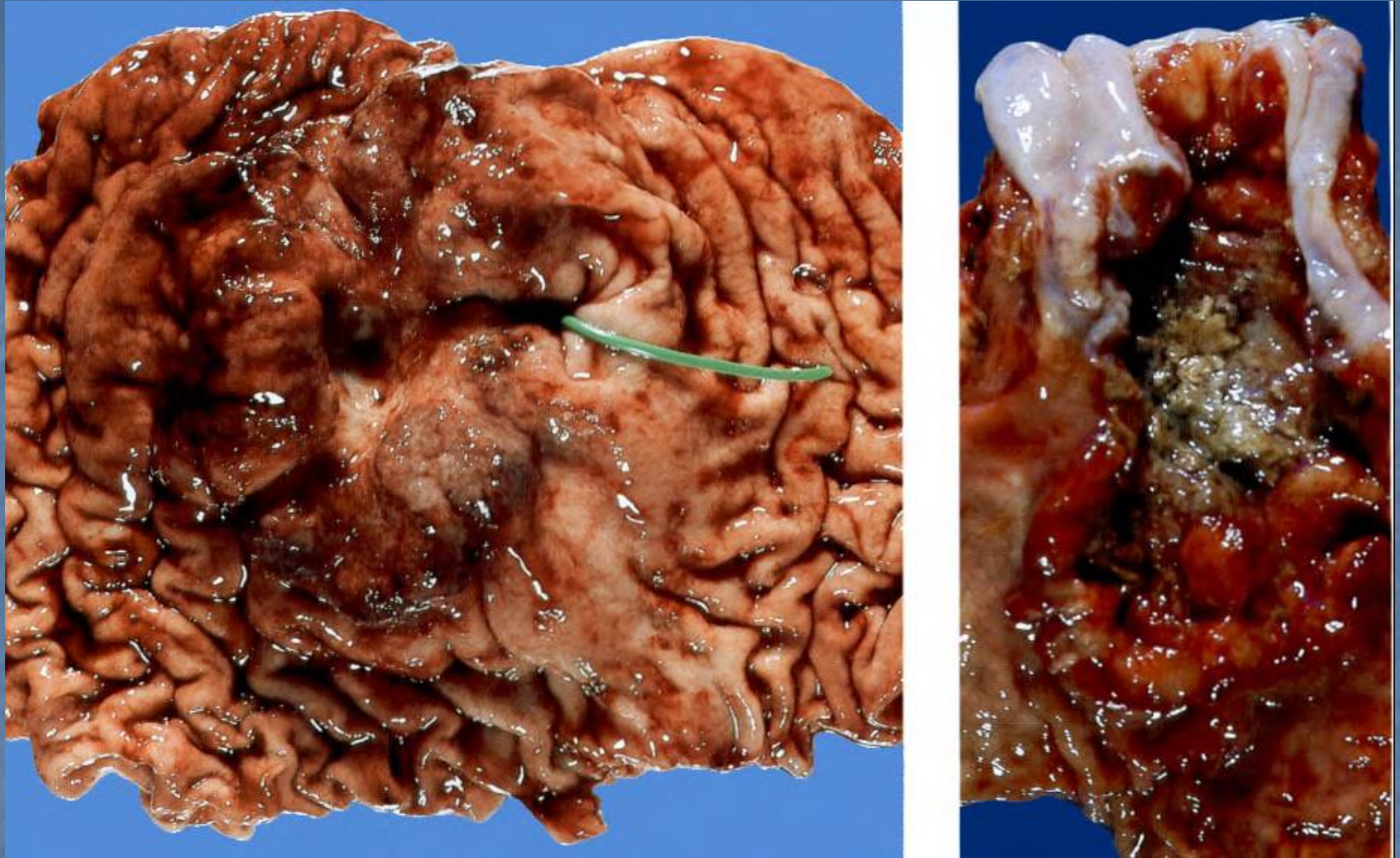
- **H. Pylori**
- **Nitrites, smoked meats, pickled, salted, chili peppers, socioeconomic, tobacco**
- **Chronic gastritis, adenomas**
- **Family history**

ADENOCARCINOMA GROWTH PATTERNS

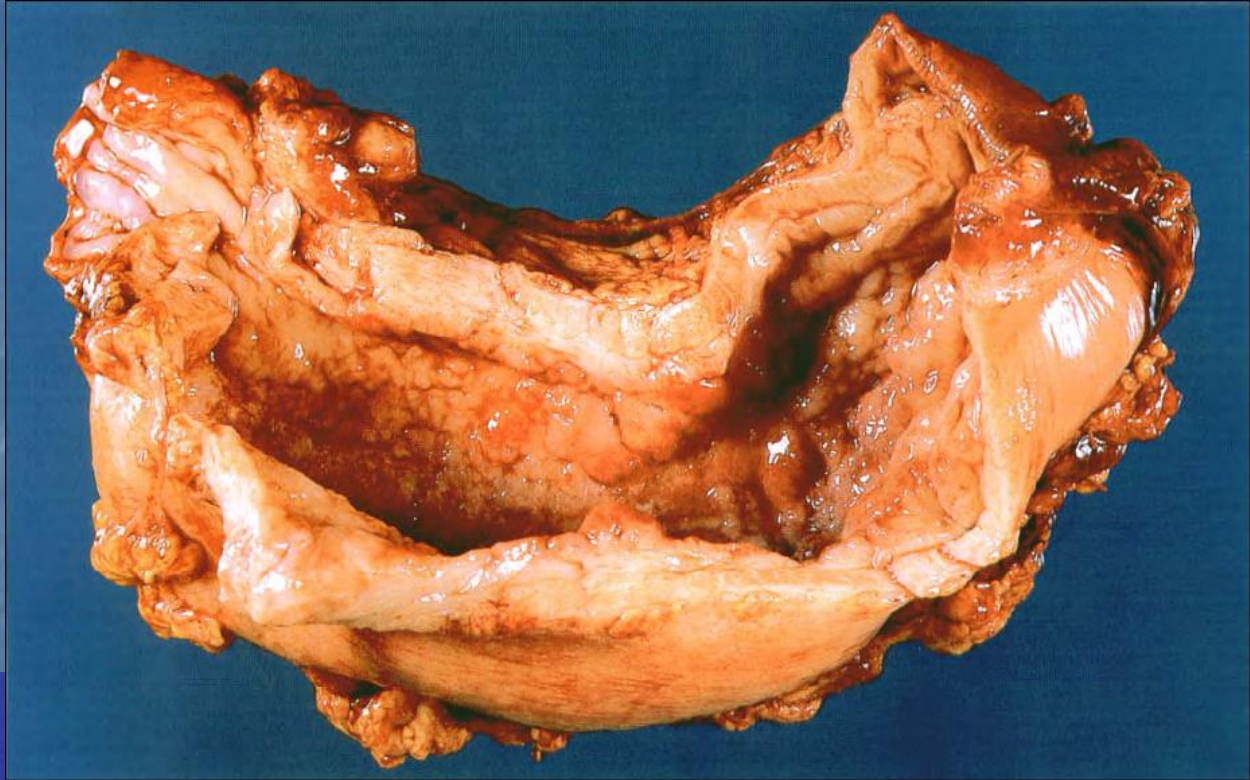


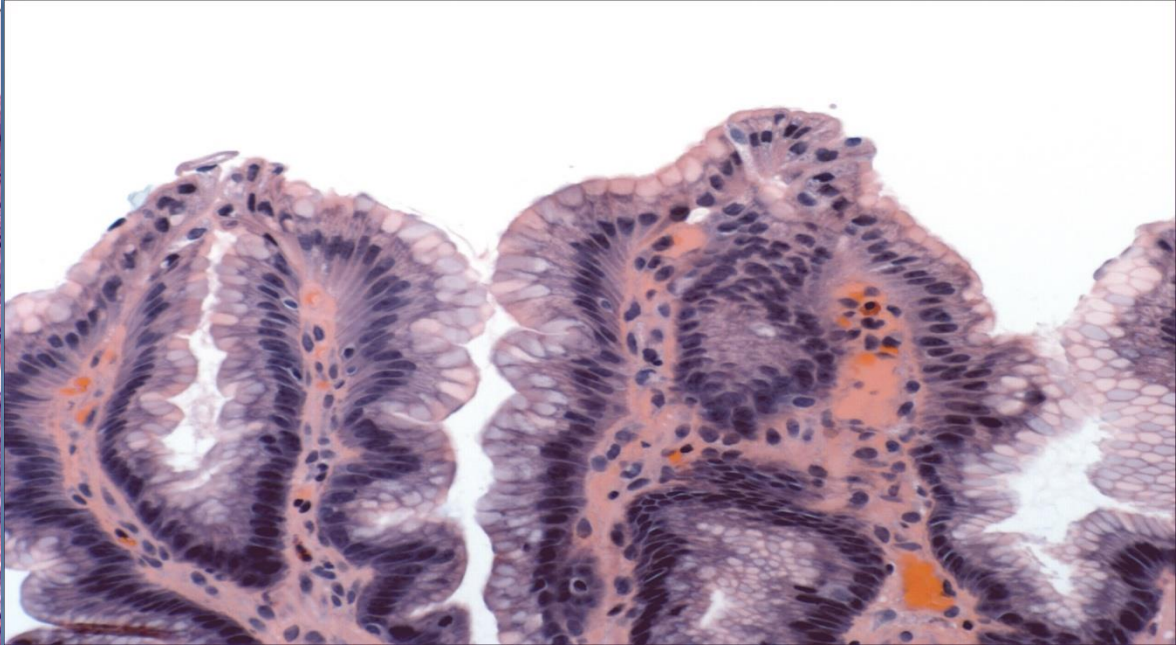
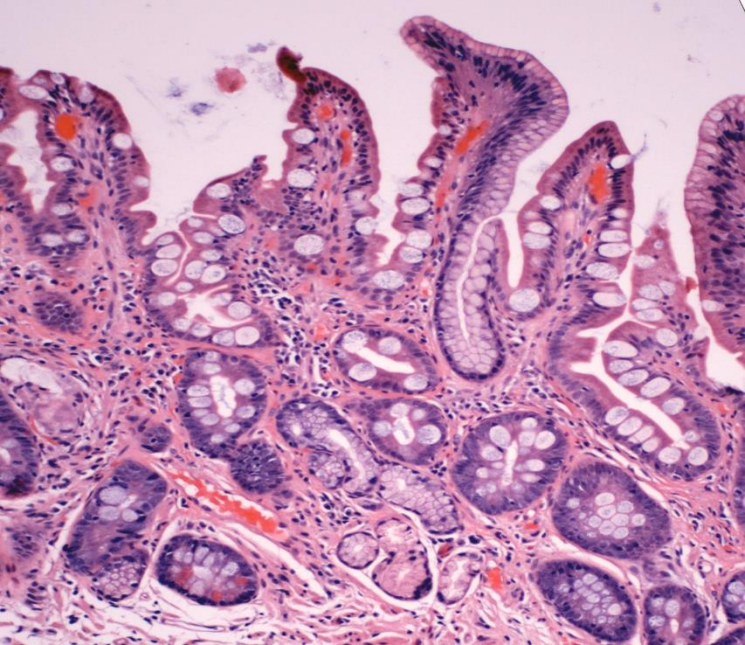




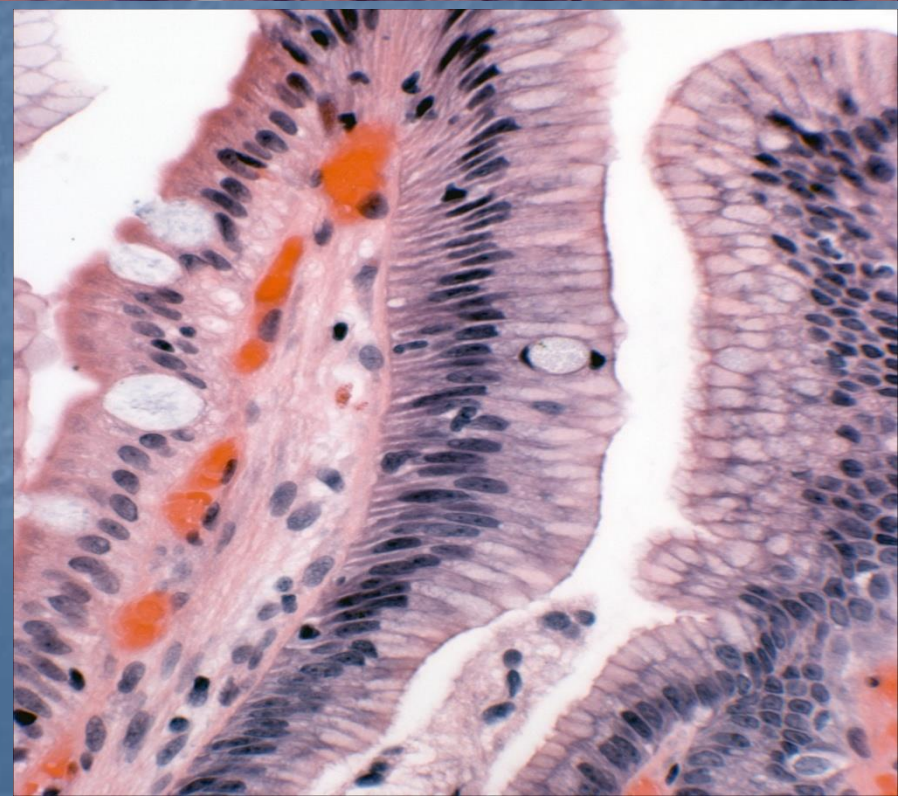


Exulcerate cancer

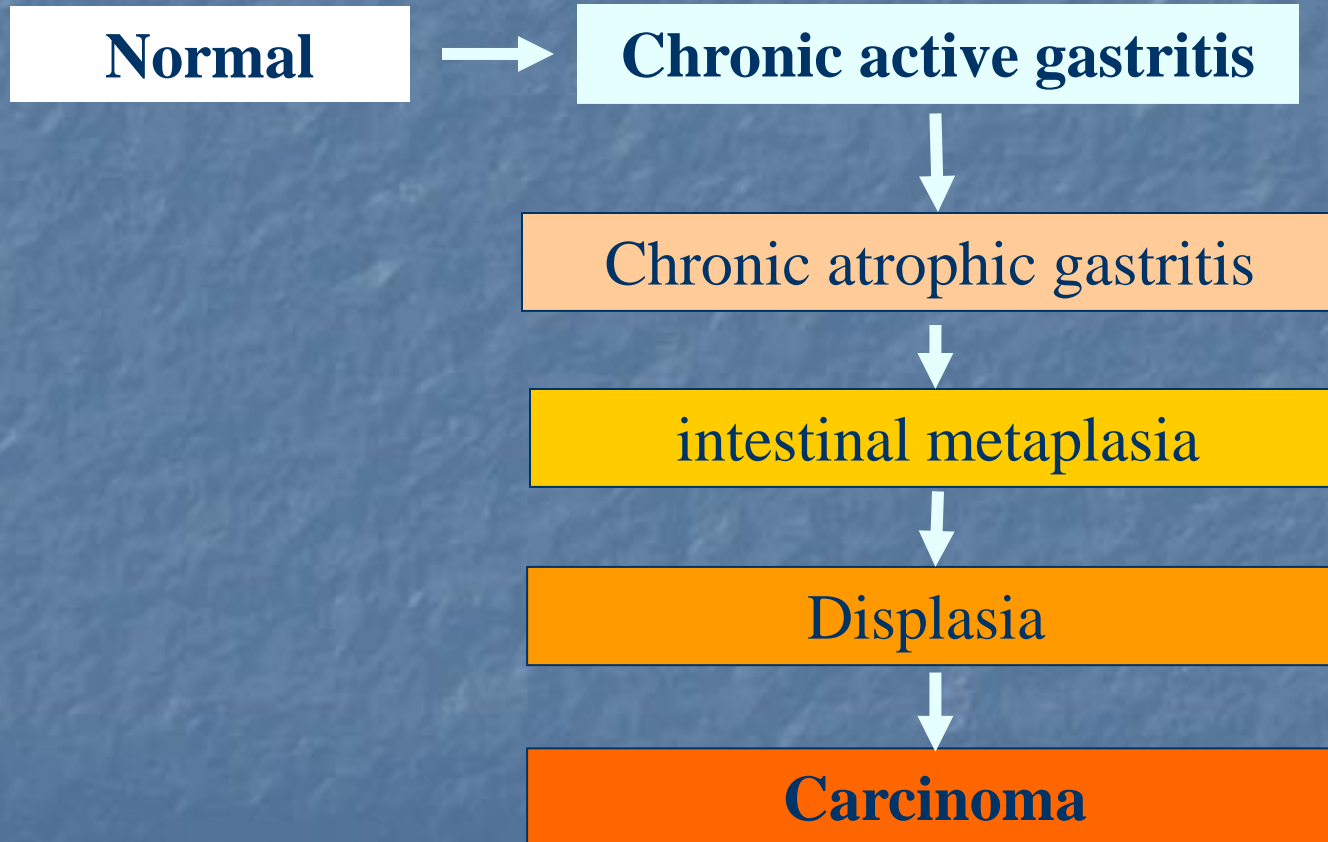


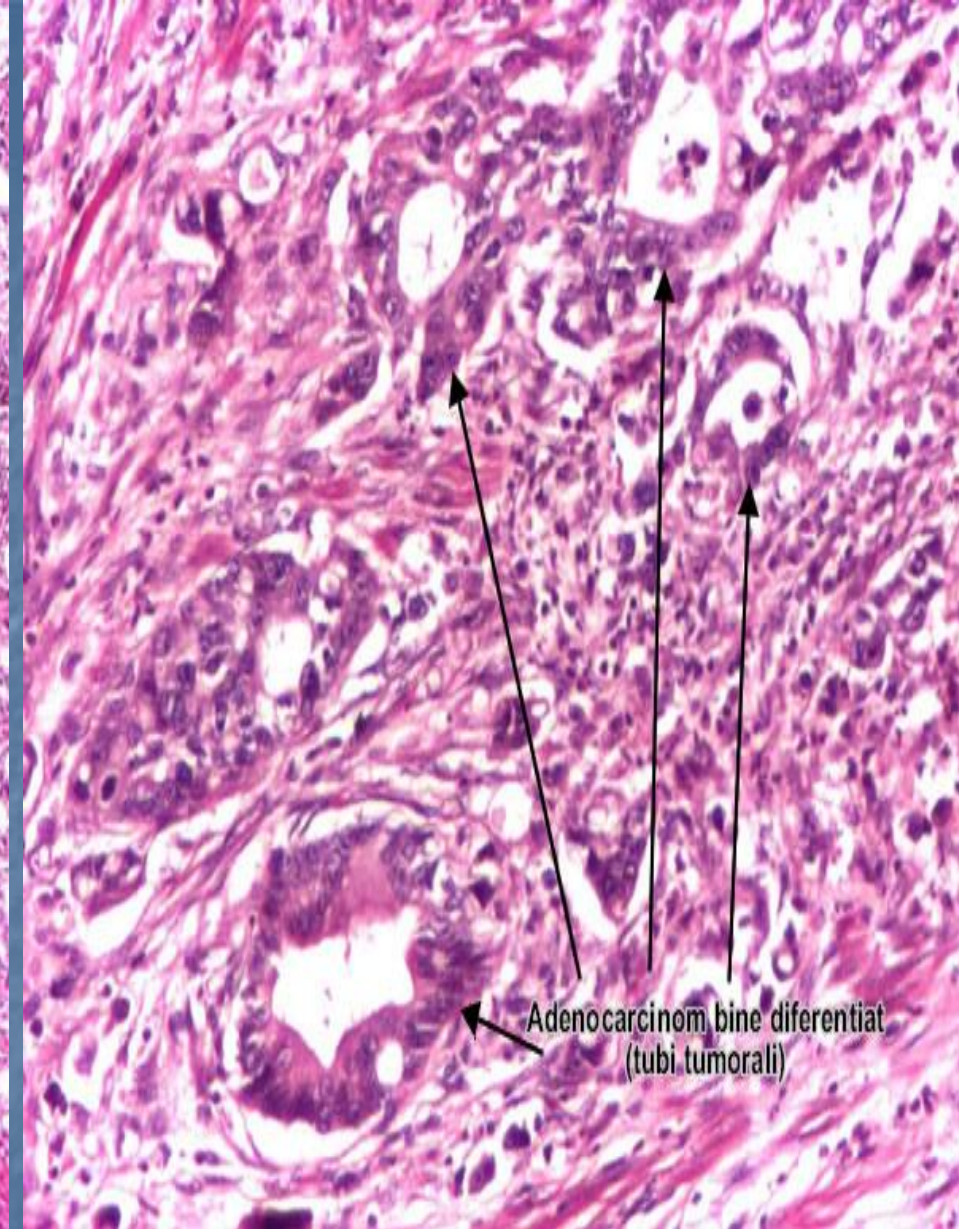
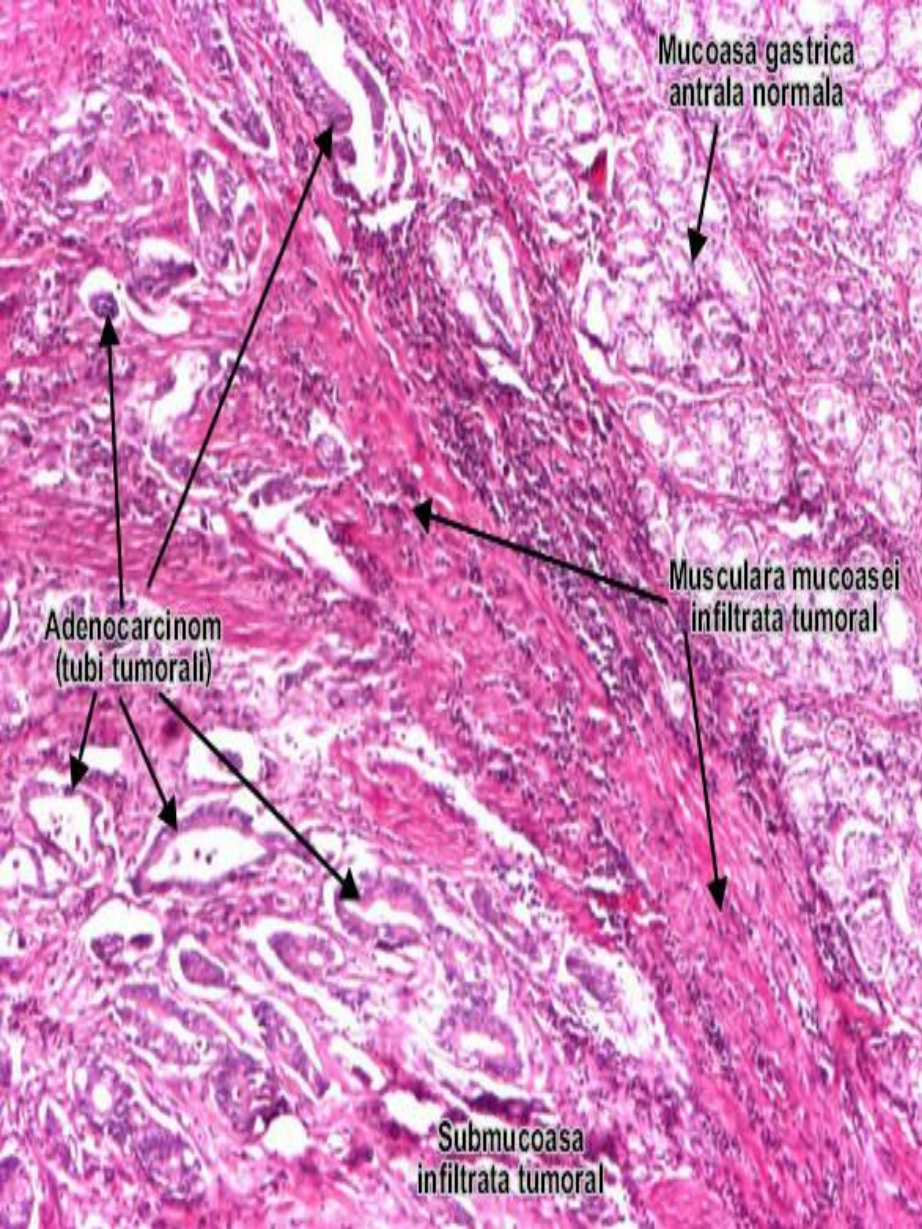


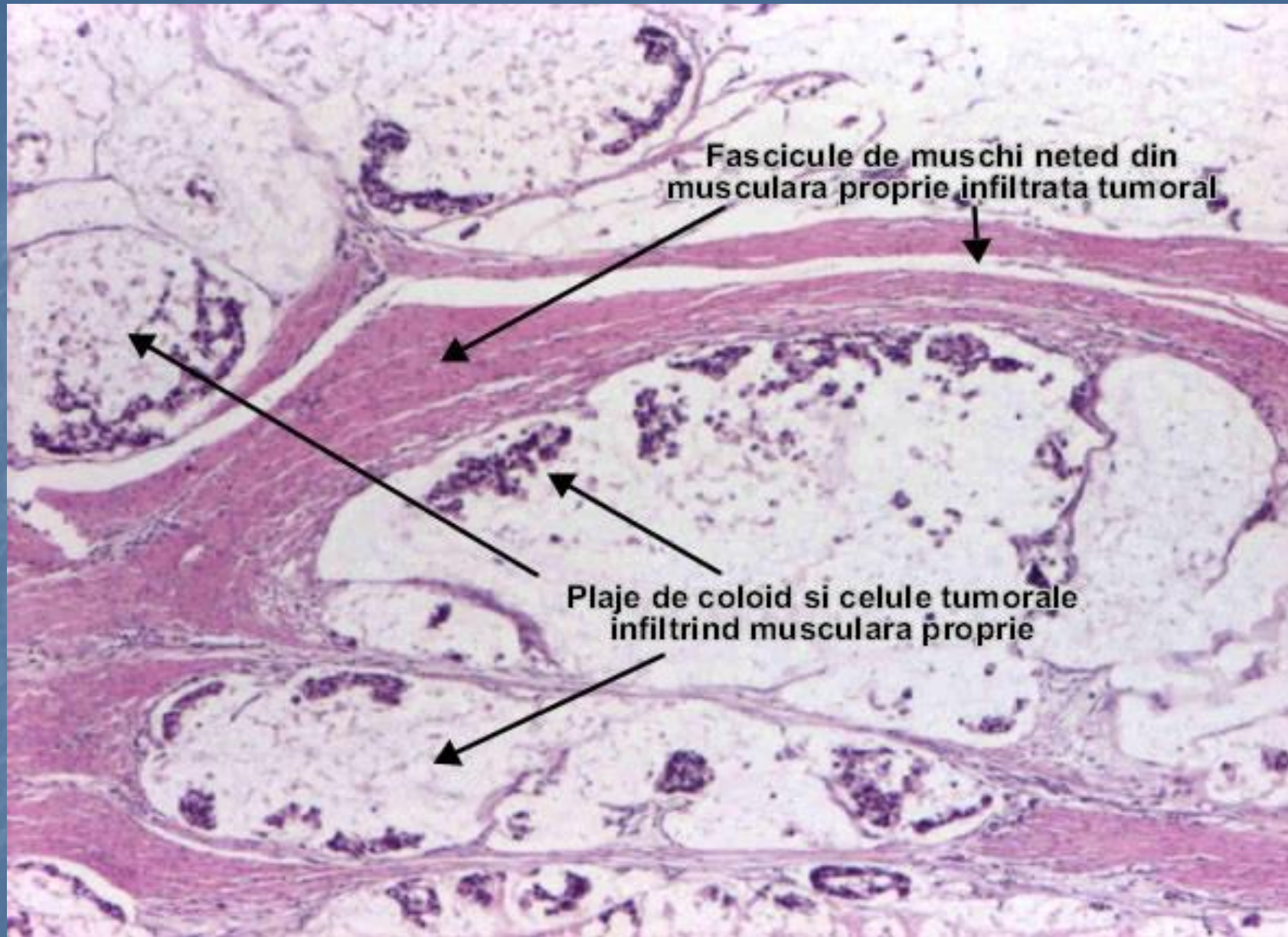
***Gastric carcinoma
de novo***

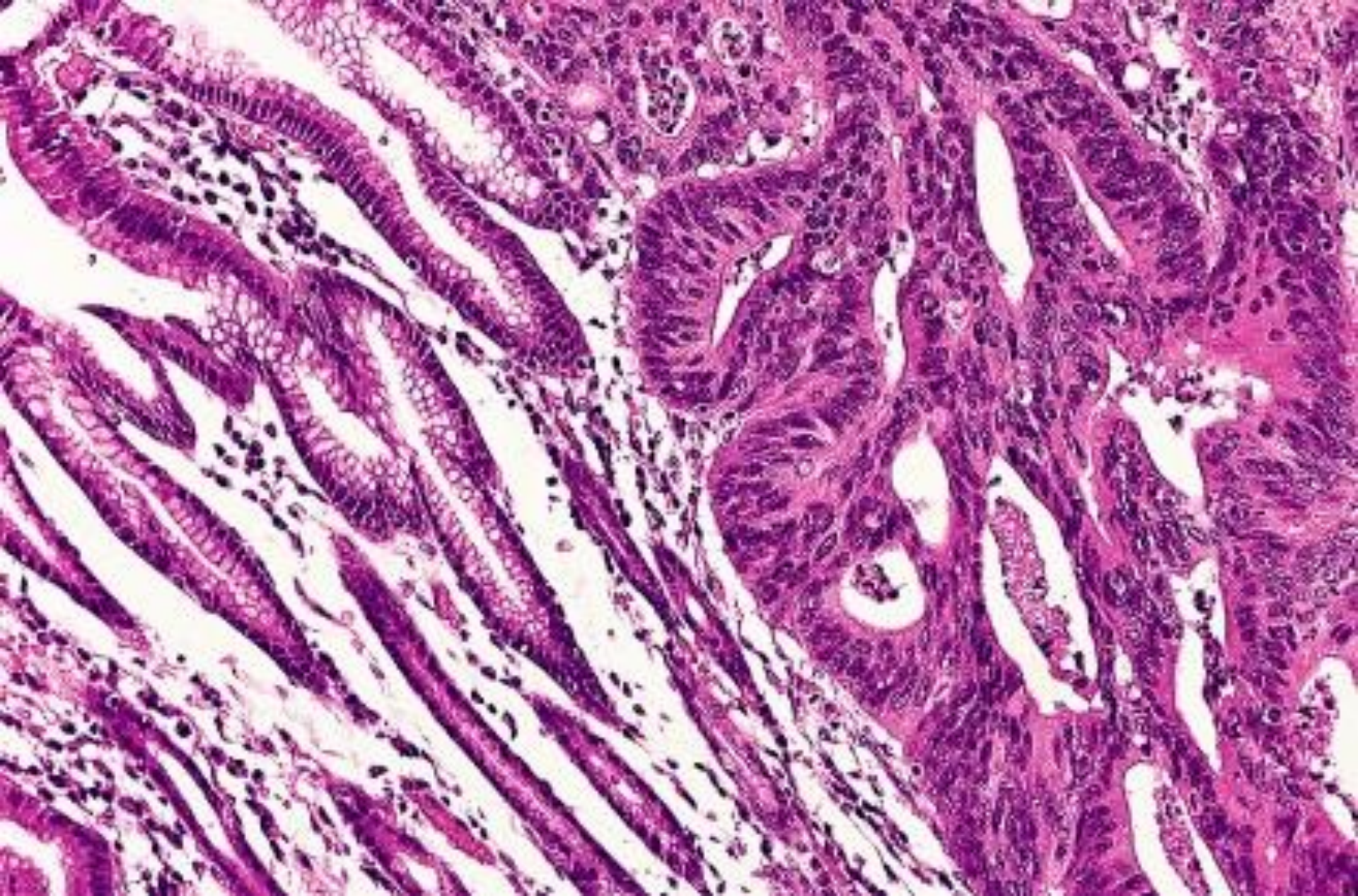


Intestinal type gastric carcinoma, the evolution of on steps.

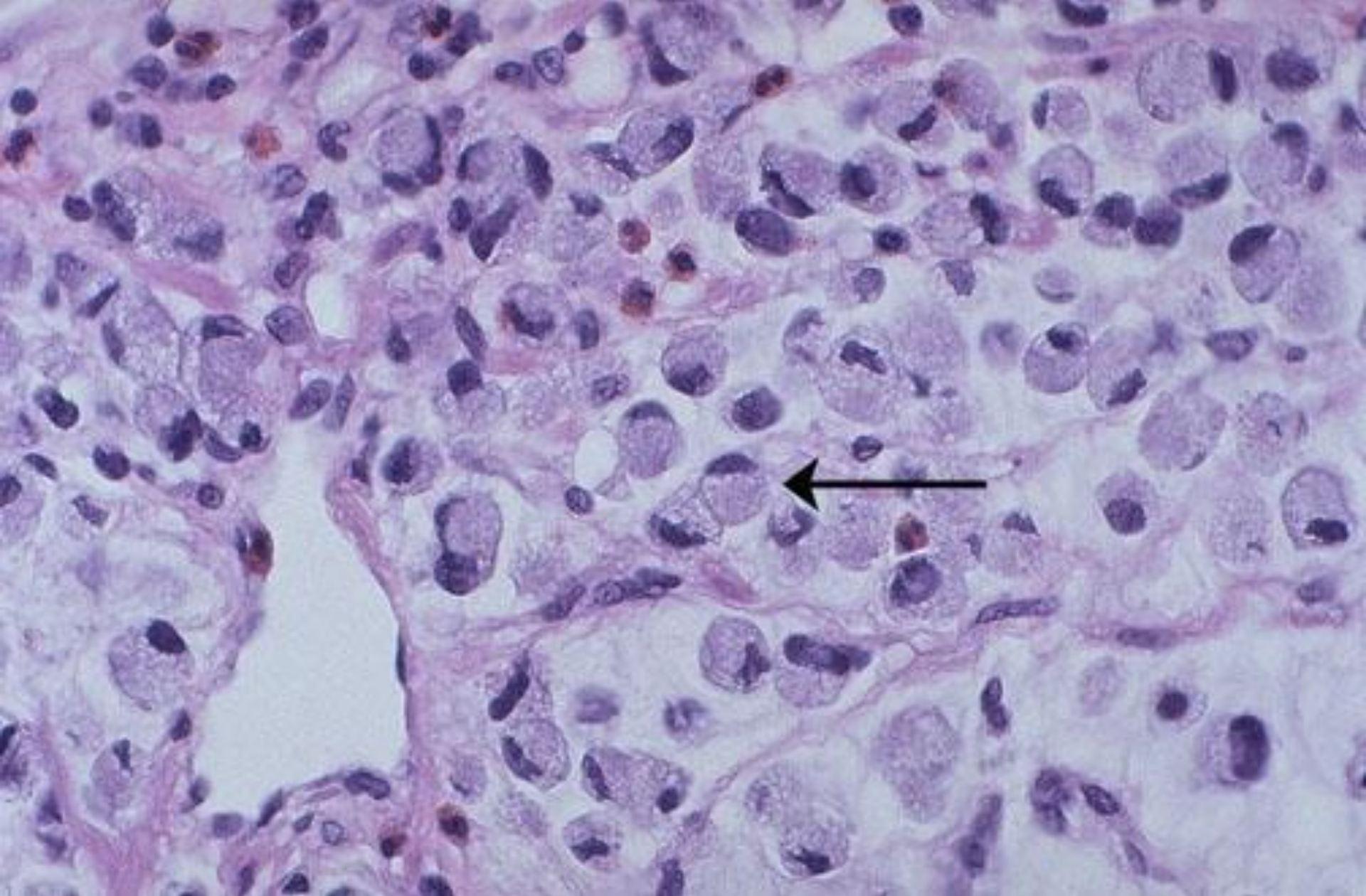








Papillary adenocarcinoma



Gastric carcinoma signet ring cells.

GASTRIC CARCINOMA - TNM staging

T – tumor comprises

T1 – mucosa and submucosa

T2 – muscle

T3 – serosa

T4 – adjacent organs

N – adenopathy:

N0 – without invasion

N1 – invaded lymph nodes in the vicinity (up to 3 cm from the tumor)

N2 – distant lymph node invasion
(Virchow mt.)

M – metastasis:

M0 – no

M1 – yes

