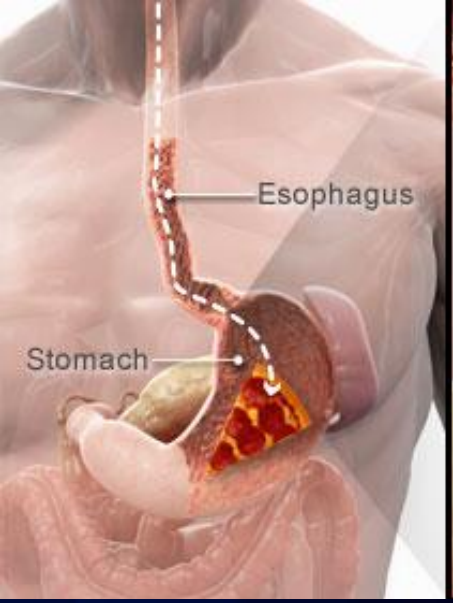
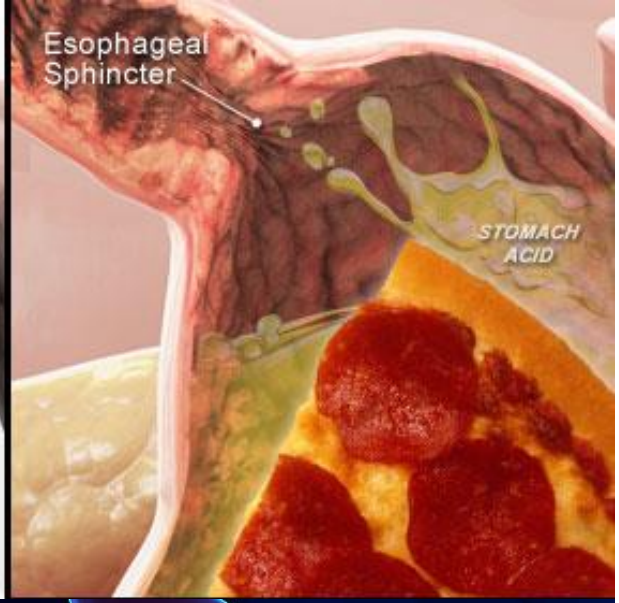
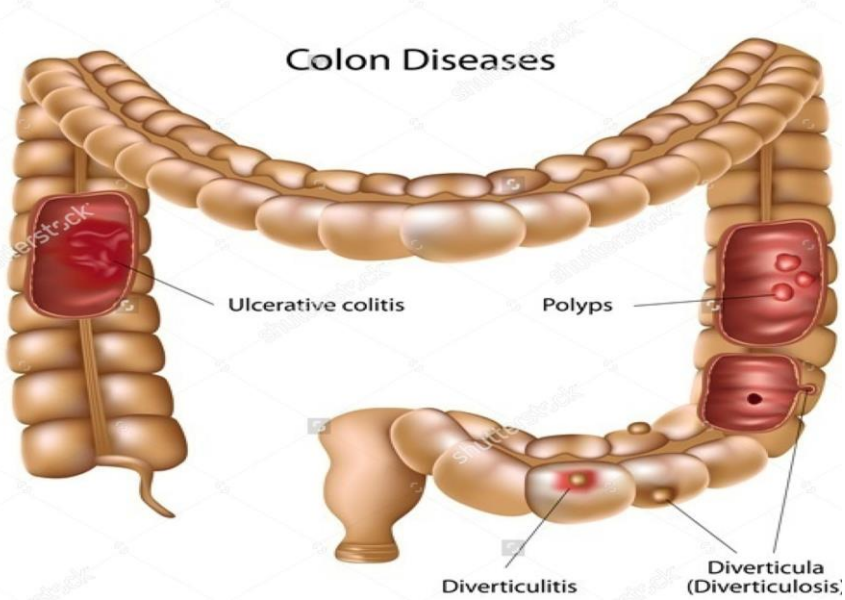






Pathology of the esophagus, stomach and intestines. Intestinal infections.

Pathology of the esophagus, stomach and intestines. Intestinal infections.

I. Microspecimens:

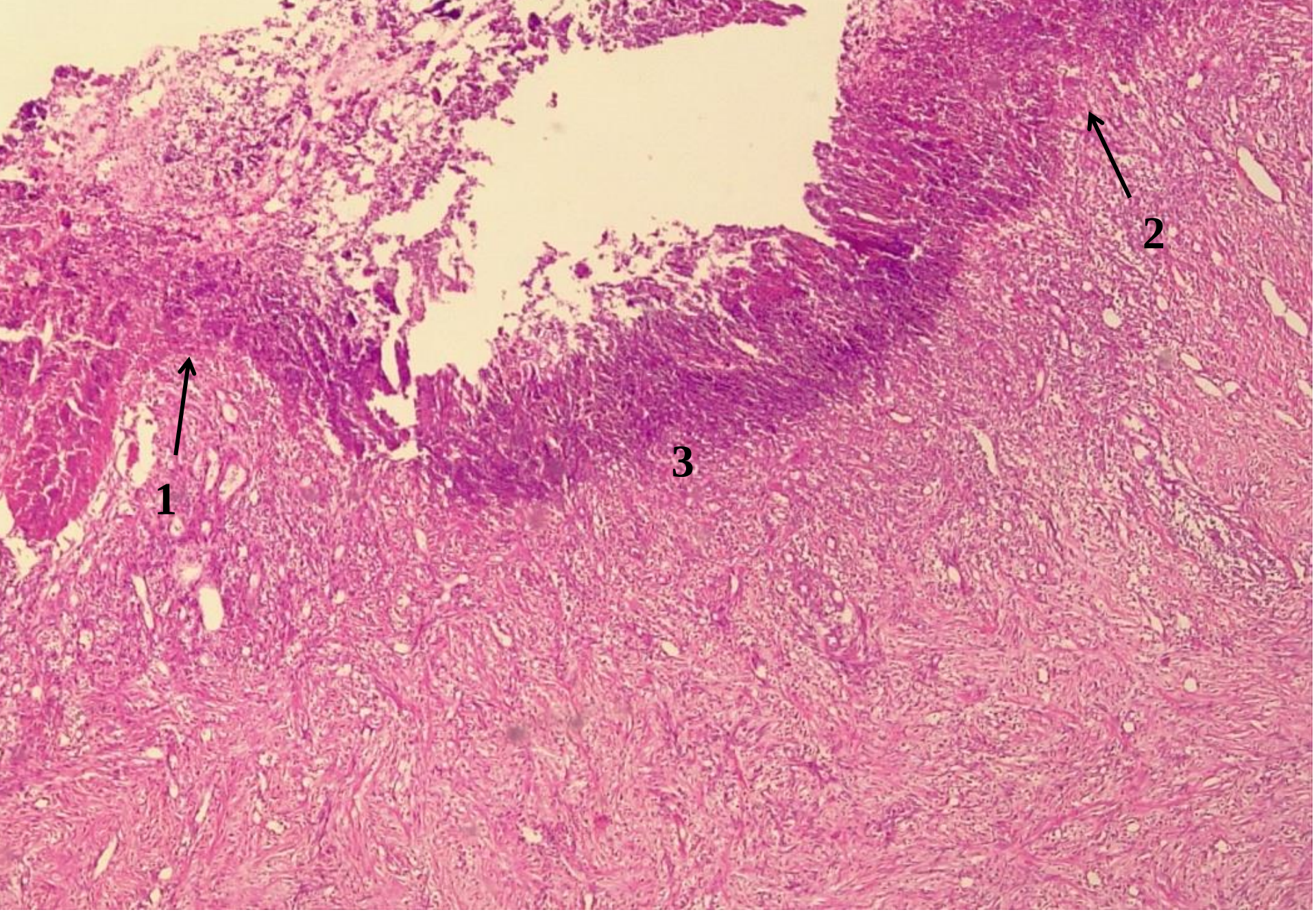
№ 87. Active chronic gastric ulcer. *(H.E. stain).*

Indications:

1. The distal edge of the ulcer.
2. The proximal edge of the ulcer.
3. The ulcer base:
   a. zone of infiltration with neutrophils
   b. zone of necrotic fibrinoid debris;
   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.



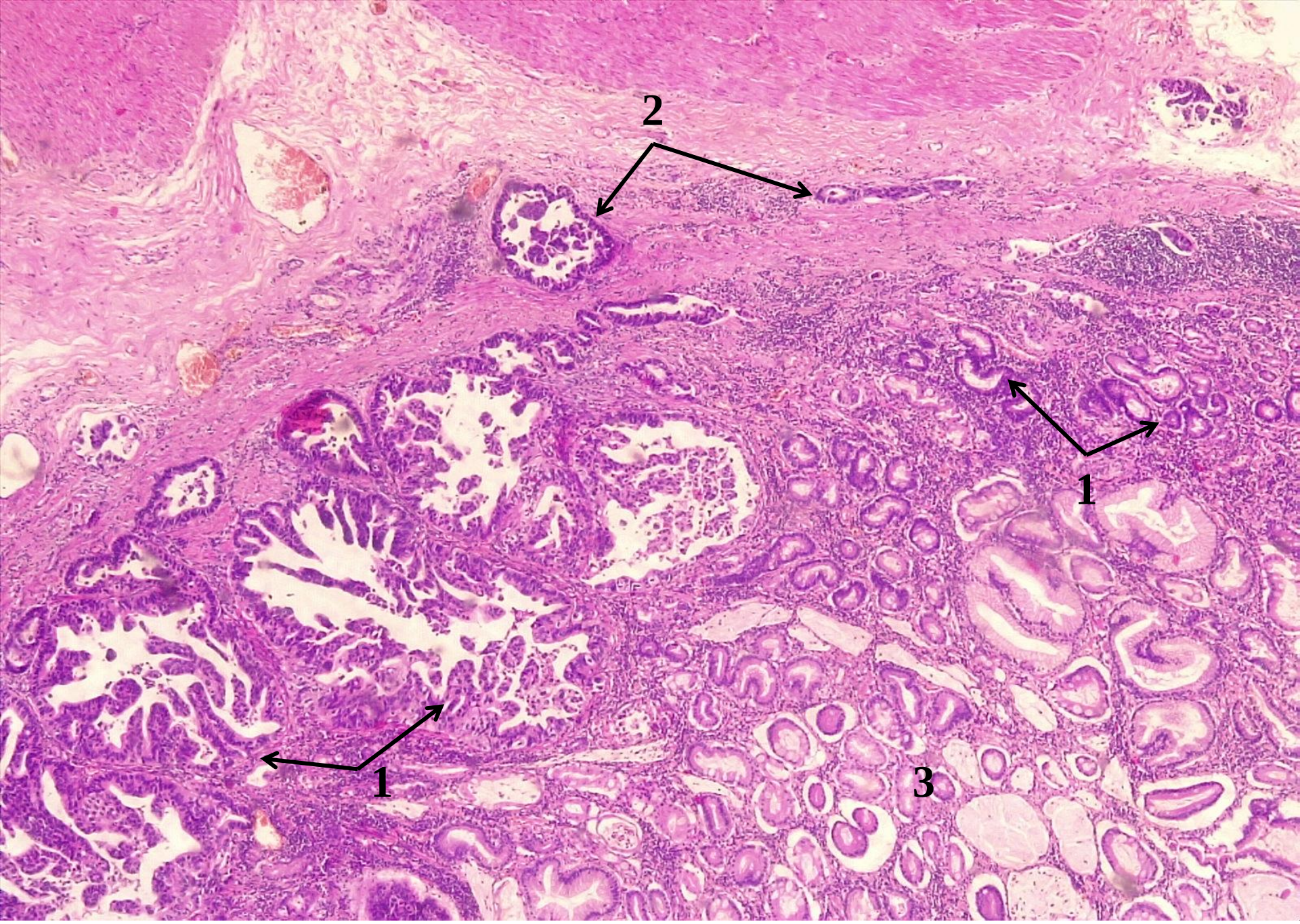
№ 87. Active chronic gastric ulcer. *(H.E. stain).*

№ 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, untill 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 lymphcyte 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).*

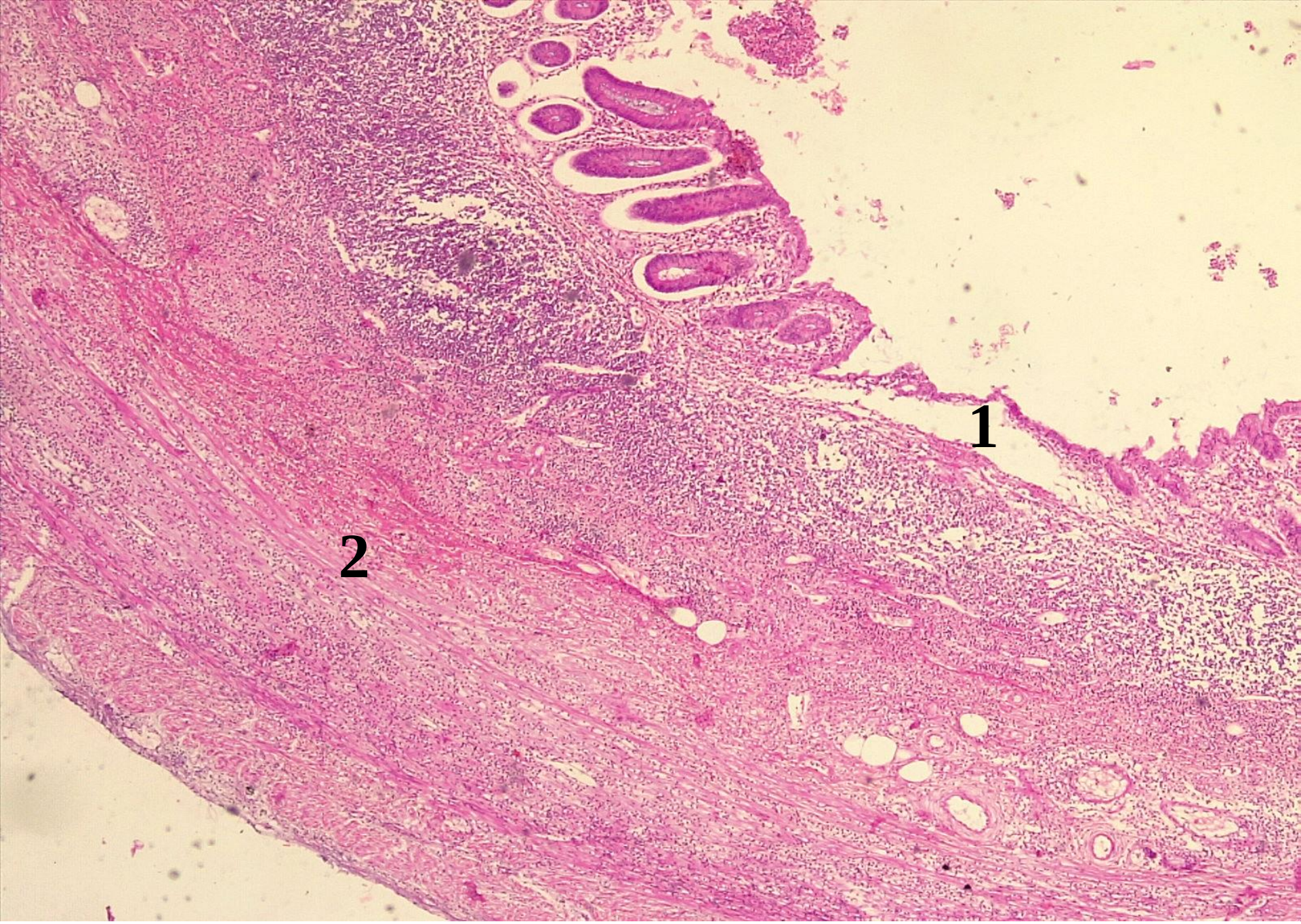
№ 88. Acute suppurative appendicitis. *(H-E. stain).*

Indications:

1. Ulcerative mucosal defects.
2. Diffuse neutrophilic infiltration of all layers of appendicular wall.

The lumen of the vermicular appendix is dilated, the wall thickened, edematous, are observed ulcerative defects in the mucosa, their bottom covered with necrotic masses and neutrophilic leukocytes, in the wall thickness diffuse infiltration with neutrophilic leukocytes is revealed, which extends in all layers, including the serous membrane, neutrophilic infiltration is more abundant in the muscular layer also there is dilatation and hyperemia of the vessels, hemorrhages, in the lumen neutrophilic leukocytes and necrotic masses.

The most common cause of acute appendicitis is obstruction of the lumen of the vermicular appendix, which can be caused by processes of fibrosis in the proximal portion, stones, including coprolites (starches), tumors, parasites, foreign bodies. These factors lead to retention of content and increased intraluminal pressure in the vermicular appendix, mucosal ischemia, epithelial damage, infection penetration, and the development of acute inflammation. The most important histological forms are: a) catarrhal appendicitis, b) phlegmonous, c) ulcero-phlegmonous and d) gangrenous. In some cases in the thickness of the appendix wall. can form microabscesses - apostematous appendicitis. Complications of acute appendicitis: a) perforation or self-amputation in gangrenous form with the development of localized or generalized peritonitis, b) spread of inflammation on the serous membrane - periapendicitis, mesenteryol - mesenteriolitis and check - peritvphlitis, c) empyema (accumulation of pus in the appendicular lumen) d) abscesses in the right iliac fossa, in the pelvis between the bladder and rectum and subdiaphragm on the right, e) pielophlebitis (inflammation of the portal vein) with abscesses in the liver. Late complications: adhesions with the large omentum, small intestine, other organs and mucocele.



№ 88. Acute suppurative appendicitis. *(H-E. stain).*

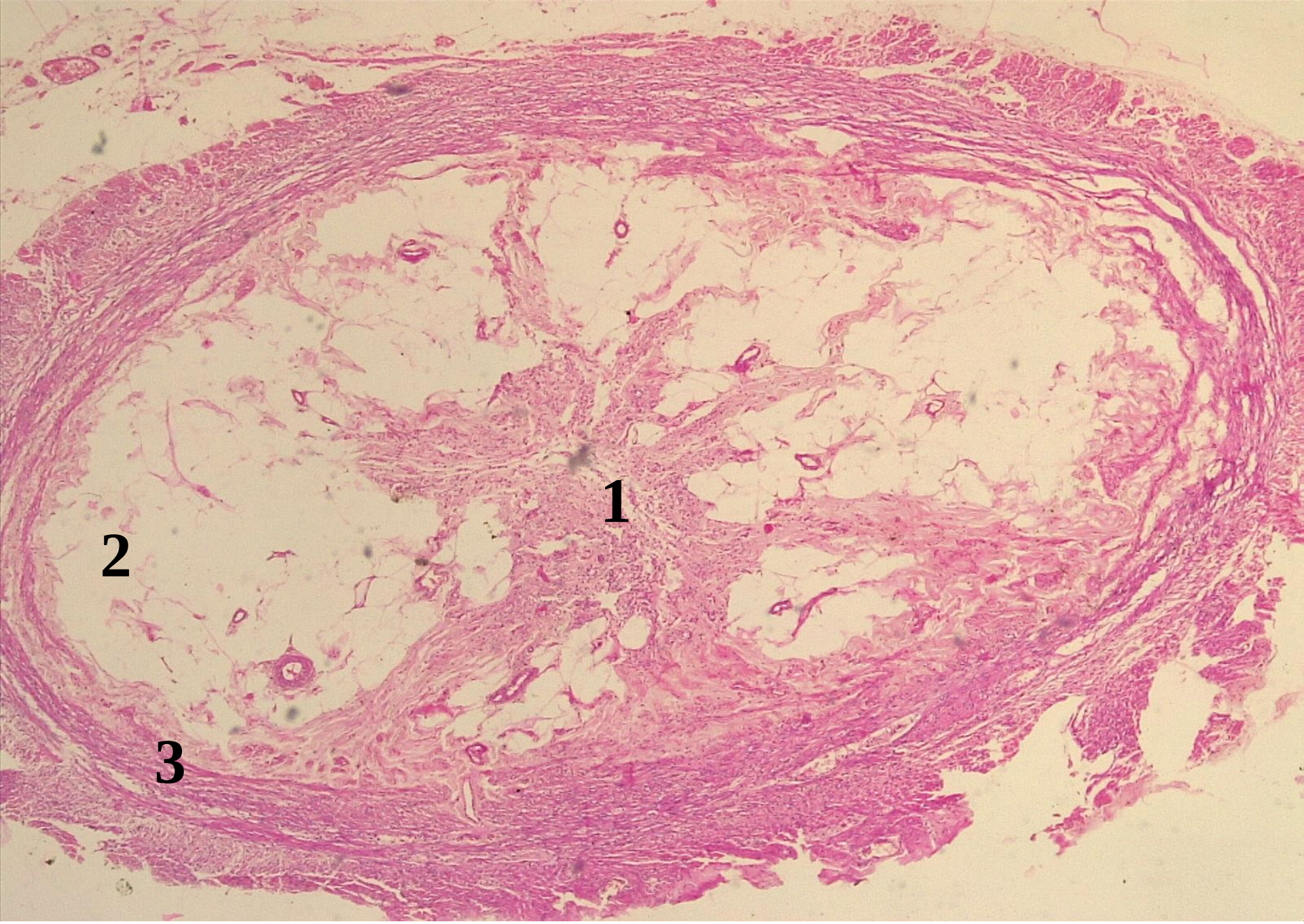
№ 88a. Chronic appendicitis. *(H-E. stain).*

Indications:

1. Occlusion of appendicular lumen.
2. Appendicular wall substituted by connective and fatty tissue.
3. Atrophied muscle layer.

The lumen of the vermicular appendix is obliterated with connective and adipose tissue, which have completely replaced the mucosa and submucosa, the muscular layer is atrophied.

Chronic appendicitis occurs as a result of acute appendicitis and is characterized by processes of sclerosis and atrophy of all layers of the wall, obliteration of the lumen may occur. In cases when the obliteration is at the level of the proximal portion of the appendix, the following may develop: a) appendicular hydrops (accumulation of serous fluid), b) mucocele (distension of the appendix with accumulation of mucus), c) myxoglobulosis (formation of mucus globules due to wall peristalsis), d) peritoneal pseudomixom in case of rupture of the mucocell wall and spread of mucus globules on the peritoneum (reminiscent of a myxoma), e) appendicular empyema in case of association of infection.



№ 88a. Chronic appendicitis. *(H-E. stain).*

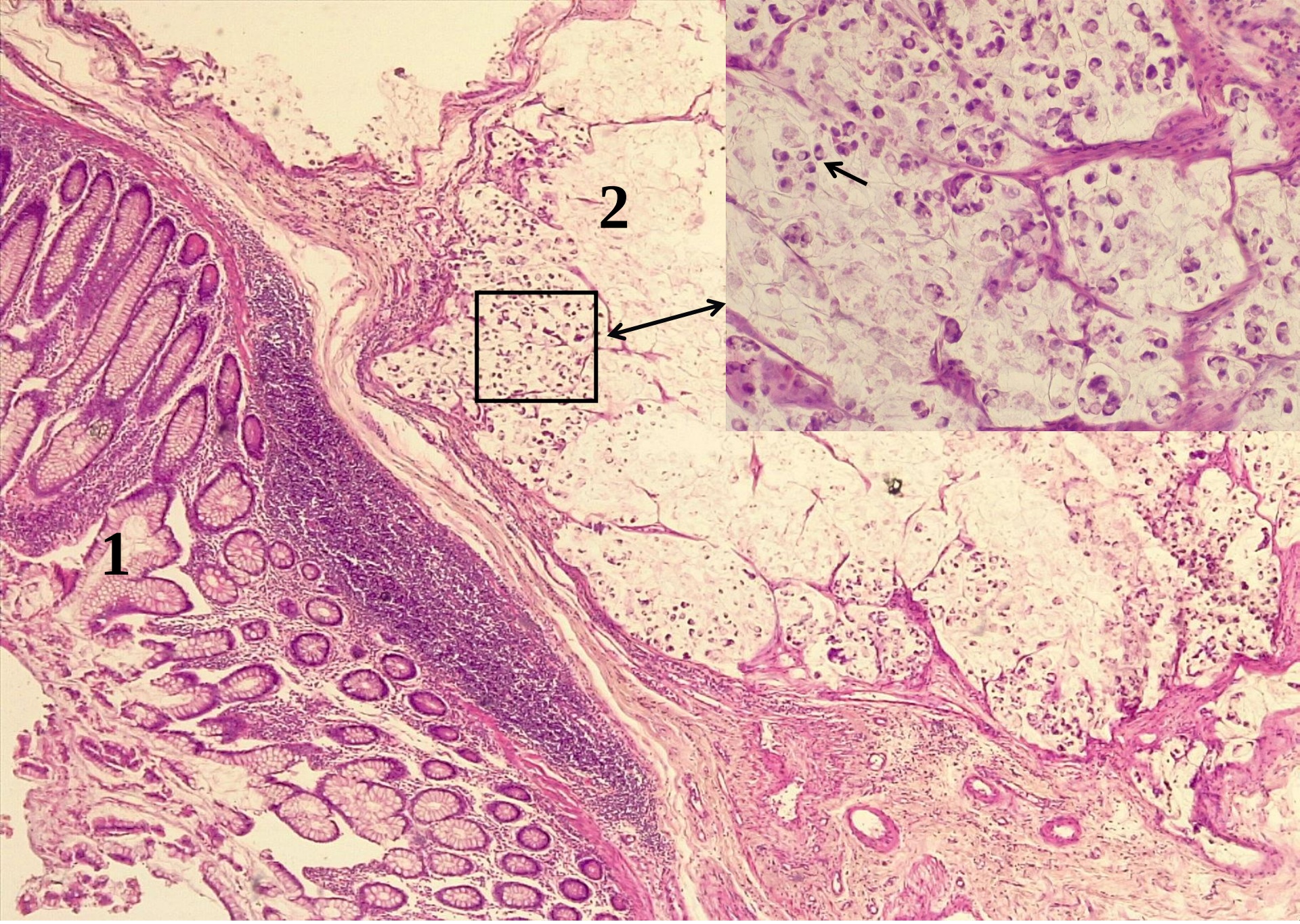
№ 48a. Mucinous carcinoma of the colon (signet-ring cell). *(H-E. stain).*

Indications:

1. Intact mucosa.
2. Clusters of "signet ring cells" and mucous substance which infiltrate the intestinal wall.
3. Muscle layer.

In the colonic wall the mucosa has a normal structure, immediately below the mucosal muscle there are "lakes" of weakly basophilic colored mucus, in the mucus "floating" isolated cells and groups of "signet ring cells", round / oval shape, with abundant cytoplasm, the nucleus displaced to the membrane and flattened; in the adjacent tissue chronic inflammatory infiltration, predominantly lymphoid.

Signet ring cell carcinoma of colon is relatively rare, in about 1% of total cases. It is localised predominantly in the right colon. Macroscopically it looks like a gelatinous mass. It is distinguished by aggressive evolution, metastases appear quickly, multiple and in several organs.



№ 48a. Mucinous carcinoma of the colon (signet-ring cell). *(H-E. stain).*

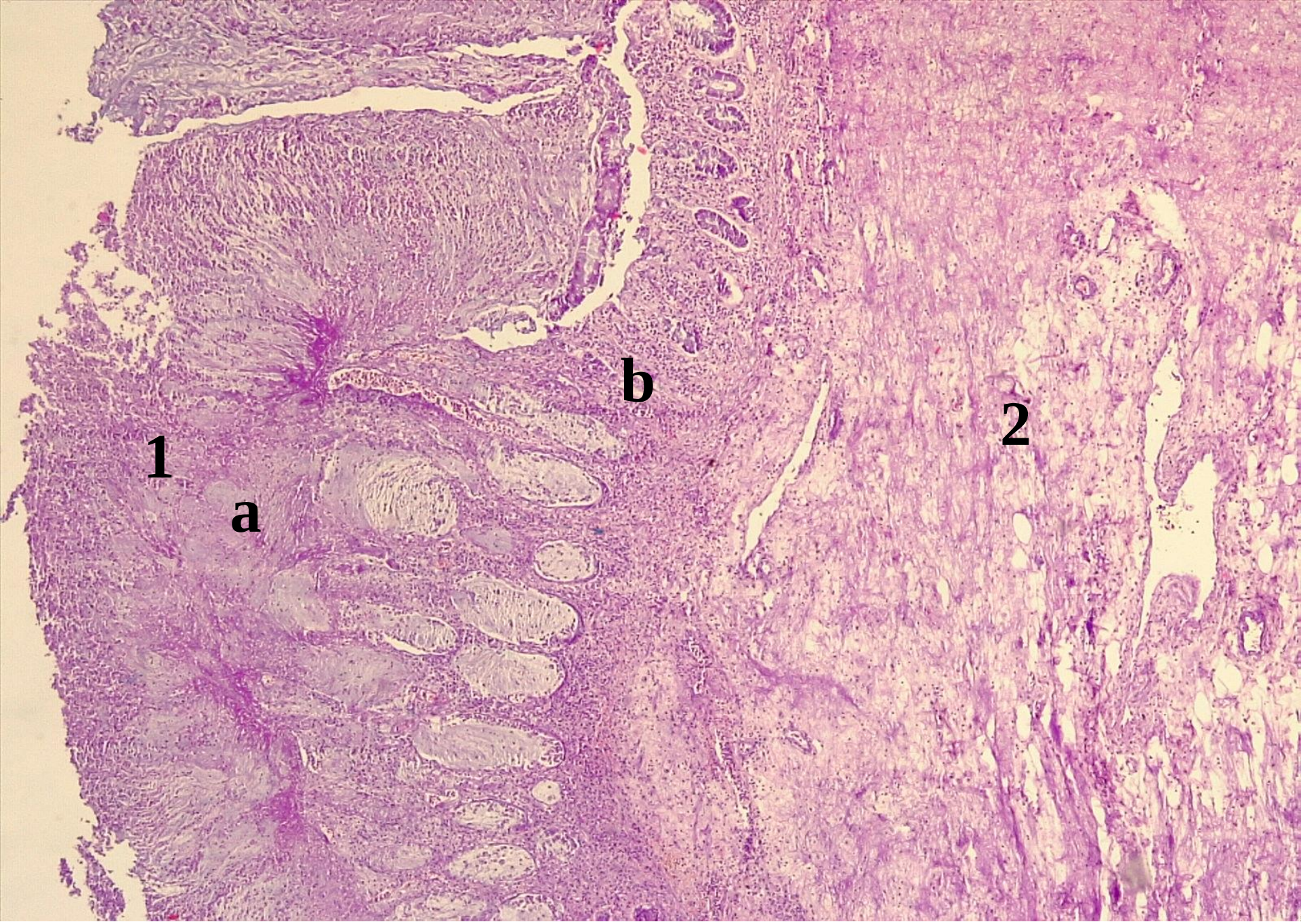
№ 15. Pseudomembranous colitis. *(H-E. stain).*

Indications:

1. Pseudomembrane:
   a. necrotic masses and fibrin with diffuse neutrophilic infiltration;
   b. underlying tissue.
2. The muscular layer of the intestinal wall.

On the surface of the colonic mucosa there is a layer of fibrin with a mixture of necrotic masses, infiltrated with neutrophilic leukocytes and mucus, which is called "pseudomembranous" so as not to be confused with true anatomical membranes; the pseudomembrane in some places is detached from the remains of the underlying mucosa, which is mostly necrotic, only the contours of the basal portions of the crypts have been preserved, filled with muco-purulent exudate; the wall of the colon is edematous, with hemorrhages, dilated vessels, hyperemia.

Pseudomembranous colitis is most commonly caused by the pathogen Clostridium difficile, which eliminates toxins, which act on the lining of the colon and / or small intestine, causing acute pseudomembranous-looking colitis / enterocolitis. Macroscopically, the colonic mucosa is covered with a whitish-gray film, sometimes with a greenish tinge due to the impregnation with bile pigments. It is usually found in patients who use antibiotics for a long time, so it is also called "colitis associated with the administration of antibiotics." Other risk factors are old age, immunosuppression and hospitalization. The prevalent clinical symptom is diarrhea and dehydration.



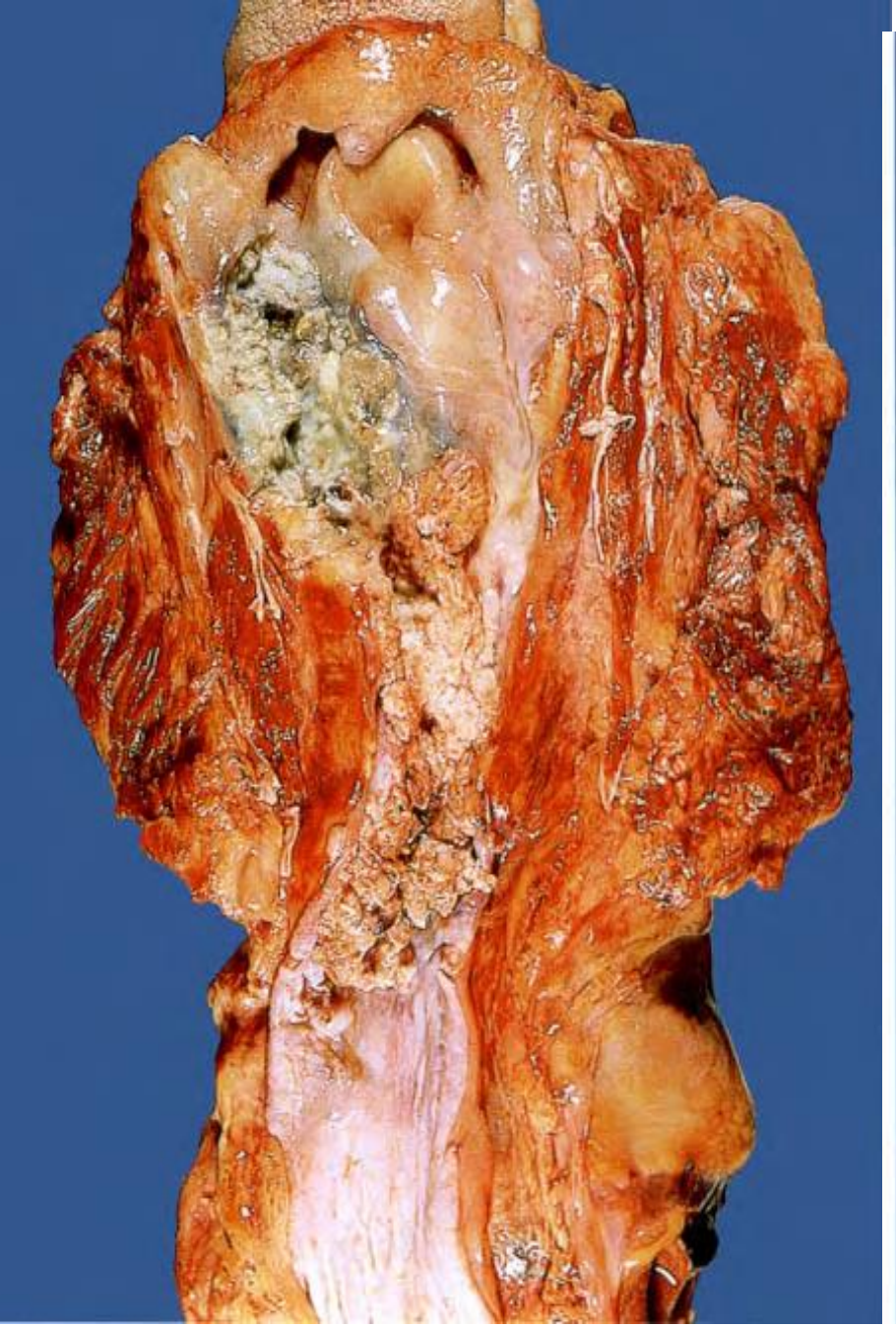
№ 15. Pseudomembranous colitis. *(H-E. stain).*

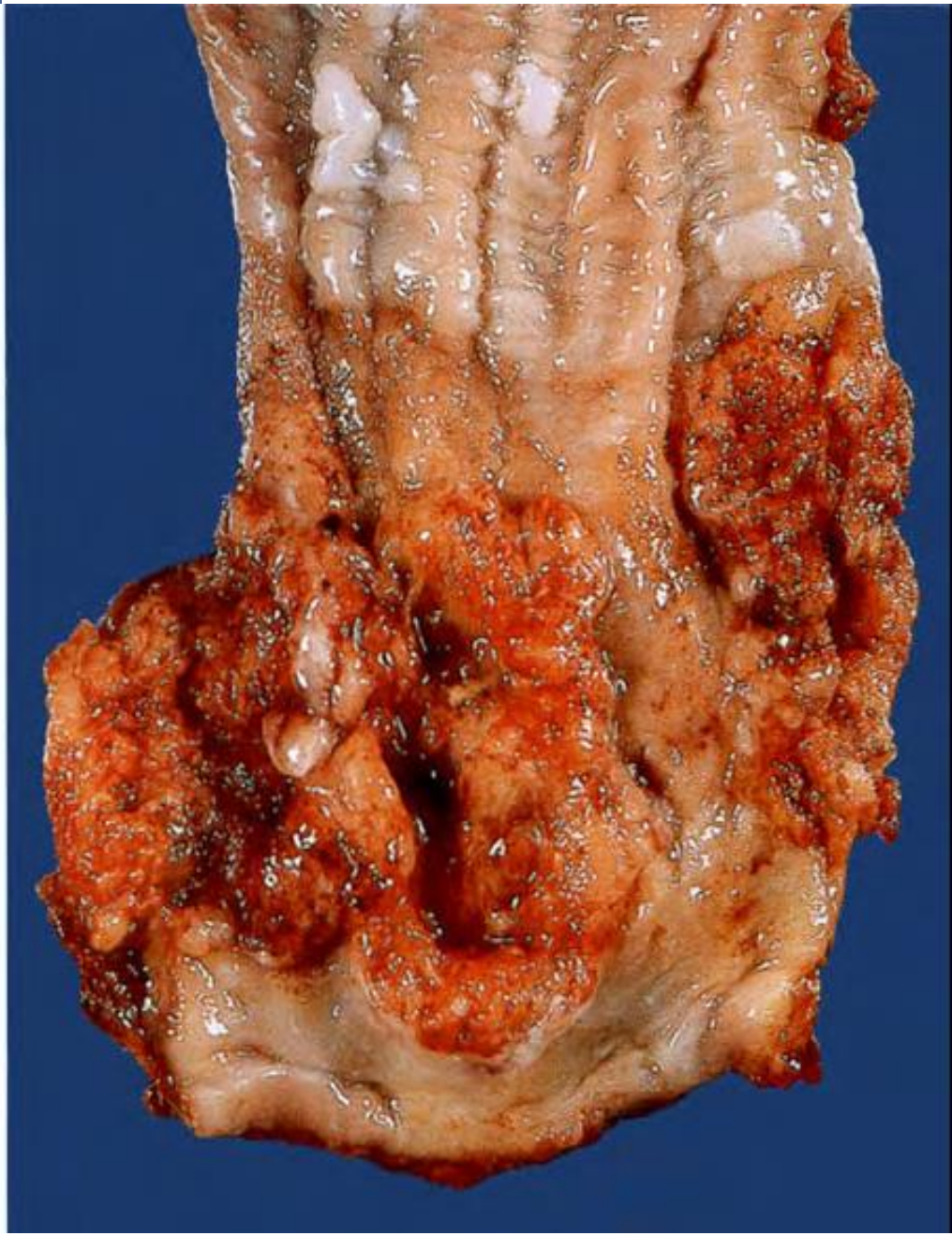
II. Macrospecimens:

№ 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 hitological 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.

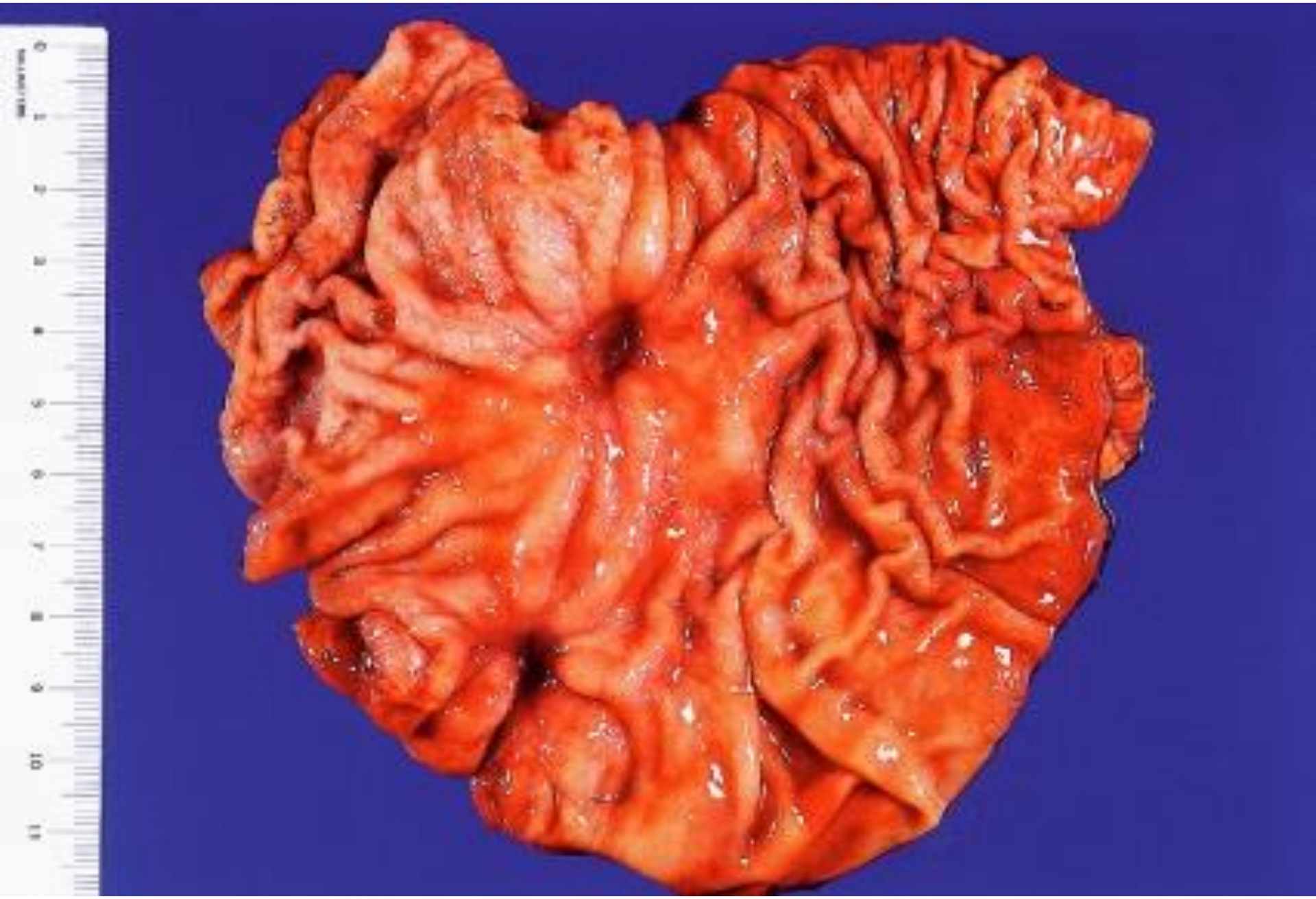
Gastric polyps are more frequently located in the anthro-pyloric region, they can be solitary or multiple. The absolute majority of gastric polyps (≈90%) are of inflammatory origin, non-neoplastic (hyperplastic polyps). Microscopically they consist of hyperplastic glands, irregularly arranged, some cystically 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.

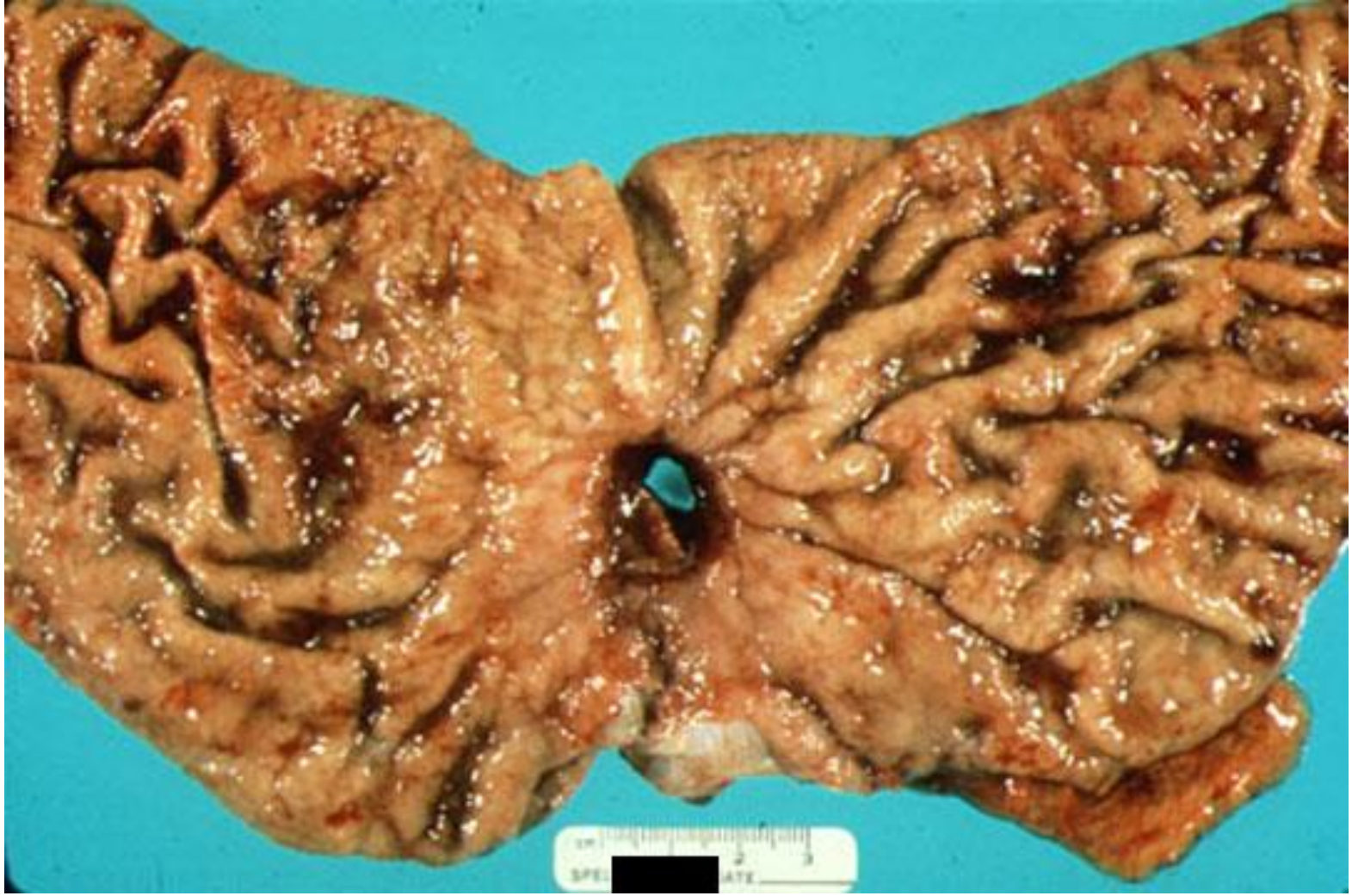
In the gastric wall a chronic ulcer can be noted, in which a perforative defect can be observed, through which the gastric contents are eliminated in the peritoneal cavity and peritonitis develops.

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.

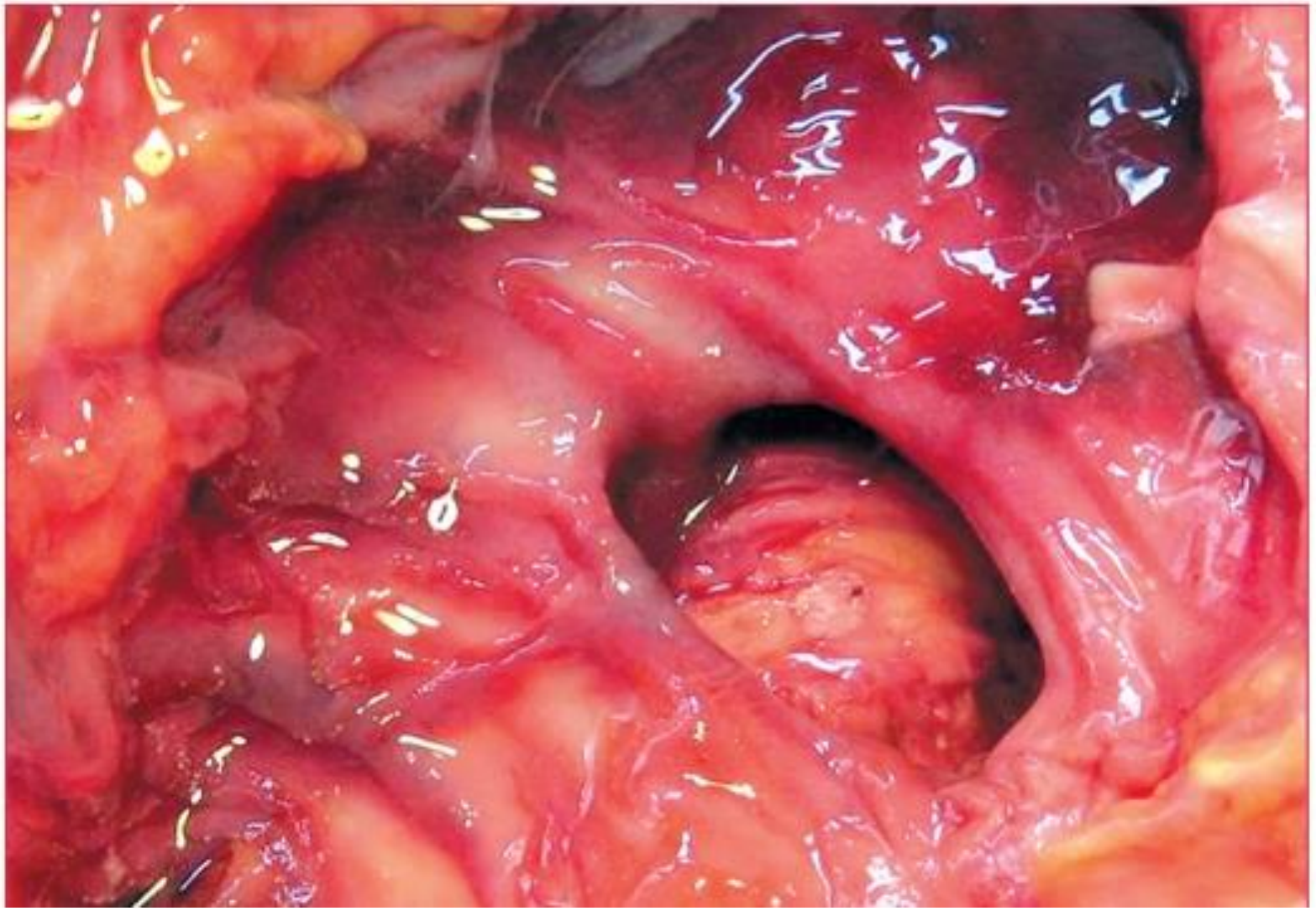
№ 54. Chronic duodenal ulcer.

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.



№ 53. Chronic gastric ulcer with perforation.

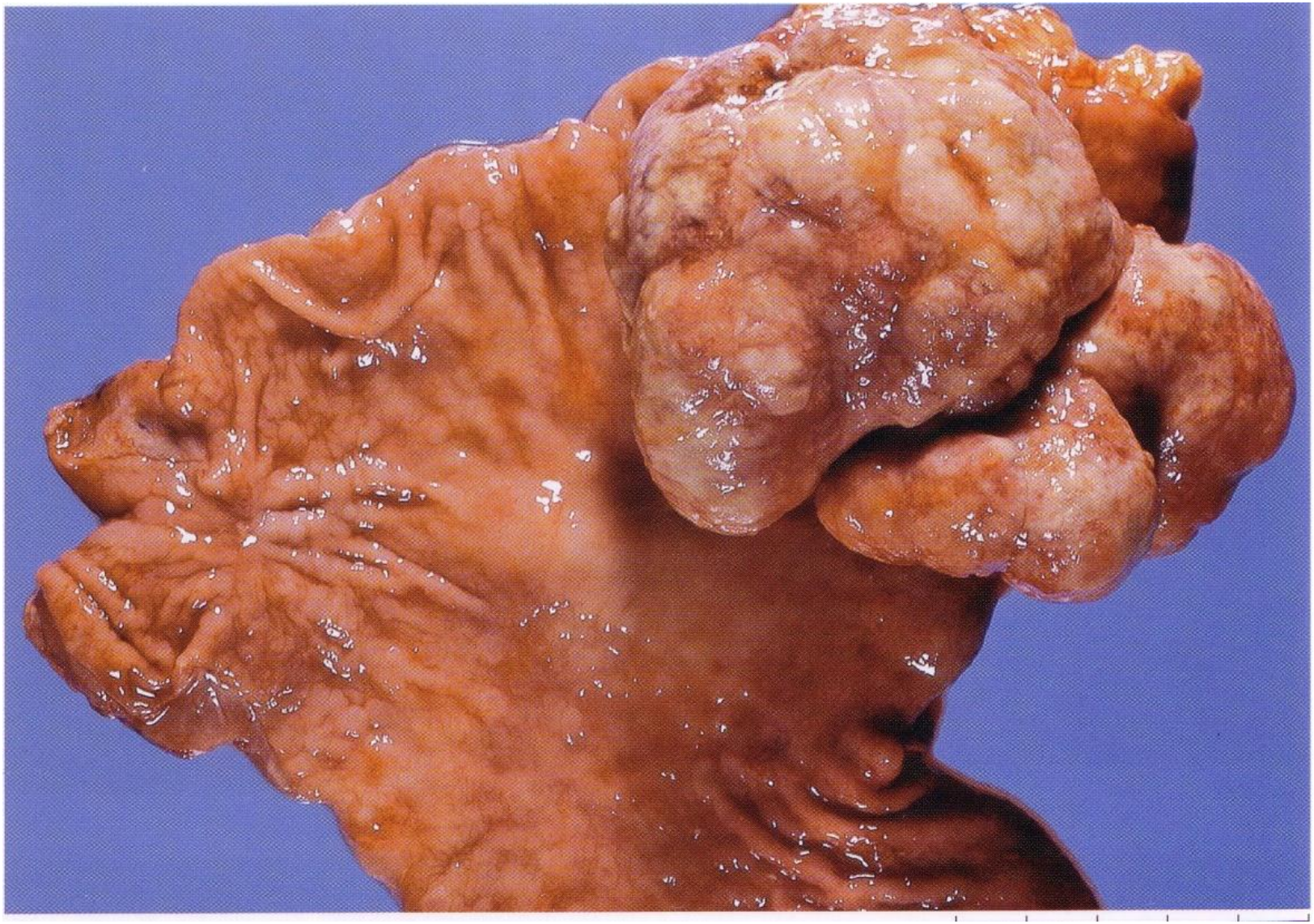


№ 54. Chronic duodenal ulcer.

№ 60. Gastric carcinoma.

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 carcinoma.

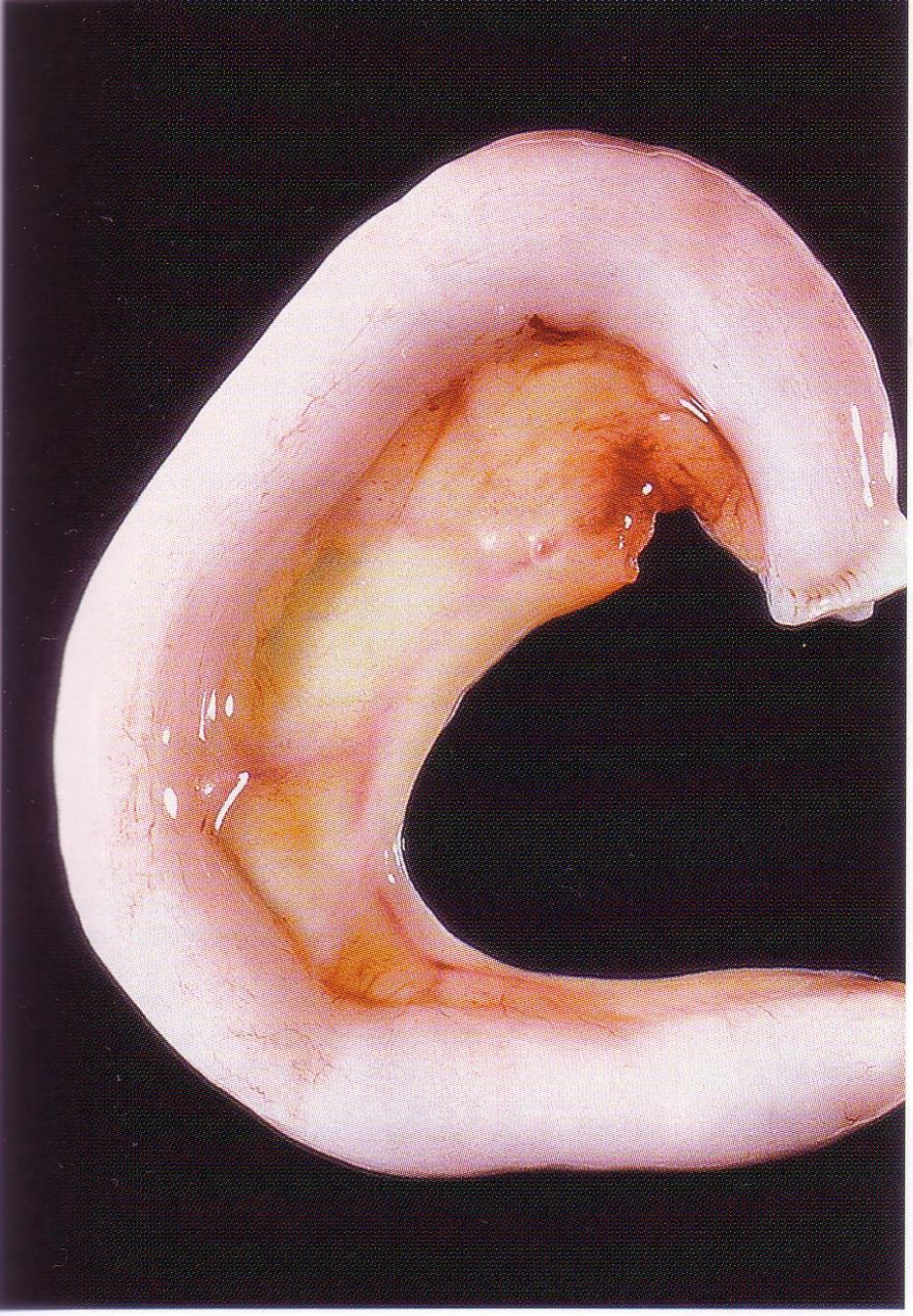
№ 55. Acute suppurative appendicitis.

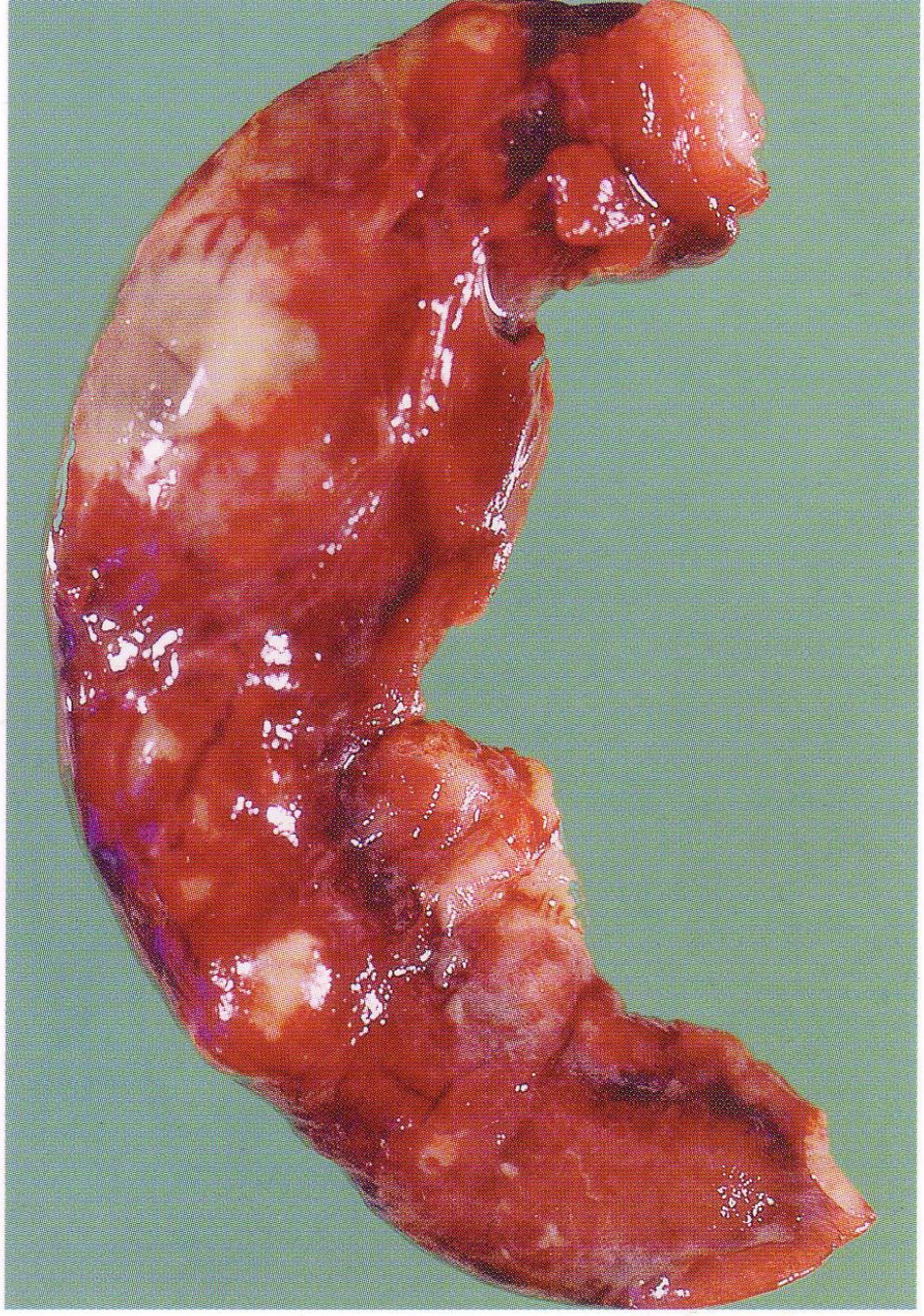
The vermicular appendix is dilated, the surface is matte, serous edematous, hyperemic, with hemorrhagic foci and whitish fibrin deposits, the mesentery is edematous, hyperemic, with hemorrhages and fibrin. *[microspecimen № 88]*

№ 61. Carcinoma of sigmoid colon.

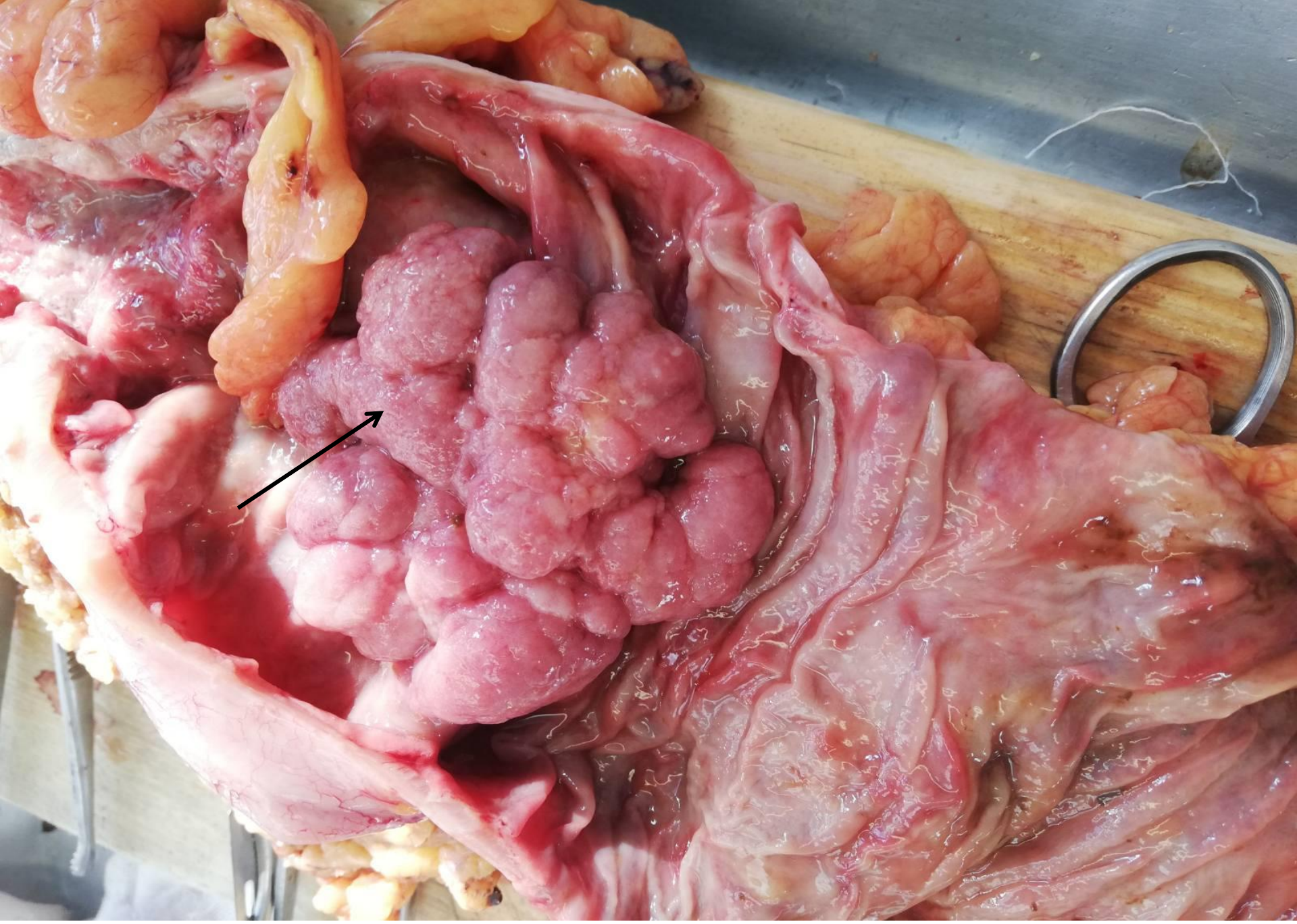
In the colon there is a large tumor node, which grows exophyte, considerably stenoses the intestinal lumen, irregular surface, with areas of necrosis and ulceration, dense-elastic consistency, pinkish-whitish color.

Colon cancer is localised more frequently in descending order: in the rectum (60%), sigma, descending colon, cecum and ileocecal region, ascending colon, hepatic flexure and spinal flexure. It can be complicated by intestinal occlusion, hemorrhage, perforation and peritonitis, infiltration of adjacent tissues / organs, phlegmon. Metastases occur primarily by lymphogenesis in regional lymph nodes, and hematogenous metastases are relatively late and are more common in the liver, lungs, brain, bones and ovaries. Microscopically in the majority of cases (90-95%) colon cancer is adenocarcinoma. Among the precursors, adenomas, familial adenomatous polyposis, nonspecific ulcerative colitis and Crohn's disease are more common.





№ 55. Acute suppurative appendicitis.



№ 61. Carcinoma of sigmoid colon.

№ 58. Encephaloid modifications of Peyer's patches in typhoid fever.

Peyer's patches are enlarged in size, protrude on the surface of the intestinal mucosa, have a gray-pink color and plicated appearance, reminiscent of the brain surface, hence the name of this stage "encephaloid intumescence of Peyer's patches"; in some patches necrotic masses are observed, which partially detach, forming ulcerations, the edges of which are slightly elevated due to edema and inflammation; the ulcers have the length and shape of the Payer patches, on average 6-8 cm and are located along the longitudinal axis of the intestine; the adjacent mucosa is edematous and hyperemic.

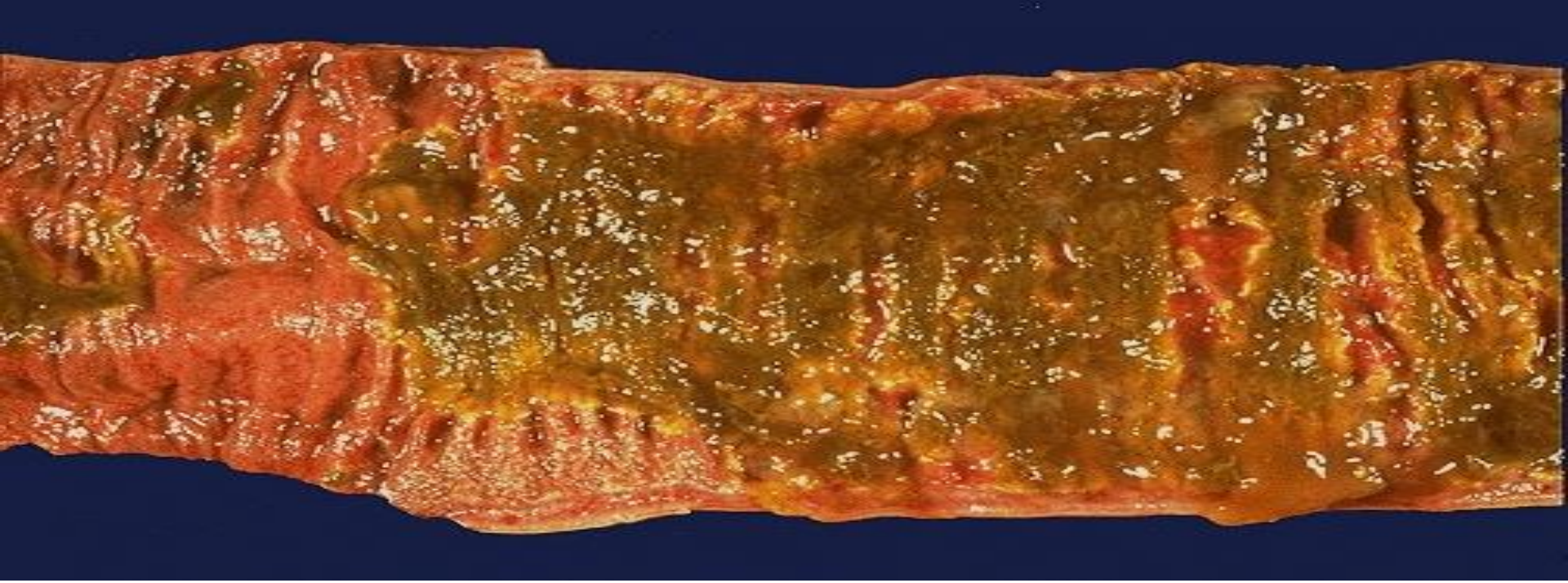
Typhoid fever is caused by Salmonella typhi, it is transmitted by food. The first morphological changes occur in the Payer plates and solitary follicles, more pronounced in the ileum. Productive inflammation develops with the proliferation of monocytes and histiocytes, which replace lymphocytes; some monocytes turn into large macrophages, with clear cytoplasm, which phagocytose typhoid bacilli and are called typhoid cells, and their agglomerations form granulomas or typhoid nodules. Due to these proliferative processes, the encephalopathy of Payer plaques develops. Subsequently, necrosis of typhoid granulomas occurs, gradual detachment of necrotic masses and the appearance of ulcers, initially covered with necrotic masses ("dirty" ulcers), which after a week become clean, with a smooth bottom, presented by the muscular layer or deeper to the peritoneum ("clean" ulcers). Ulcers can be complicated by bleeding and perforation. Fine scars form on the site of the ulcers, which have the shape of Payer patches. General complications: pneumonia, cholecystitis, waxy necrosis of skeletal muscles, osteomyelitis, meningitis, sepsis.

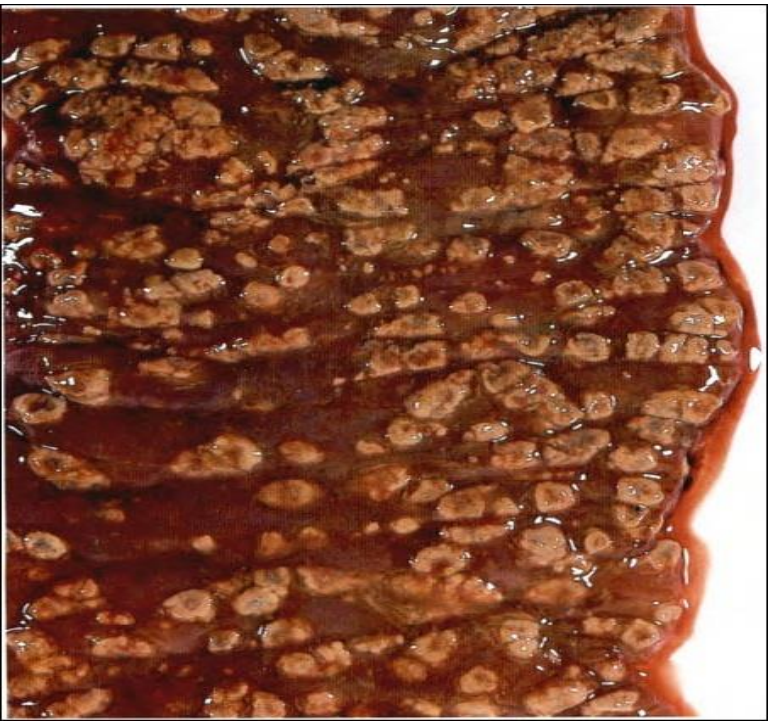
№ 58. Encephaloid modifications of Peyer's patches in typhoid fever.

№ 57. Fibrinous ulcerative colitis in schigellosis.

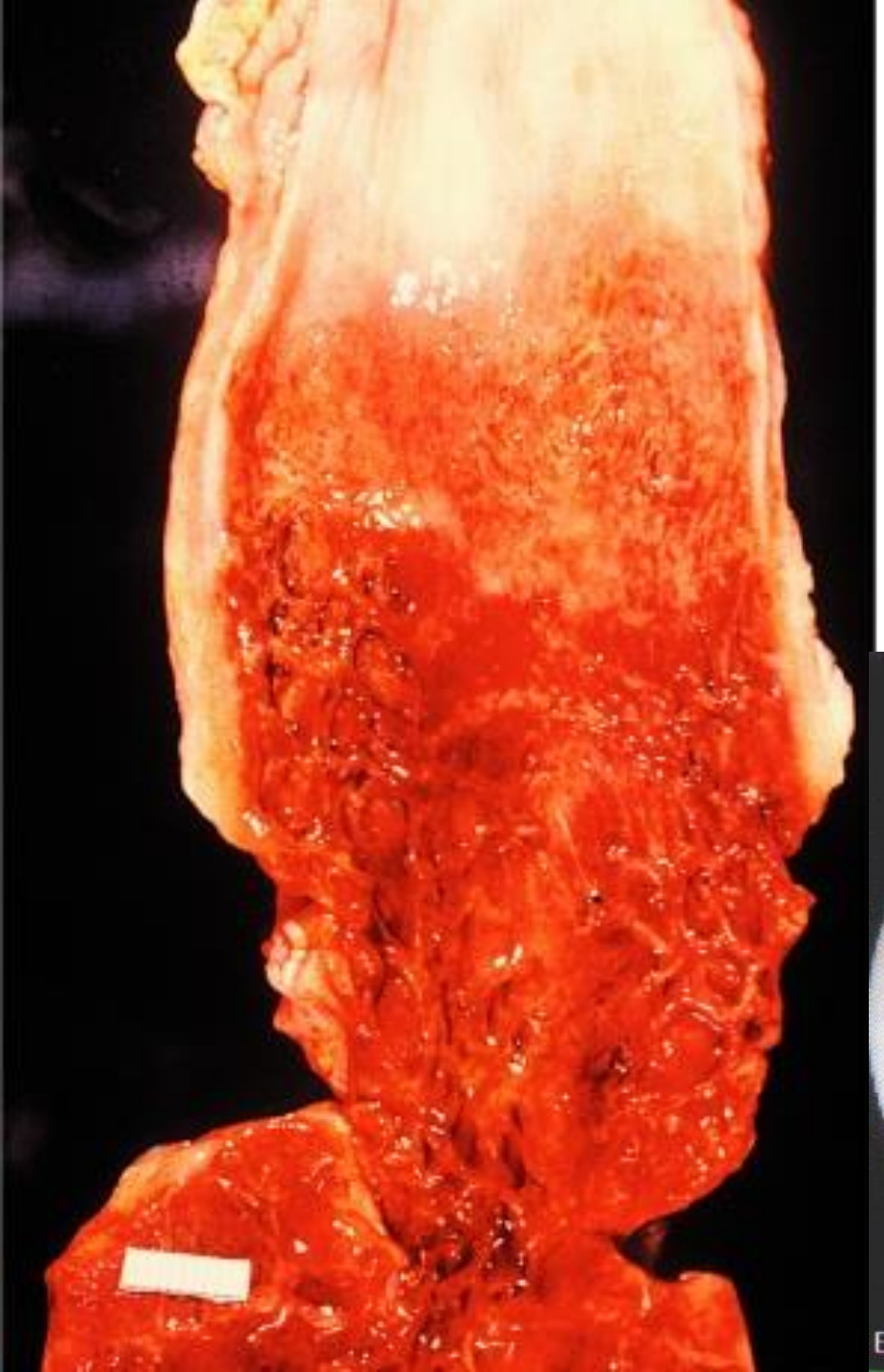
On the surface of the colonic mucosa there is a brown-gray fibrin film, the adjacent mucosa is edematous, hyperemic.

Schigellosis is caused by the pathogen Schigella, the infection occurs through food (fecal-oral). It affects the left colon more, especially the recto-sigmoid portion. Locally, there are 4 stages of dysenteric colitis: a) catarrhal colitis, b) fibrinous colitis, c) ulcerative colitis and d) healing of ulcers. Fibrinous colitis replaces catarrhal colitis, usually in the 2nd week after the onset of the disease. Ulcers occur following the detachment of fibrinous pseudomembranes, are located more frequently in the rectum and sigmoid colon, have an irregular shape and can be extensive and deep. Local complications are perforation with the development of paraproctitis and peritonitis and intestinal hemorrhage. The most common general complications: reactive arthritis, conjunctivitis, urethritis. In patients with chronic Schigellosis may develop secondary amyloidosis.

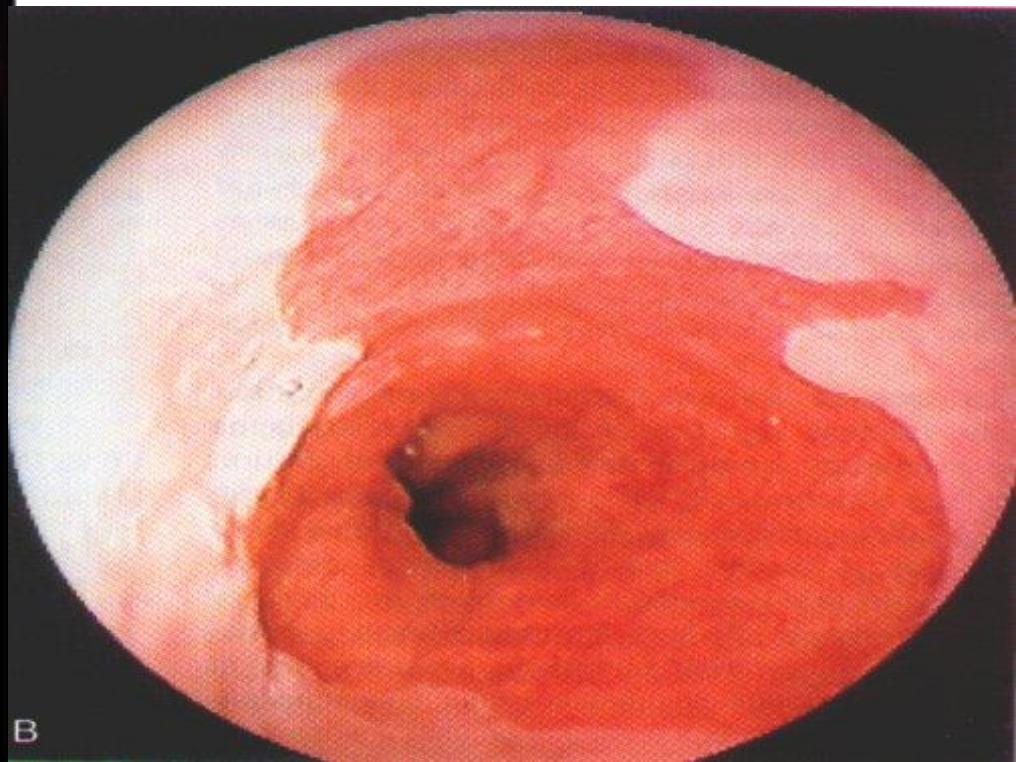




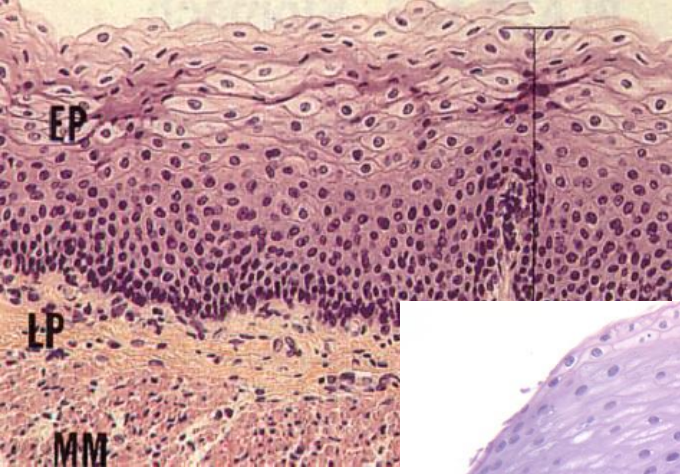
№ 57. Fibrinous ulcerative colitis in schigellosis.

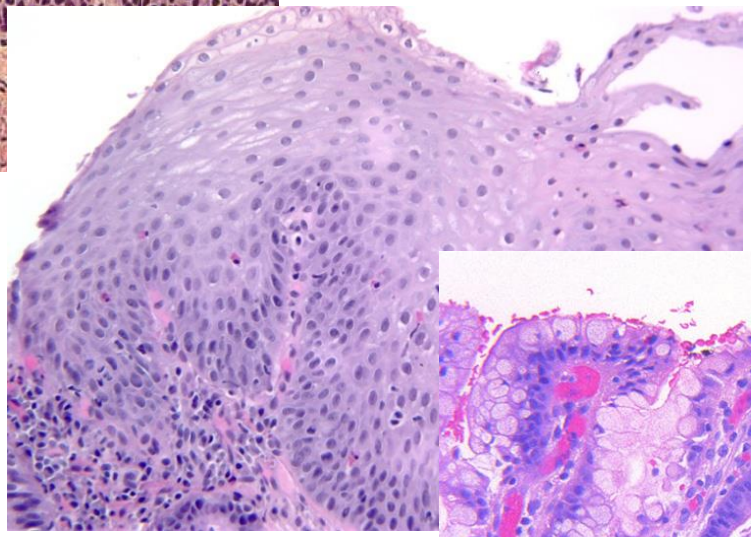


Reflux esophagitis: macroscopic and endoscopic pattern.

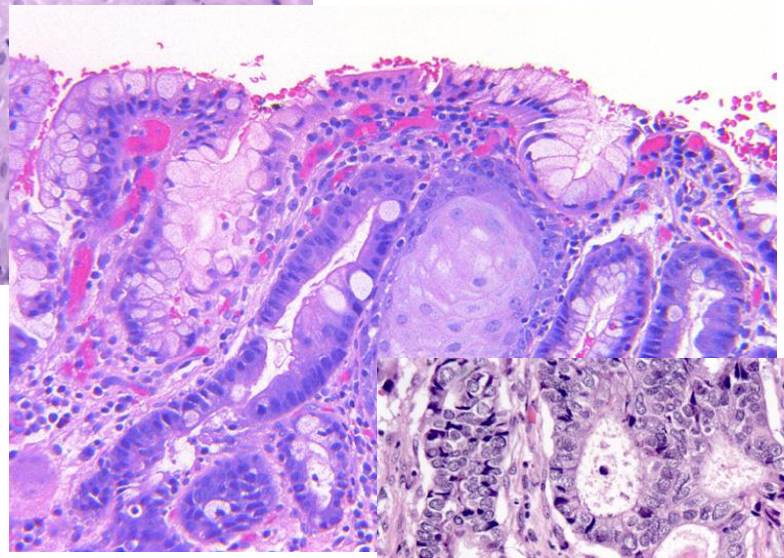


Morphopathogenesis of esophageal adenocarcinoma.



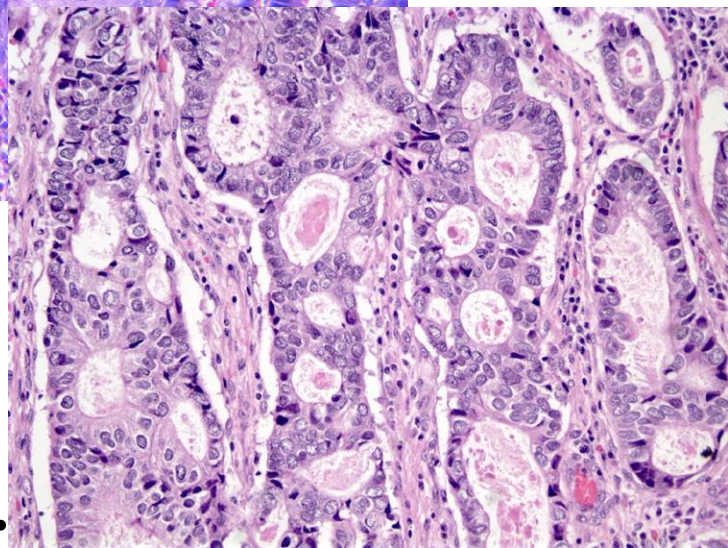


Reflux esophagitis.

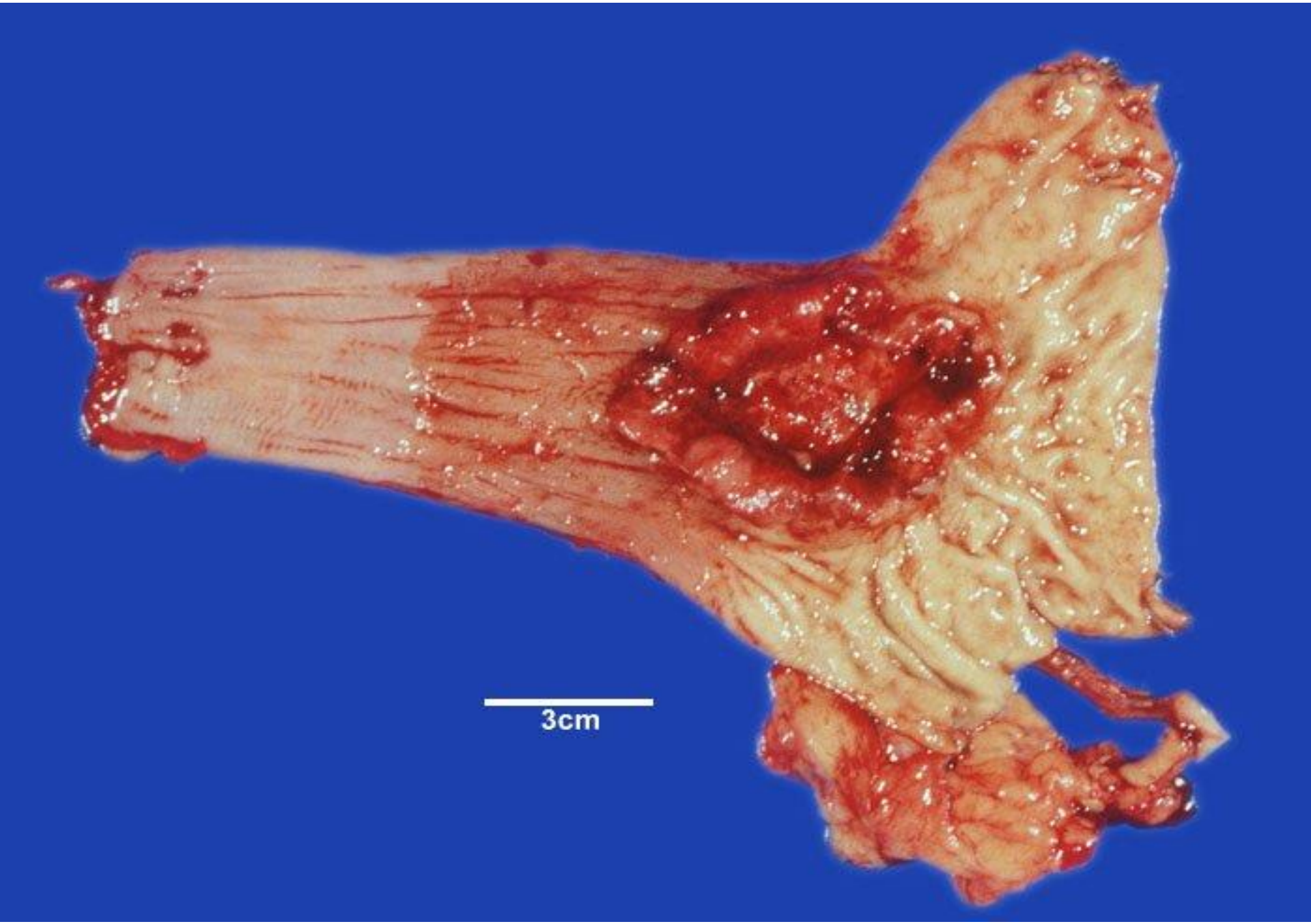


Barrett esophagus (intestinal type).

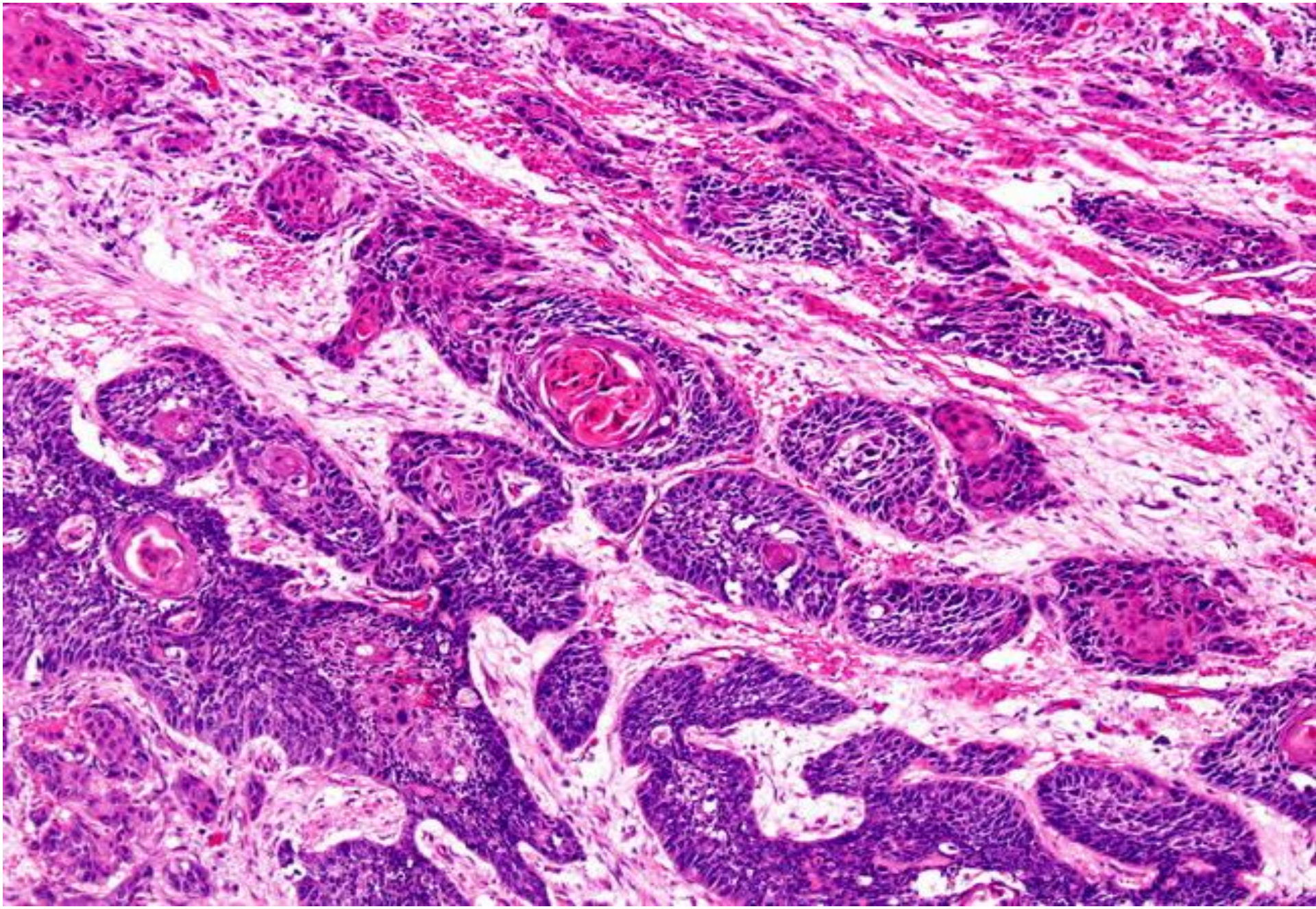
Barrett with dysplasia (carcinoma in-situ).



Invasive adenocarcinoma.

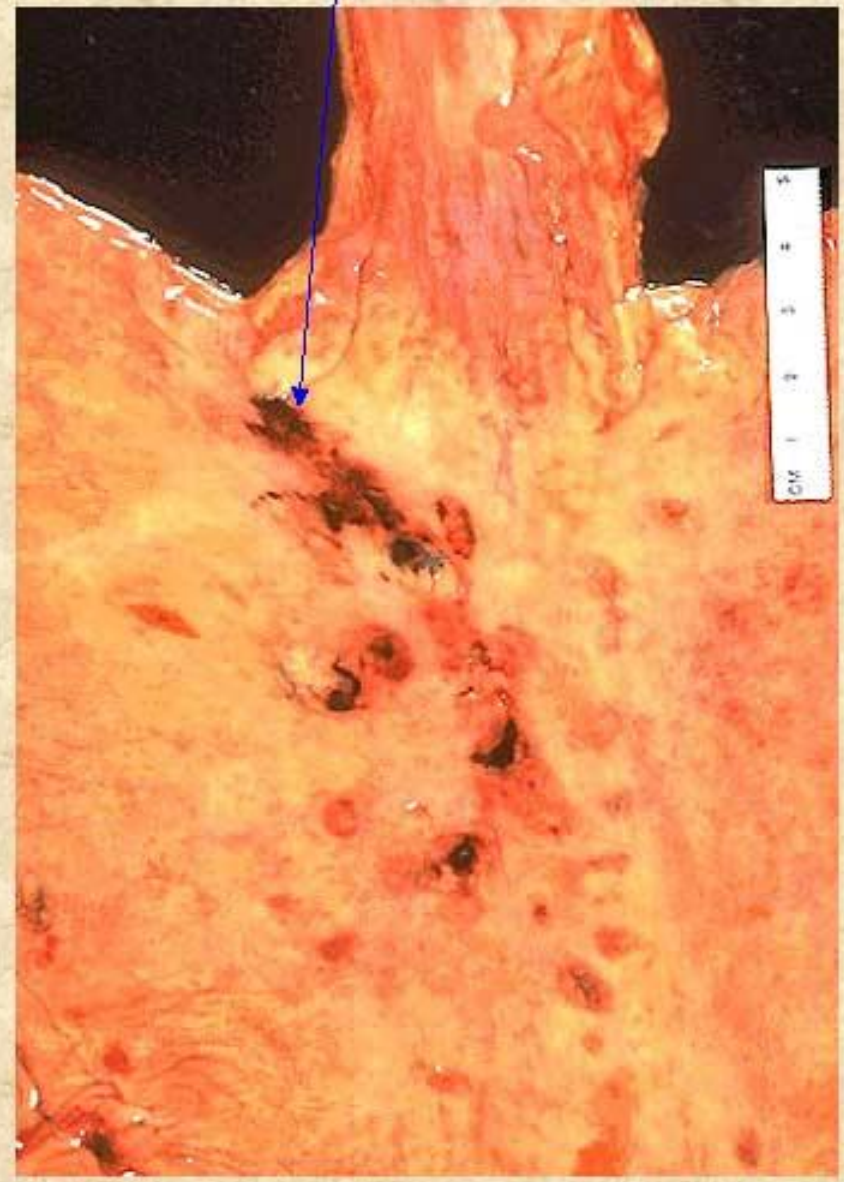


Esophageal adenocarcinoma.



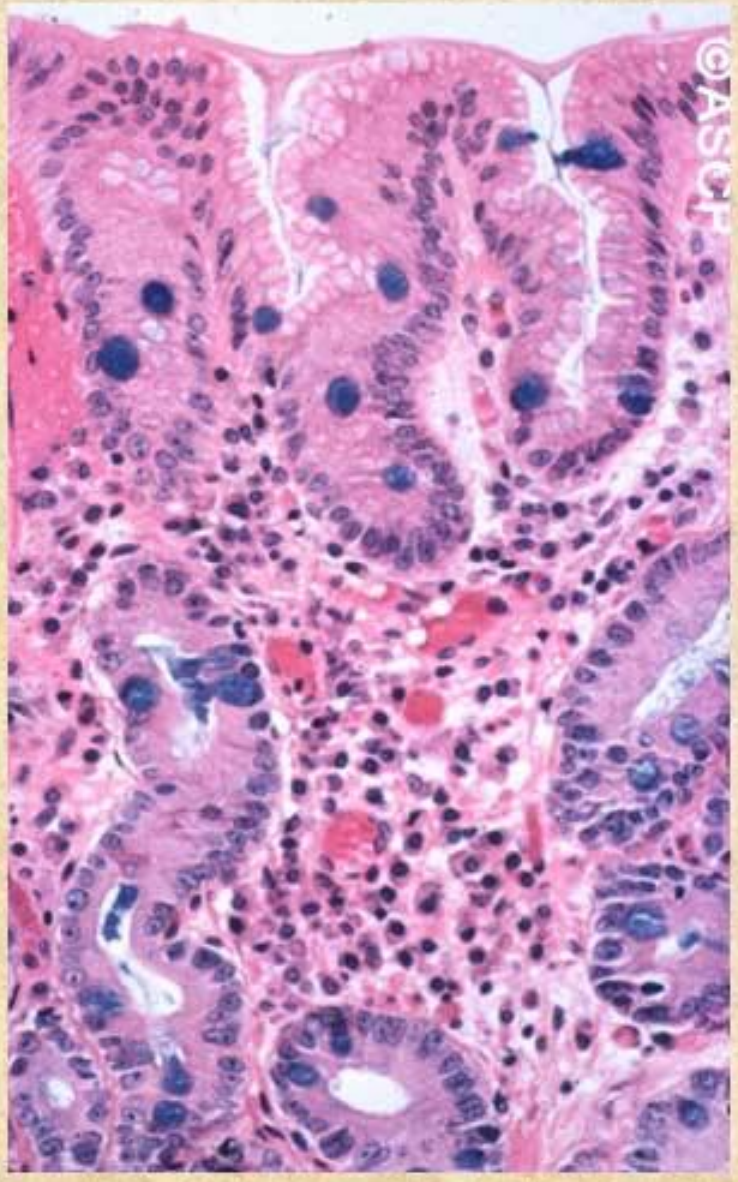
Esophageal keratinizing squamous cell carcinoma.

Acute erosion

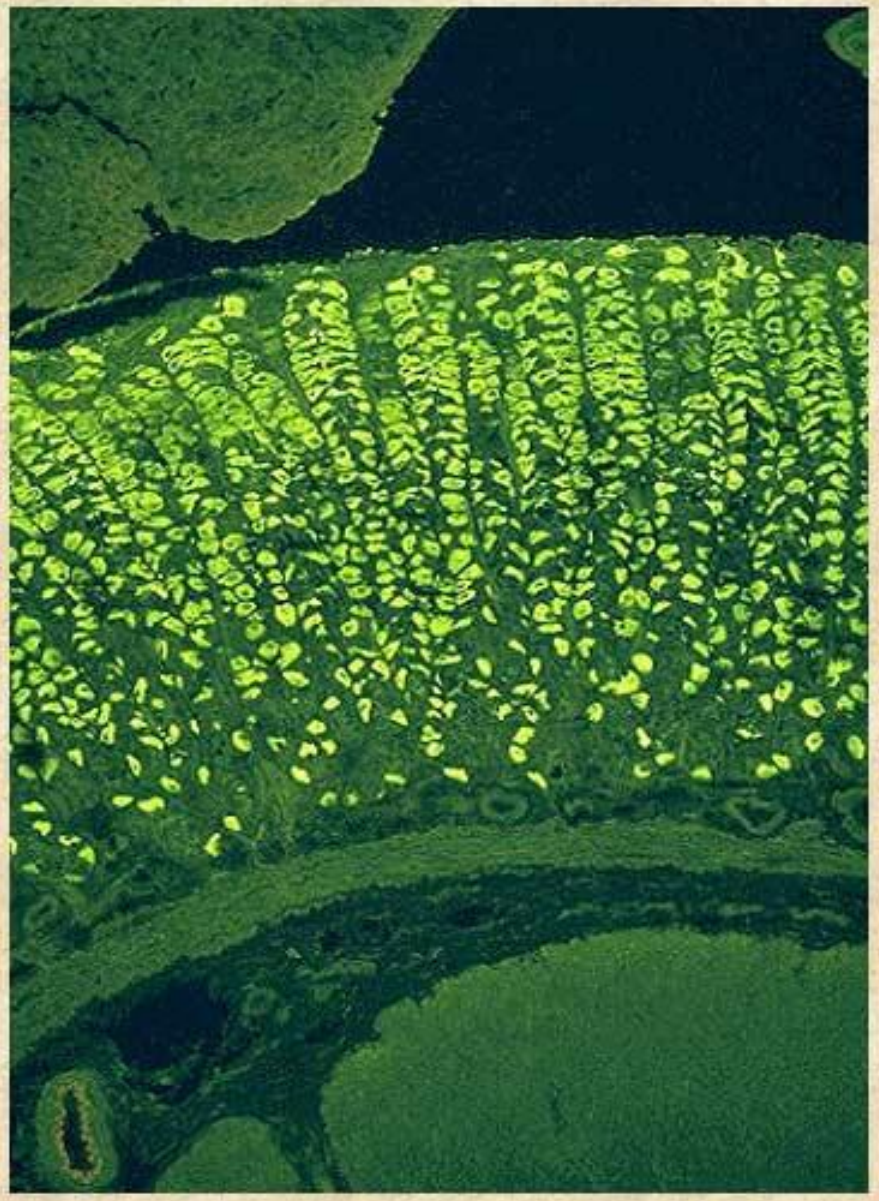


Acute ulcer

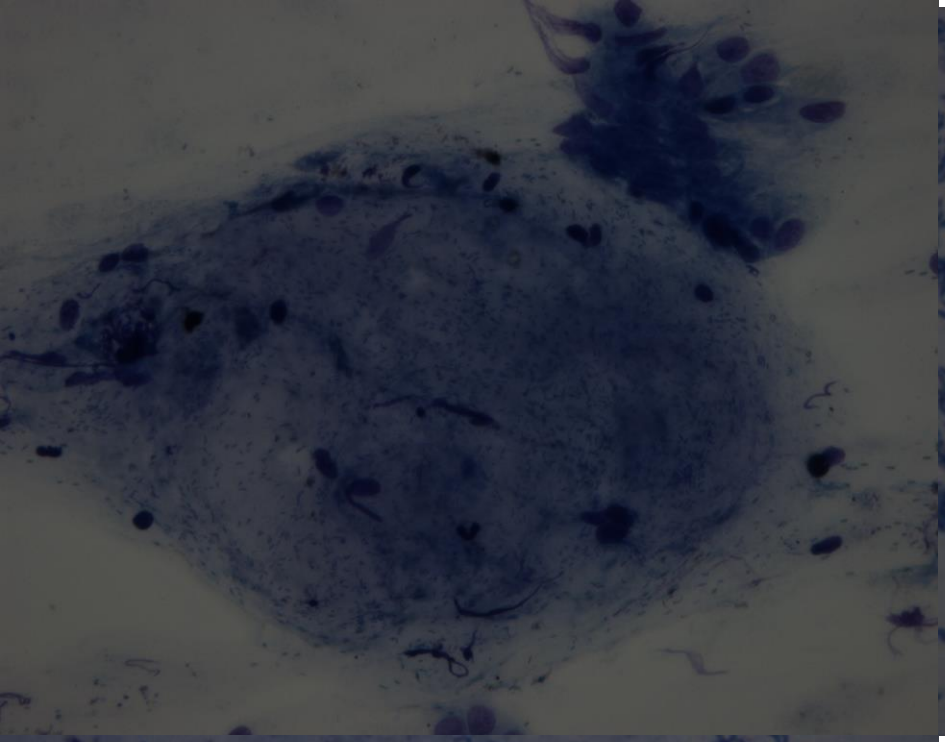
Autoimmune chronic gastritis

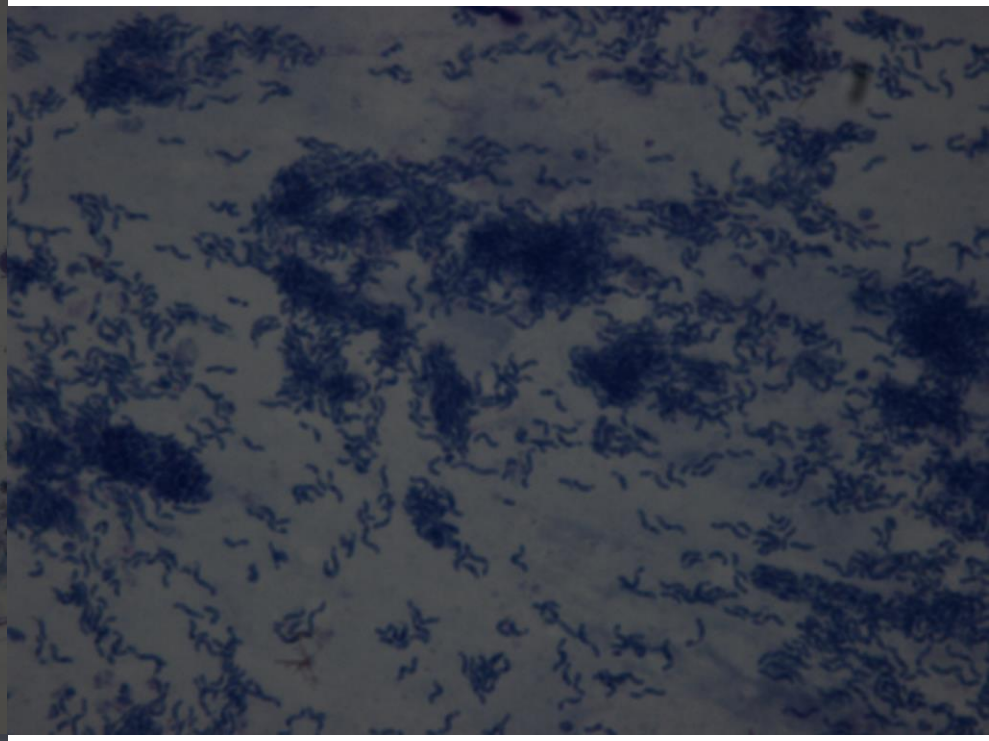


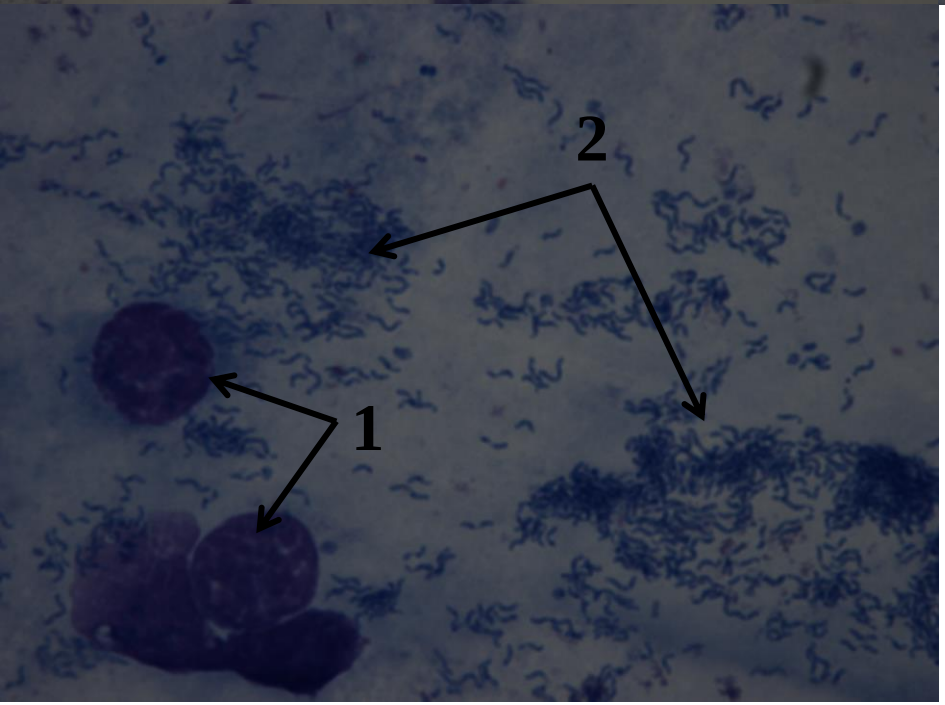
H & E



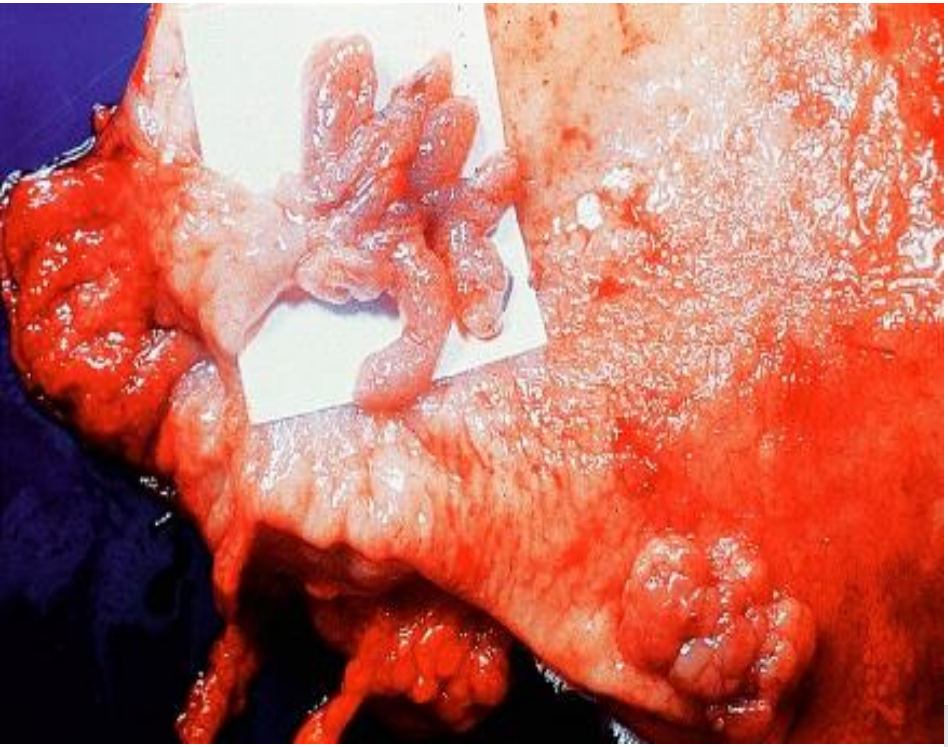
IF Anti-parietal cell Ab



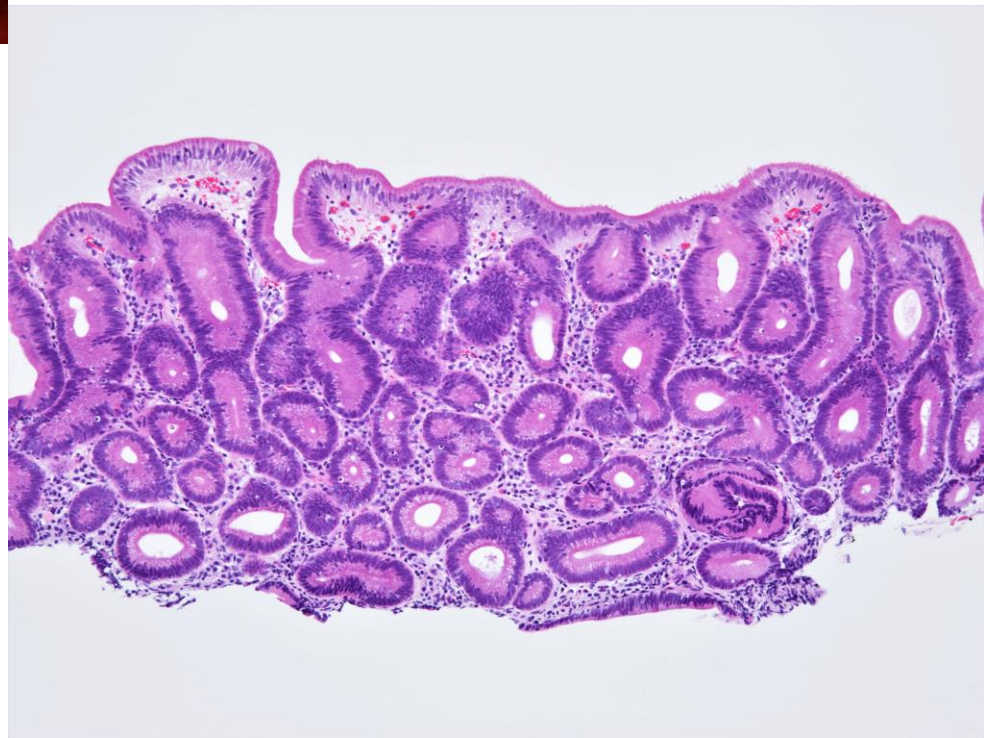


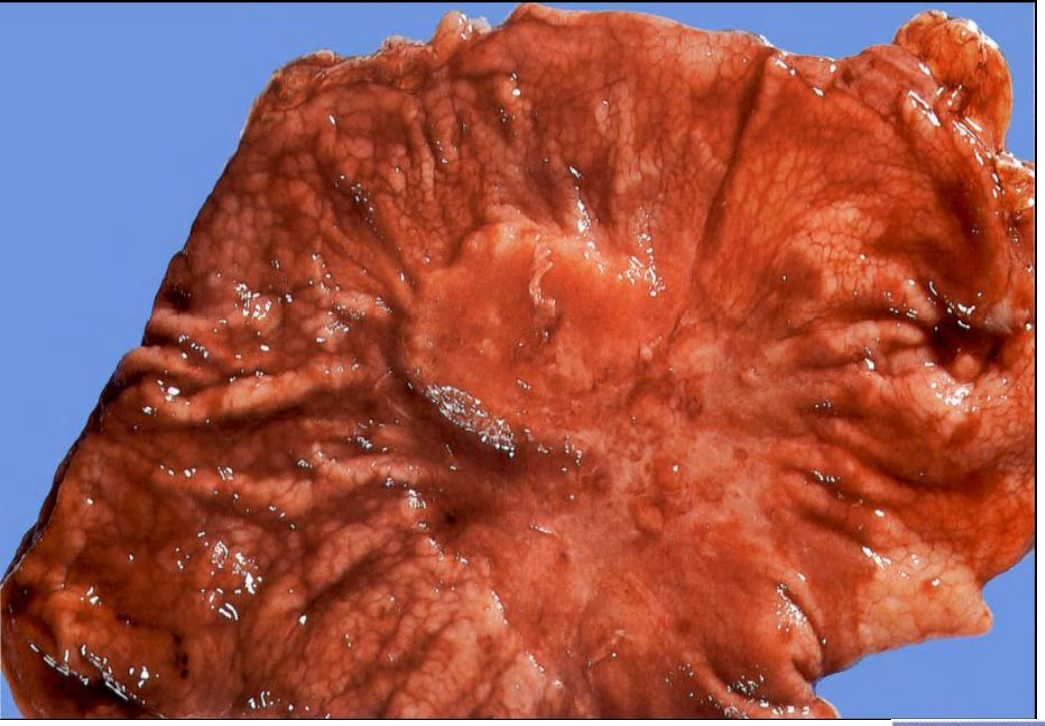


Cytology of the gastric mucosa (smear).
1. Chief cells
2. Helicobacter pylori.



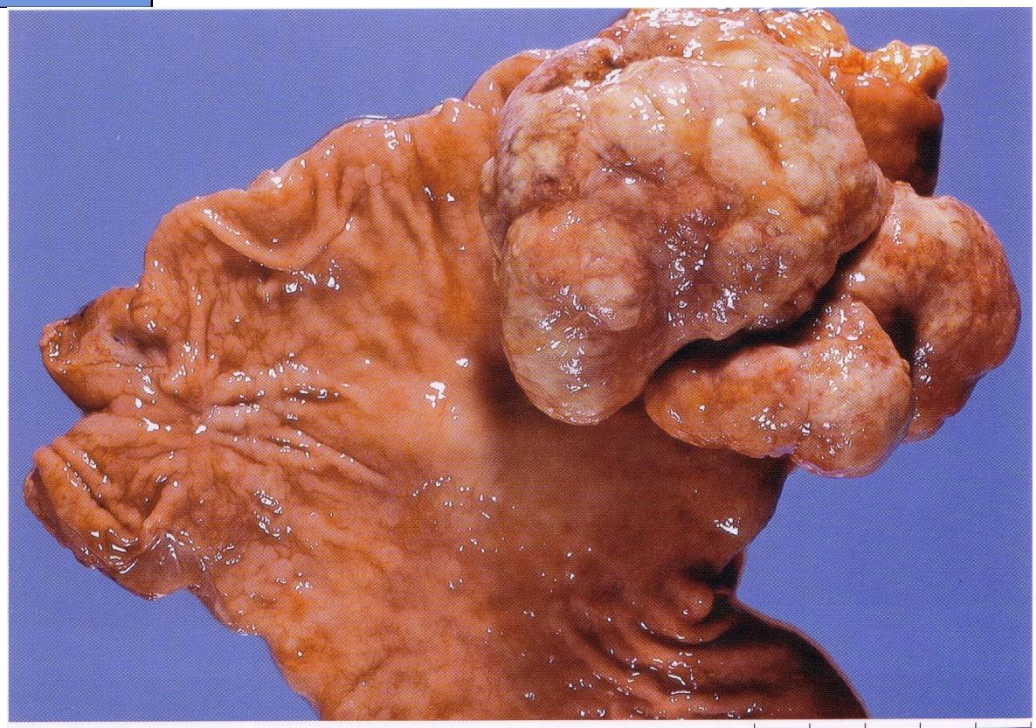
Gastric adenoma.

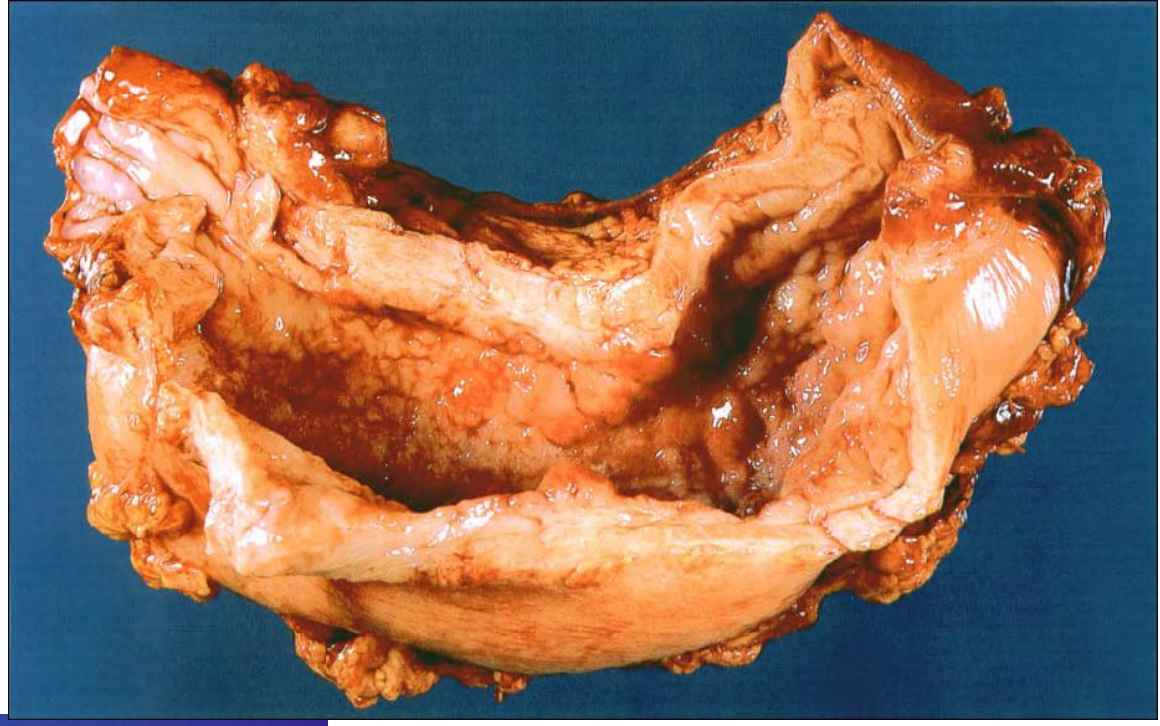




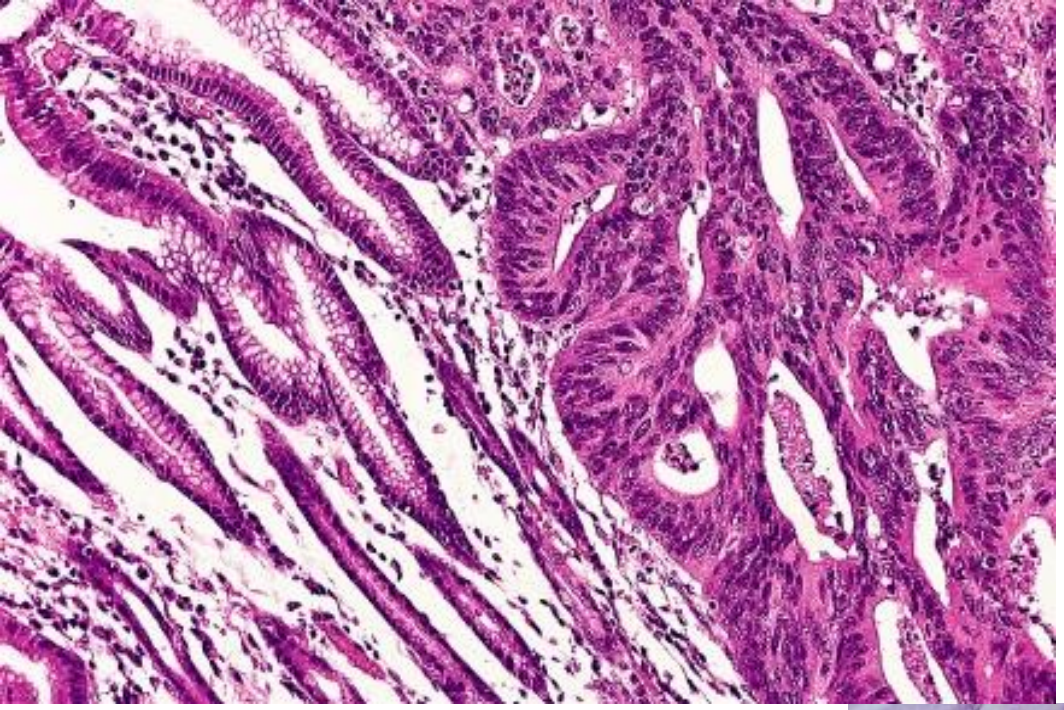
Polypoid gastric carcinoma

Gastric carcinoma plaque-like lesion



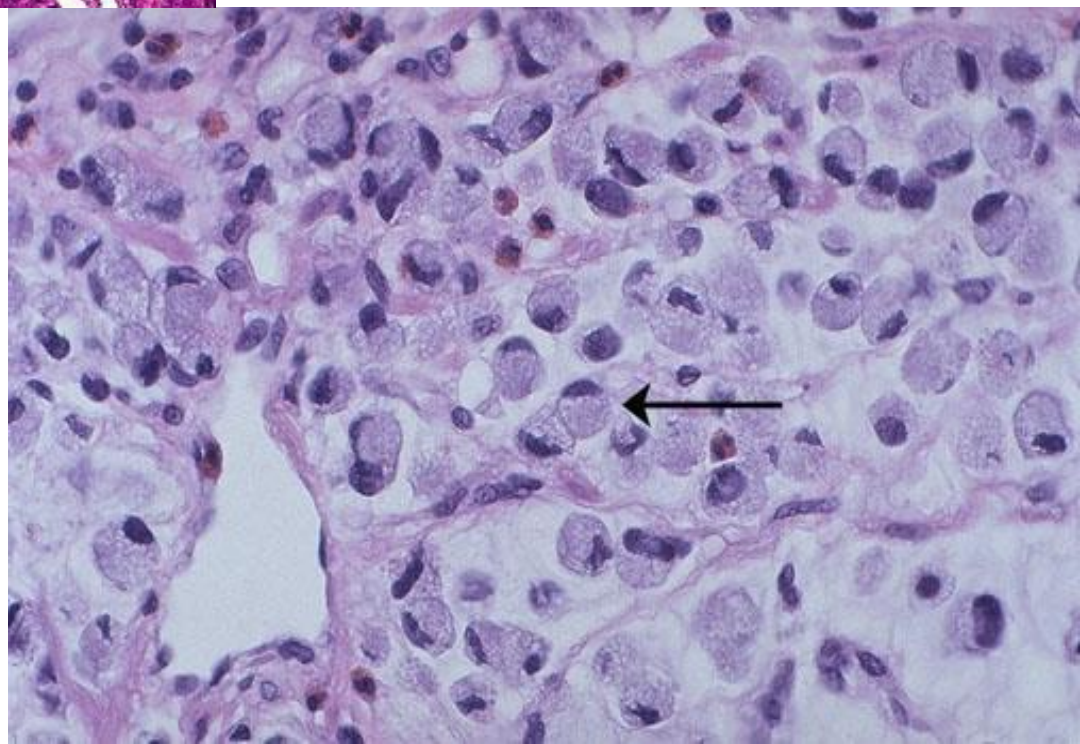


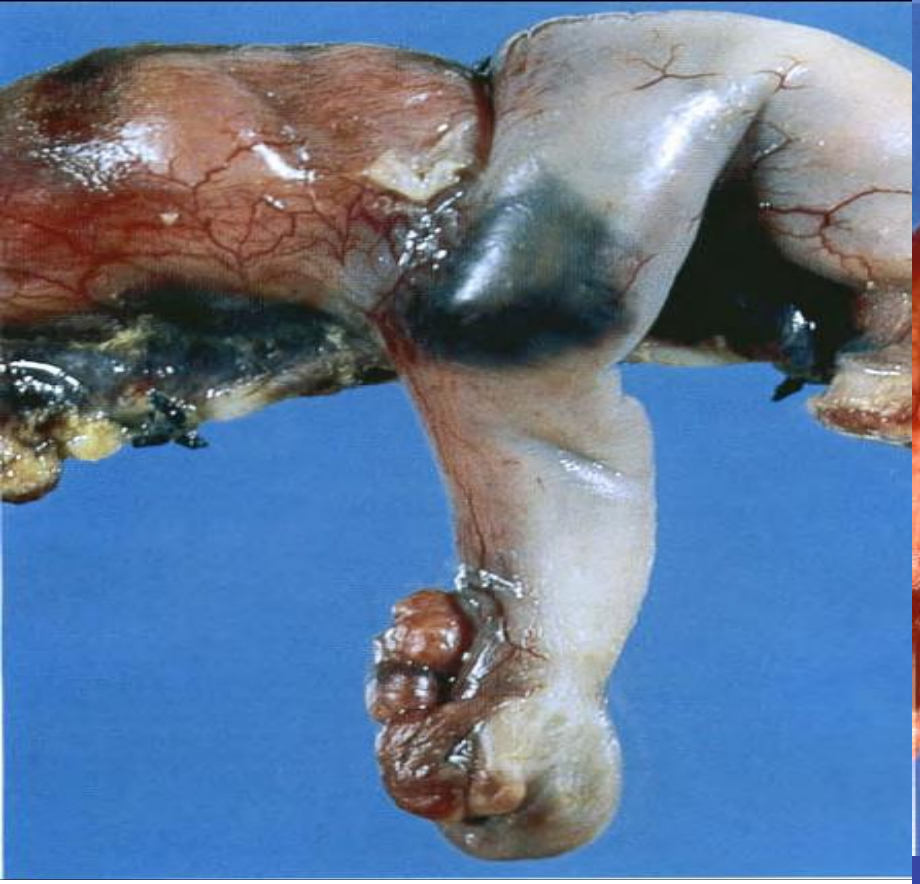
Infiltrative gastric carcinoma.

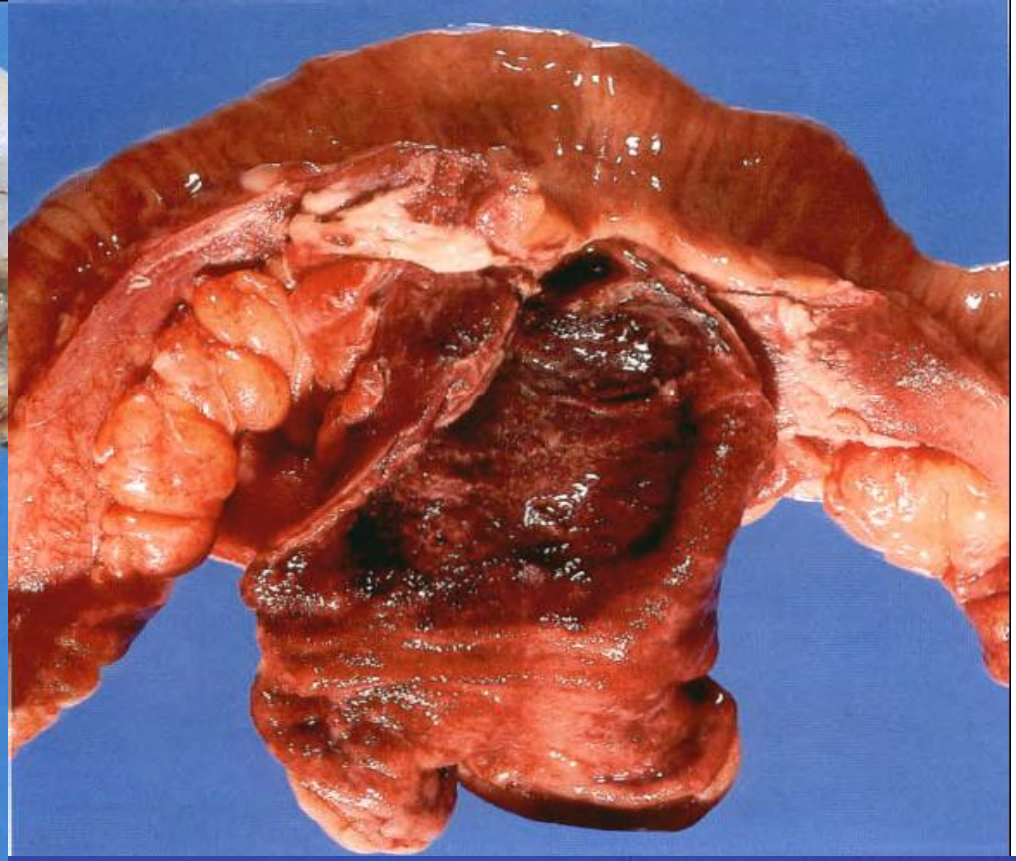


Gastric signet ring cell carcinoma

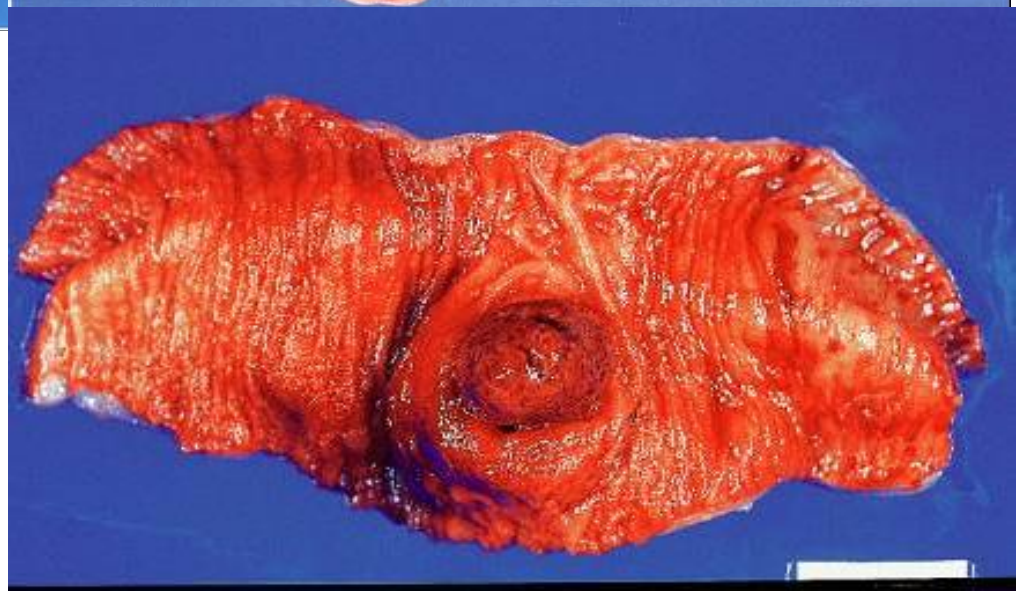
Gastric papillary adenocarcinoma.

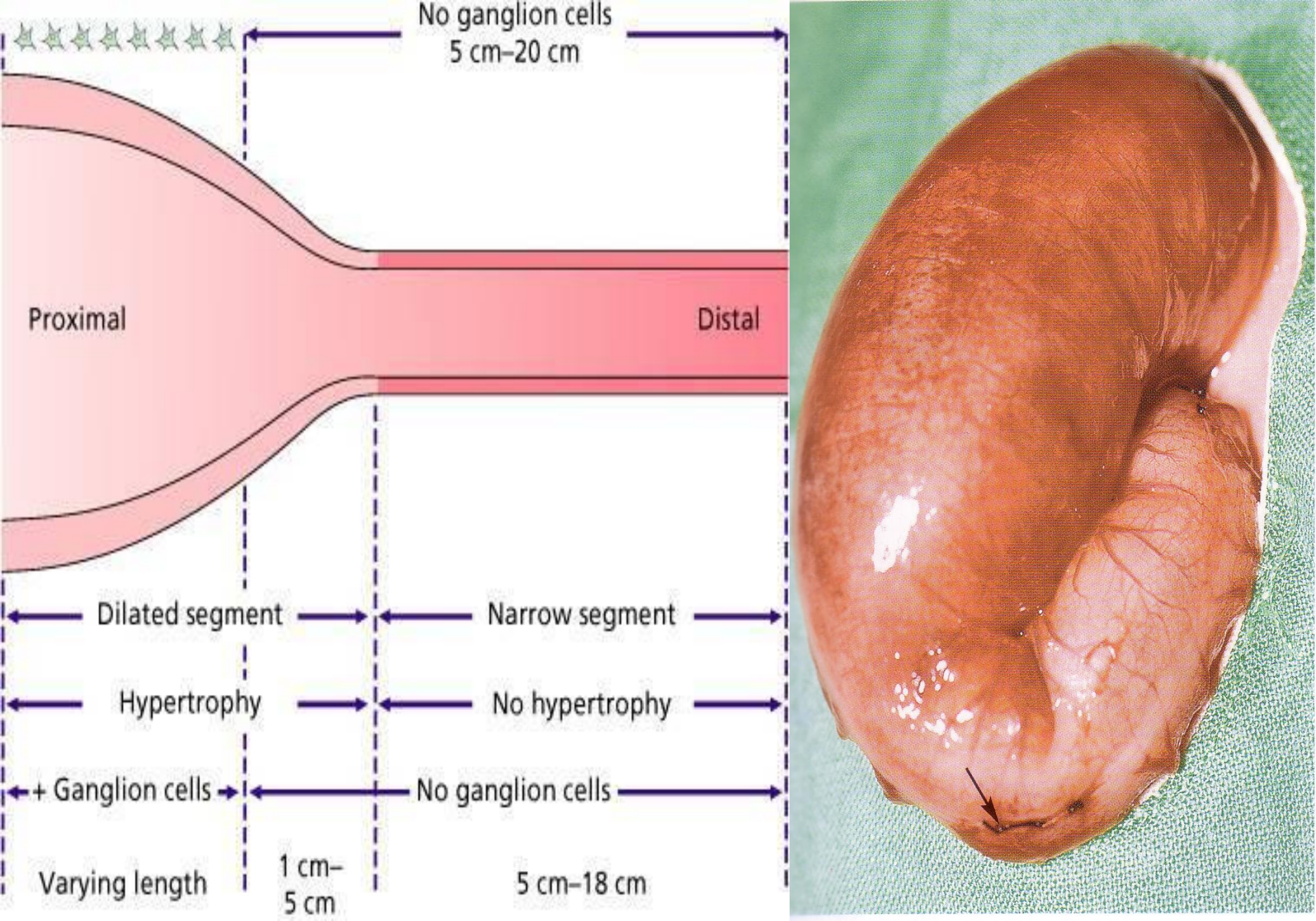




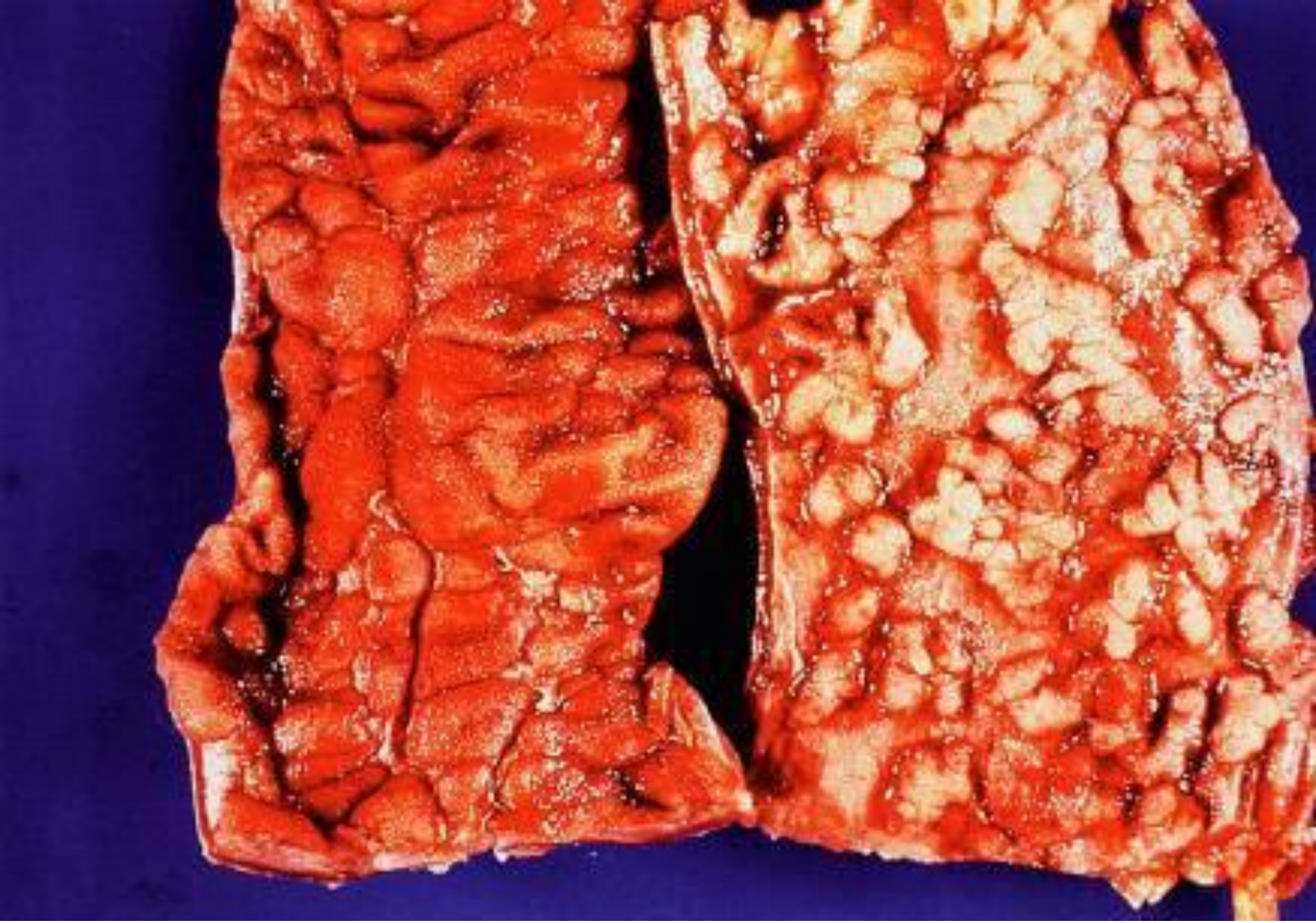


Meckel's diverticulum.

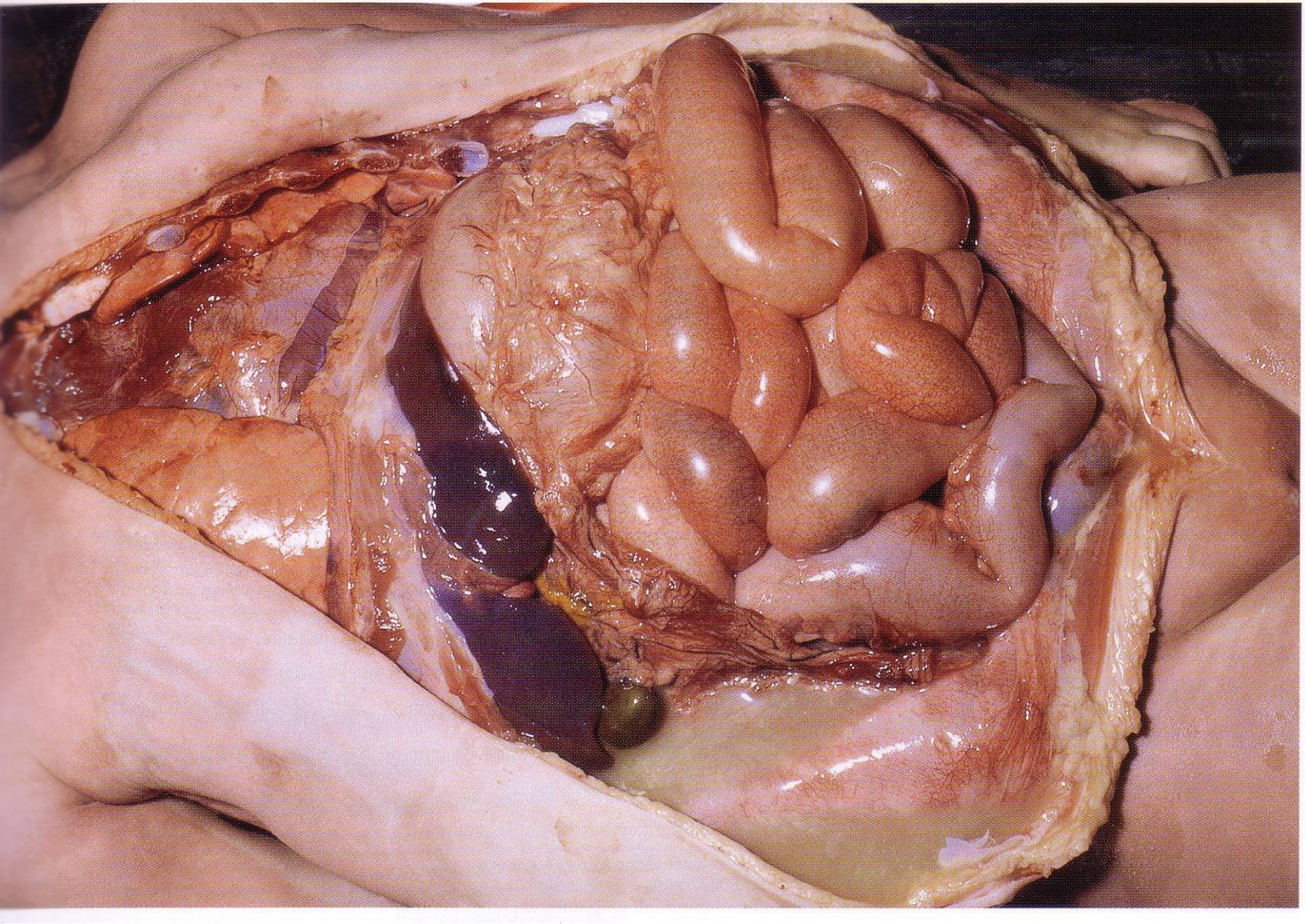




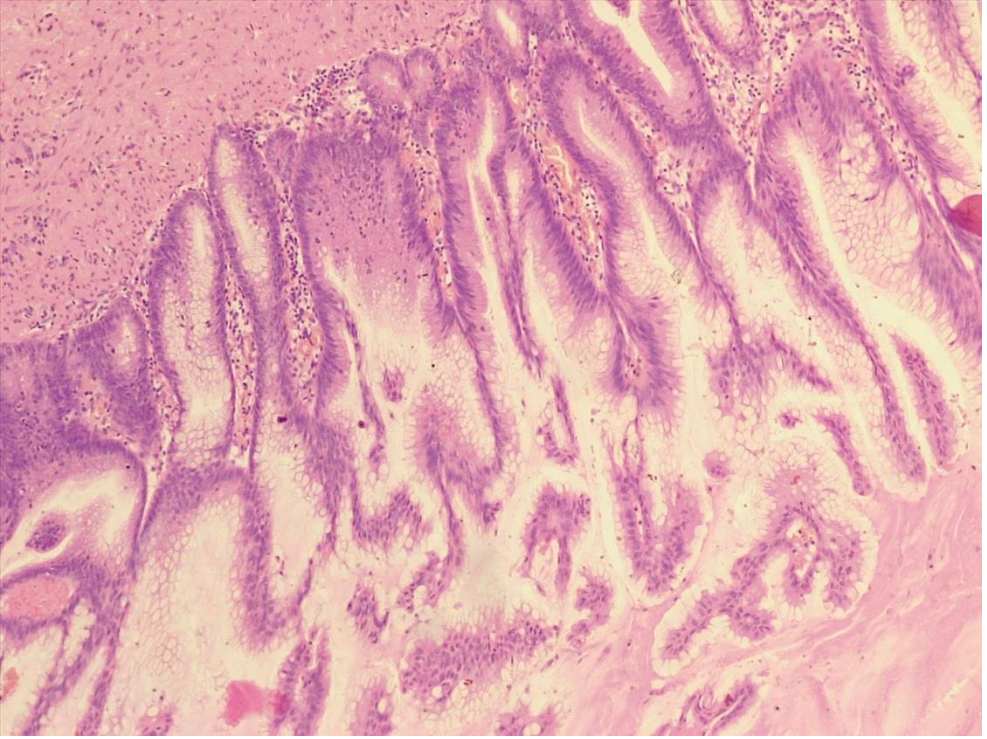
Scheme of large bowel involvement in Hirschprung's disease.



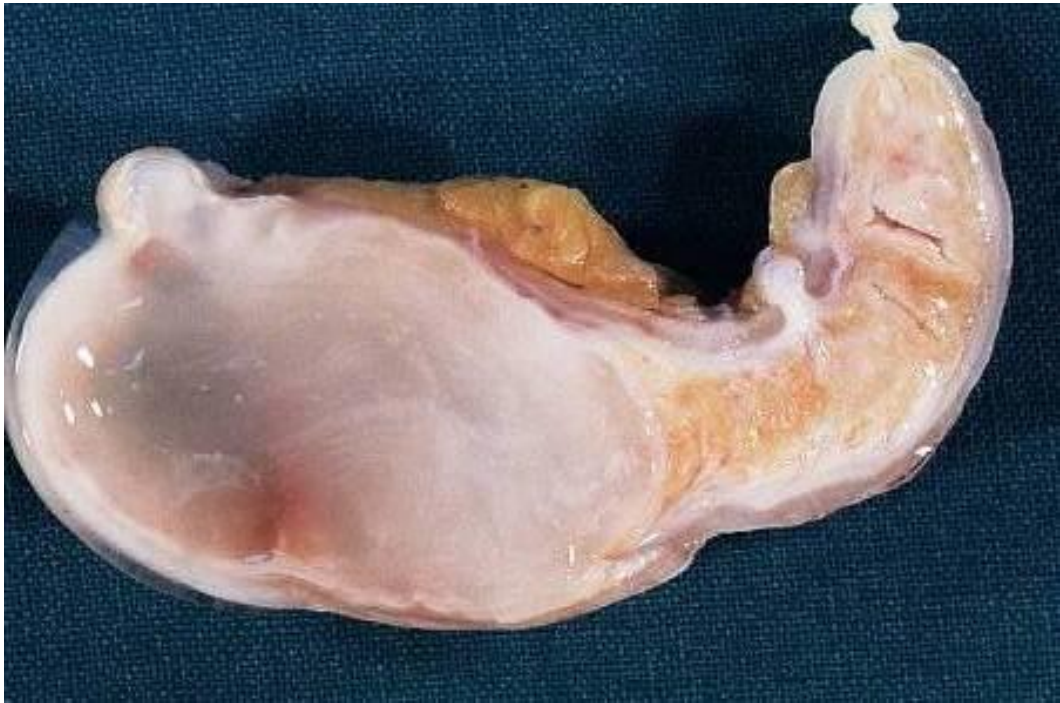
Crohn's disease vs. Nonspecific ulcerative colitis.

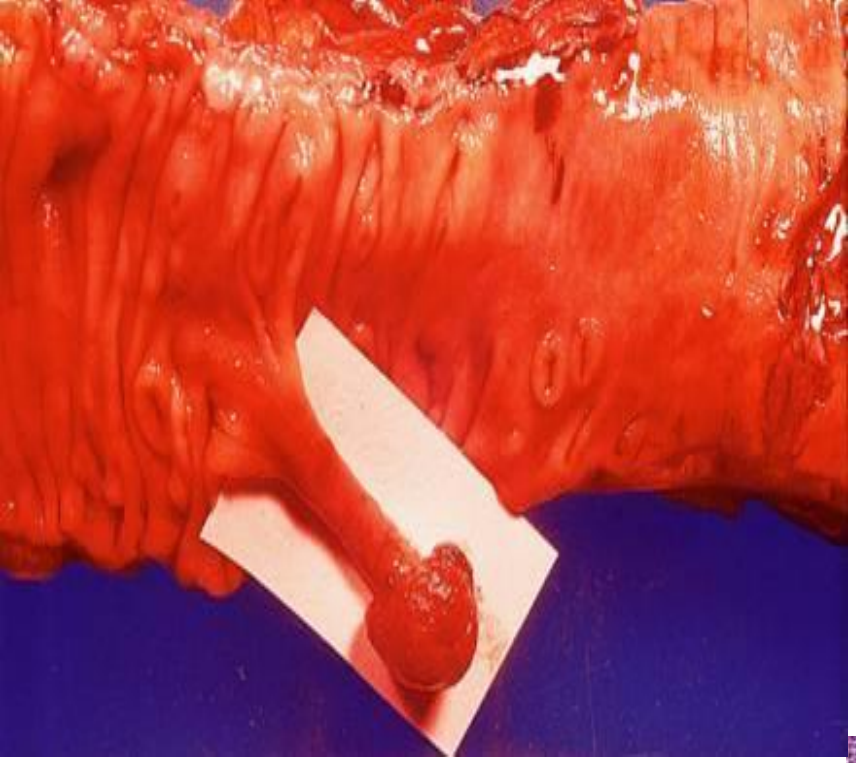


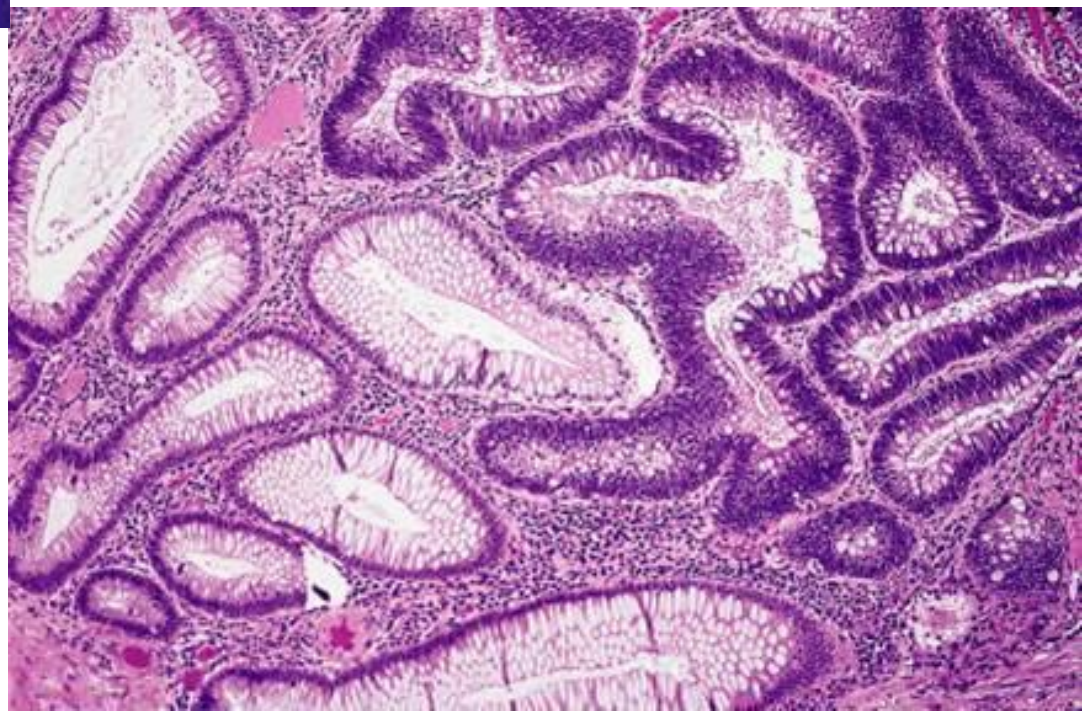
Purulent peritonitis - complication of acute appendicitis.



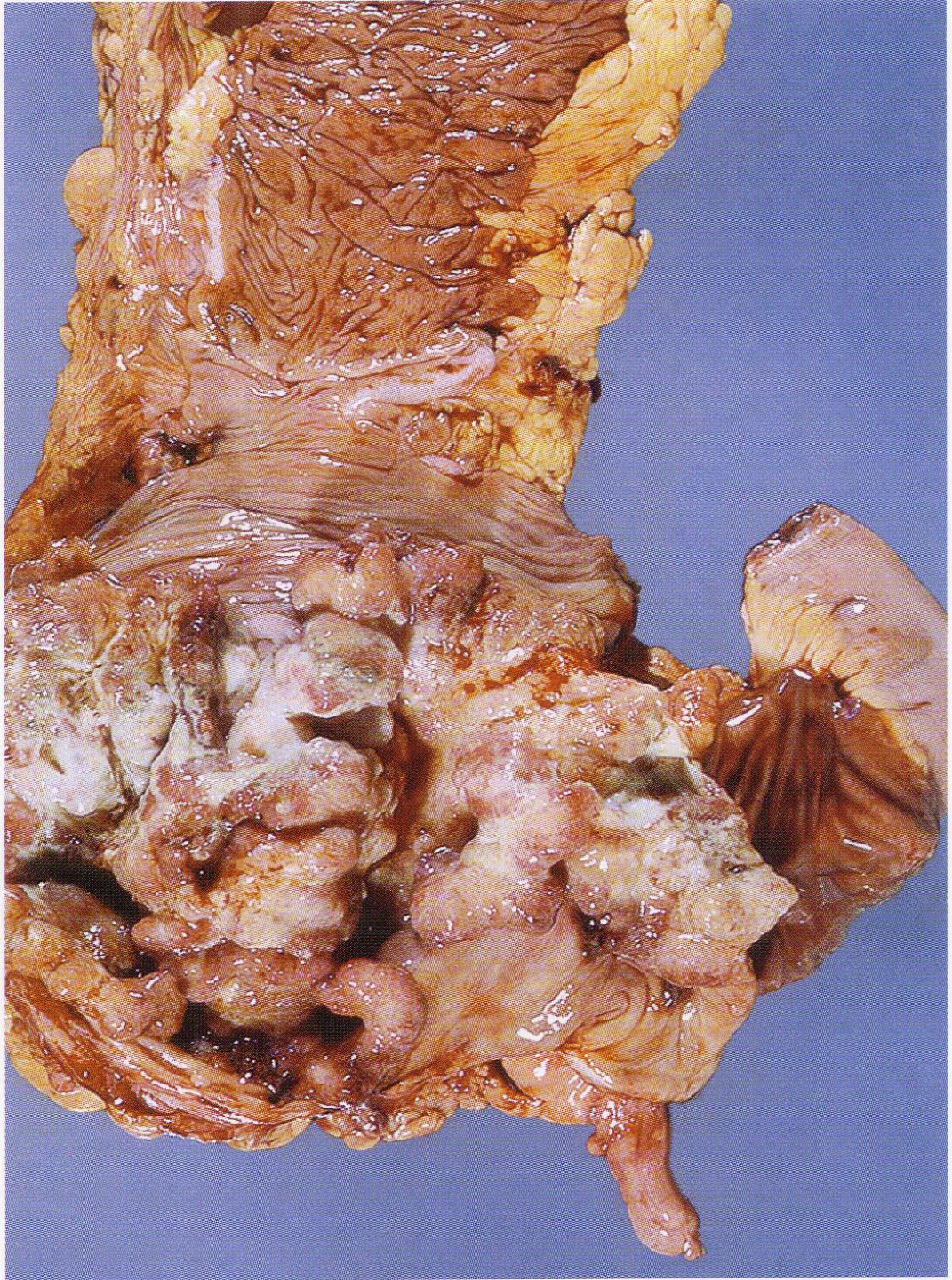
Mucinous appendiceal neoplasms.

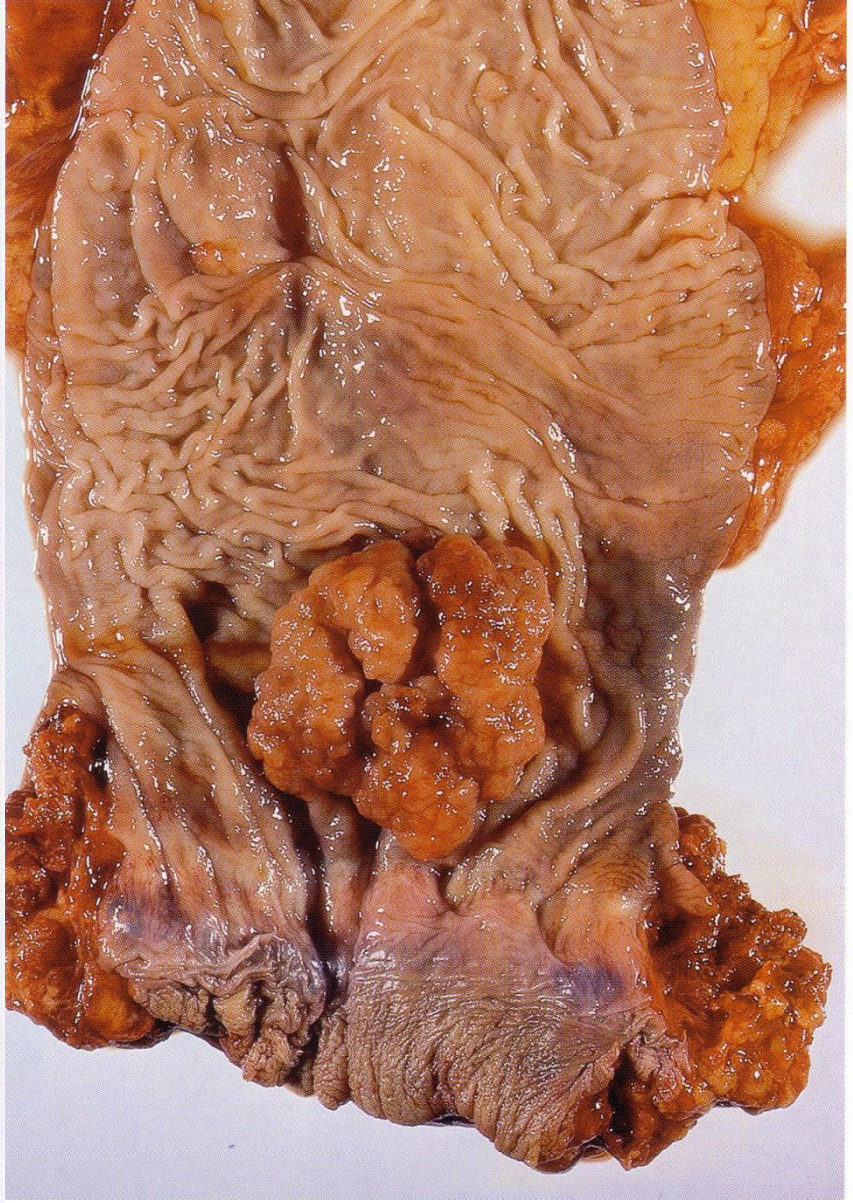






Tubulo-villous adenoma of the colon.

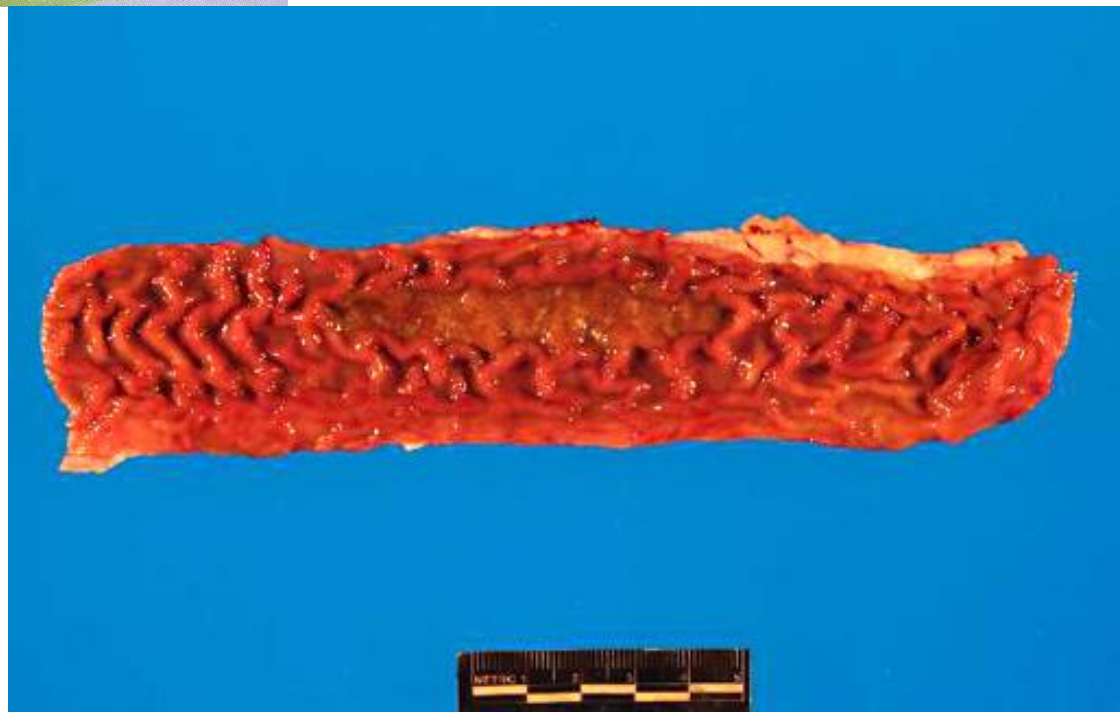




Carcinoma of the cecum and rectum

Peyer's patches necrosis in typhoid fever

Encephaloid modifications of Peyer's patches in typhoid fever.



The stage of ulcerations in typhoid fever.

ESOPHAGUS

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

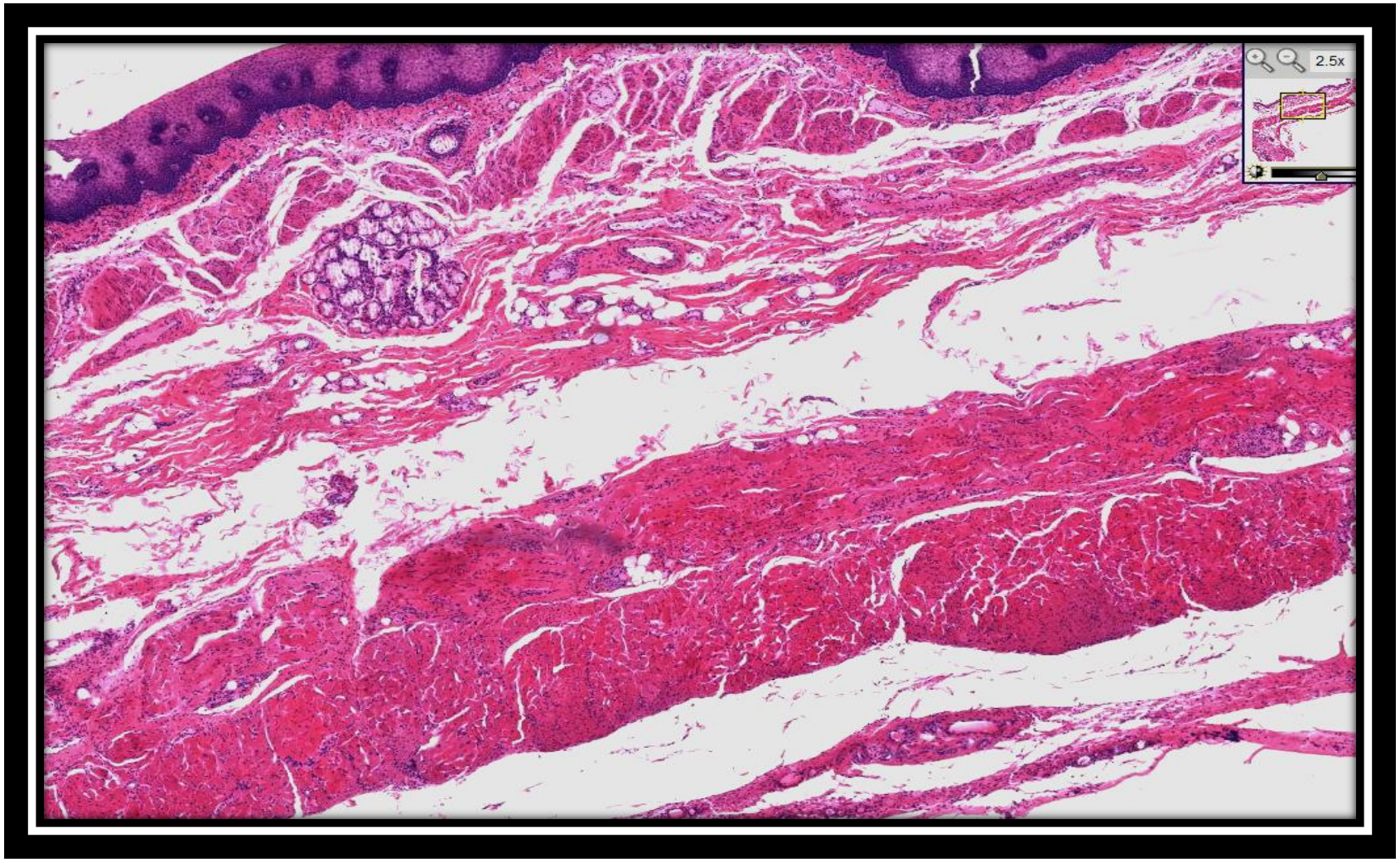
ANATOMY

25 cm.

UES/LES

Mucosa/Submucosa/Muscularis/Adventitia*

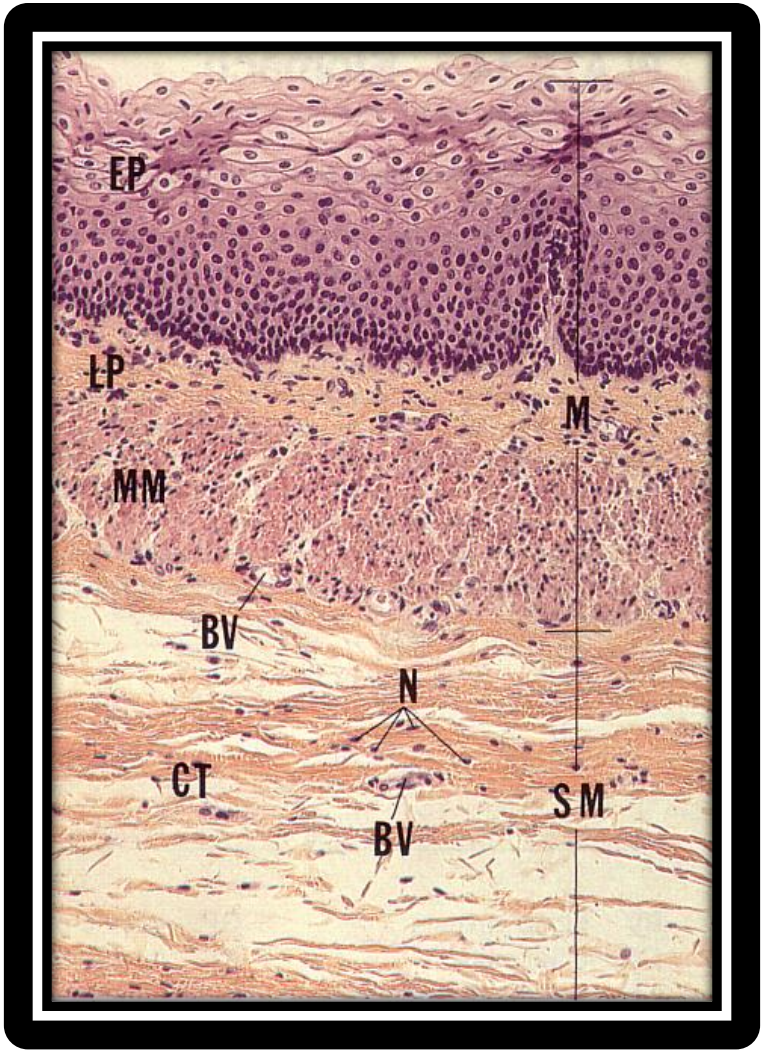
Histologic structure of esophagus



DEFINITIONS

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

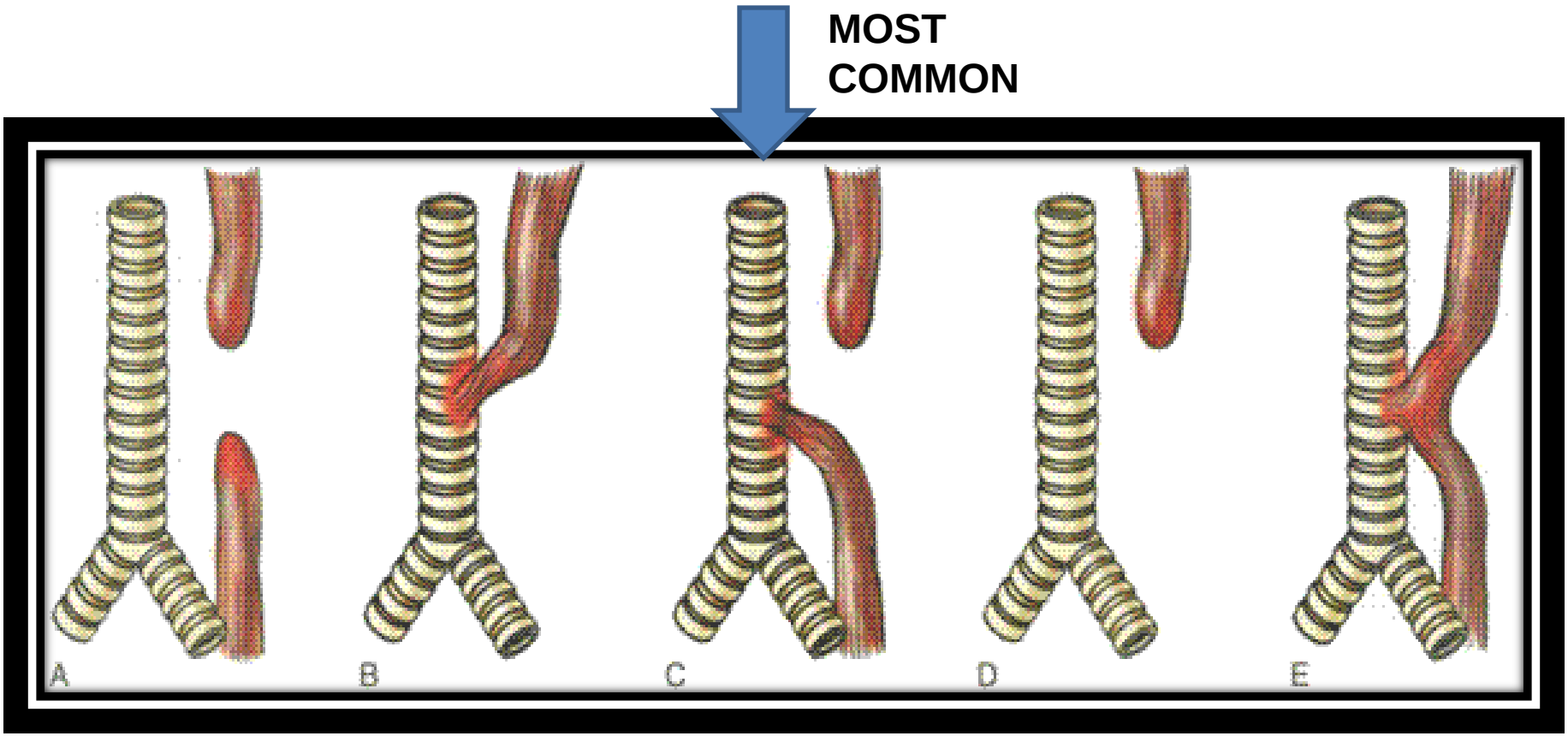
Esophagus Pathology

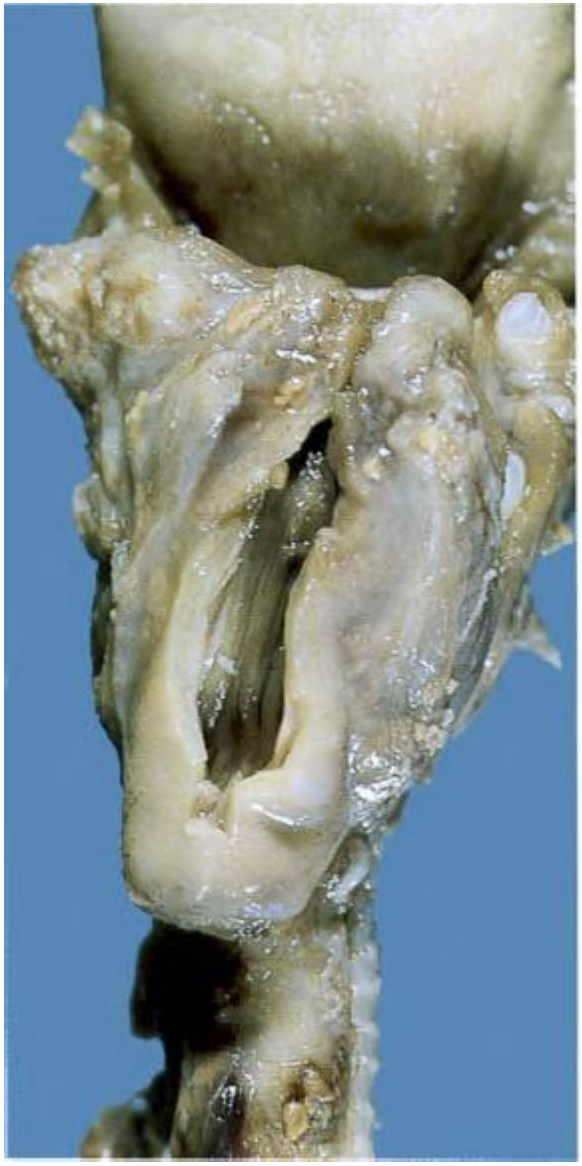
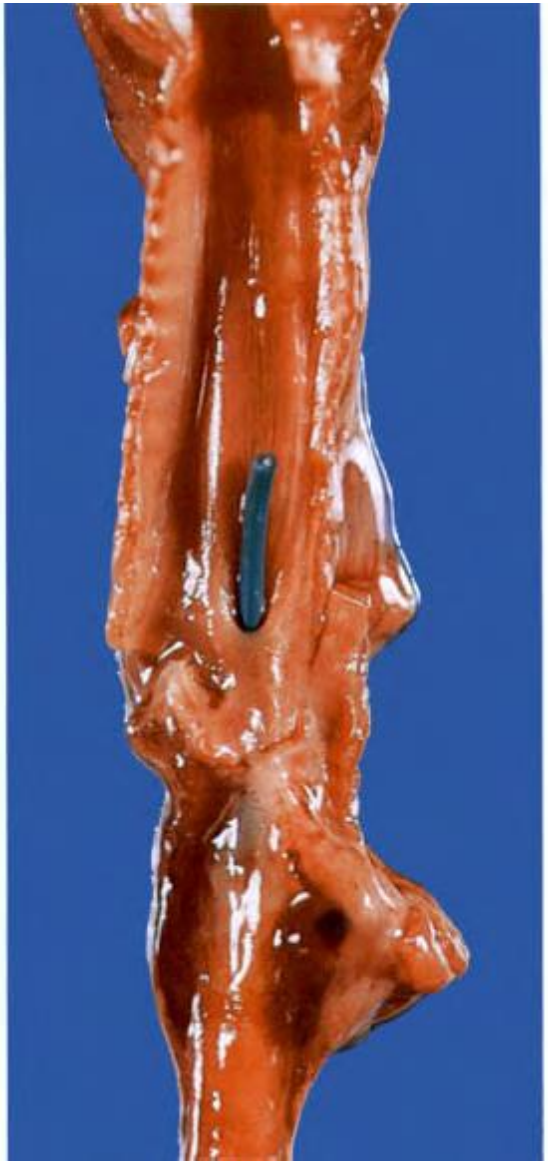
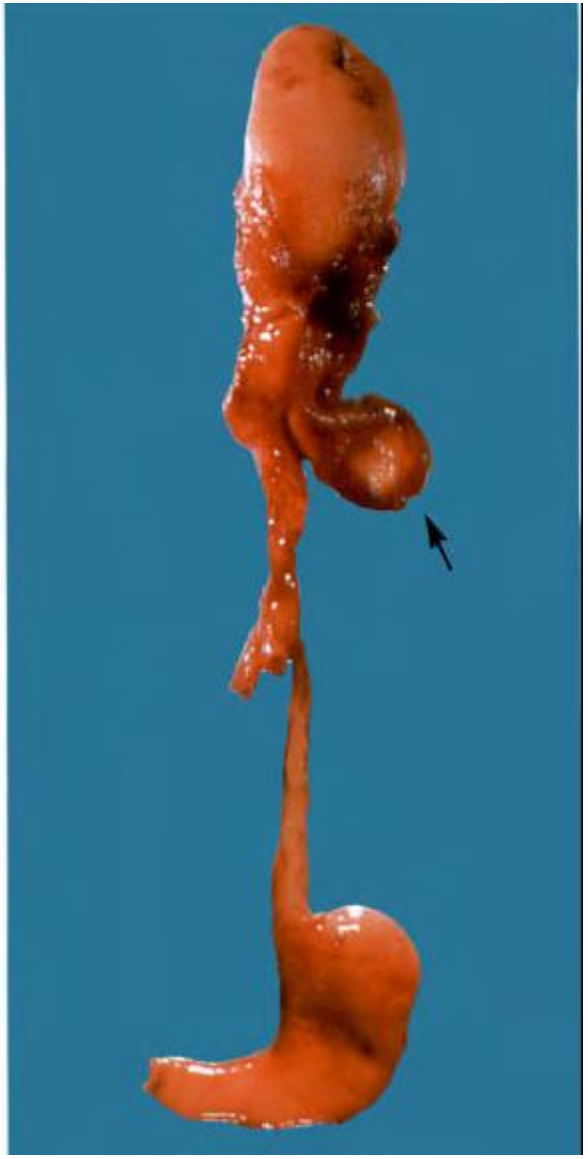


- **Esophageal motor disorders**
  - Achalasia
  - Hiatal hernia
  - Mallory-Weiss syndrome
- **Esophagitis**
  - Reflux esophagitis
  - Infectious esophagitis
  - Chemical esophagitis
- **Esophageal varices**
- **Tumors**
  - B – leiomyoma,papiloma,
  - fibroma, lipoma, angioma
  - Malignant
    - Squamous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

CONGENITAL ANOMALIES

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



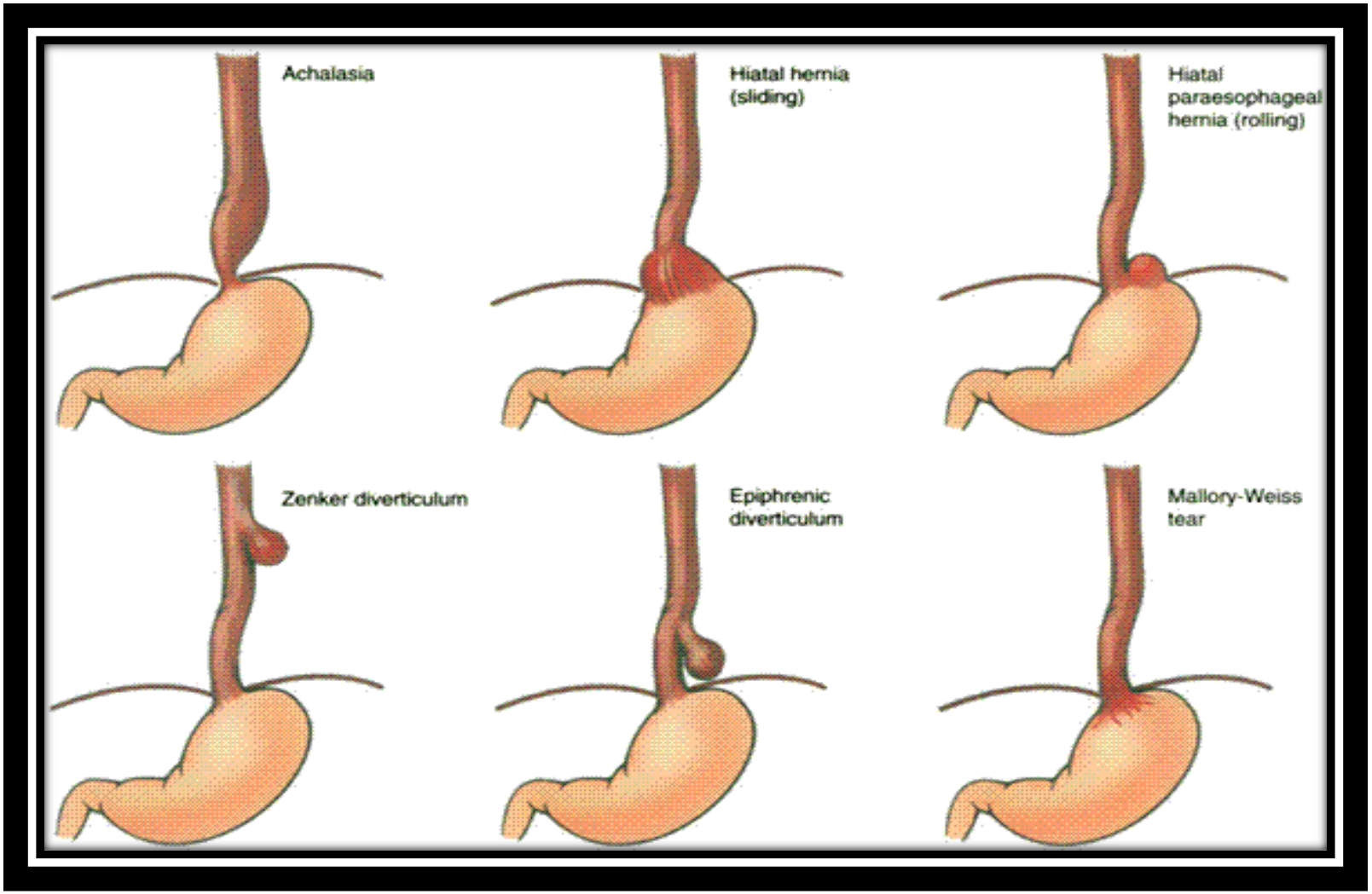
MOTOR DISORDERS

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

Achalasia

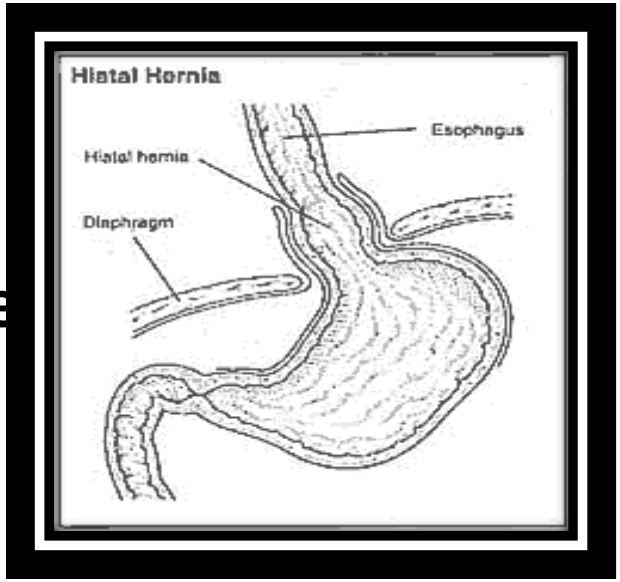
- "Failure to relax"
  - Aperistalsis
  - Incomplete relaxation of the LES
  - Increased LES tone
    - INCREASE: Gastrin, serotonin, acetylcholine, Prostaglandin F2α, motulin, Substance P, histamine, pancreatic polypeptide
  - Progressive dysphagia starting in teens
  - Mostly UNCERTAIN etiology

MOTOR DISORDERS



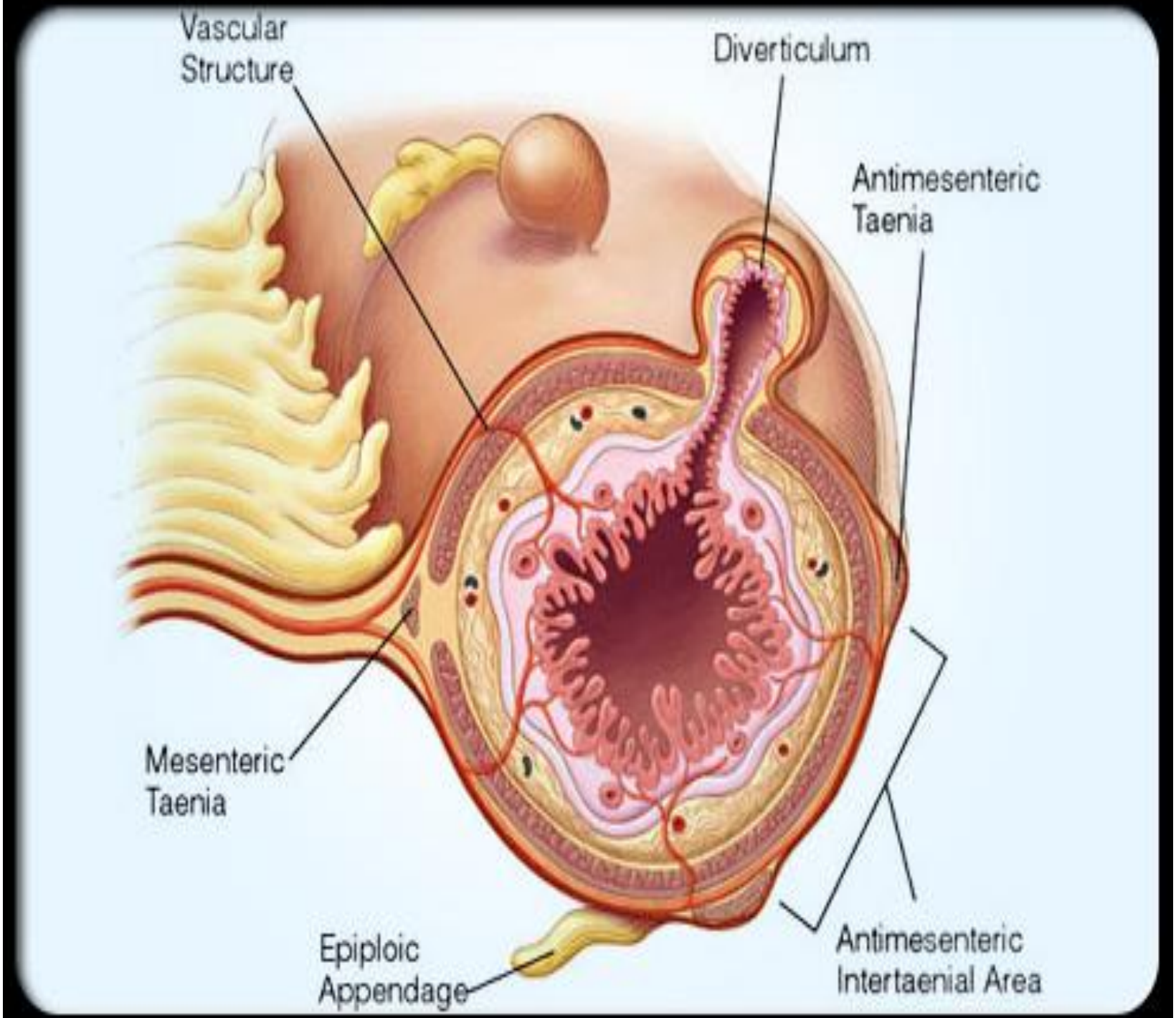
HIATAL HERNIA

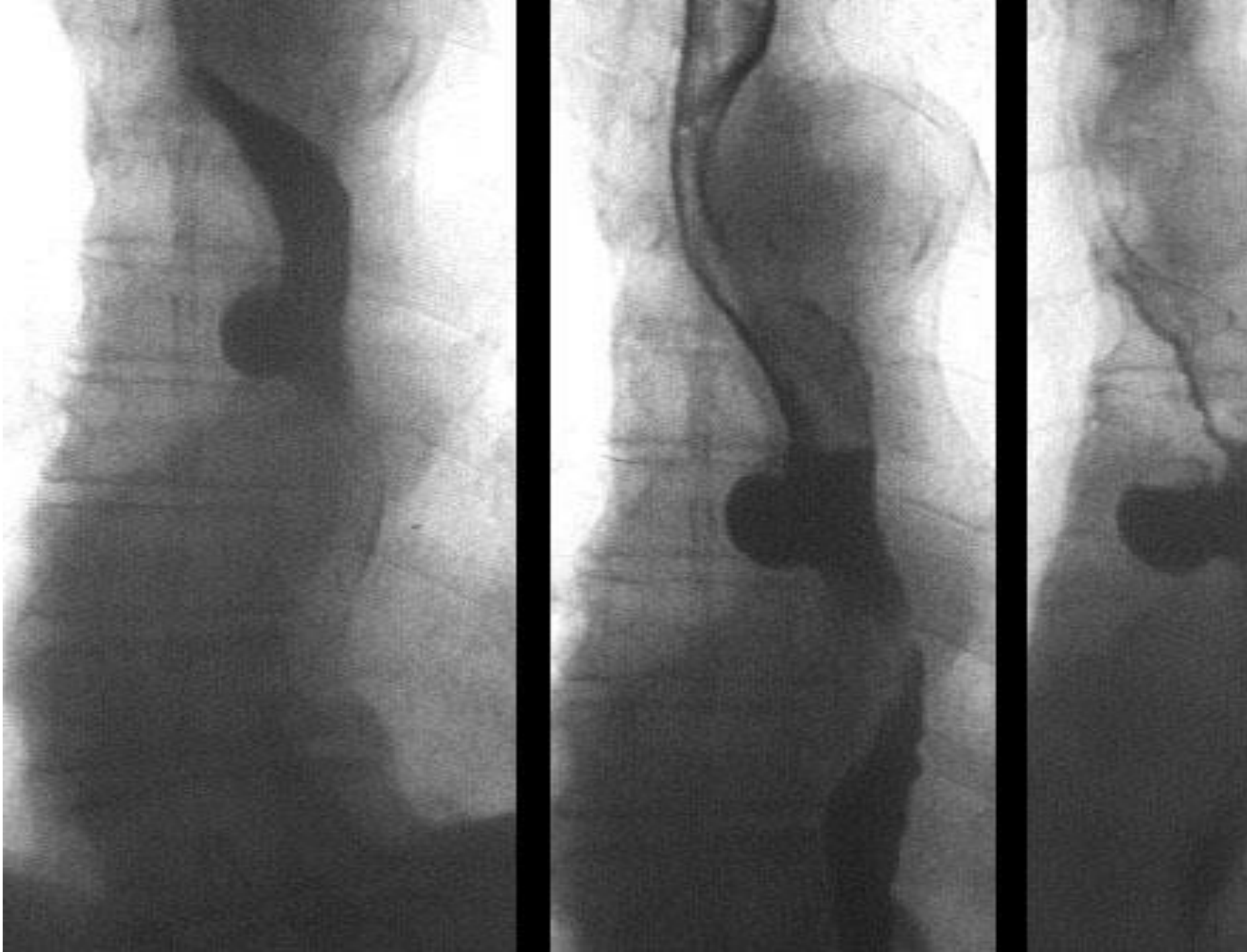
- Diaphragmatic muscular defect
- WIDENING of the space which the lower esophagus passes through
- IN ALL cases, STOMACH above diaphragm
- Usually associated with reflux
- Very common→ Increases with age
- Ulceration, bleeding, perforation, strangulation



DIVERTICULA

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

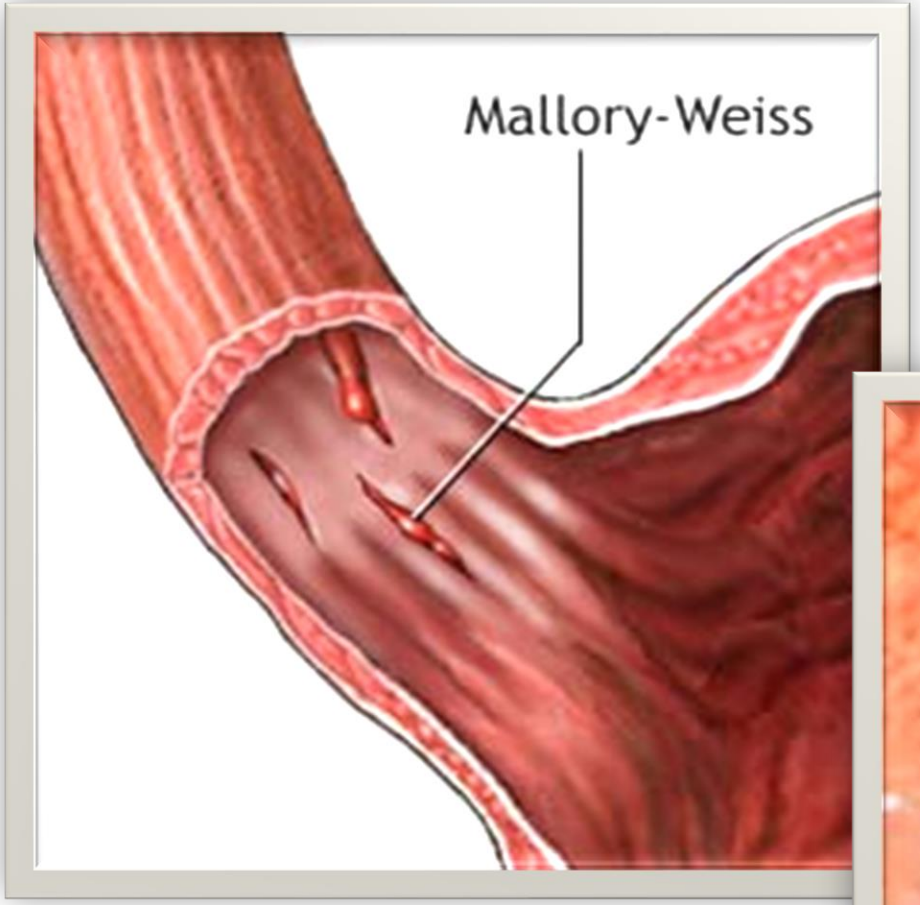

Vascular Structure
Diverticulum
Antimesenteric Taenia
Mesenteric Taenia
Epiploic Appendage
Antimesenteric Intertaenial Area

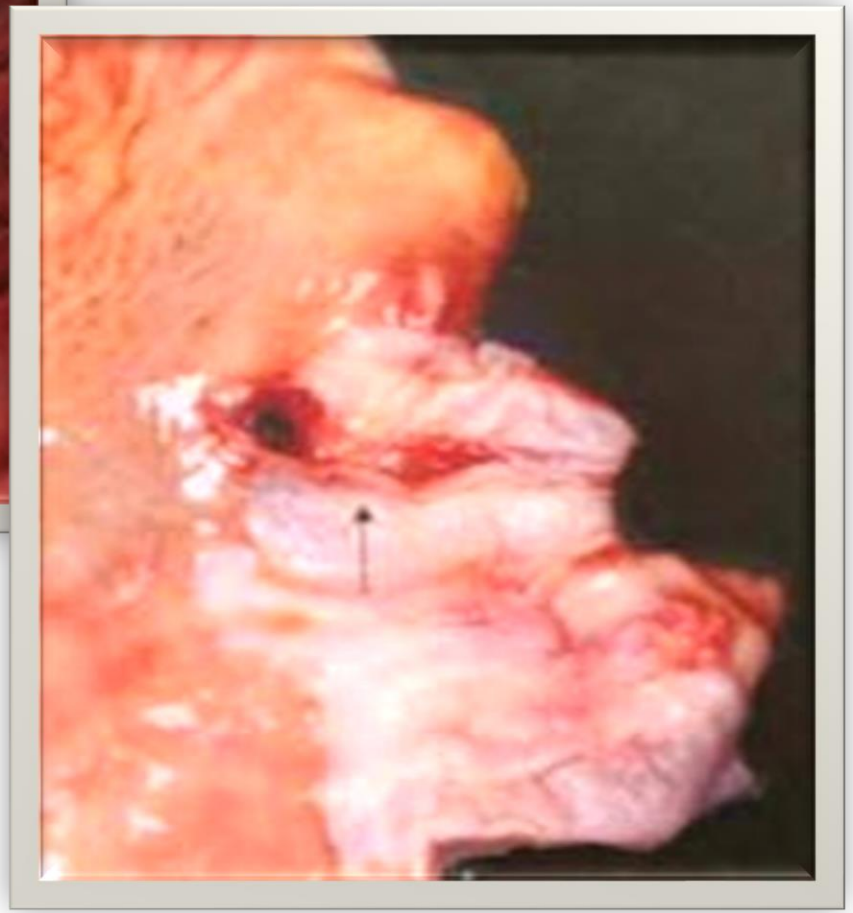


LACERATION

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

MALLORY-WEISS

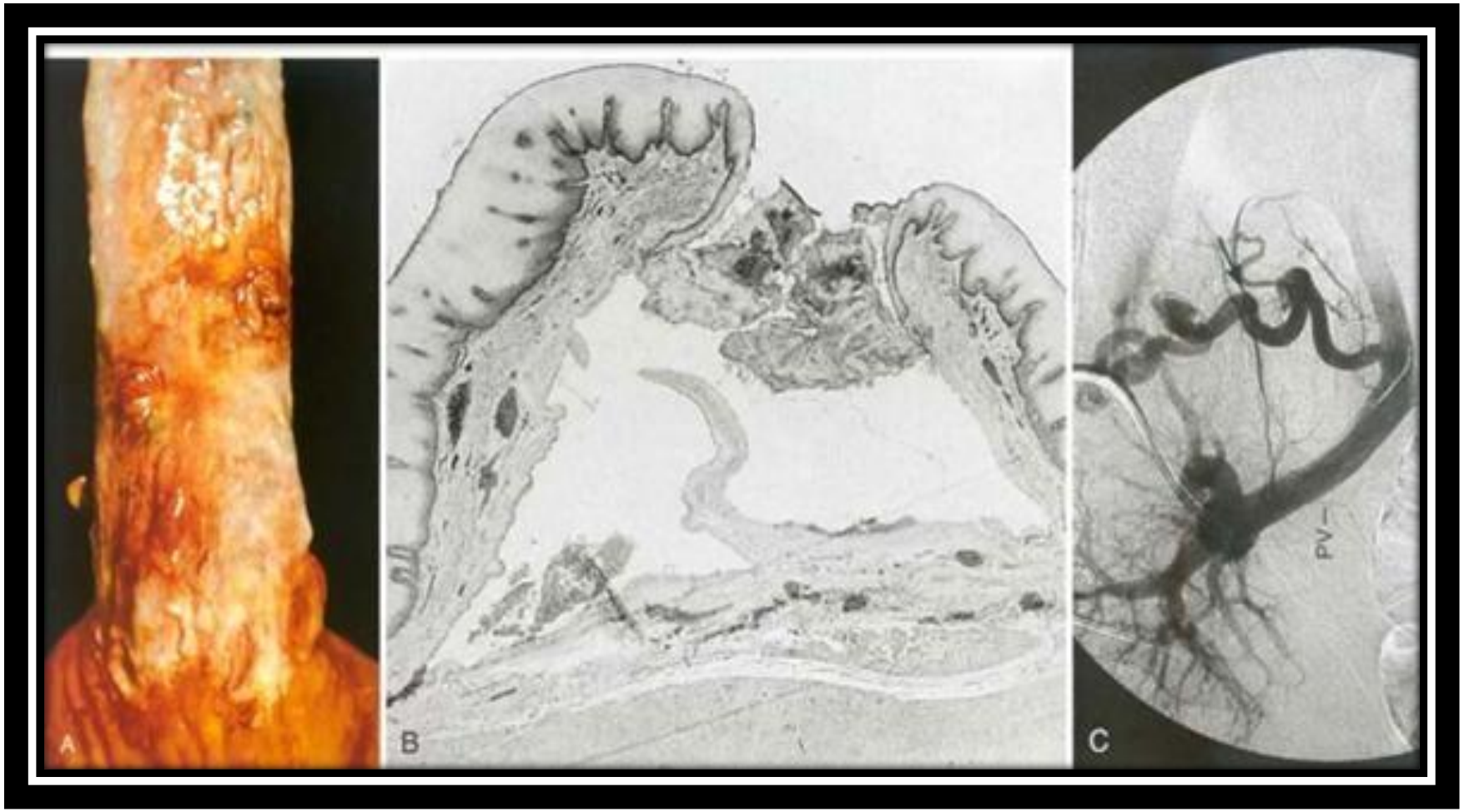

Mallory-Weiss



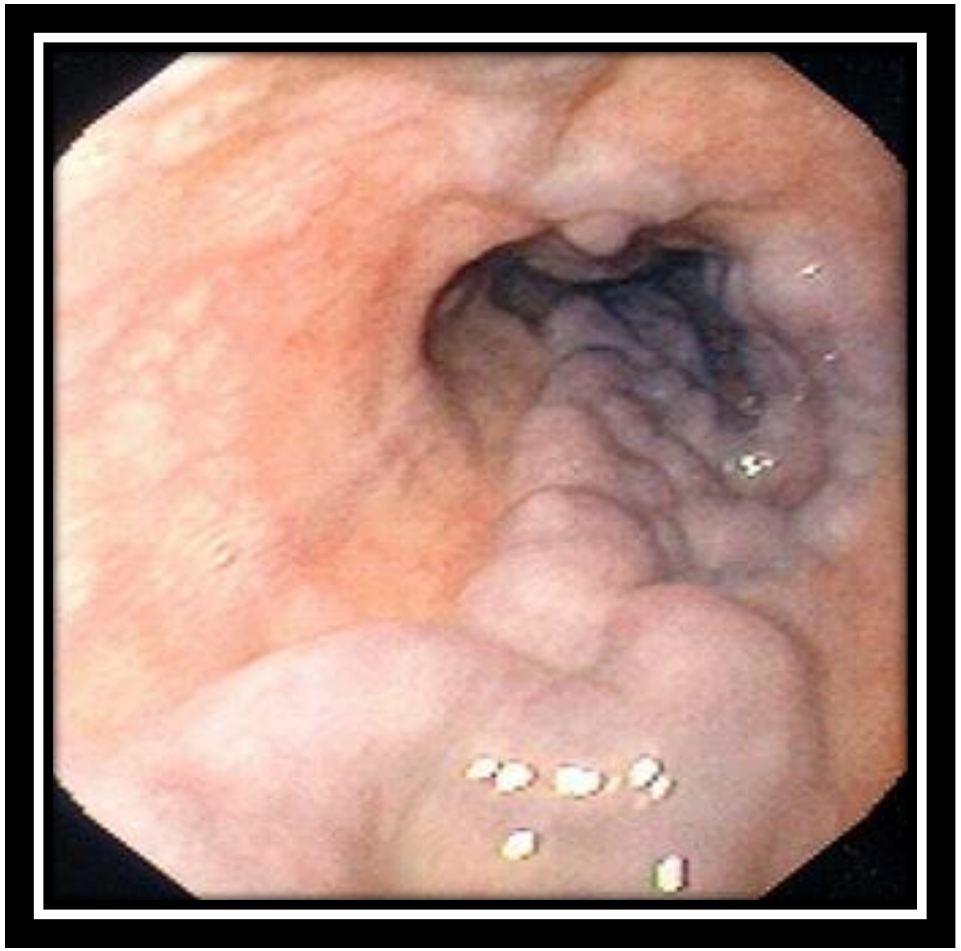
VARICES

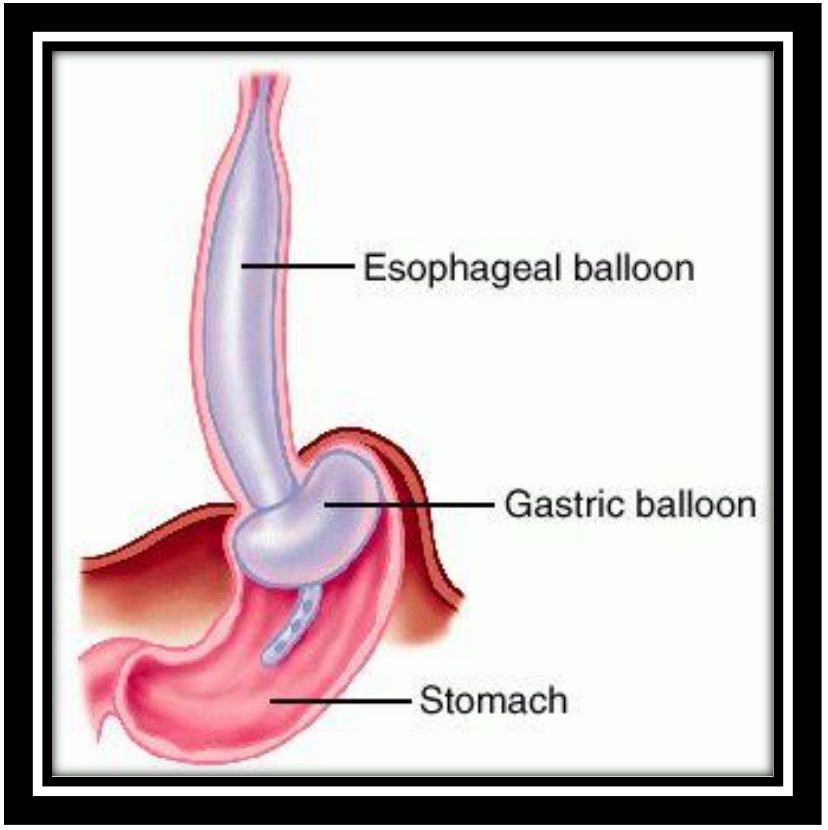
- **THREE common areas of portal/caval anastomoses**
  - **Esophageal**
  - **Umbilical**
  - **Hemorrhoidal**
- **100% related to portal hypertension**
- **Found in 90% of cirrhotics**
- **MASSIVE, SUDDEN, FATAL hemorrhage is the most feared consequence**

VARICES



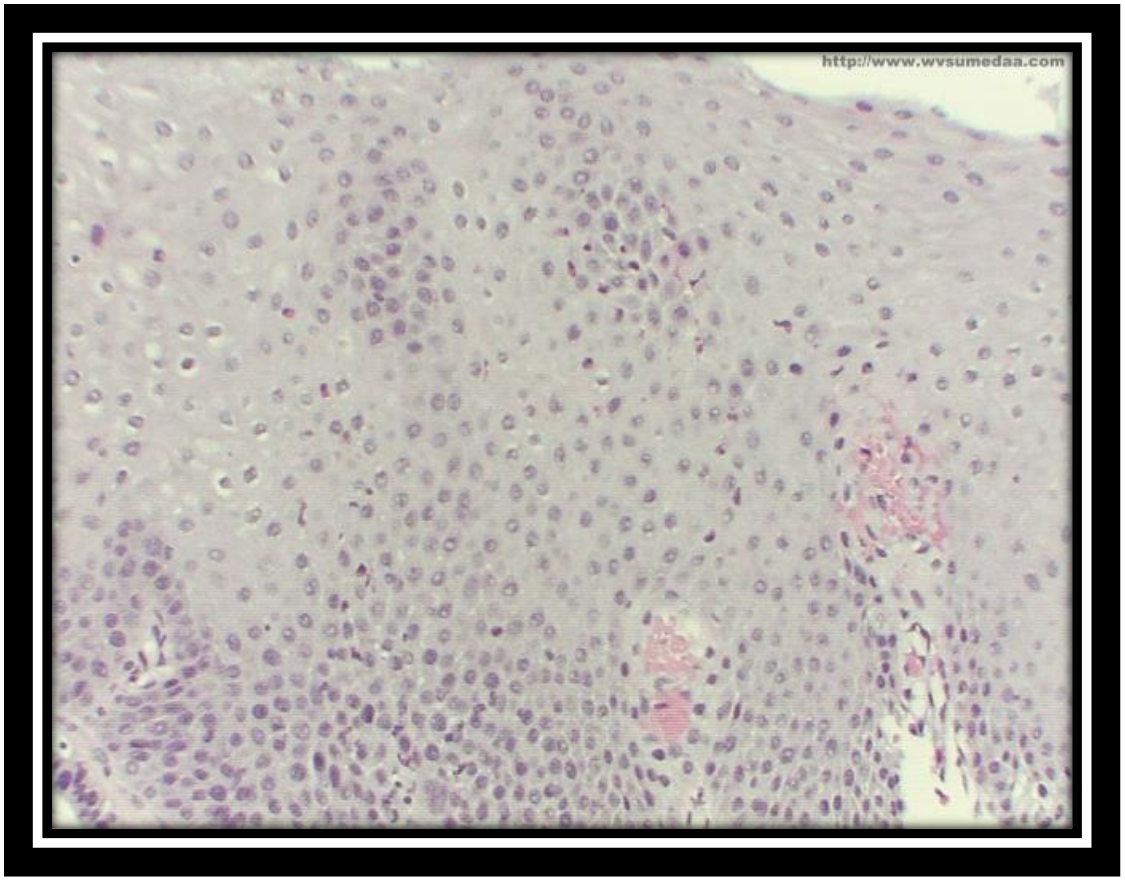
VARICES





ESOPHAGITIS

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



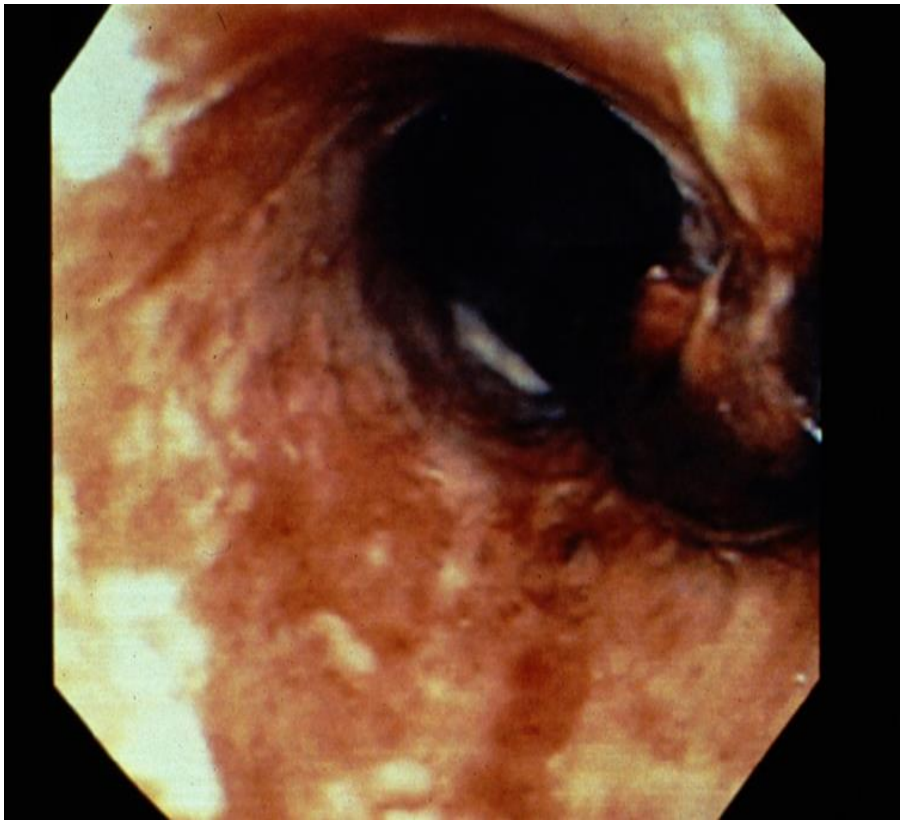
REFLUX/GERD

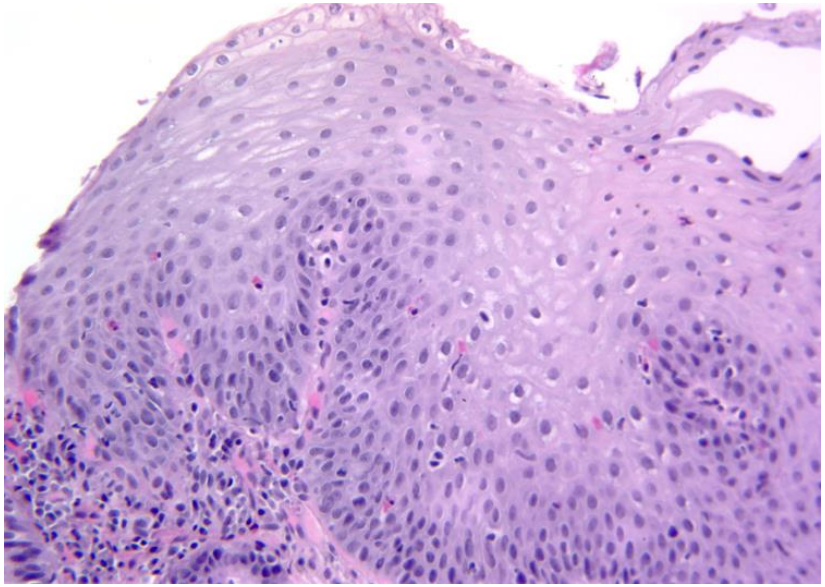
- DECREASED LES tone
- Hiatal Hernia
- Slowed reflux clearing
- Delayed gastric emptying

REFLUX/GERD

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

REFLUX/GERD

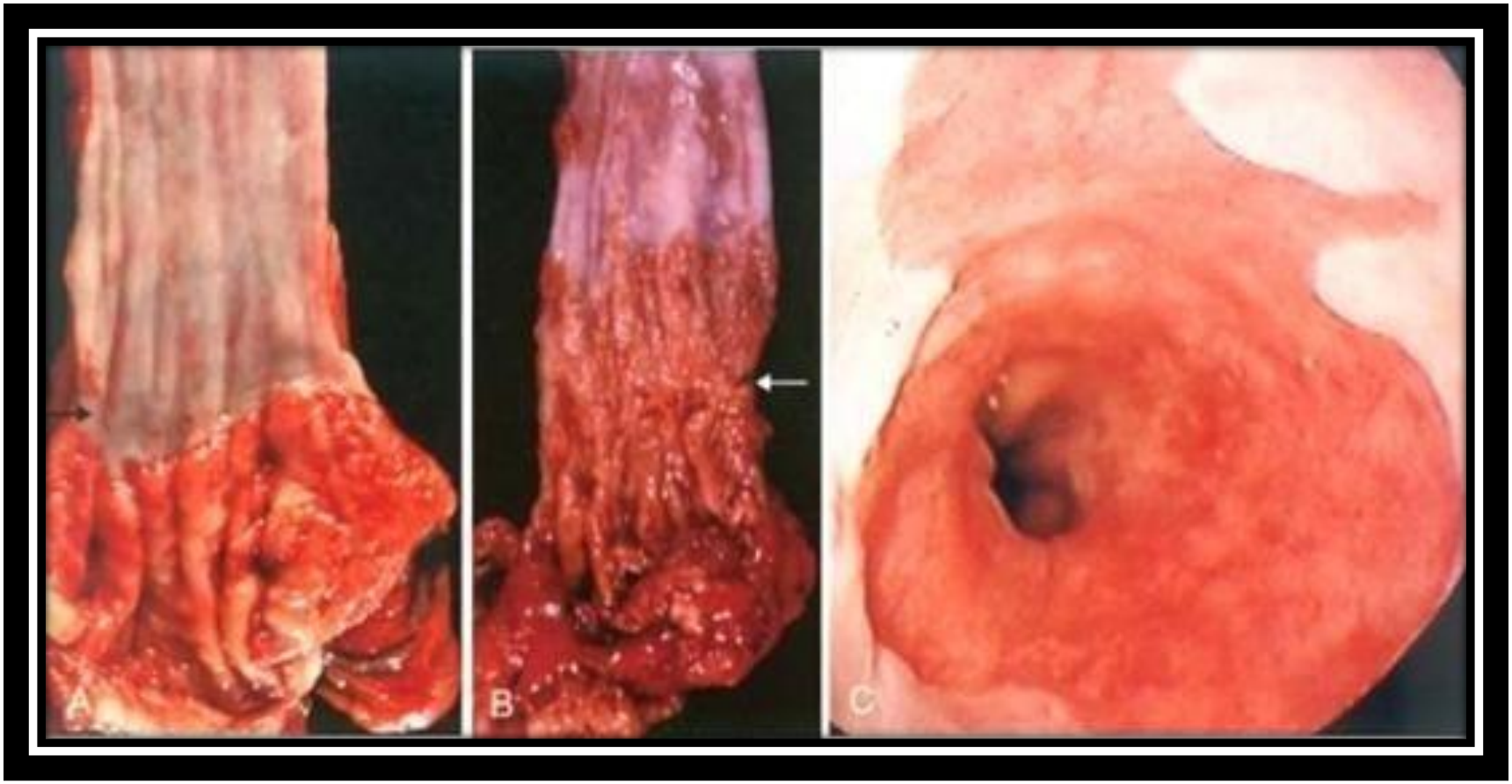


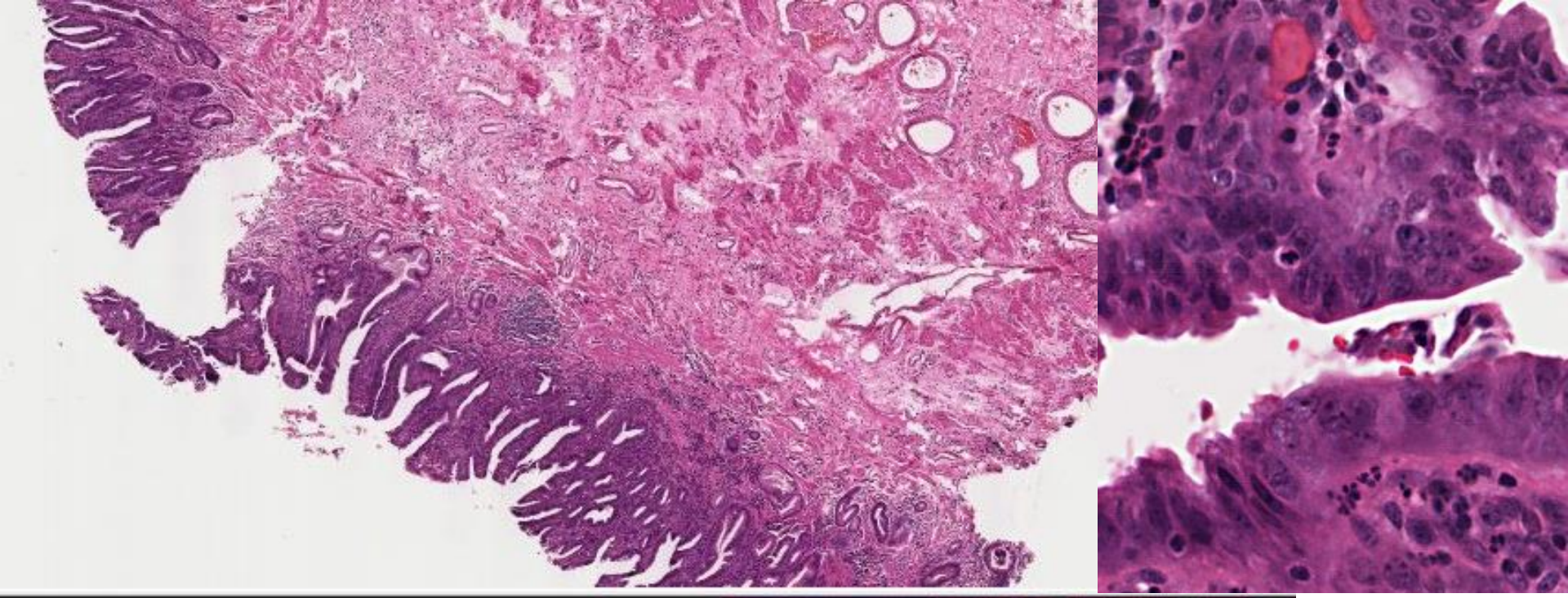


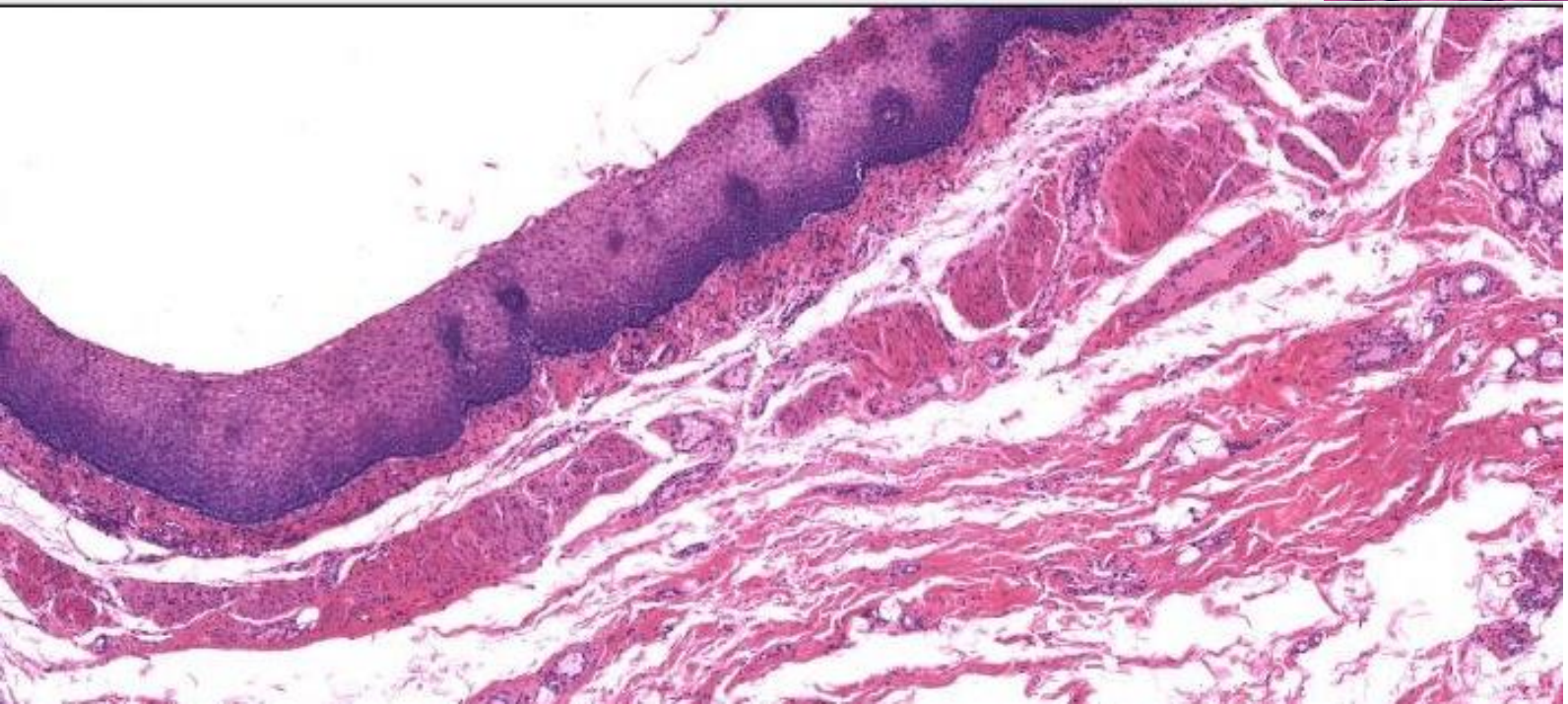
BARRETT'S ESOPHAGUS

- 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

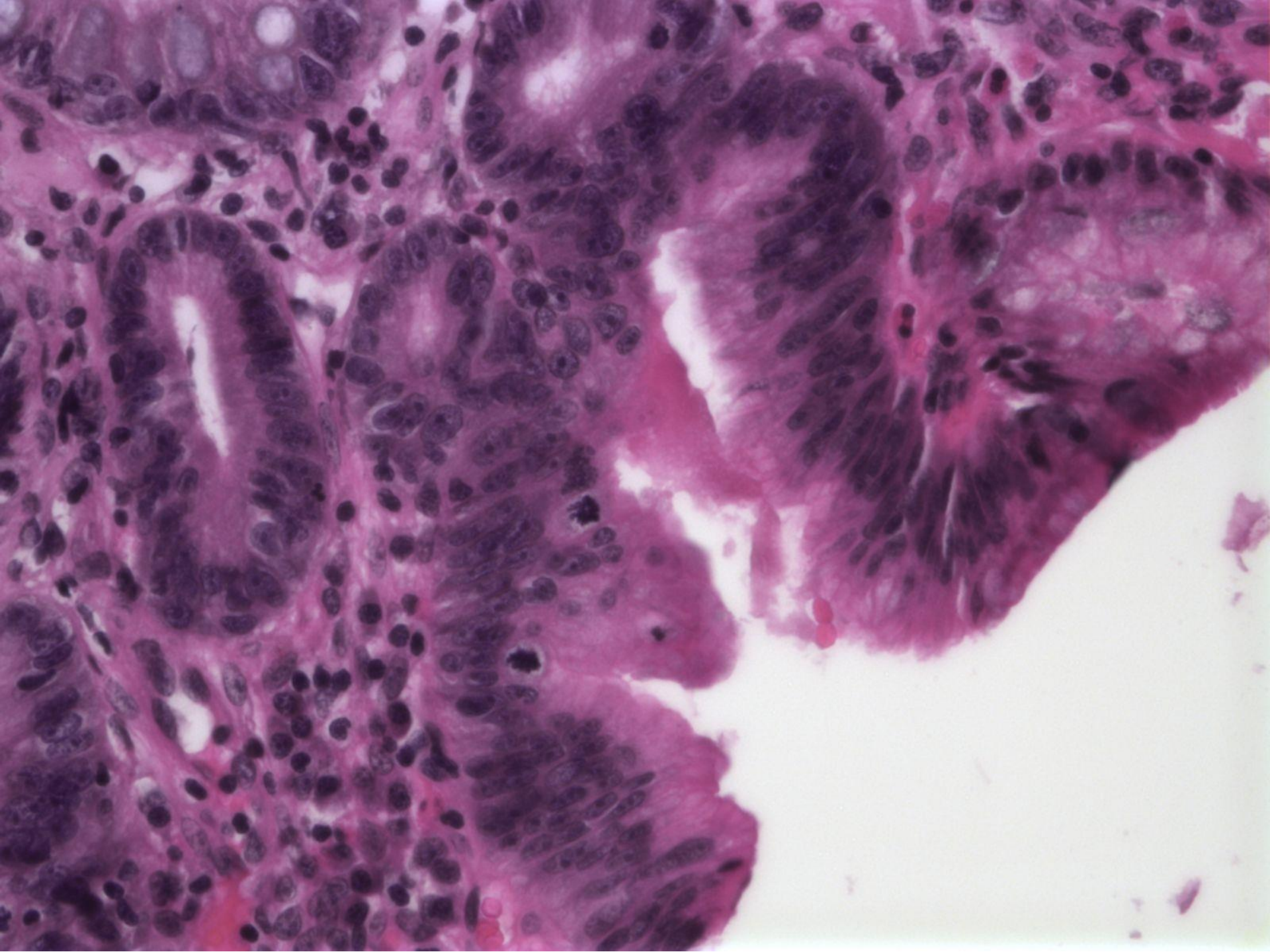






BARRETT'S ESOPHAGUS

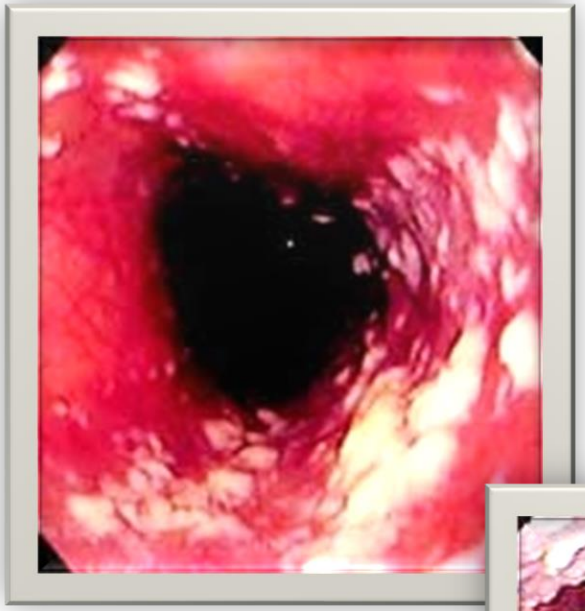
- **INTESTINALIZED (GASTRICIZED) mucosa is AT RISK for glandular dysplasia.**
- **Searching for dysplasia when BARRETT's is present**
- **MOST/ALL adenocarcinomas arising in the esophagus arise from previously existing BARRETT's**



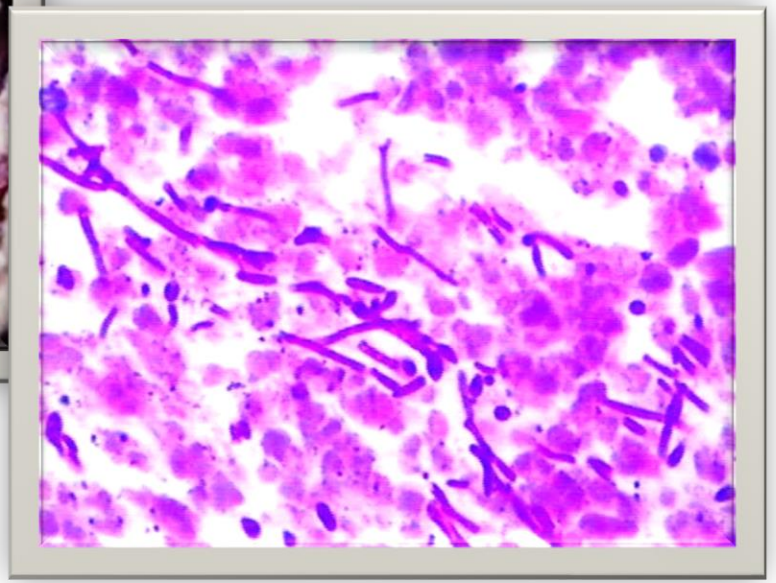
ESOPHAGITIS

- **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.

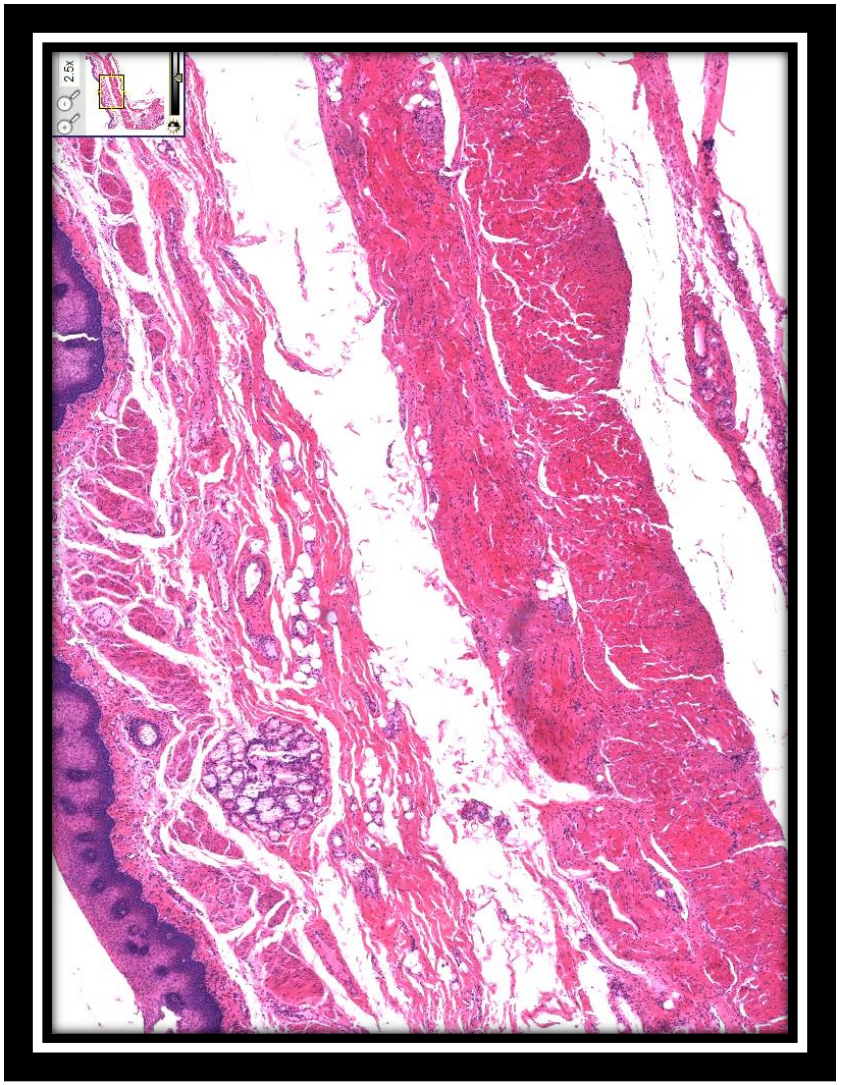


TUMORS

- **BENIGN**
- **MALIGNANT**
  - **Squamous cell carcinoma**
  - **Adenocarcinoma**

BENIGN TUMORS

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

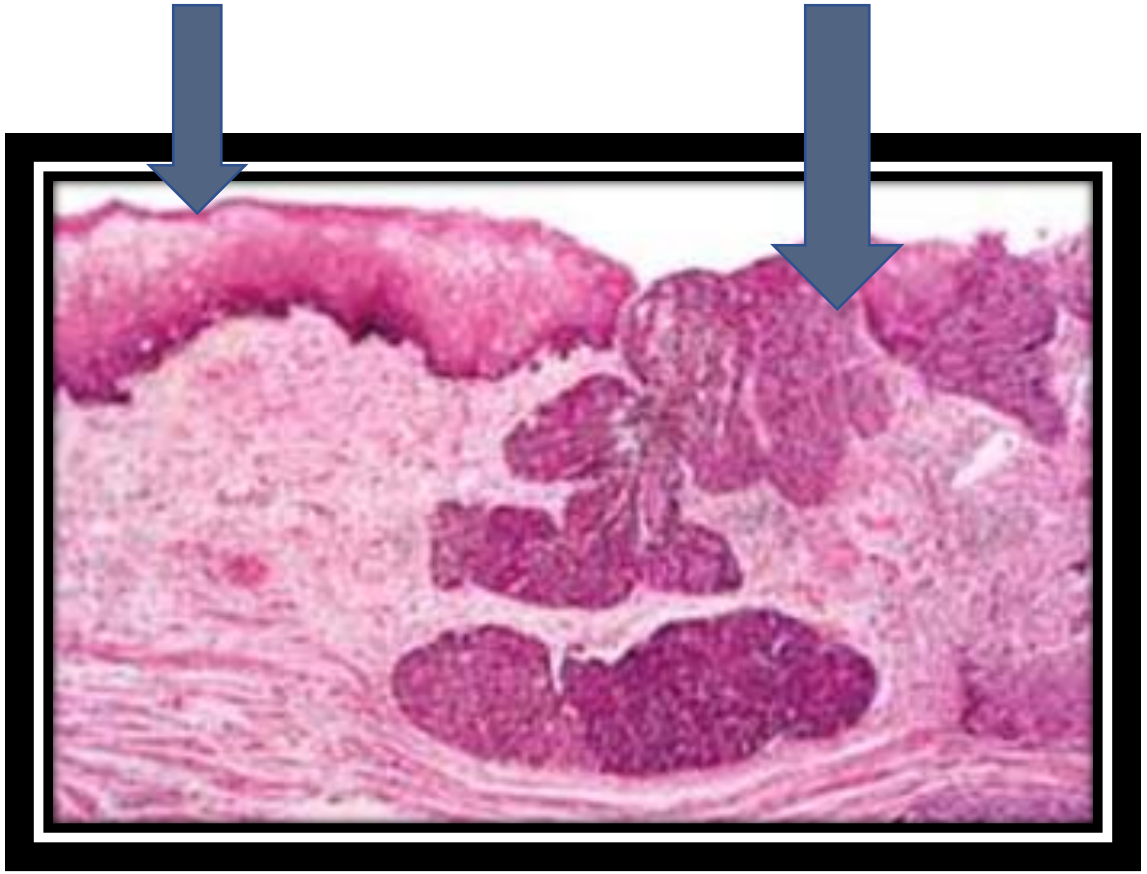


SQUAMOUS CARCINOMA

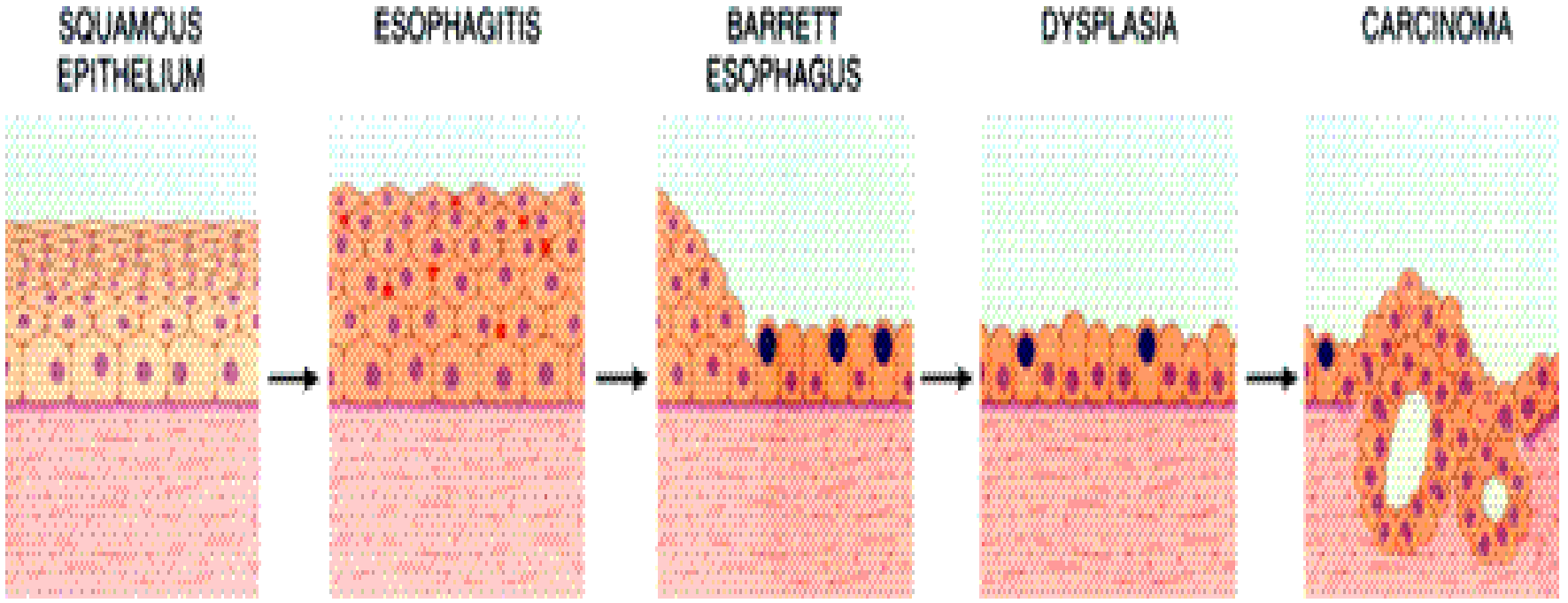
- Nitrites/Nitrosamines
- Fungi in food
- Tobacco
- Alcohol
- Esophagitis

SQUAMOUS CARCINOMA

- **DYSPLASIA→IN-SITU→INFILTRATION**



ADENOCARCINOMA



Pathogenesis of Adenocarcinoma

- Reflux Esophagitis

↓

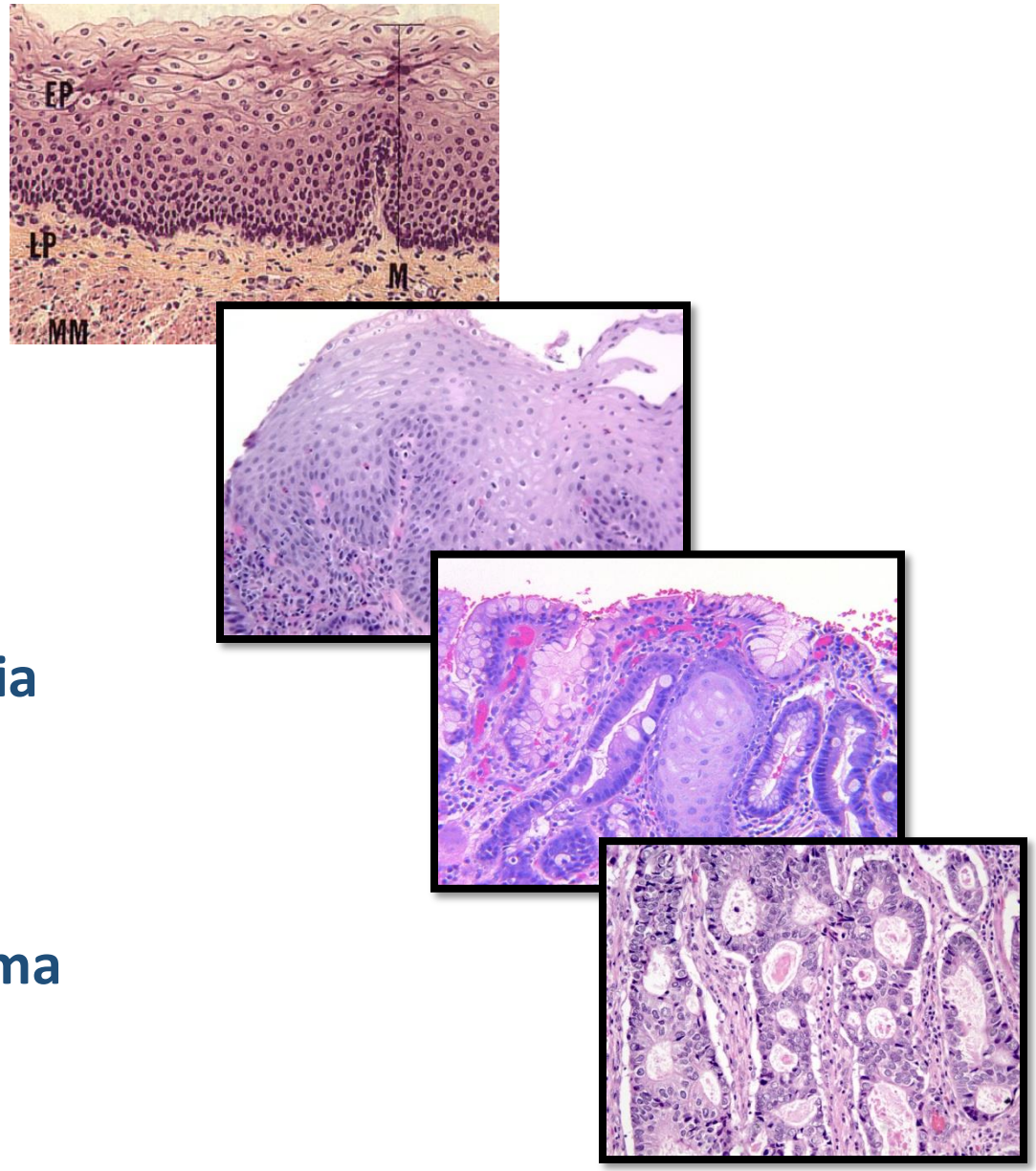
Barrett's esophagus
(intestinal type)

↓

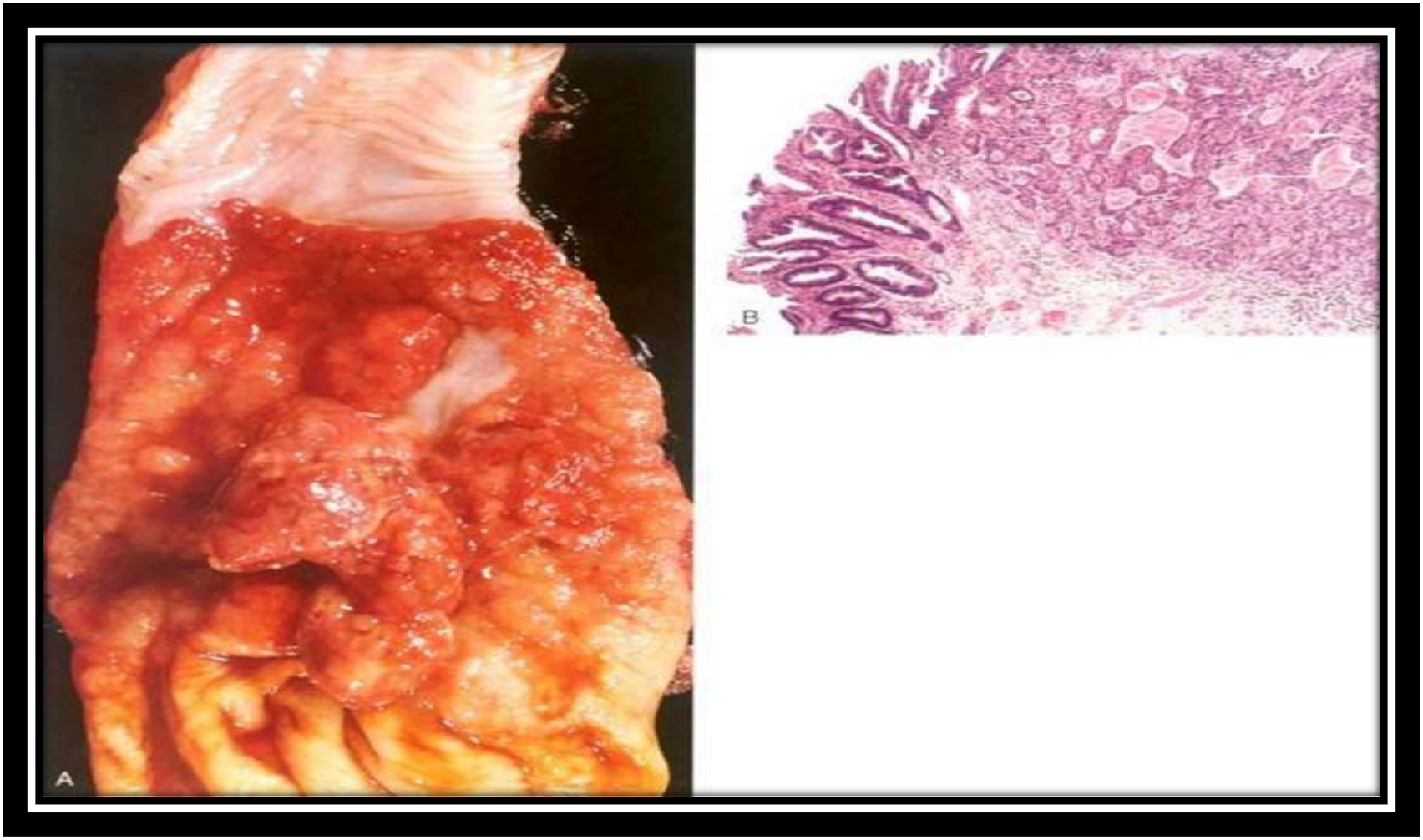
Barrett's with dysplasia
(Carcinoma in-situ)

↓

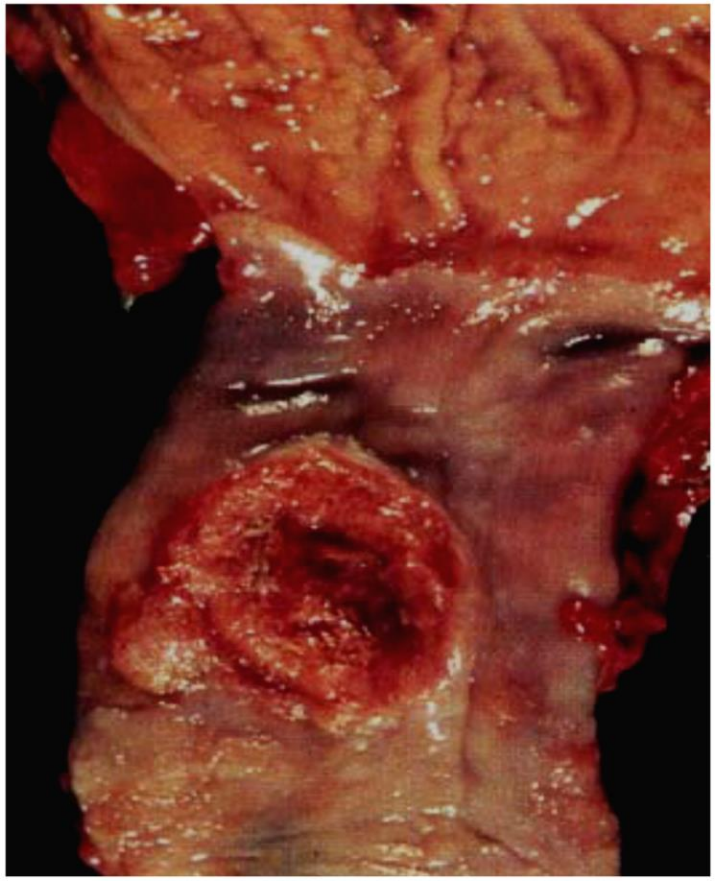
Invasive adenocarcinoma

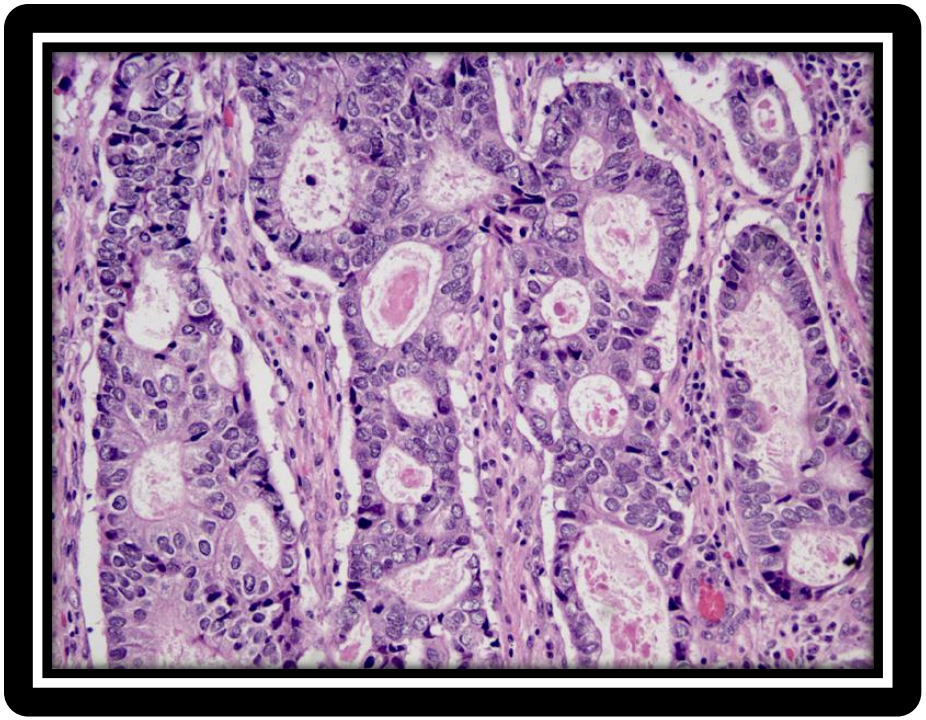


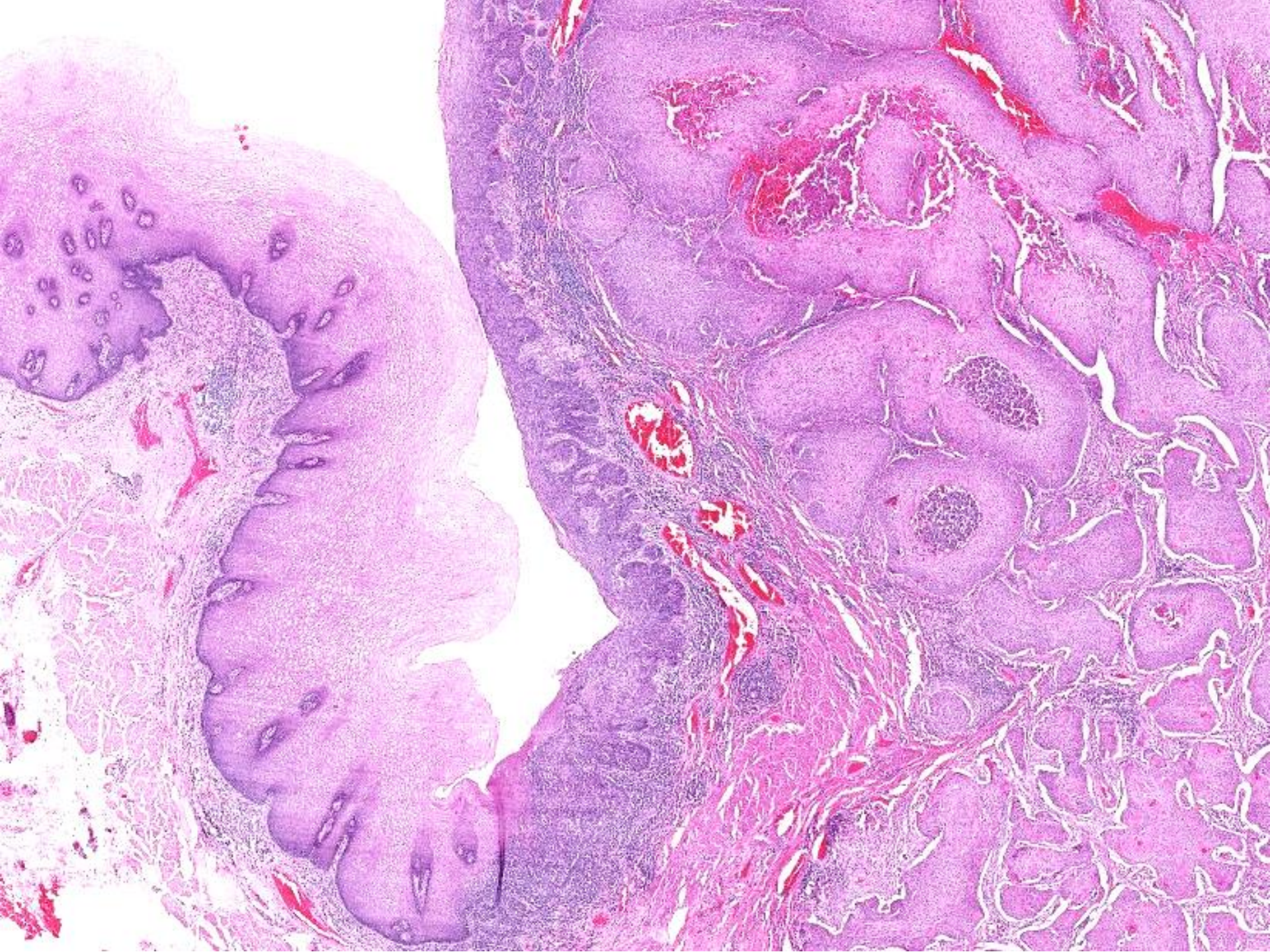
ADENOCARCINOMA



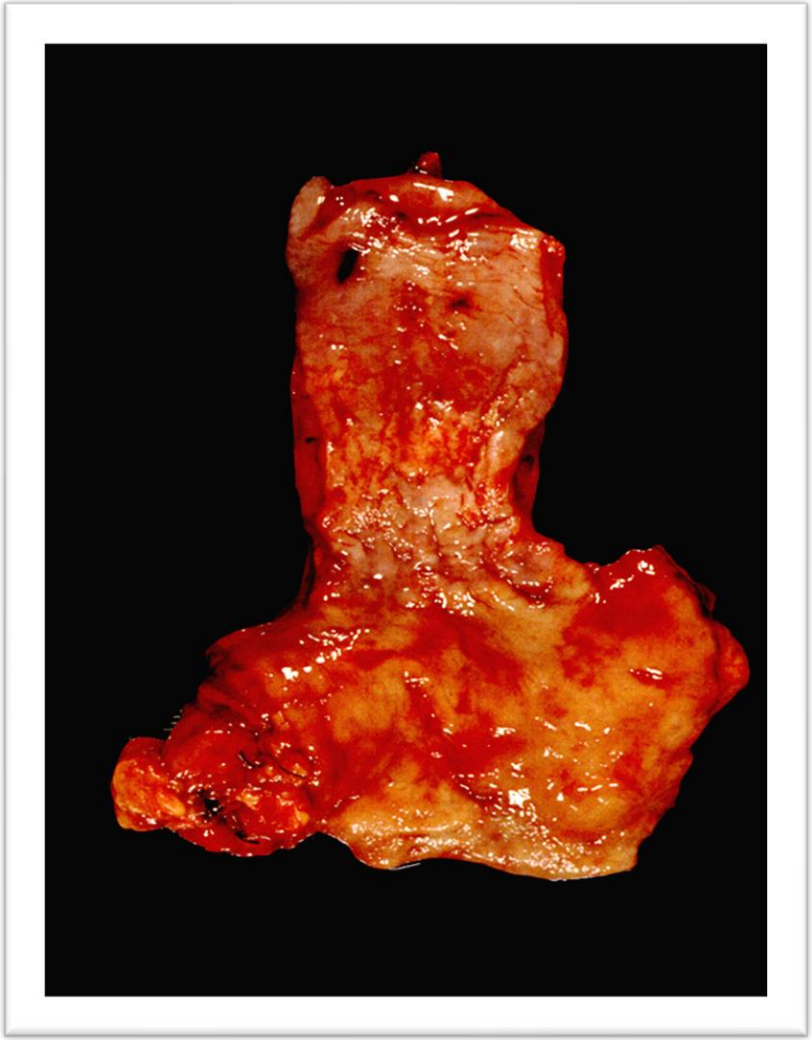
Esophageal Adenocarcinoma

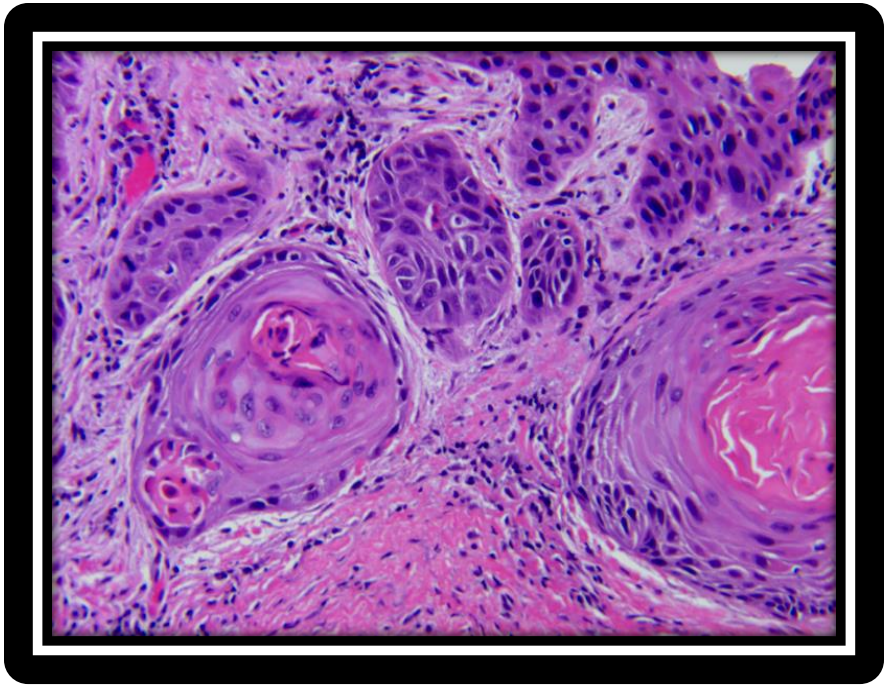




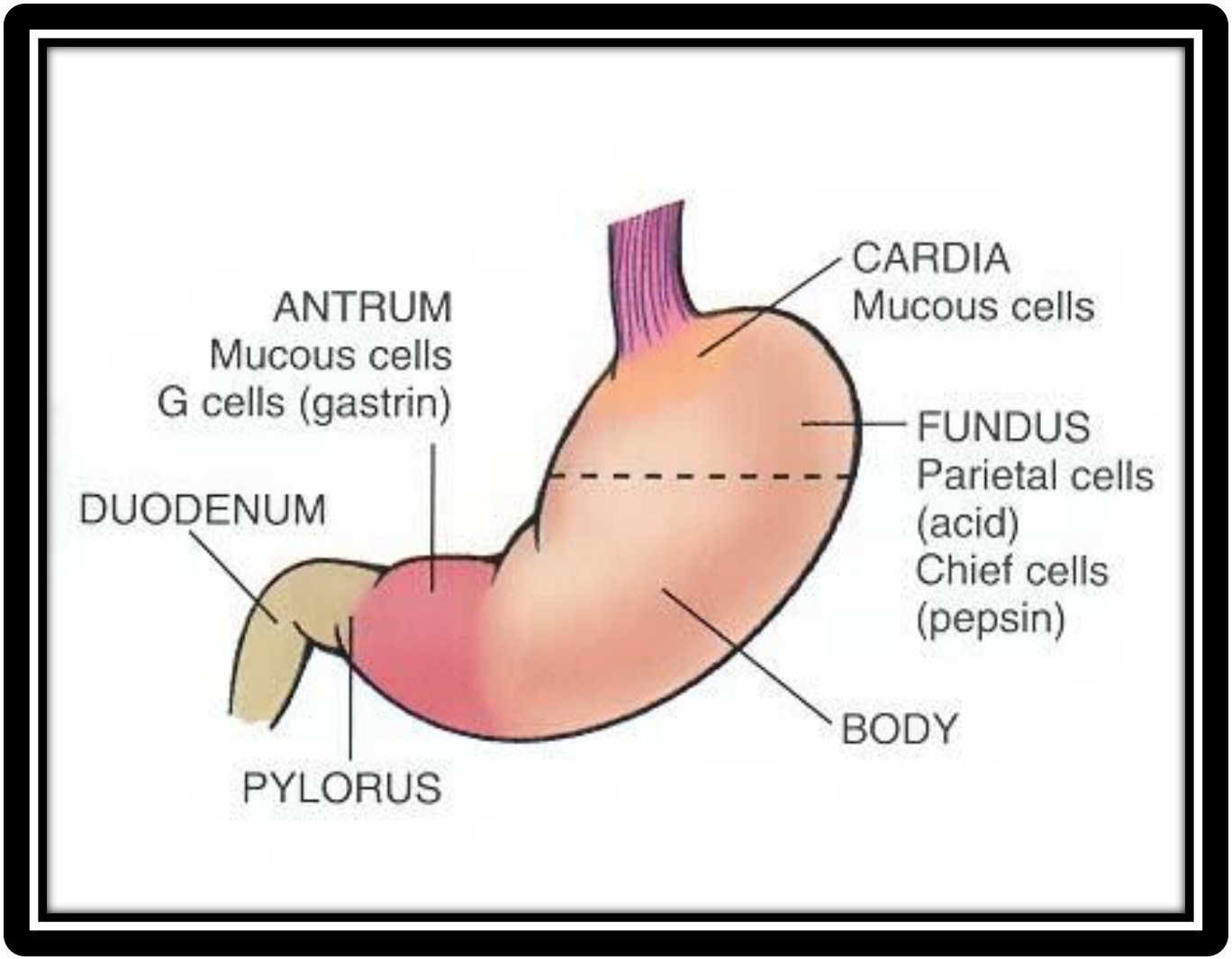


Esophageal Squamous Cell Carcinoma





Stomach


CARDIA
Mucous cells
ANTRUM
Mucous cells
G cells (gastrin)
FUNDUS
Parietal cells
(acid)
Chief cells
(pepsin)
DUODENUM
BODY
PYLORUS

ANATOMY

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

Greater/Lesser Curvatures

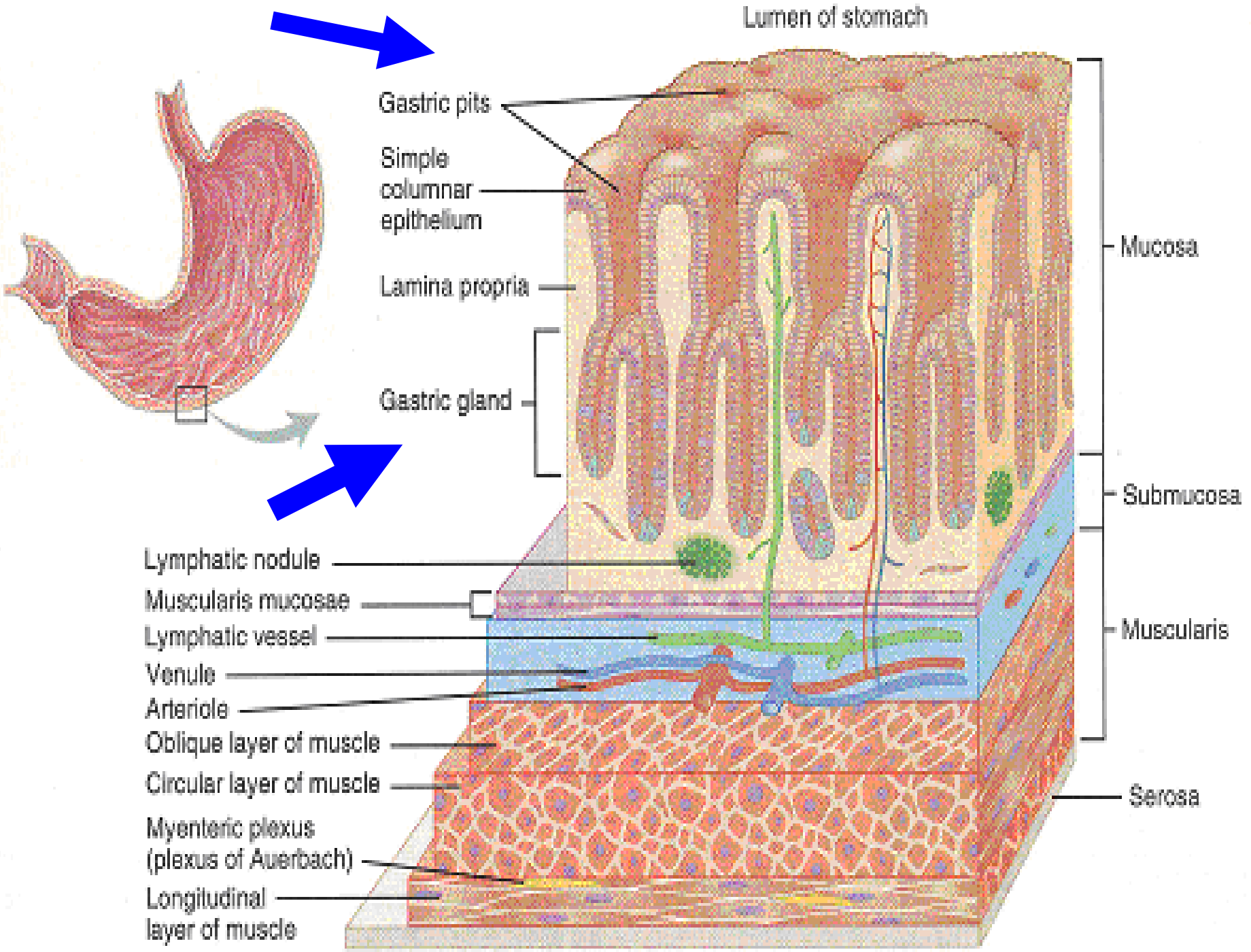
1500-3000 ml

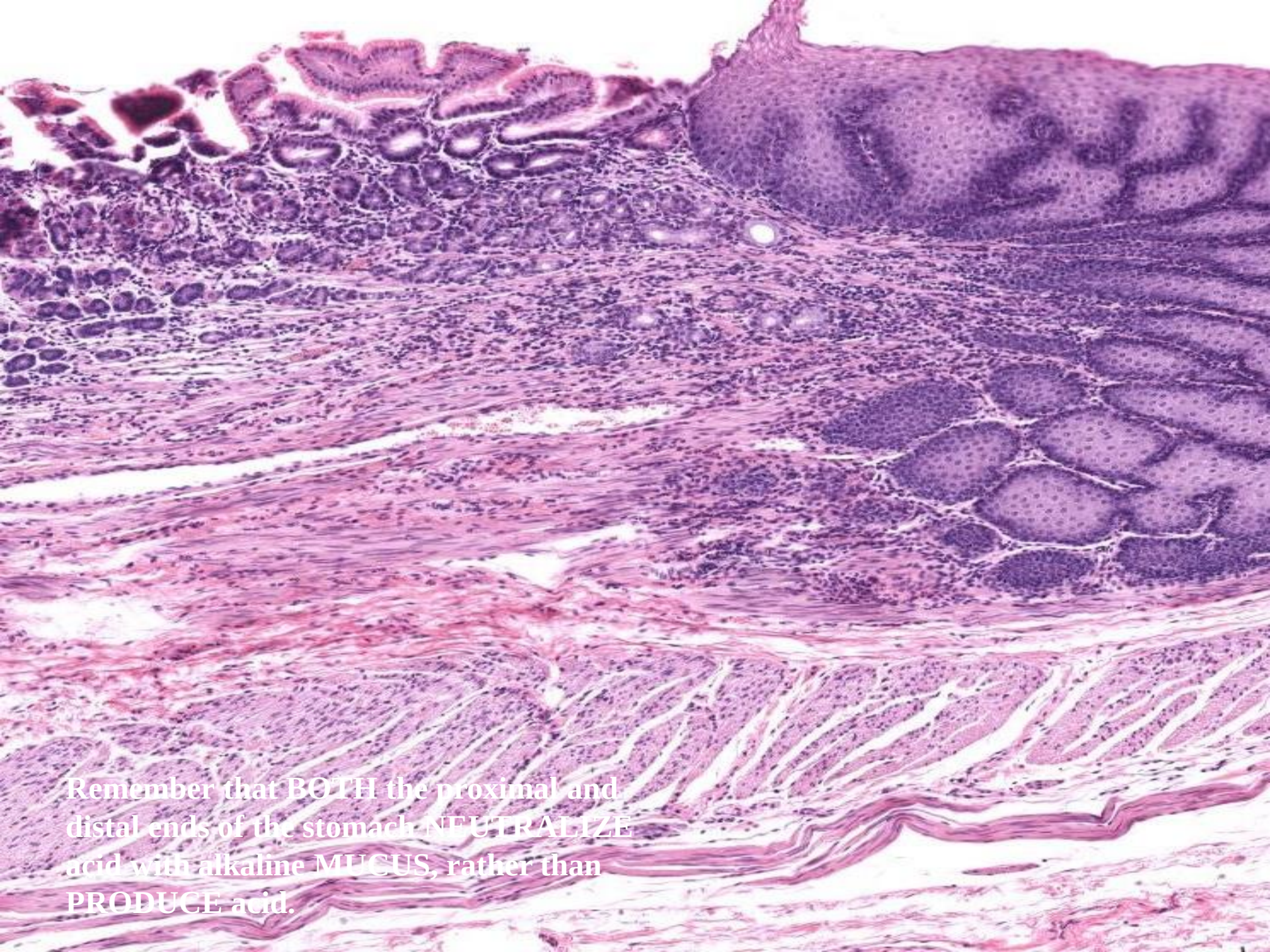
Rugae

INNERVATION: VAGUS, Sympathetic

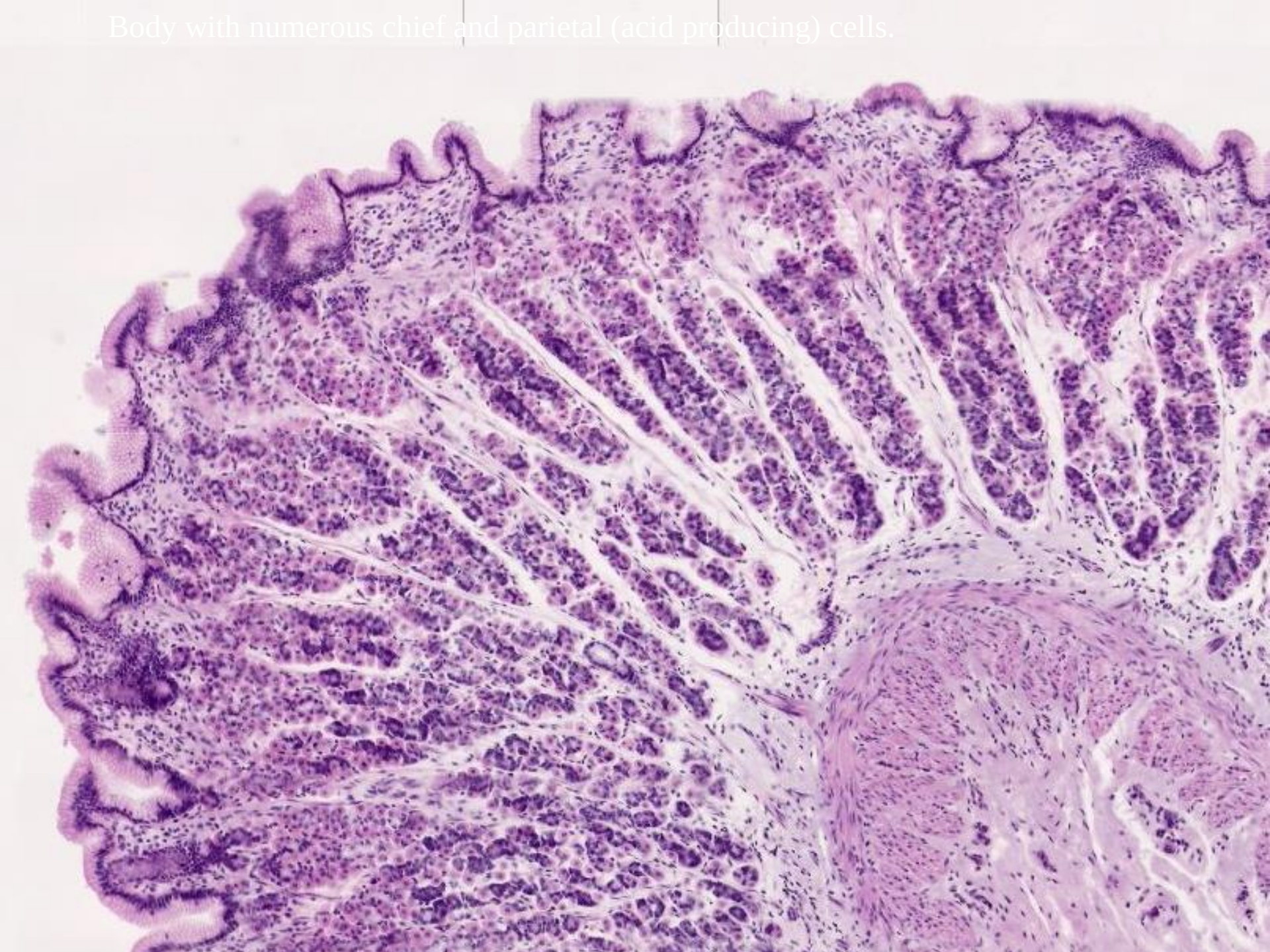
VEINS: Portal

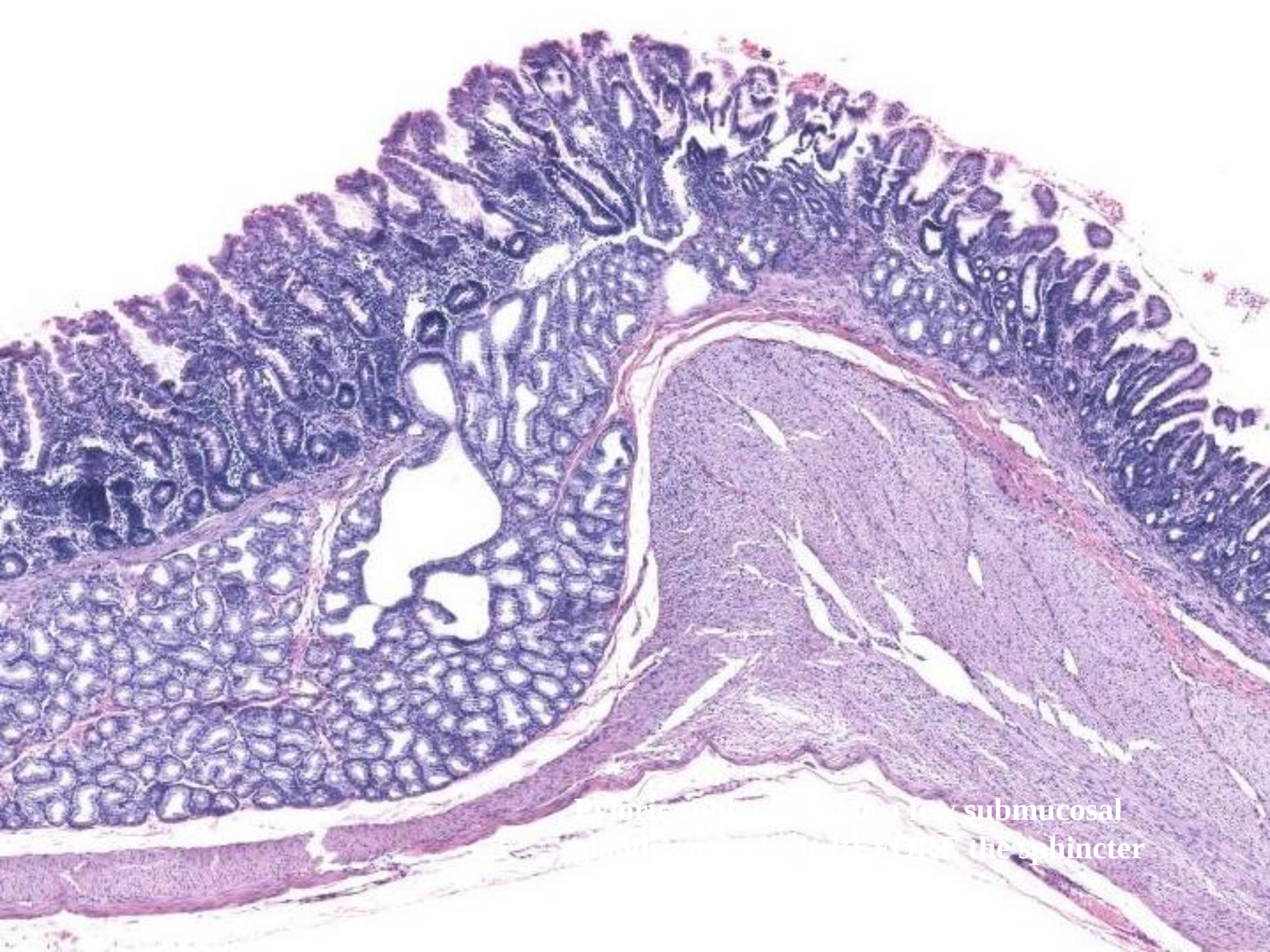
Blood Supply: **RG, LG, RGE(O), LGE(O), SG, <u>ALL</u> 3 branches of the celiac, no matter what the variations may be.**


Lumen of stomach
Gastric pits
Simple columnar epithelium
Lamina propria
Gastric gland
Lymphatic nodule
Muscularis mucosae
Lymphatic vessel
Venule
Arteriole
Oblique layer of muscle
Circular layer of muscle
Myenteric plexus (plexus of Auerbach)
Longitudinal layer of muscle
Mucosa
Submucosa
Muscularis
Serosa


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.





CELLS

MUCOUS: MUCUS, PEPSINOGEN II

CHIEF: PEPSINOGEN I, II

PARIETAL: ACID

ENTEROENDOCRINE: HISTAMINE, SOMATOSTATIN, ENDOTHELIN

ACID PROTECTION

MUCUS

HCO_3^-

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

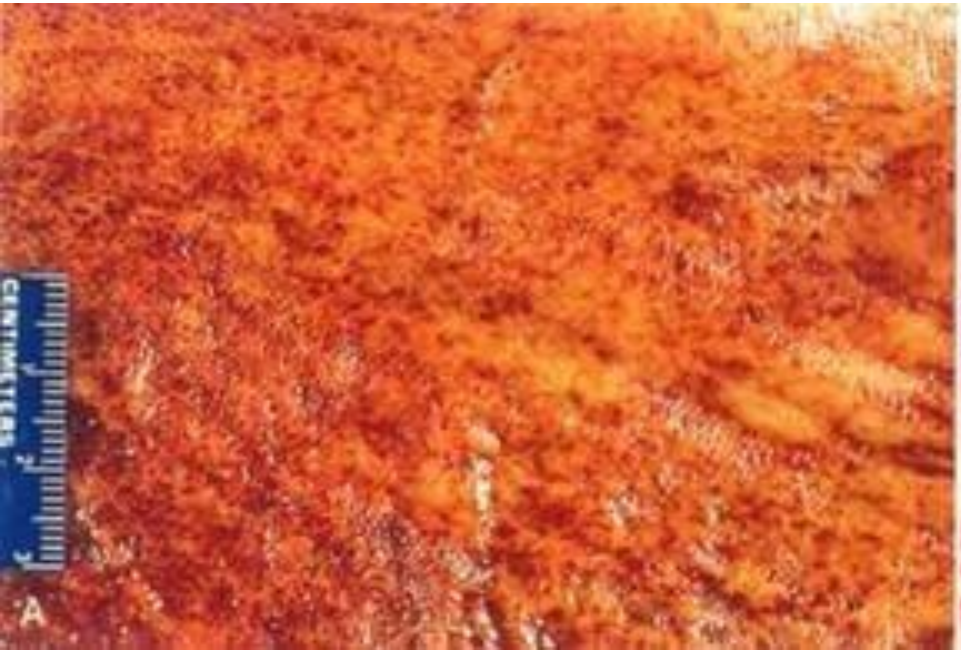
Acute Gatritis Causes

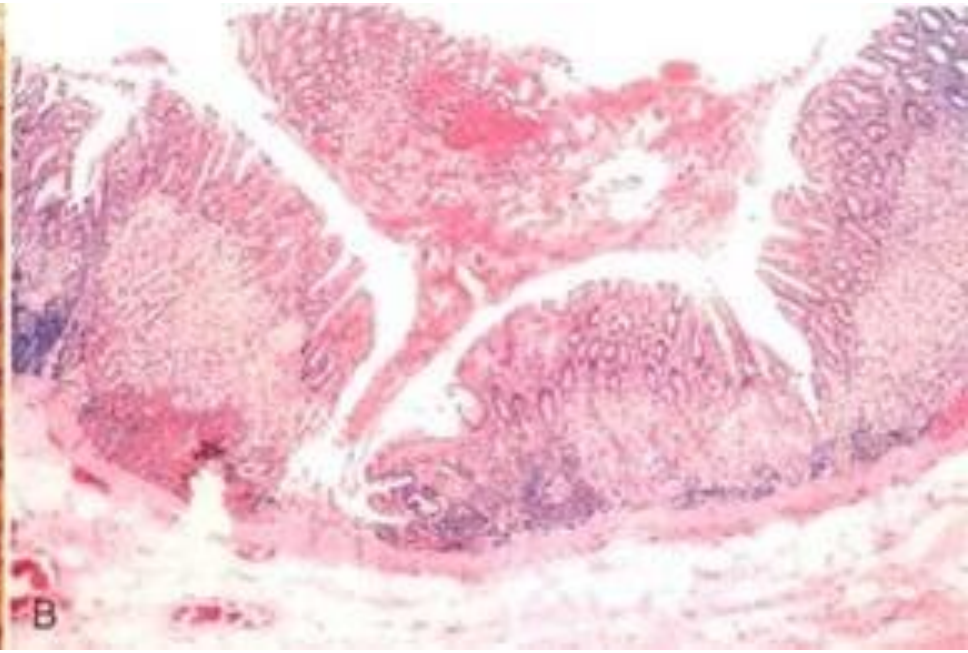
Heavy use of nonsteroidal anti-inflammatory drugs (NSAIDs), particularly aspirin
Excessive alcohol consumption
Heavy smoking
Treatment with cancer chemotherapeutic drugs
Uremia
Systemic infections (e.g., salmonellosis)
Severe stress (e.g., trauma, burns, surgery)
Ischemia and shock
Suicide attempts with acids and alkali
Mechanical trauma (e.g., nasogastric intubation)
After distal gastrectomy with reflux of bilious material

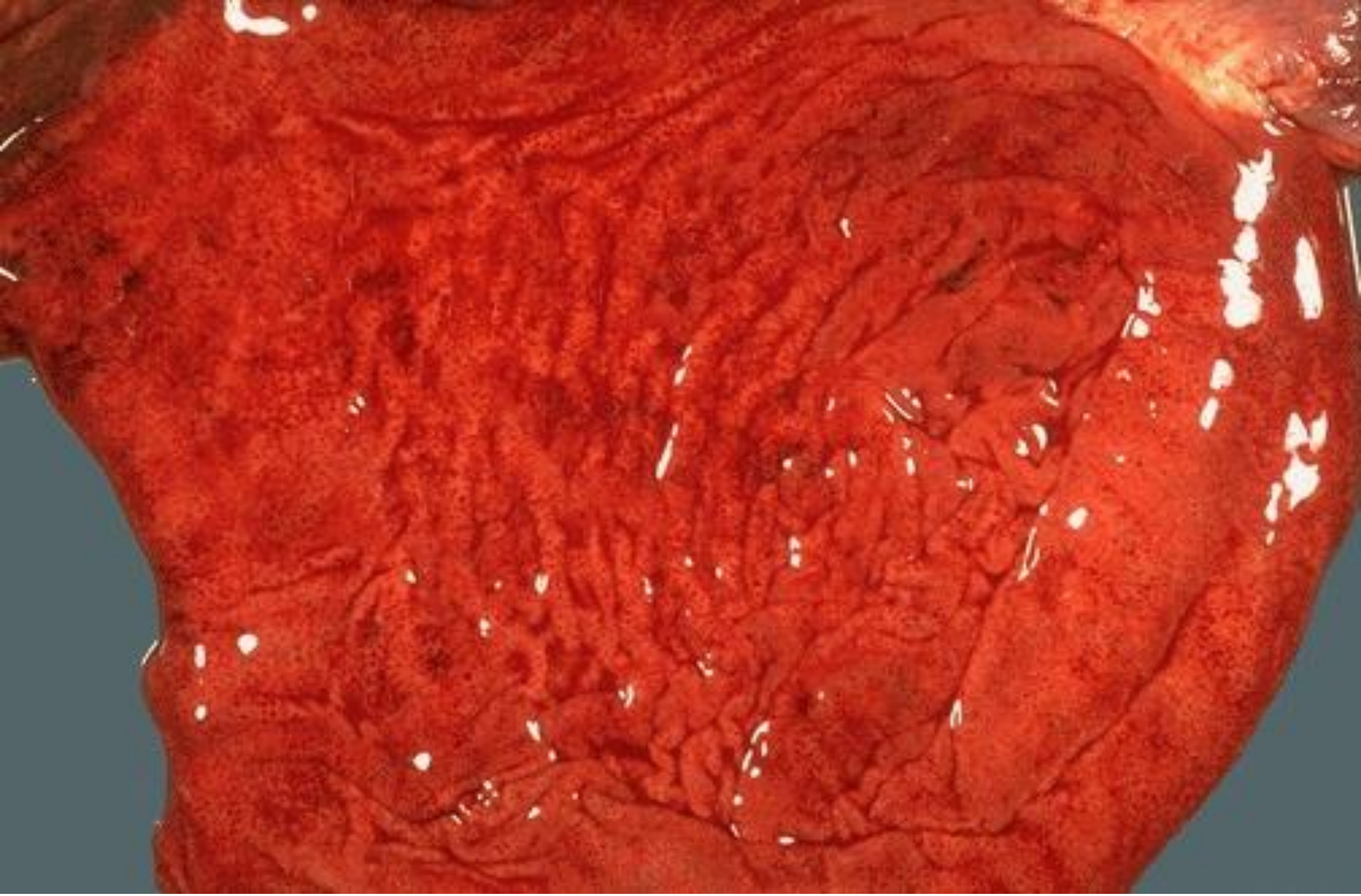
The main CLINICAL differentiation between acute and chronic gastritis would be the presence or absence of blood, respectively.

GASTRITIS

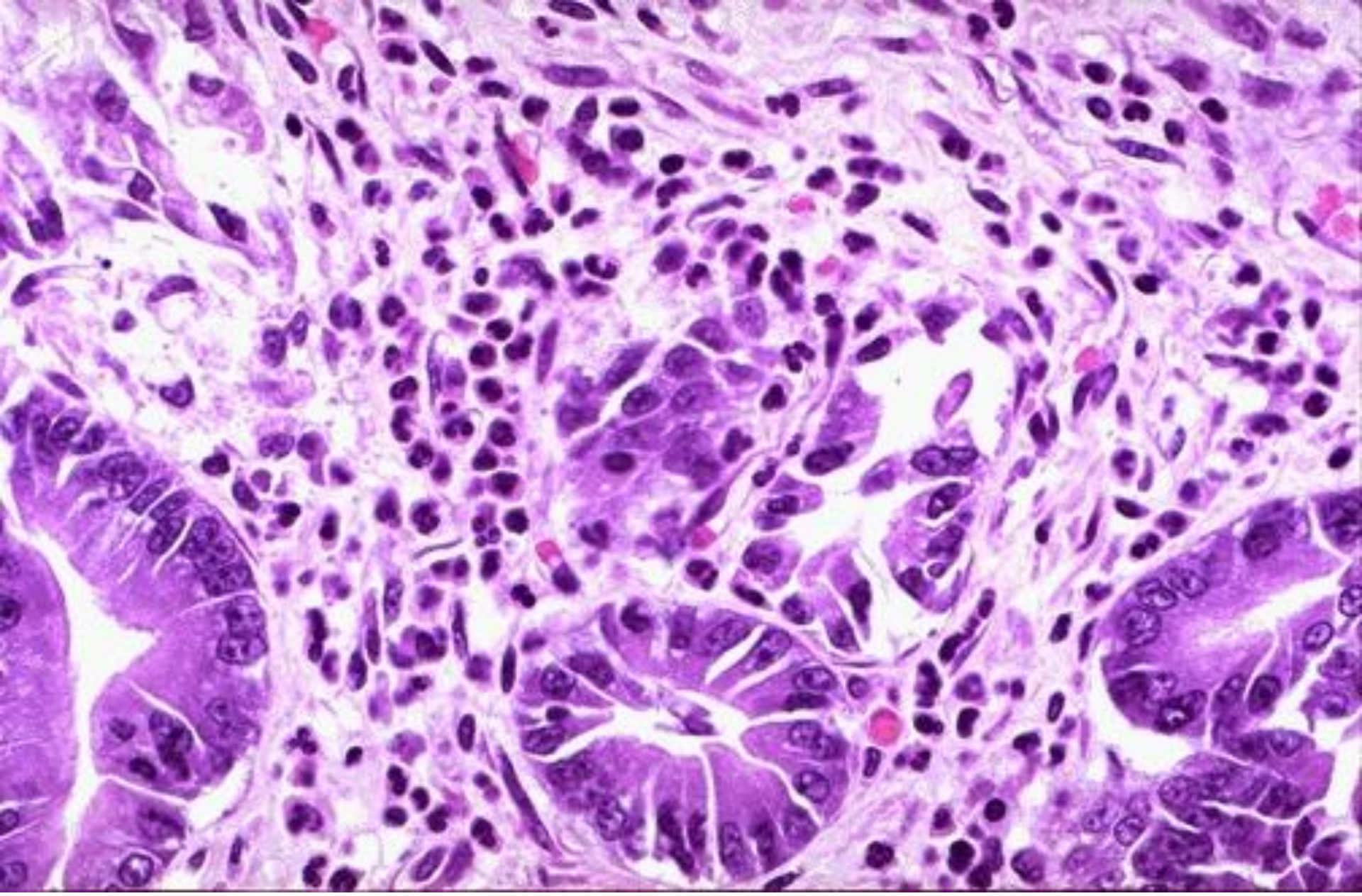
- **ACUTE, HEMORRHAGIC**
- **HISTOLOGY: Erosion, Hemorrage, NEUTROPHILS**



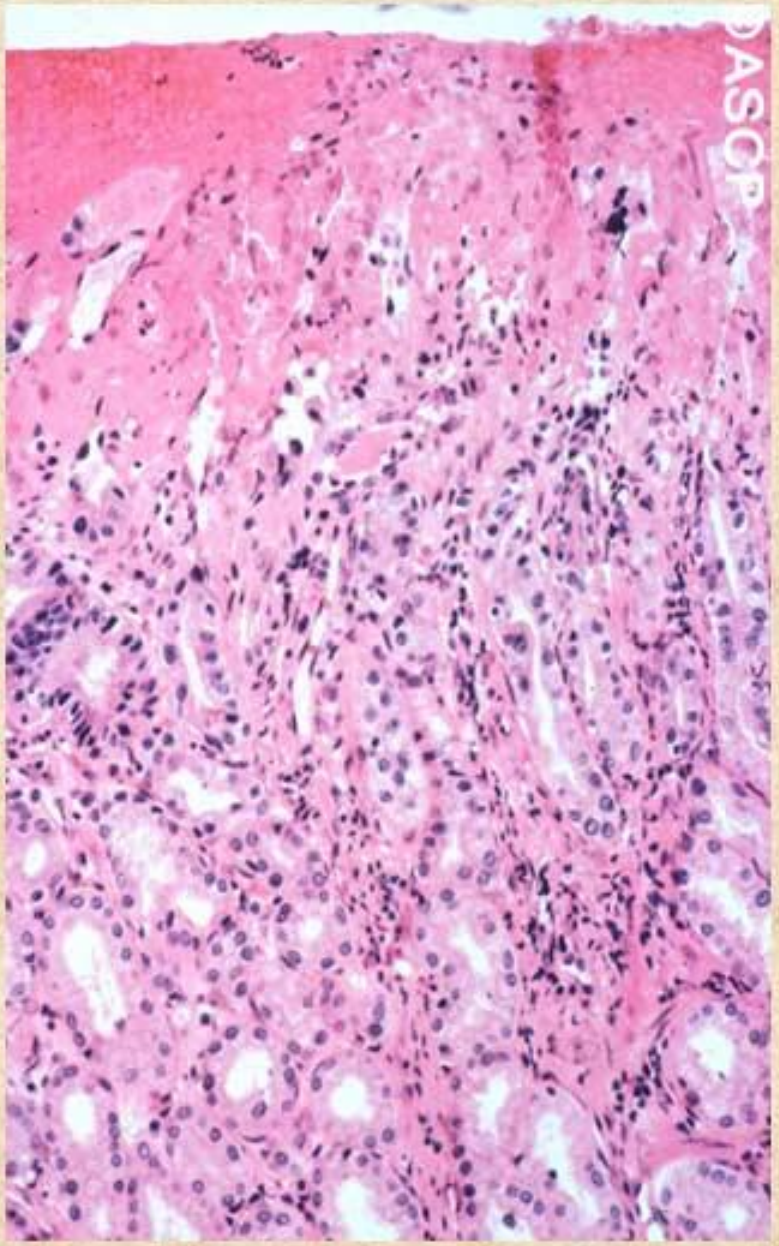




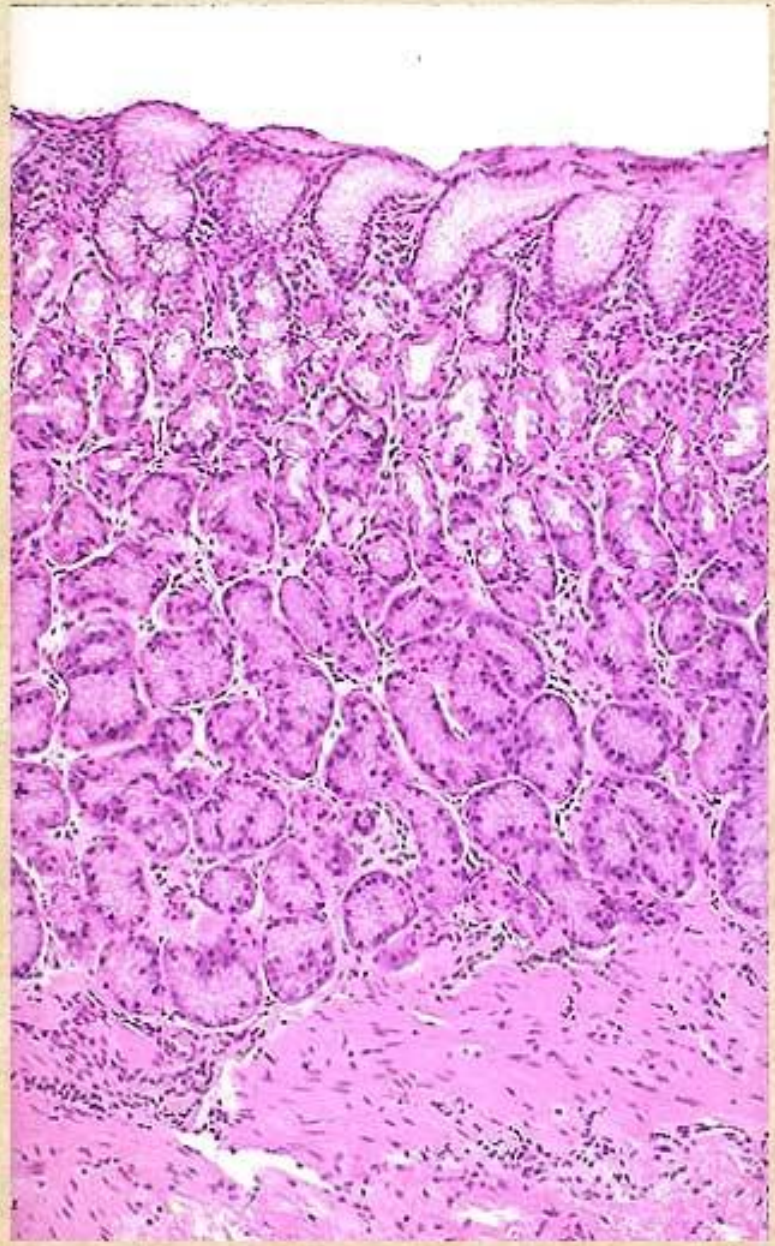
Acute gastritis



Acute erosive gastritis



Normal fundus

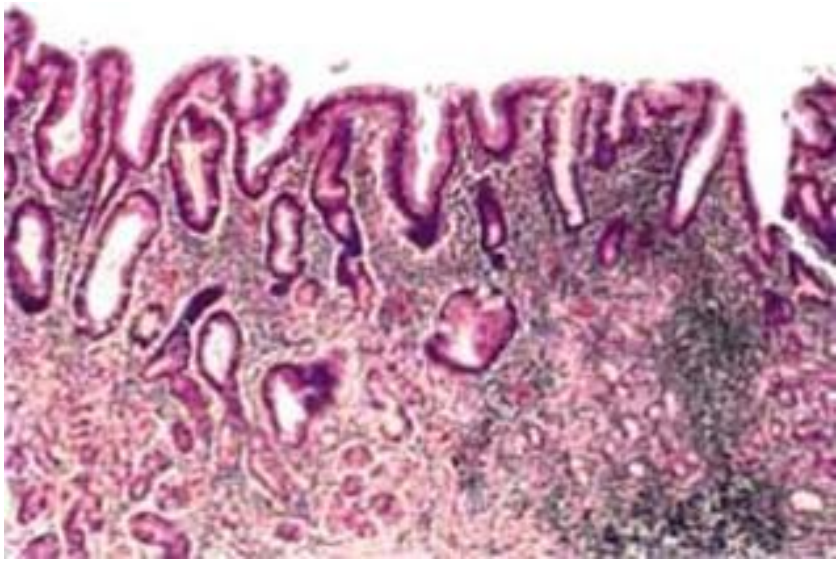


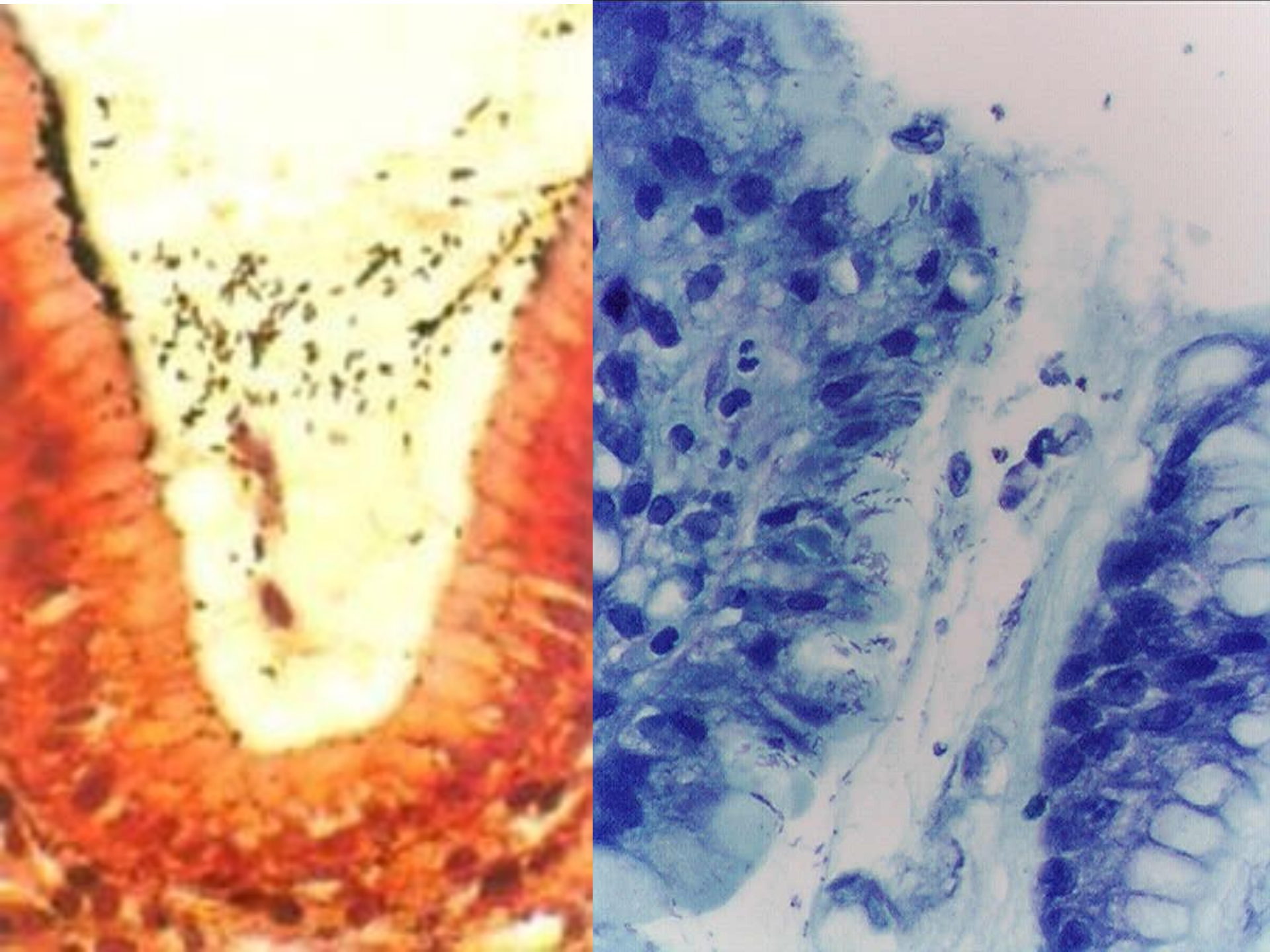
GASTRITIS

- **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**

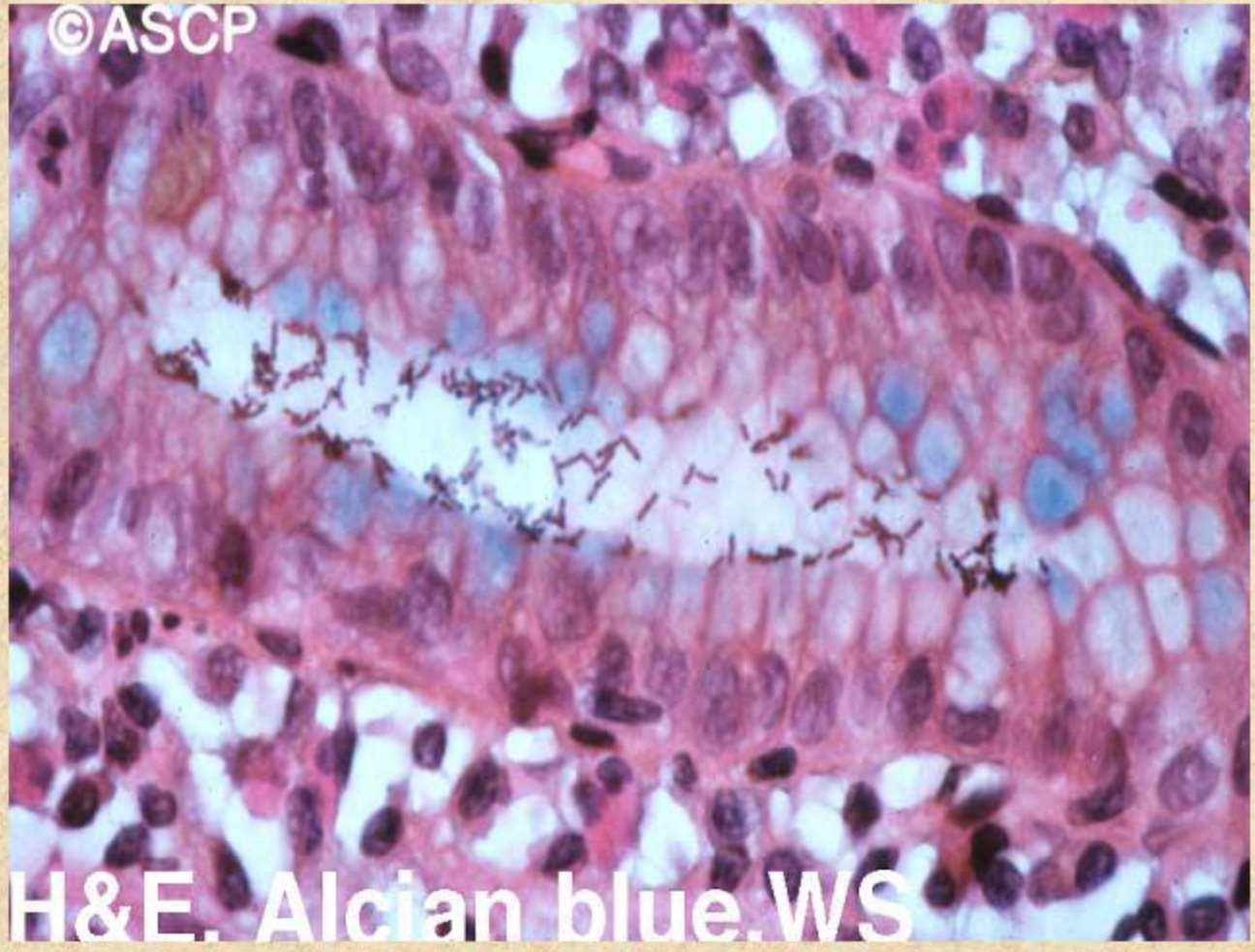
GASTRITIS

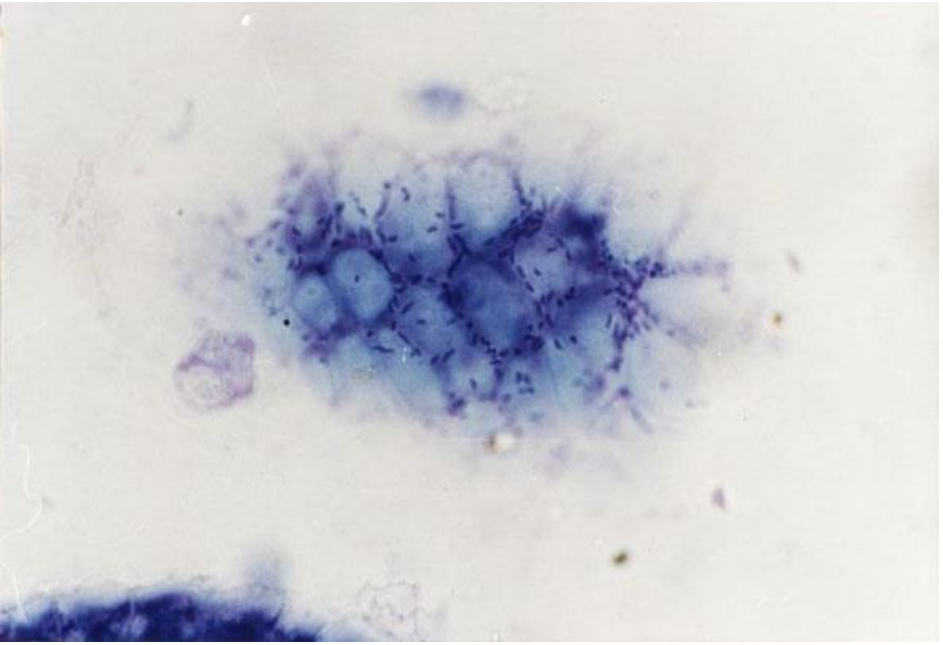
- **CHRONIC, NO EROSIONS, NO HEMORRHAGE**
- **Perhaps some neutrophils**
- **Lymphocytes, lymphoid follicles**
- **REGENERATIVE CHANGES**
  - **METAPLASIA, intestinal**
  - **ATROPHY, mucosal hypoplasia, “thinning”**
  - **DYSPLASIA**

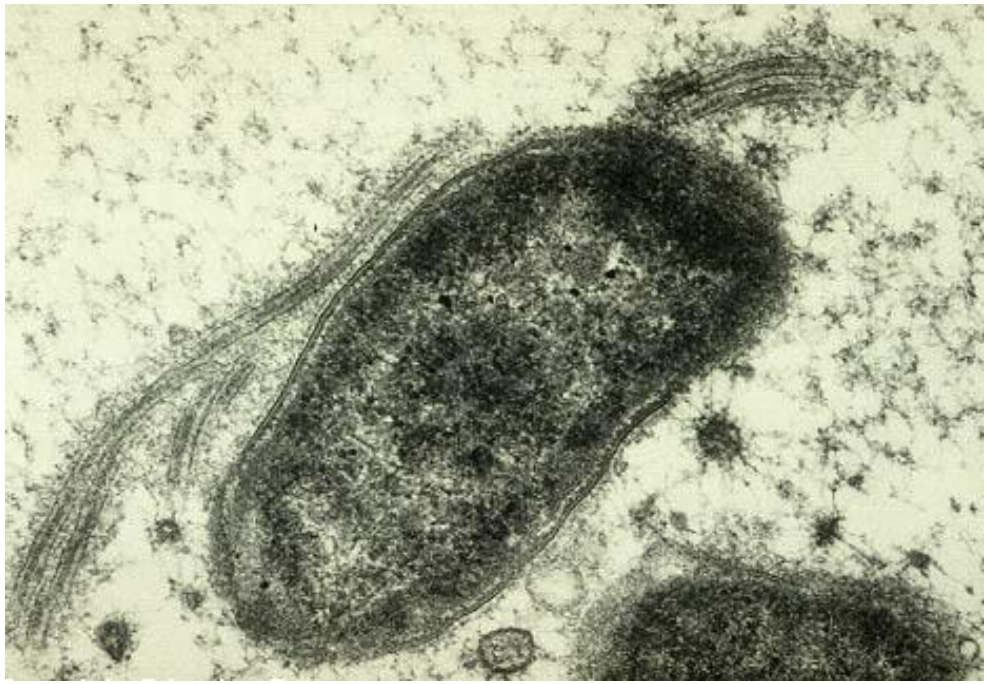




H. pylori Gastritis







GASTRITIS

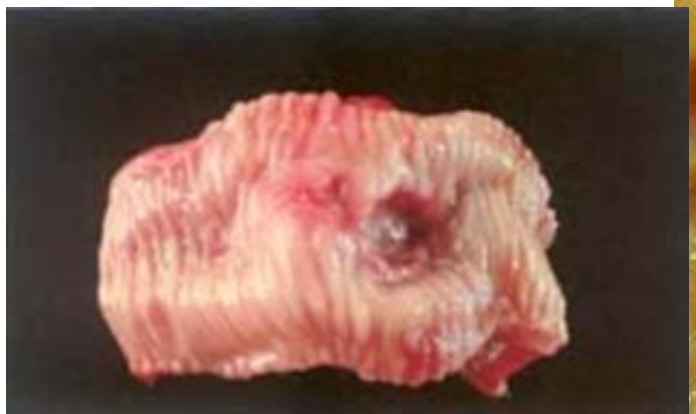
- **AUTOIMMUNE (10%)**
- **ANTIBODIES AGAINST→**
  - **acid producing enzyme H^+**
  - **K^+ -ATPase**
  - **gastrin receptor**
  - **and intrinsic factor**

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→SEROSA**
- **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

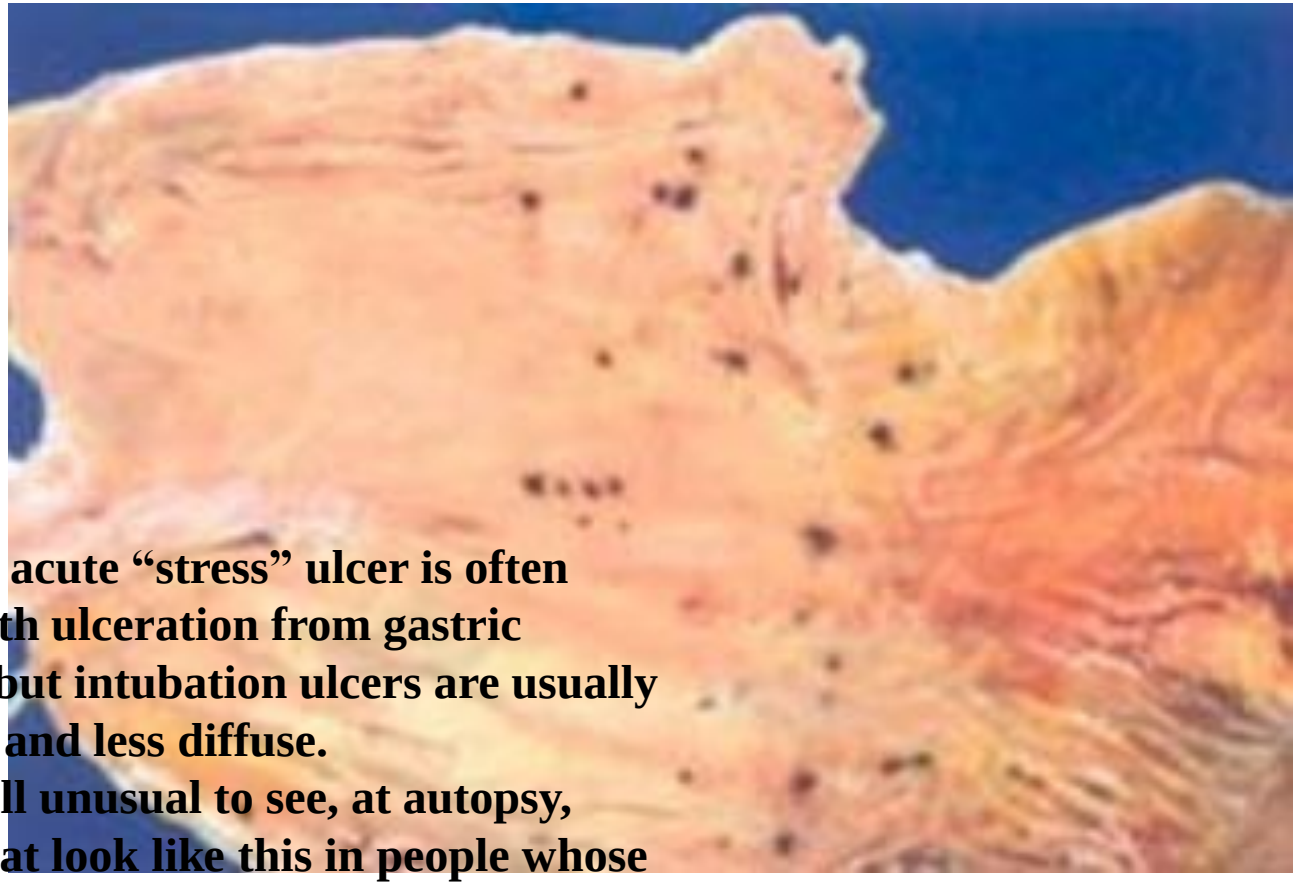
- **Bleeding**
  - **Occurs in 15% to 20% of patients**
  - **Most frequent complication**
  - **May be life-threatening**
  - **Accounts for 25% of ulcer deaths**
  - **May be the first indication of an ulcer**
- **Perforation**
  - **Occurs in about 5% of patients**
  - **Accounts for two thirds of 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**

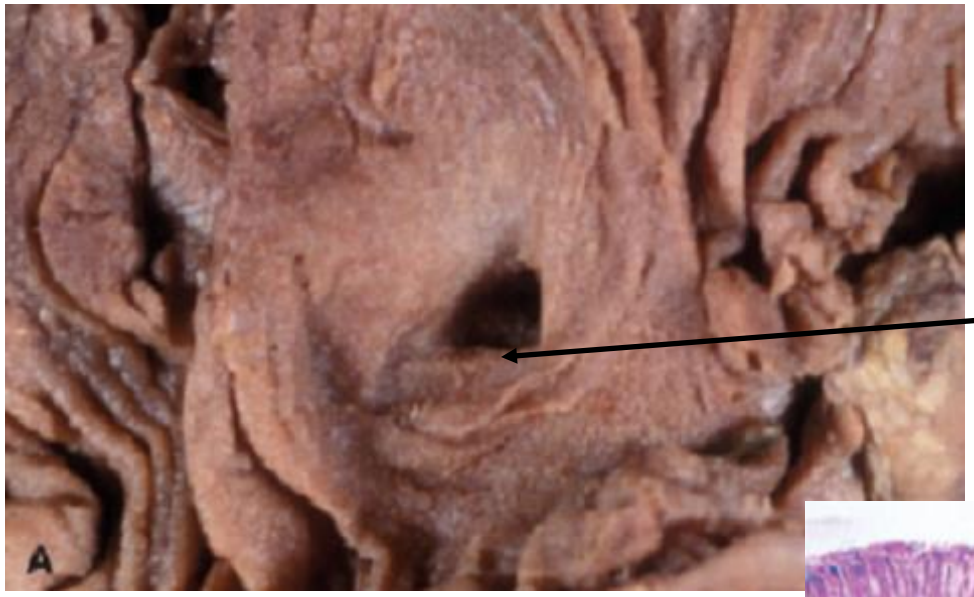
"ACUTE" ULCERS

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

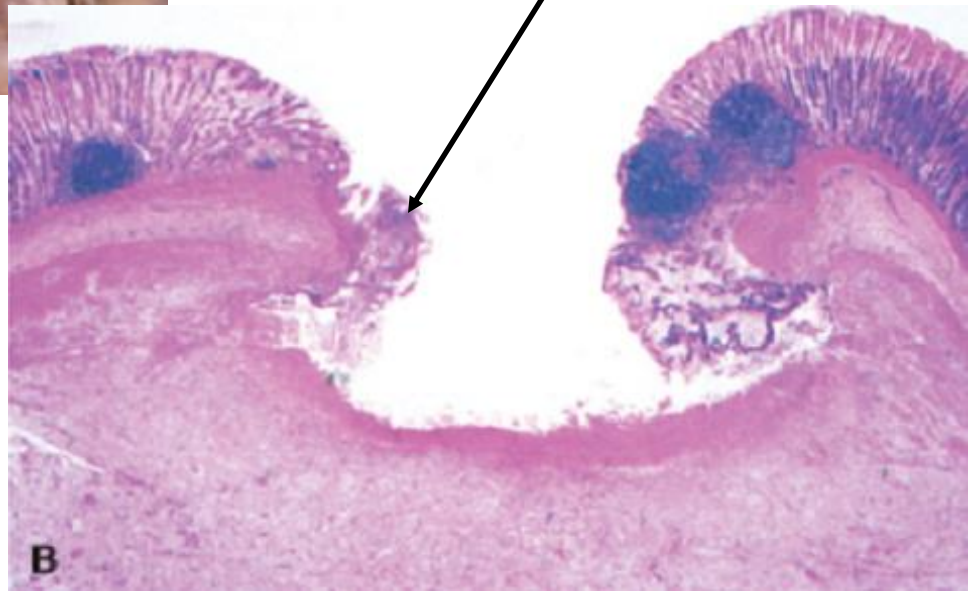


This type of acute "stress" ulcer is often confused with ulceration from gastric intubation, but intubation ulcers are usually more linear and less diffuse.
It is not at all unusual to see, at autopsy, stomachs that look like this in people whose death was preceded by extreme physiologic stress.

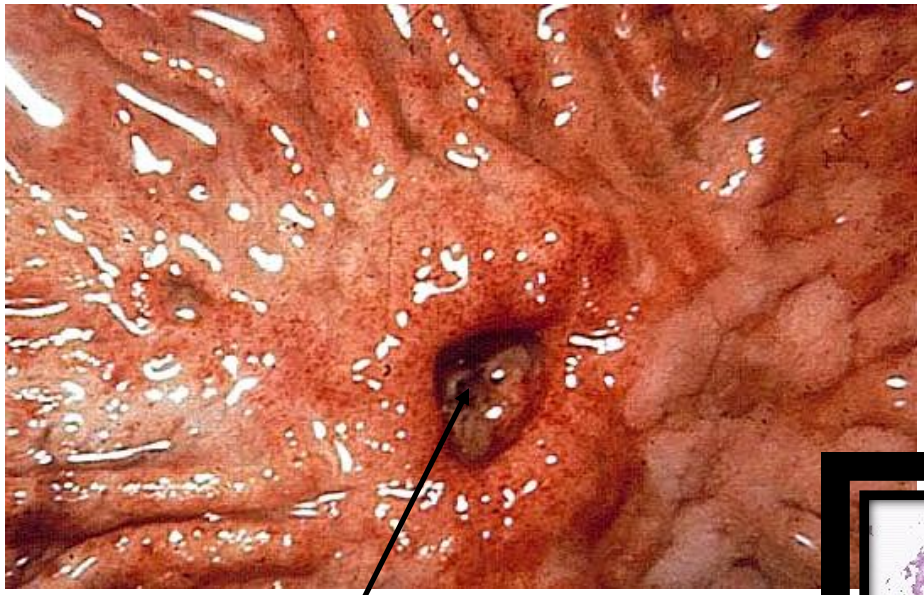
Duodenal Ulcer - Peptic Ulcer

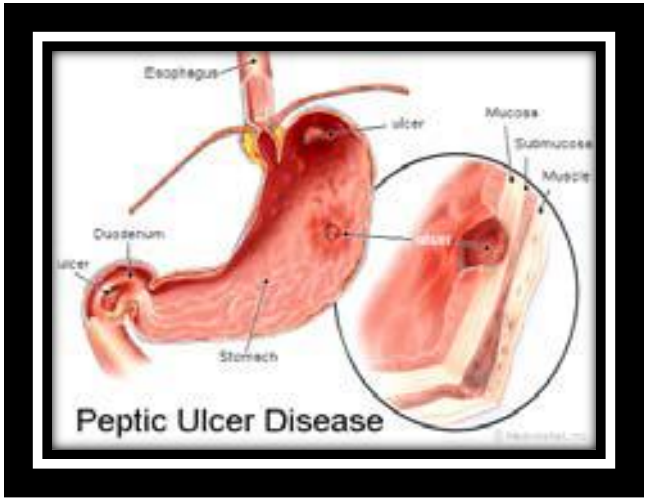


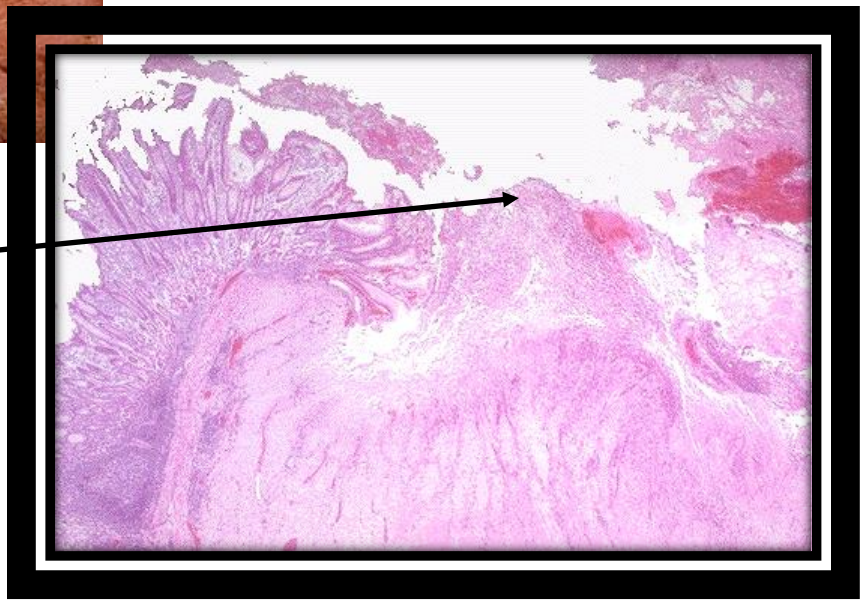
Loss of mucosal layer



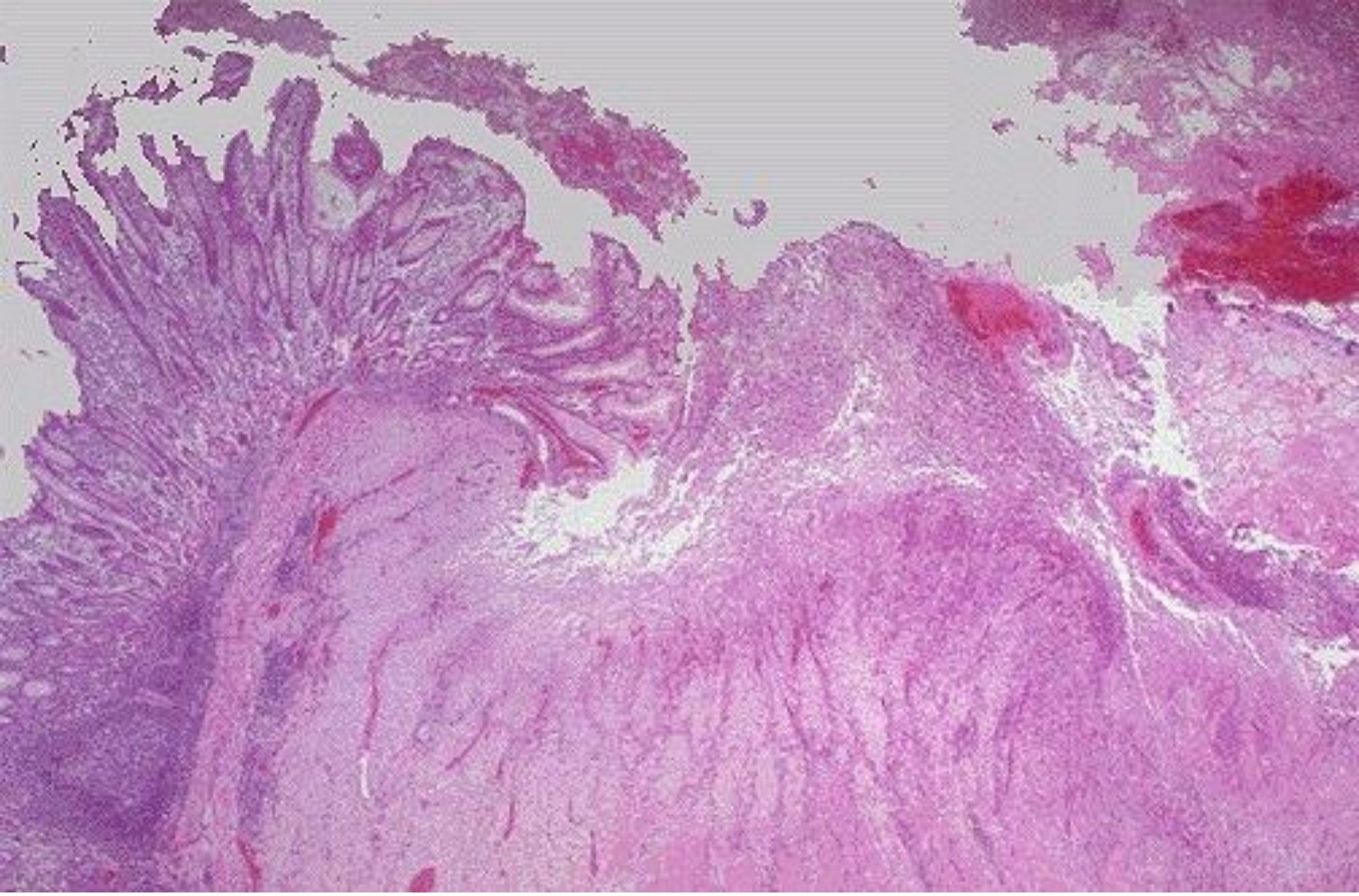
Gastric Ulcer

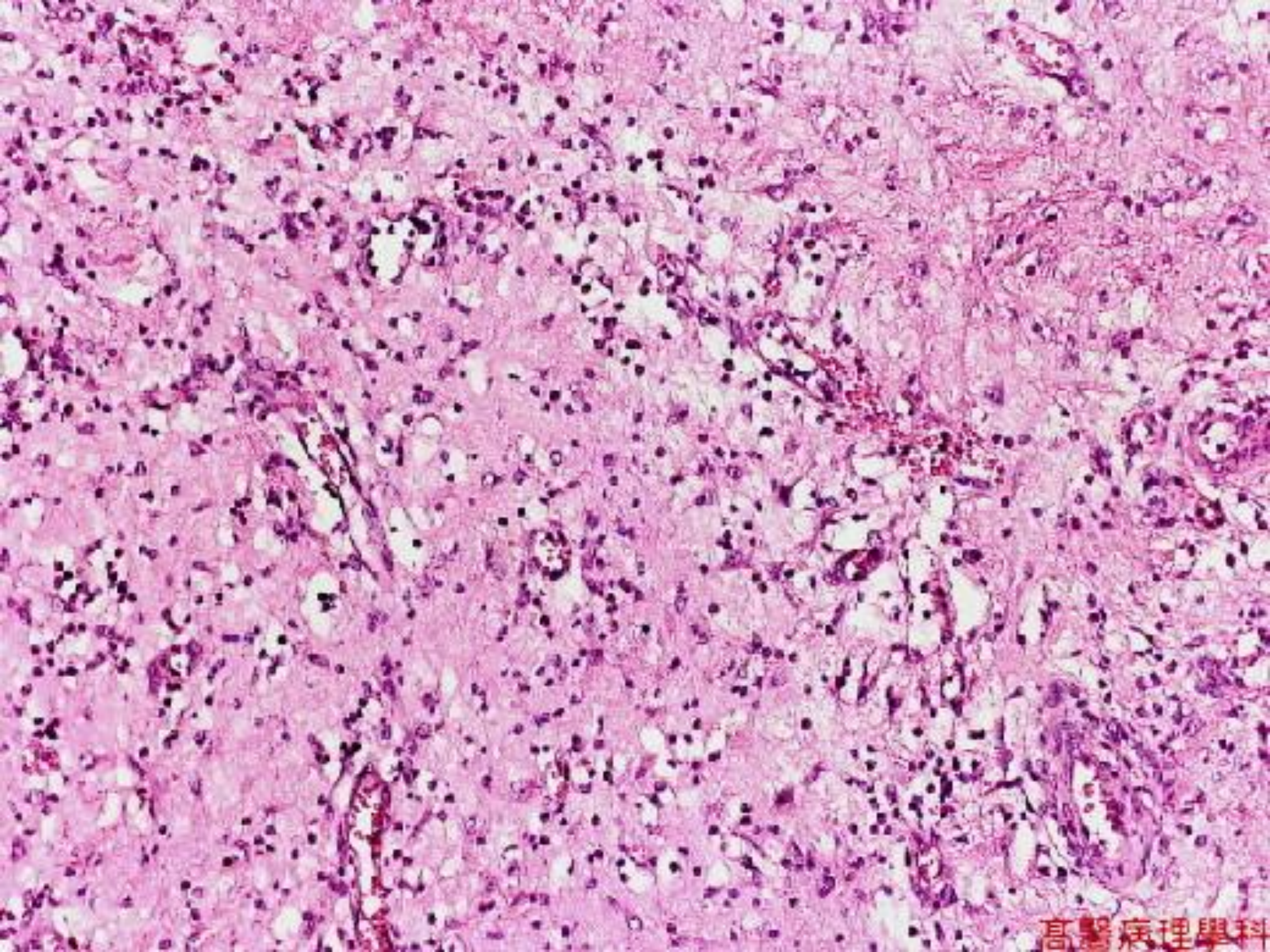


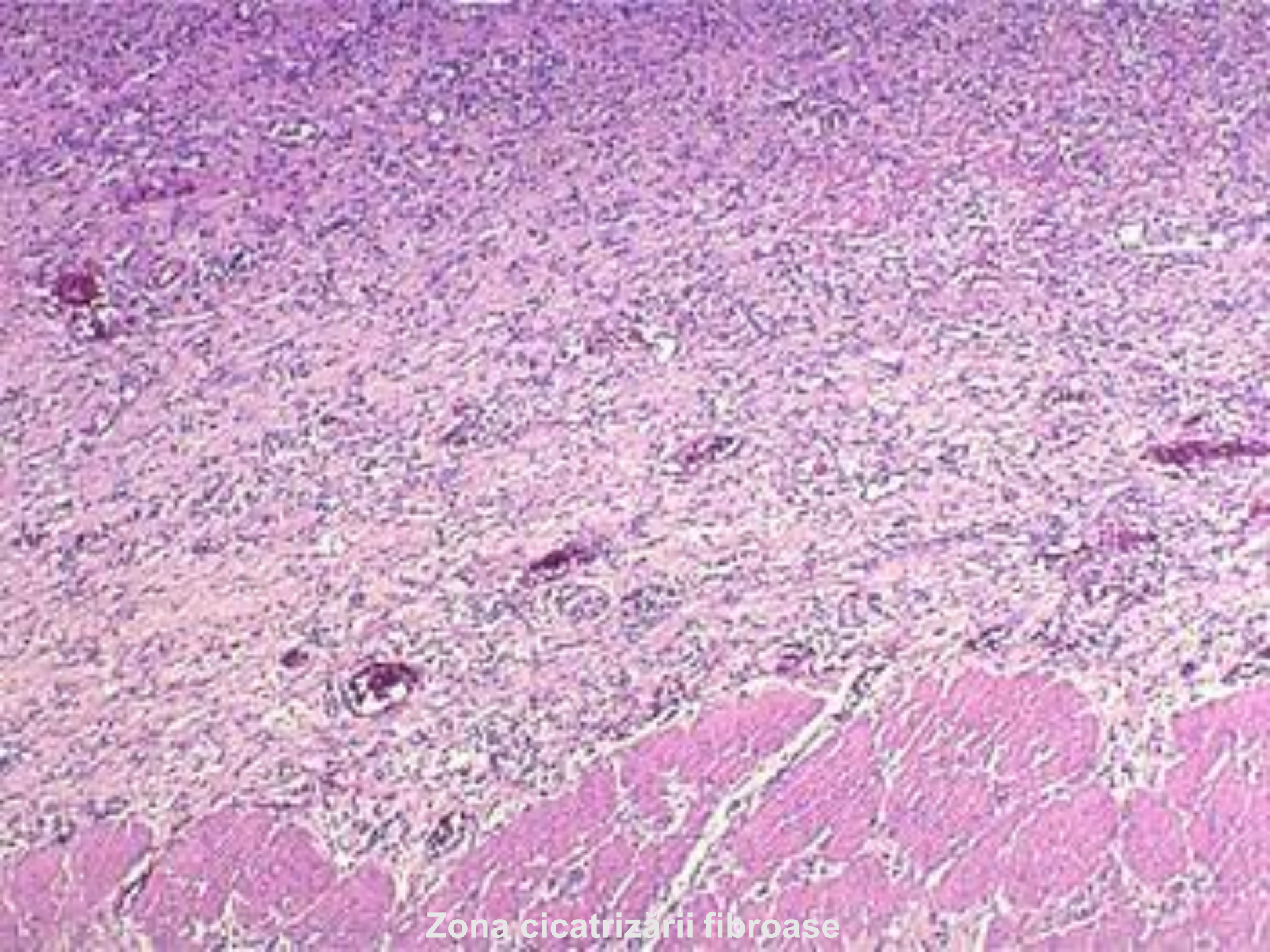




Loss of mucosa




高醫病理學科


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

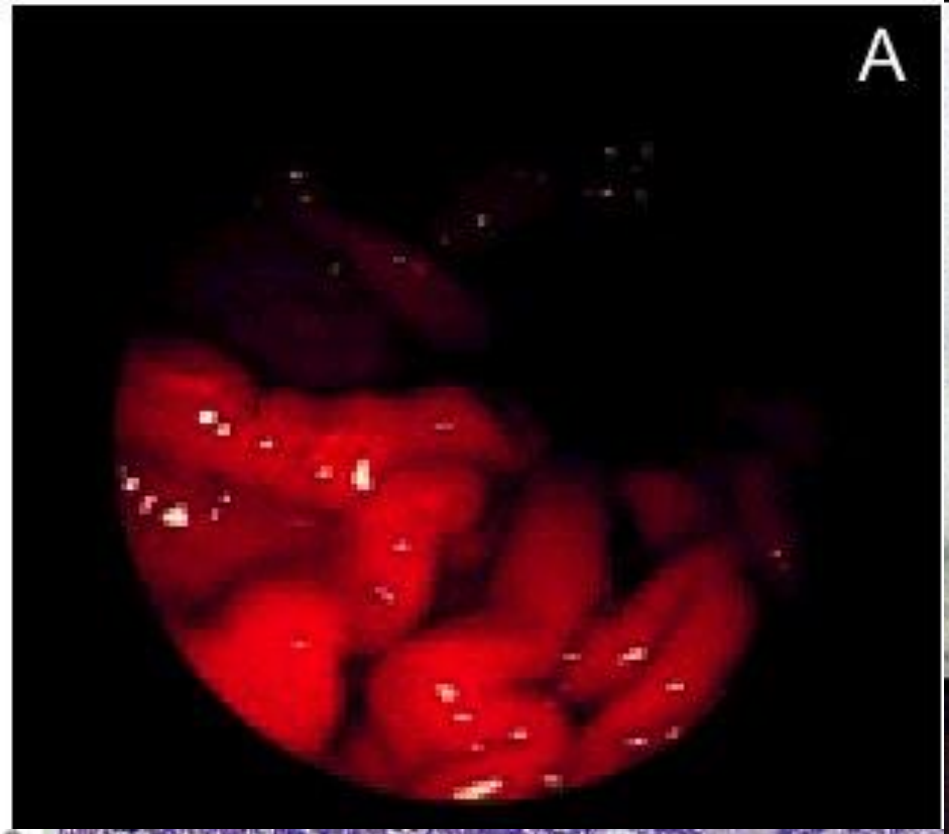
RUGAL PROMINENCE (cerebriform)

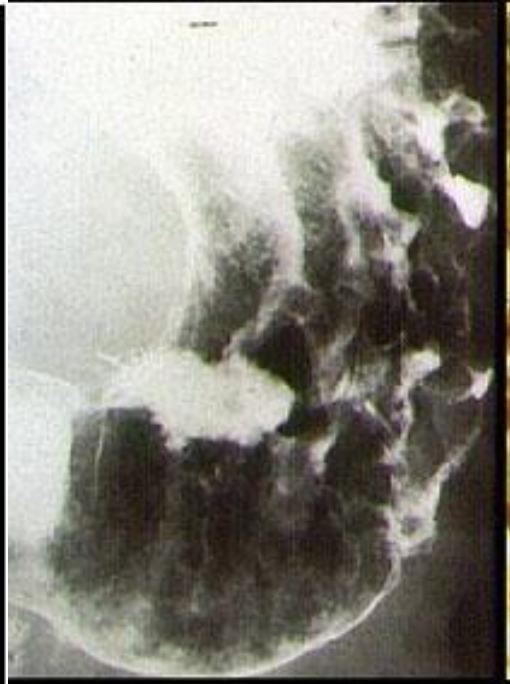
NO INFLAMMATION

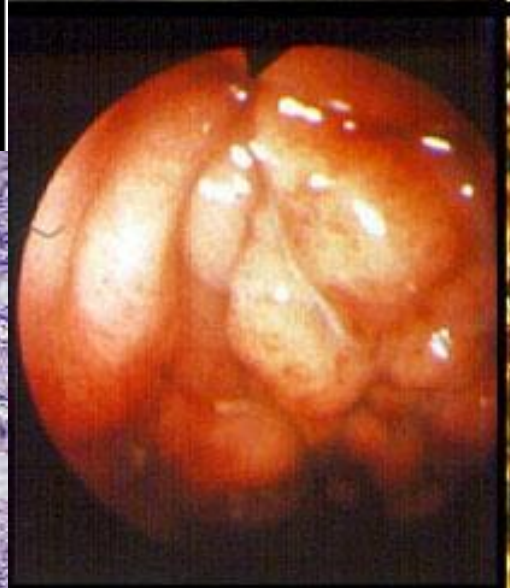
HYPERPLASIA of MUCOSA

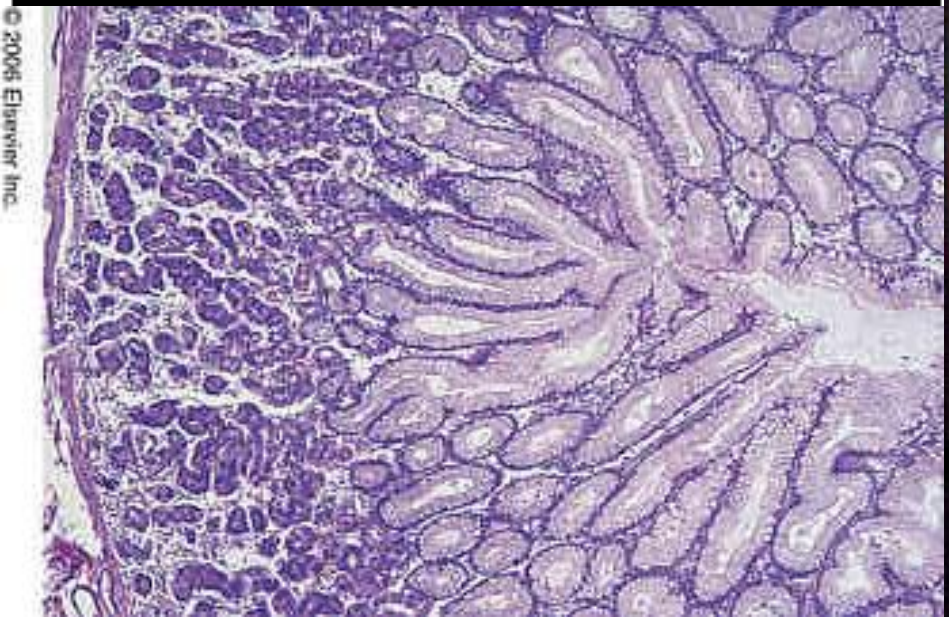
“HYPERTROPHIC” GASTROPATHY

- **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)***



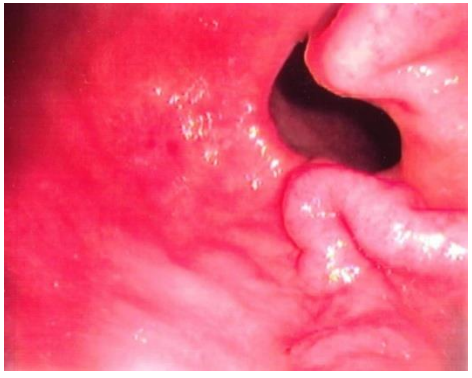


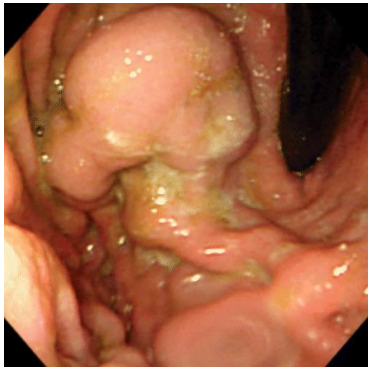




GASTRIC "VARICES"

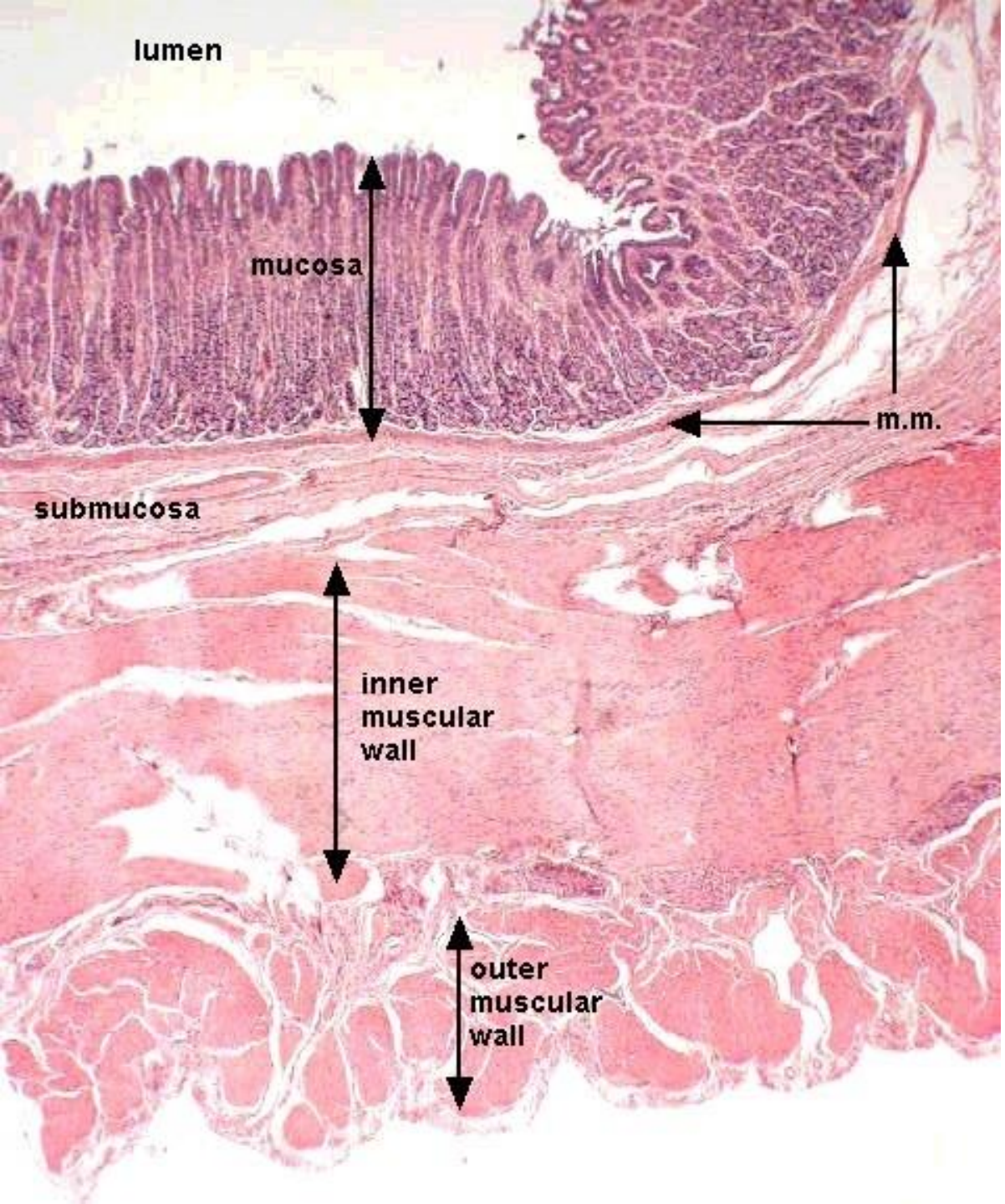
- **SAME SETTING AND ETIOLOGY AS ESOPHAGEAL VARICES, i.e., PORTAL HYPERTENSION**
- **NOT AS COMMON AS ESOPHAGEAL VARICES**
- **MAY LOOK LIKE PROMINENT RUGAE**
- **IF A PATIENT HAS GASTRIC VARICES, HE ALSO PROBABLY HAS ESOPHAGEAL, (but probably not vice versa)**





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)**
  - **CARCINOID (NEUROENDOCRINE)**



BENIGN TUMORS

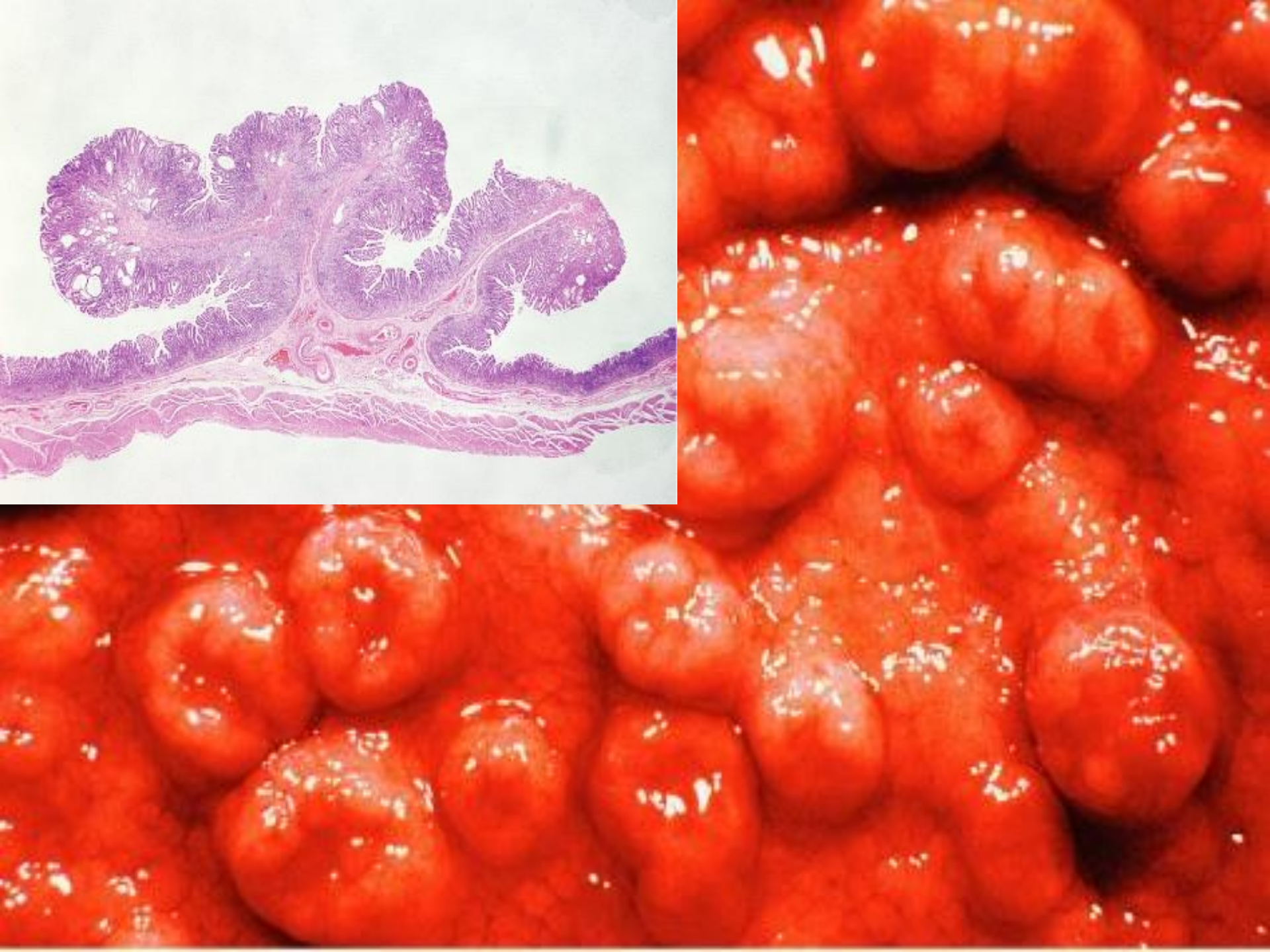
BEBNIBGNB

MUCOSA (POLYPS)

---HYPERPLASTIC

---Fundic

---ADENOMATOUS



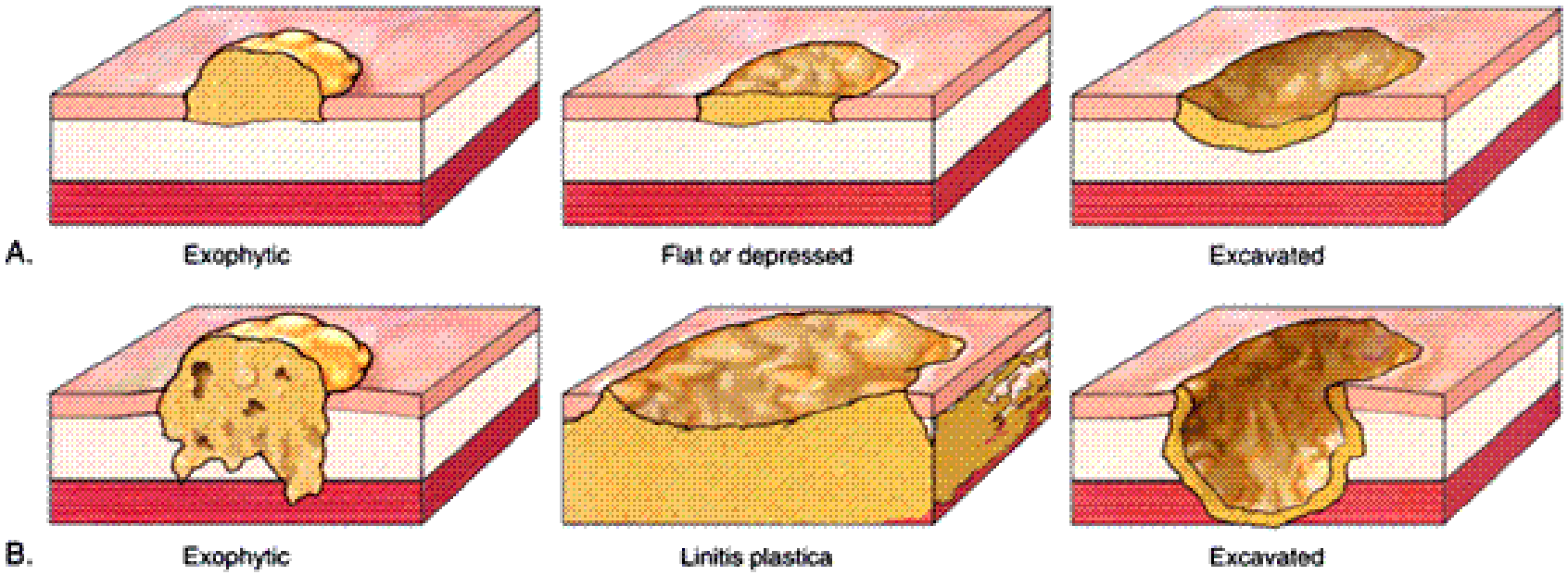
WHO GASTRIC NEOPLASMS

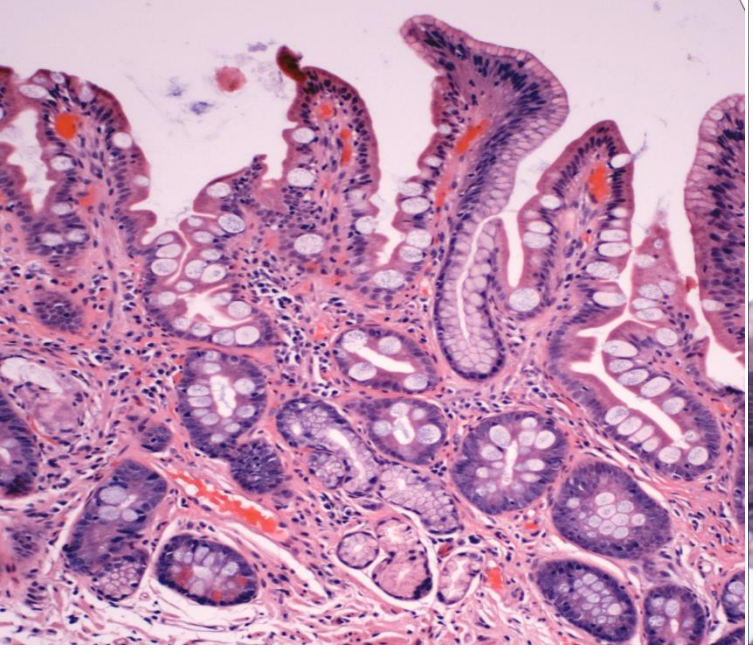
- ***Epithelial Tumors: Adenomatous polyps, Adenocarcinoma (papillary, tubular, mucinous, signet ring, adenosquamous, unclassified), Small cell, Carcinoid (neuroendocrine)***
- ***Nonepithelial Tumors: Leiomyo(sarc)oma, Schwannoma, GIST, Granular Cell Tumor, Kaposi sarcoma***
- ***Malignant Lymphomas:***

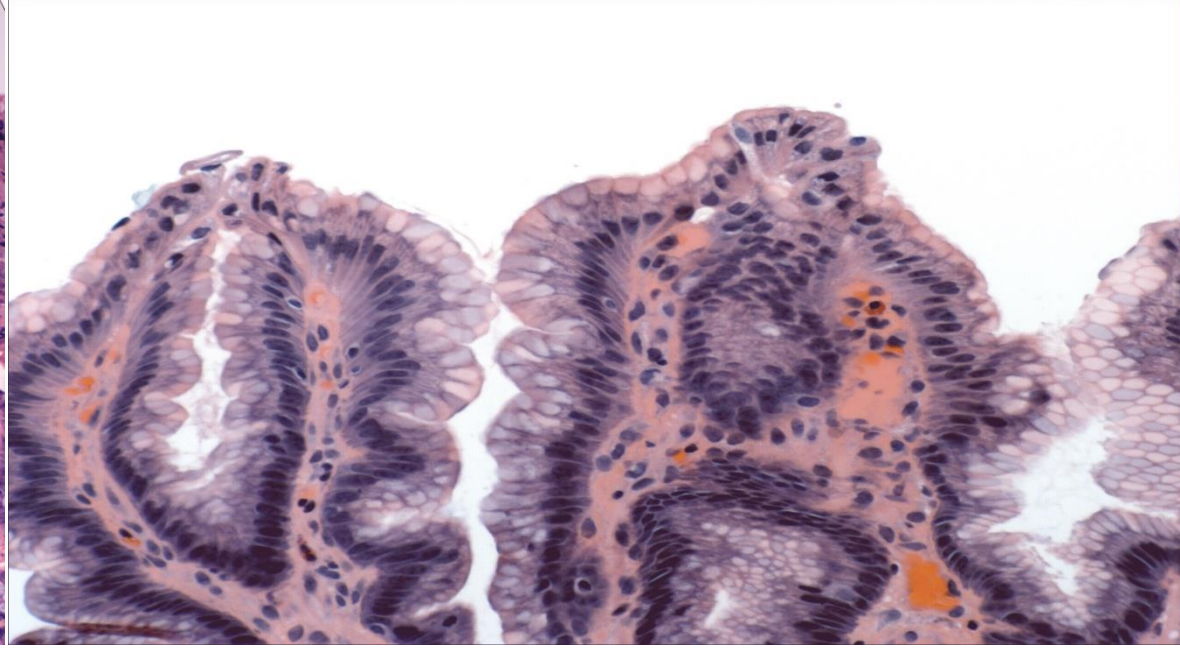
ADENOCARCINOMA RISK FACTORS

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

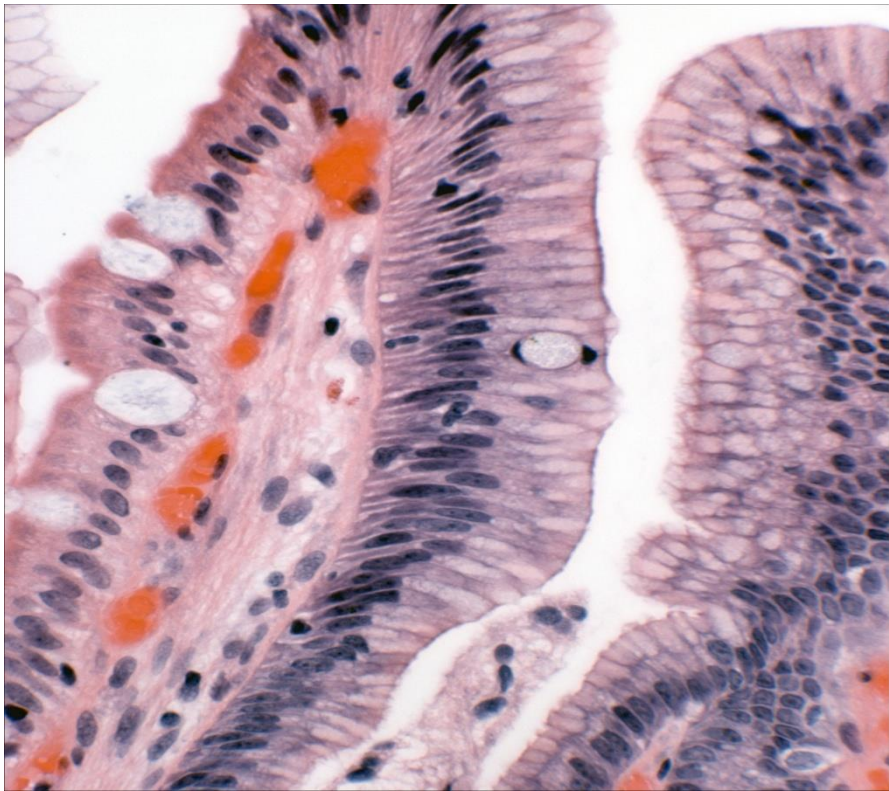
ADENOCARCINOMA GROWTH PATTERNS



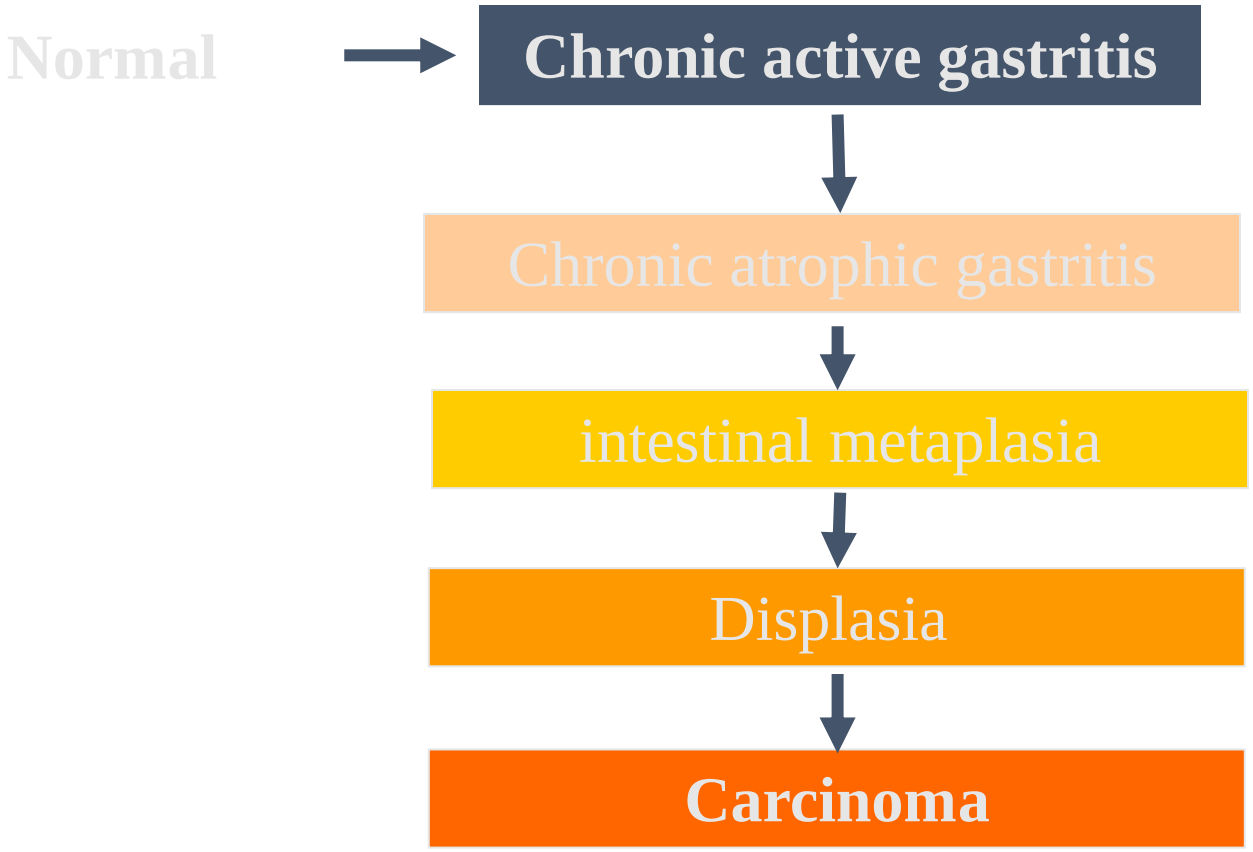


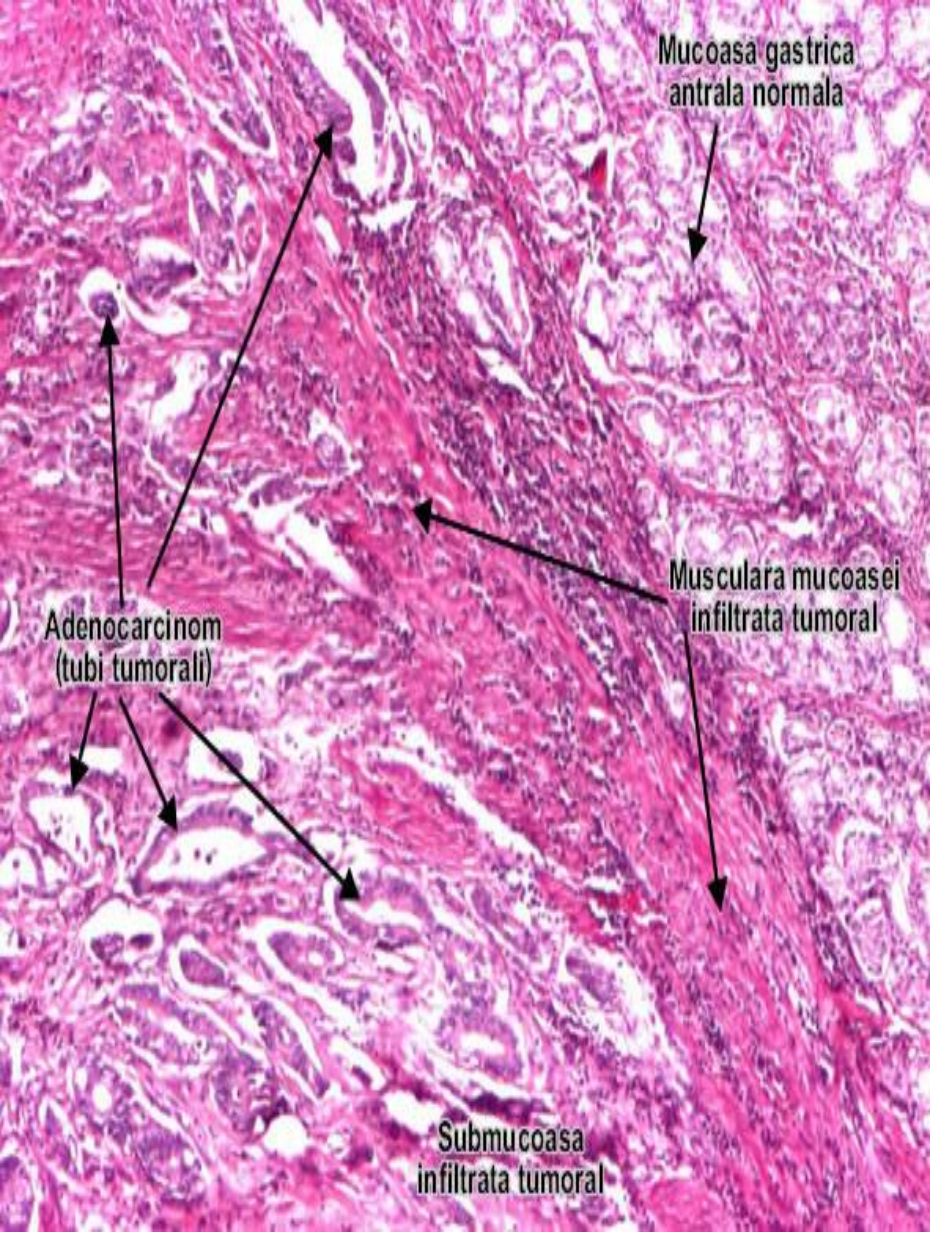


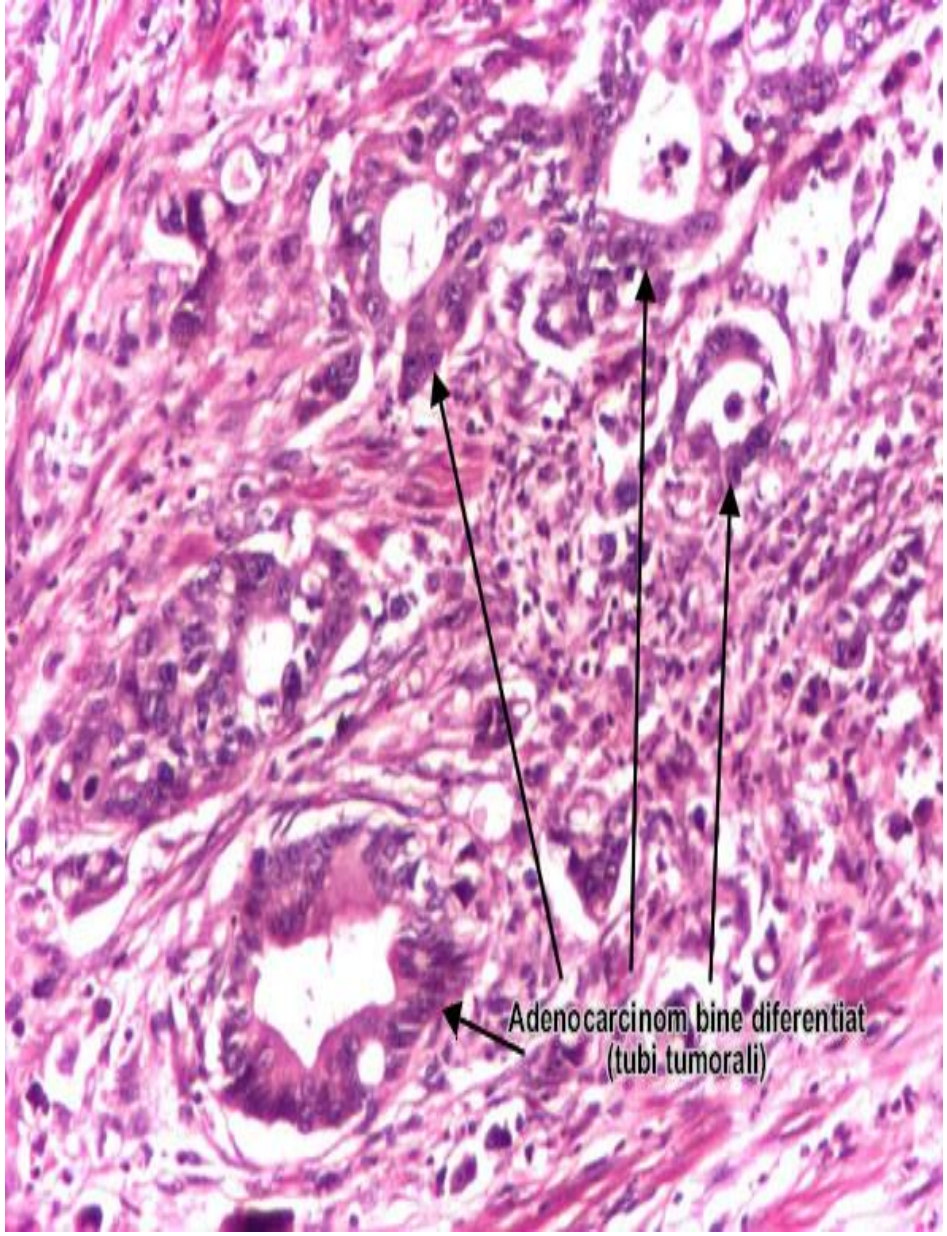
Gastric carcinoma de novo

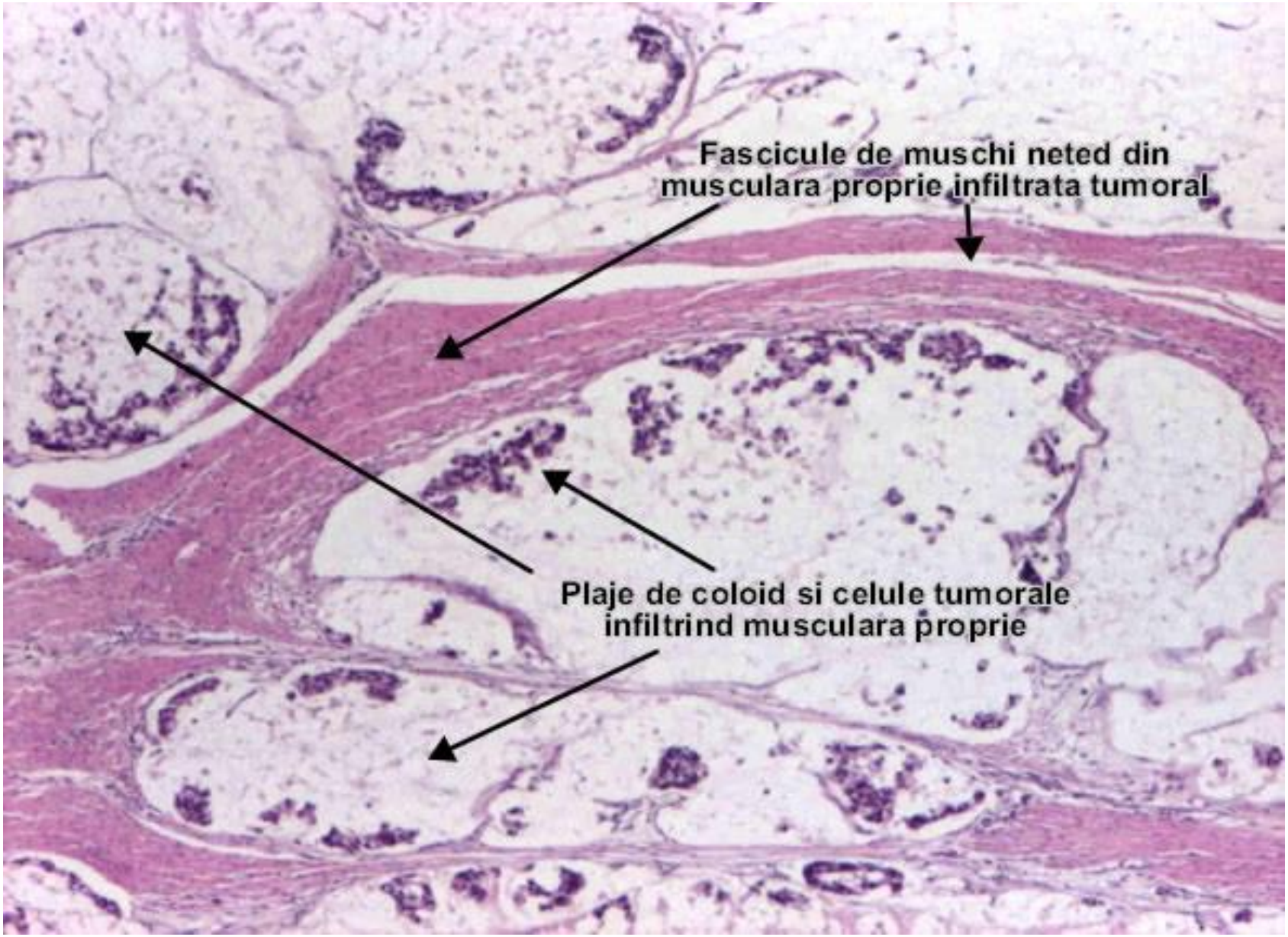


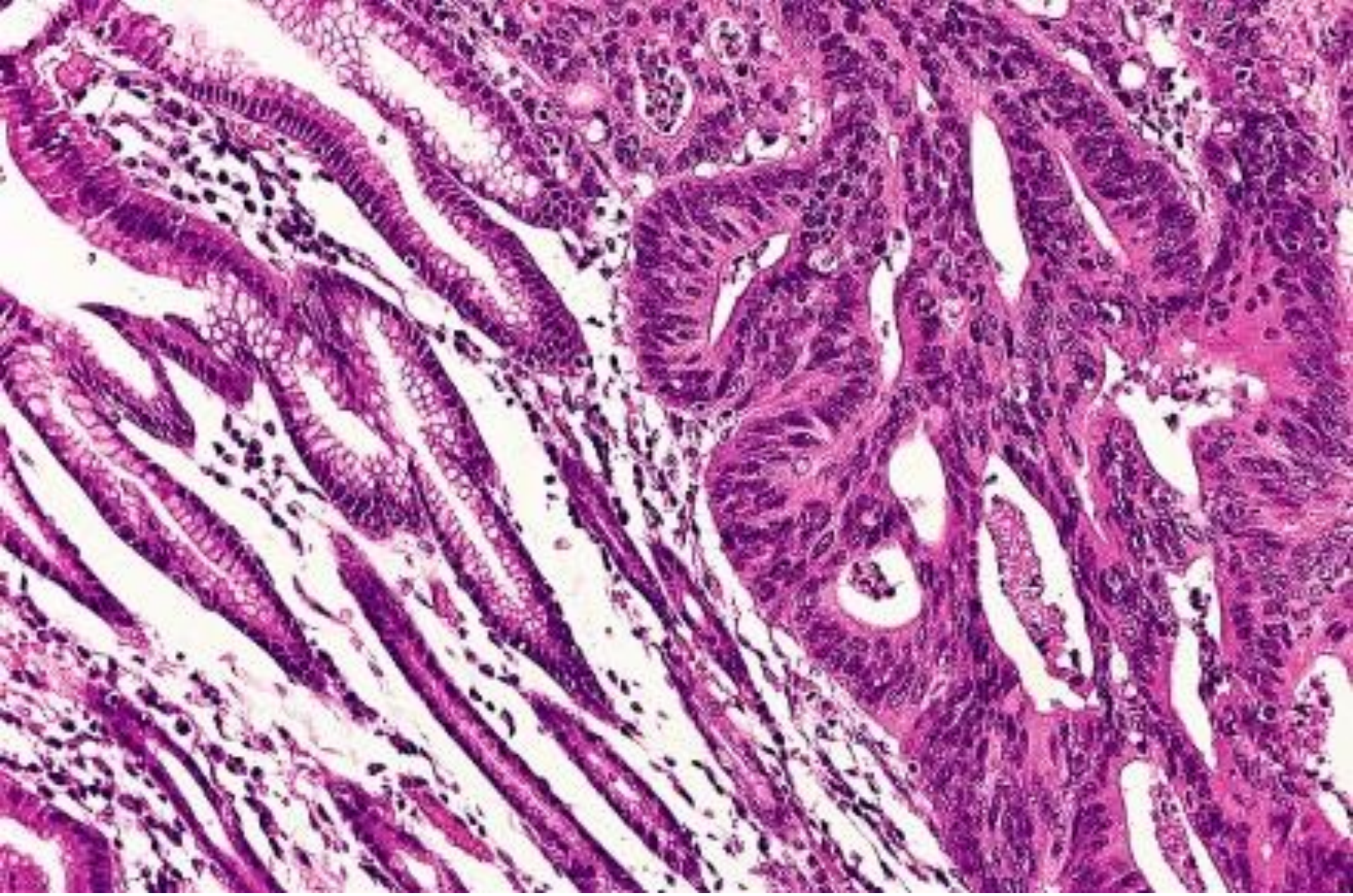
Intestinal type gastric carcinoma, the evolution of on steps.



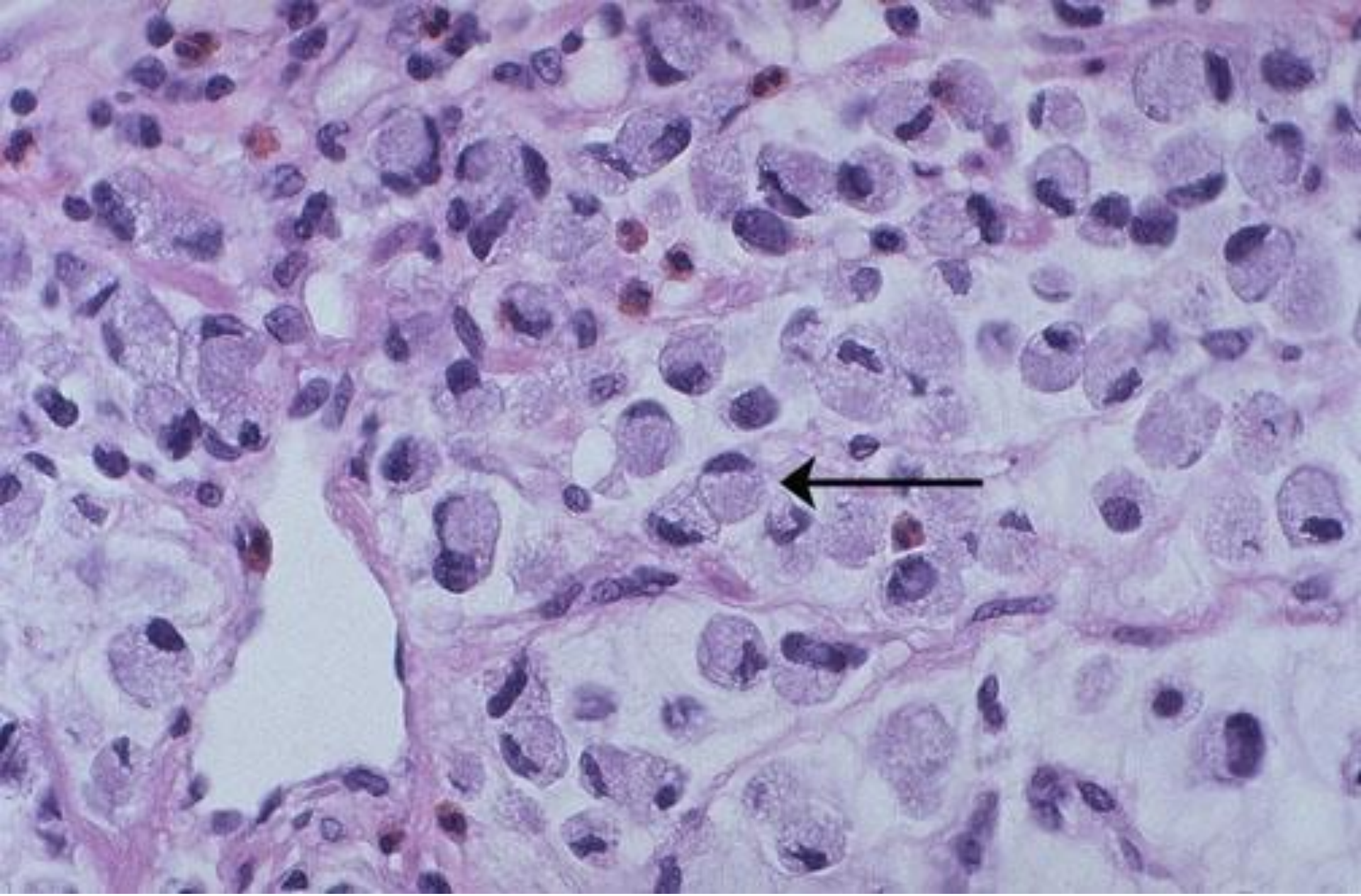

Mucoasa gastrica
antrala normala
Musculara mucoasei
infiltrata tumoral
Adenocarcinom
(tubi tumorali)
Submucoasa
infiltrata tumoral


Adenocarcinom bine diferentiat
(tubi tumorali)


Fascicule de muschi neted din
musculara proprie infiltrata tumoral
Plaje de coloid si celule tumorale
infiltrind musculara proprie



Papillary adenocarcinoma



Gastric carcinoma signet ring cells.

GASTRIC CARCINOMA - TNM staging

T – tumor comprises

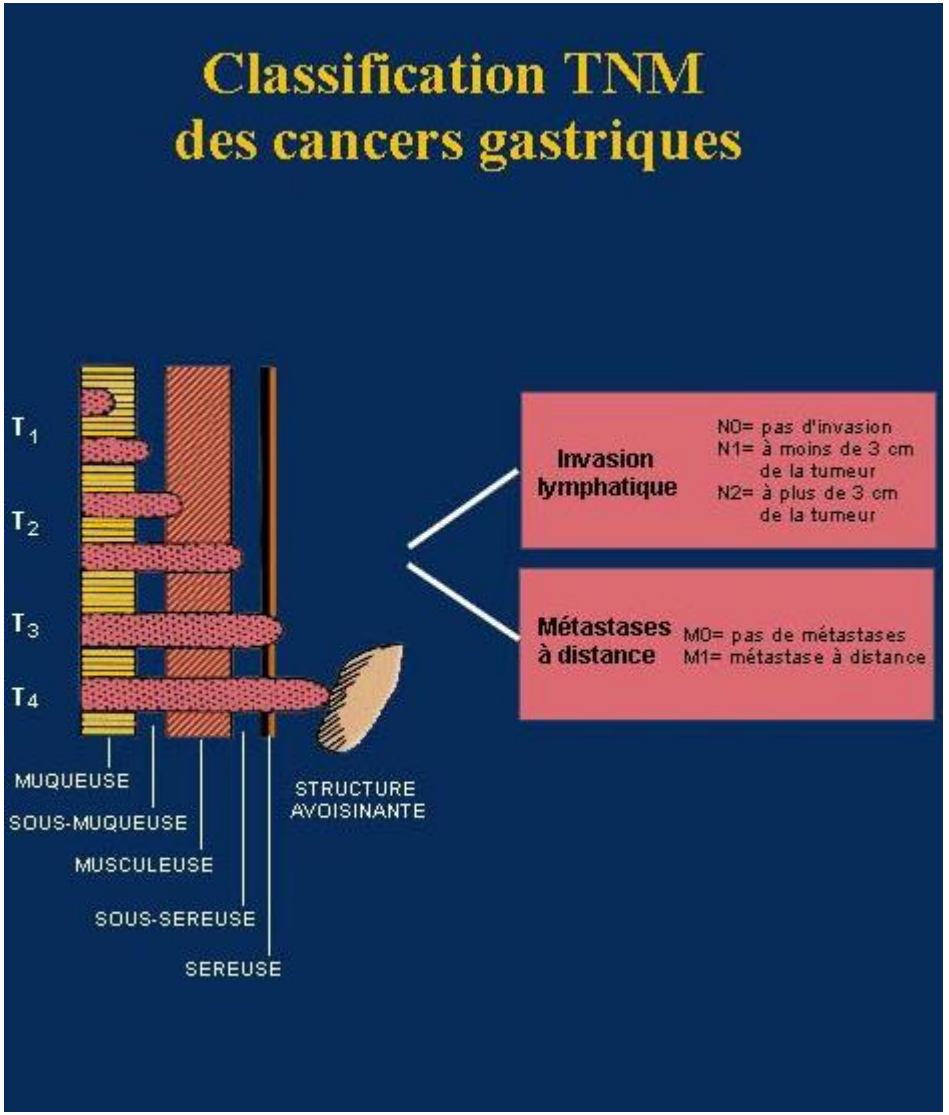
- T1 – mucosa and submucosa
- T2 – muscle
- T3 – serosa
- T4 – adjacent organs

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

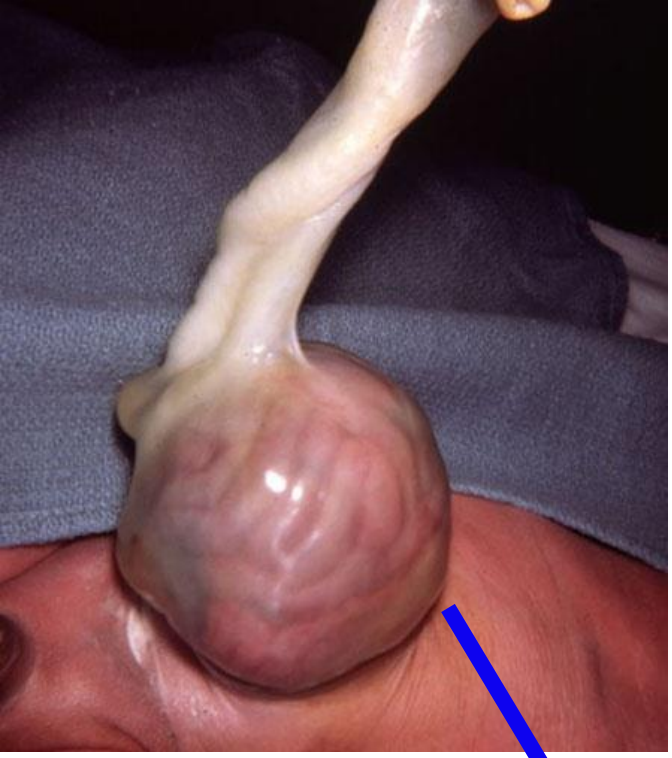


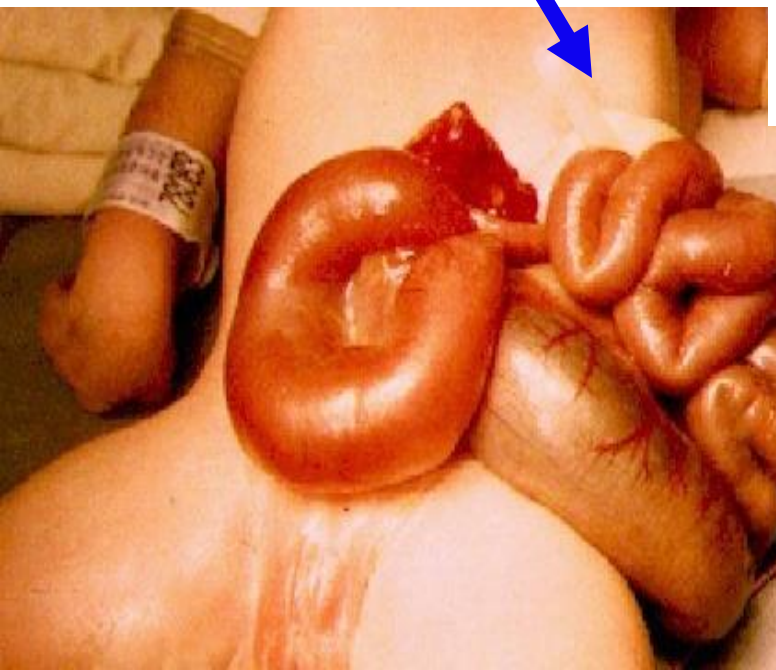
SMALL/LARGE INTESTINE

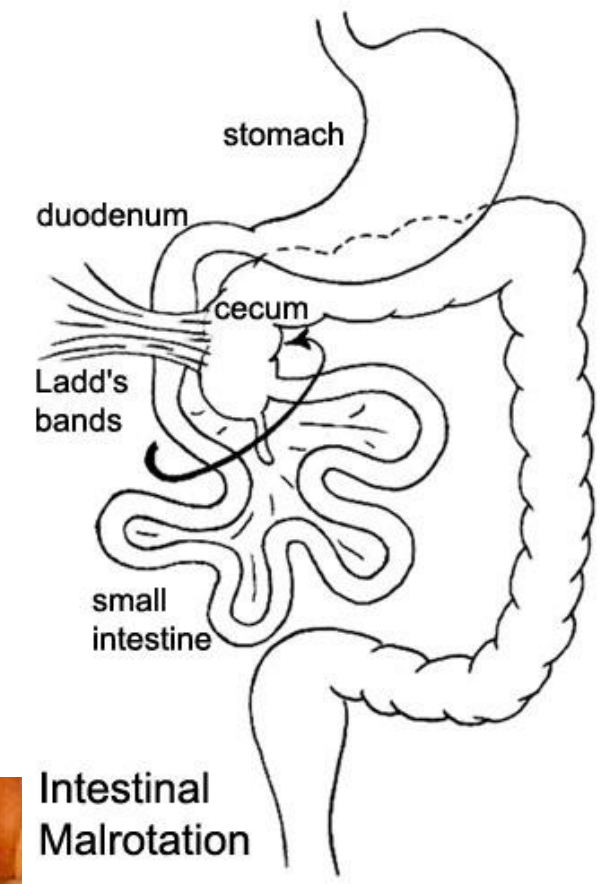
- **NORMAL: Anat., Vasc., Mucosa, Endocr., Immune, Neuromuscular.**
- **PATHOLOGY:**
  - **CONGENITAL**
  - **ENTEROCOLITIS: DIARRHEA, INFECTIOUS, OTHER**
  - **MALABSORPTION: INTRALUMINAL, CELL SURFACE, INTRACELL.**
  - **(I)IBD: CROHN DISEASE and ULCERATIVE COLITIS**
  - **VASCULAR: ISCHEMIC, ANGIODYSPLASIA, HEMORRHAGIC**
  - **DIVERTICULOSIS/-ITIS**
  - **OBSTRUCTION: MECHANICAL, PARALYTIC (ILEUS) (PSEUDO)**
  - **TUMORS: BENIGN, MALIGNANT, EPITHELIAL, STROMAL**

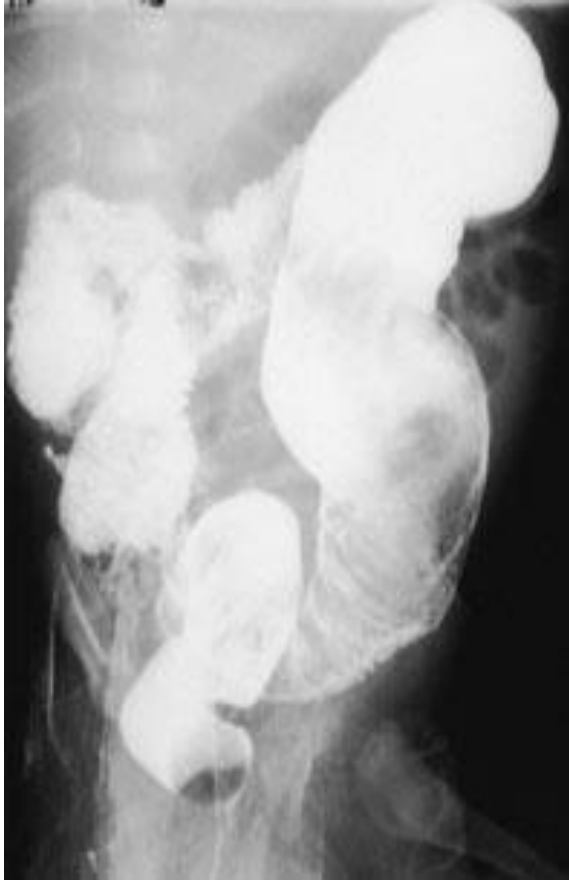
CONGENITAL

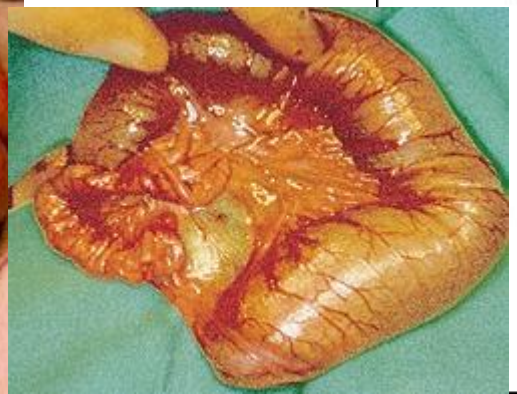
- **DUPLICATION**
- **MALROTATION**
- **OMPHALOCELE**
- **ATRESIA/STENOSIS SPECTRUM**
- **MECKEL (terminal ileum, “vitelline” duct)**
- **AGANGLIONIC MEGACOLON (HIRSCHSPRUNG DISEASE)**

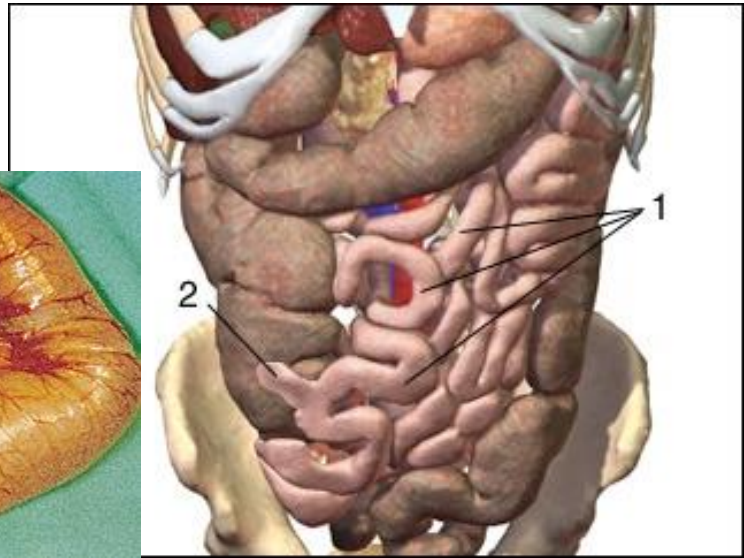


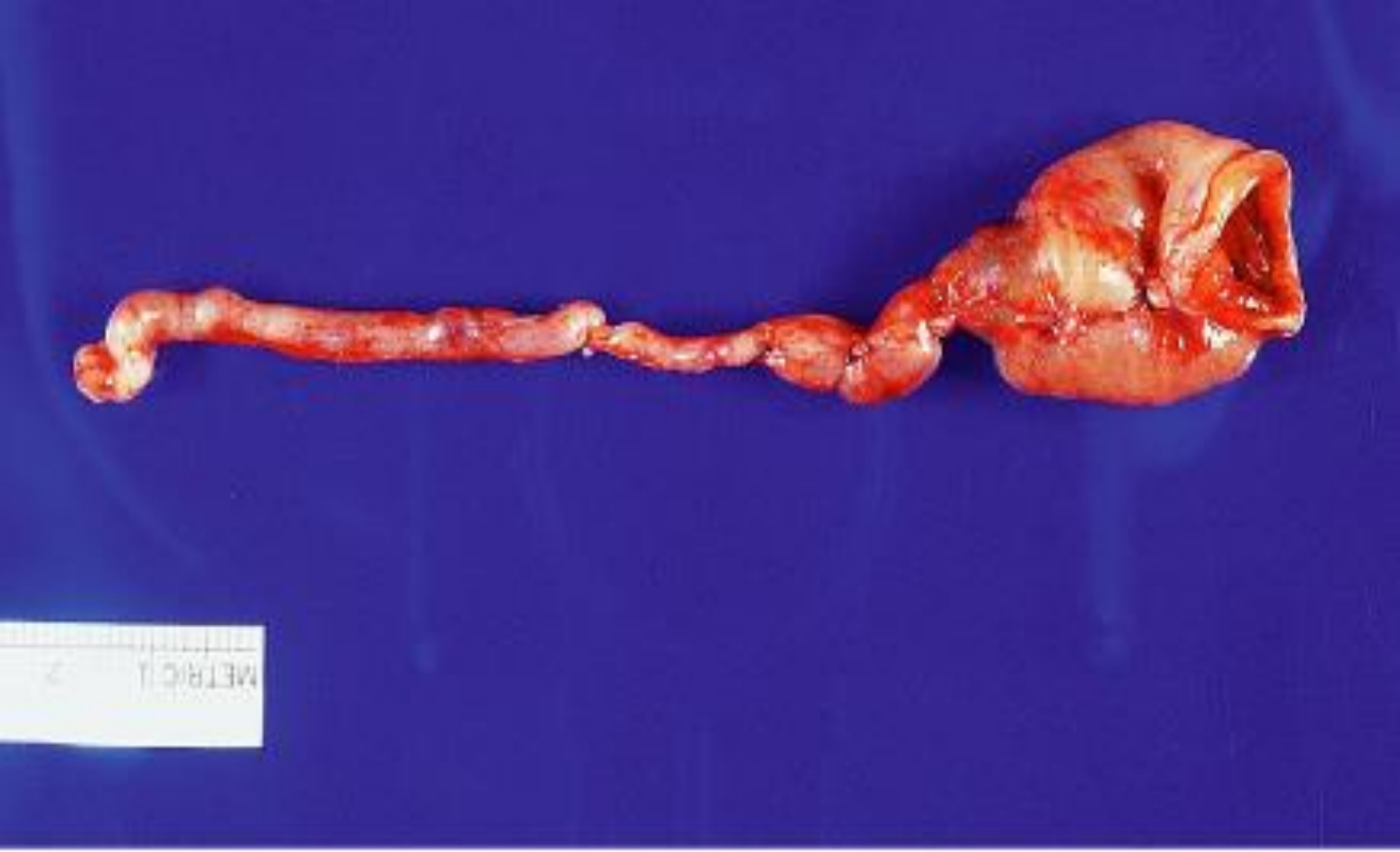


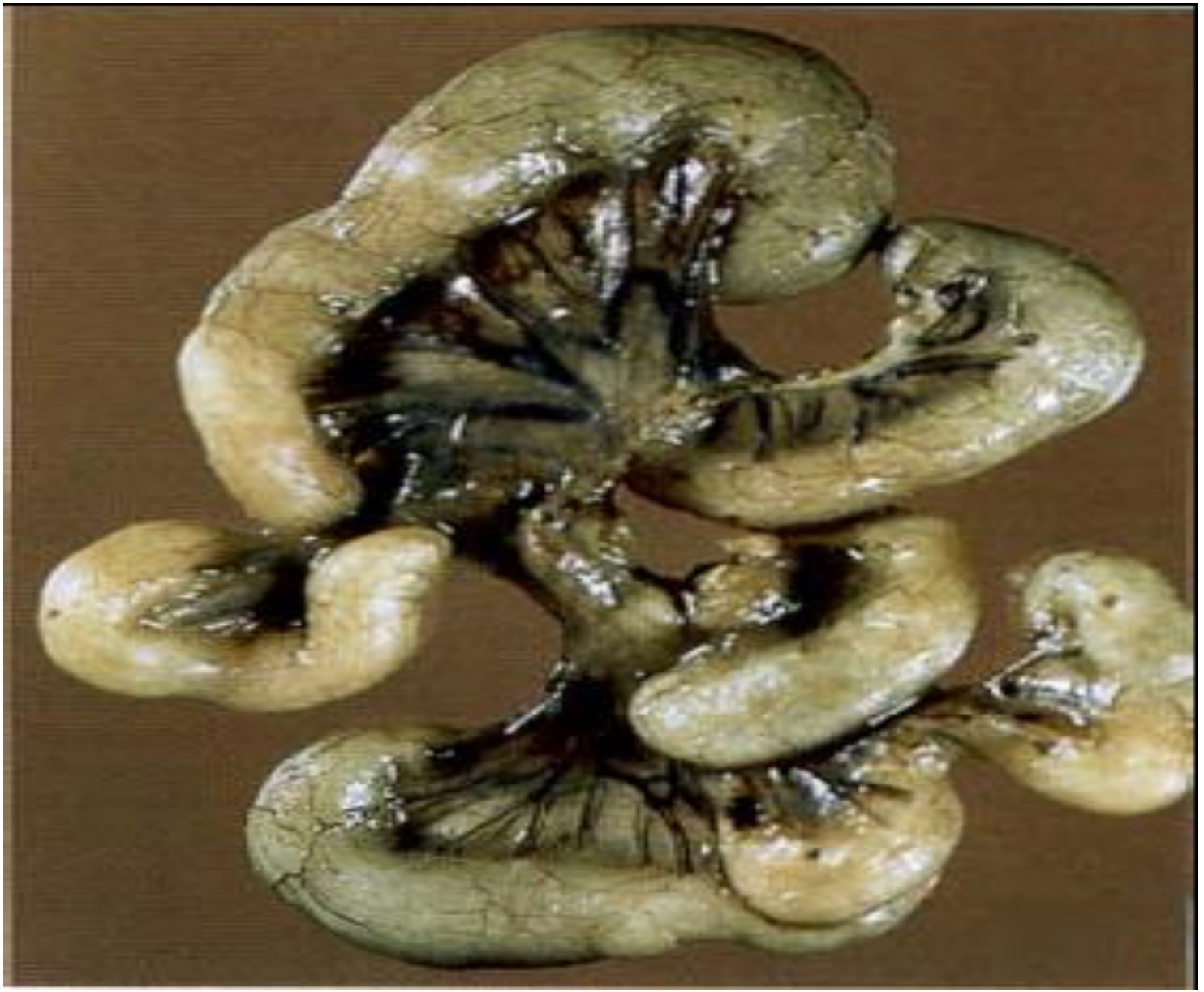

stomach
duodenum
cecum
Ladd's
bands
small
intestine
Intestinal
Malrotation

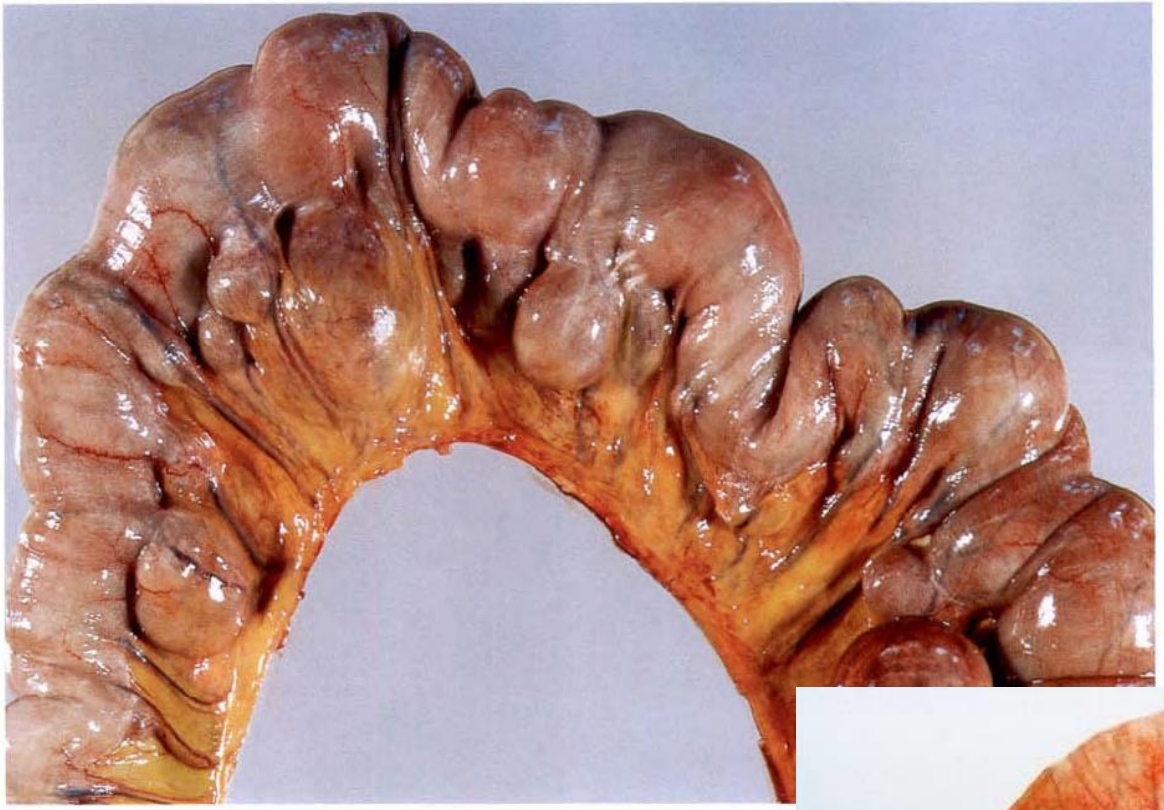


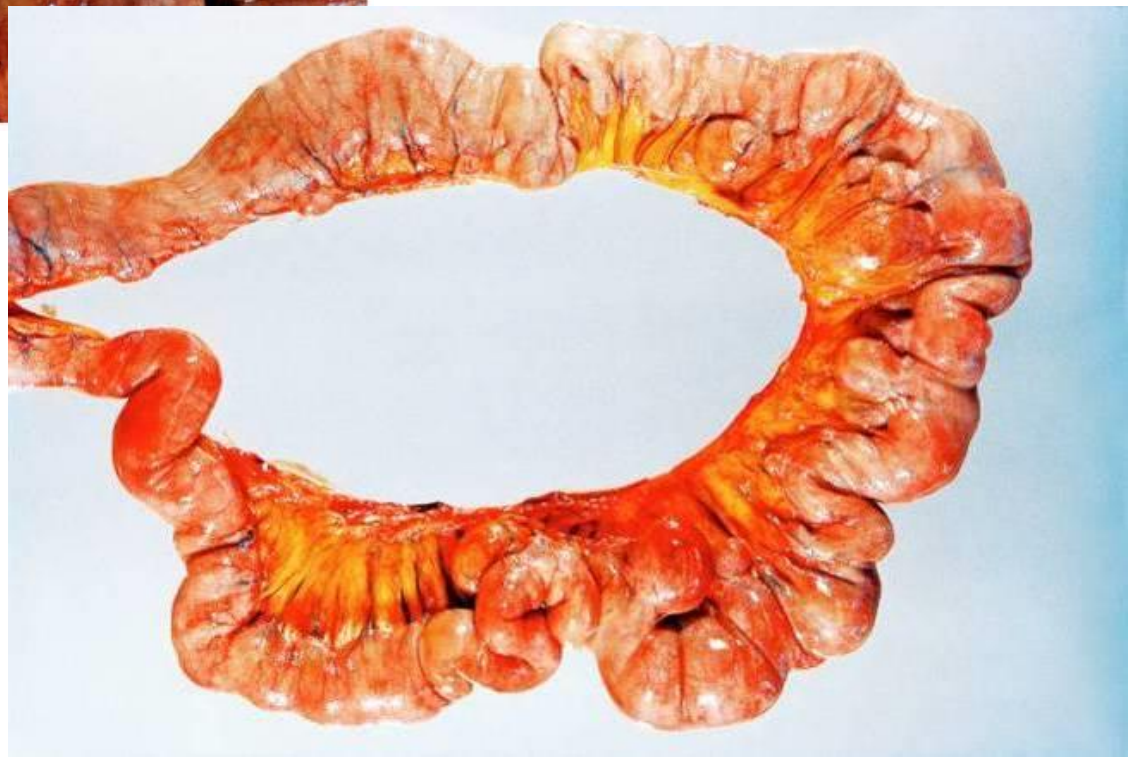


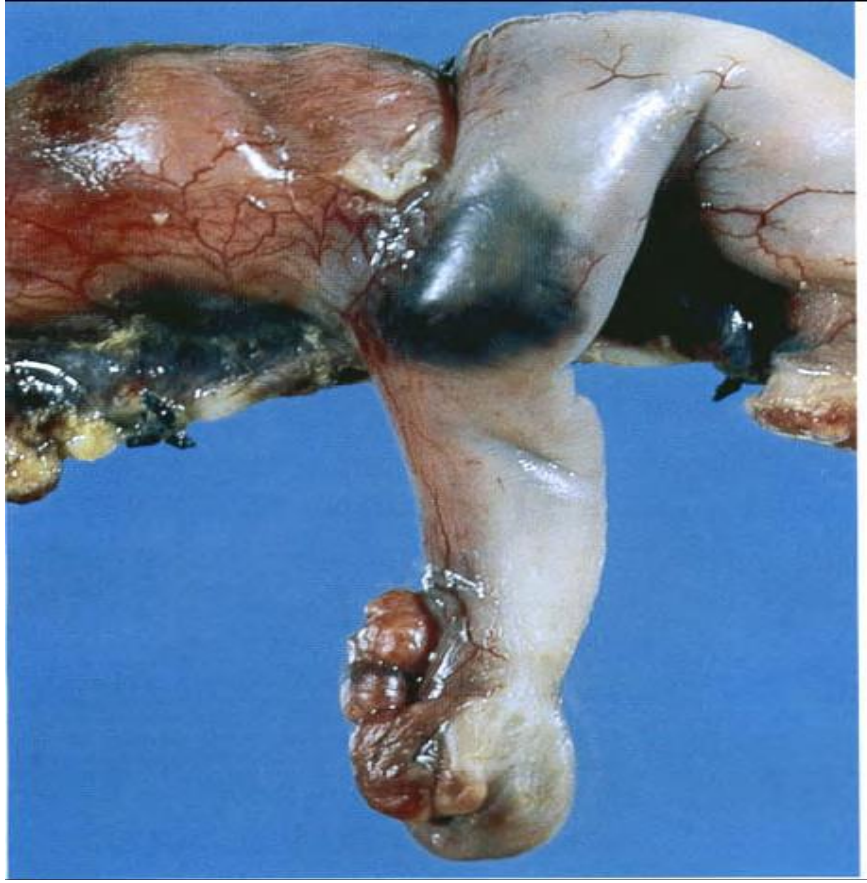

1
2

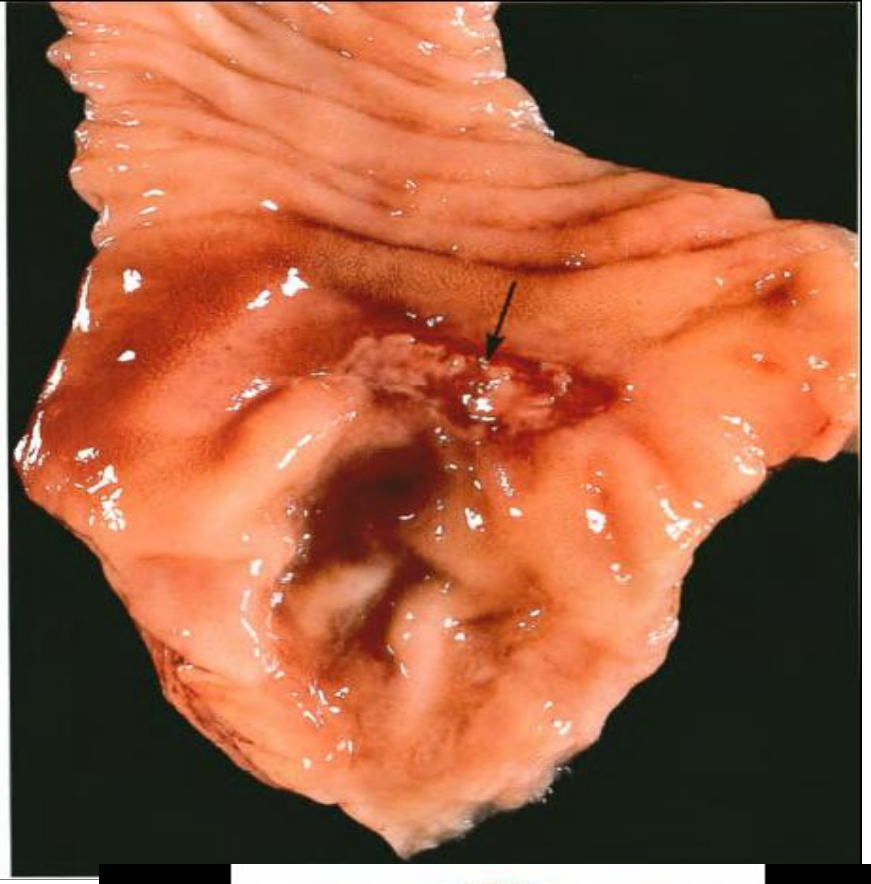


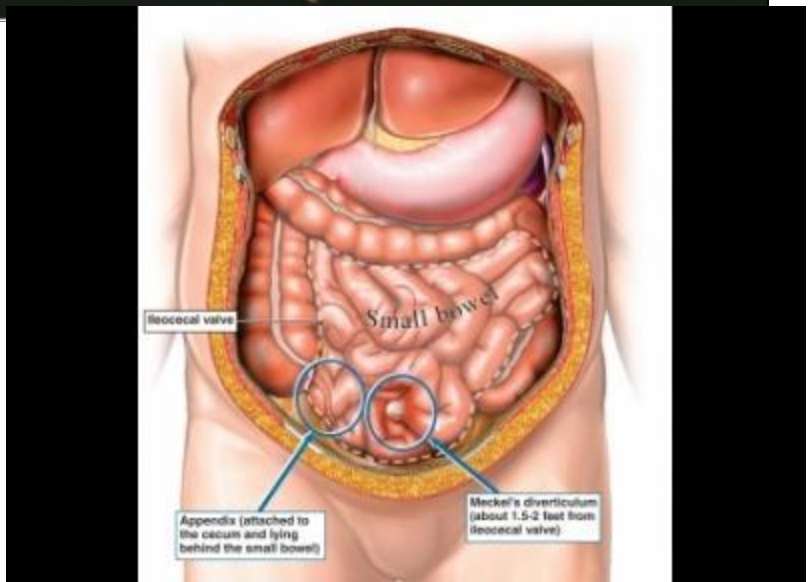


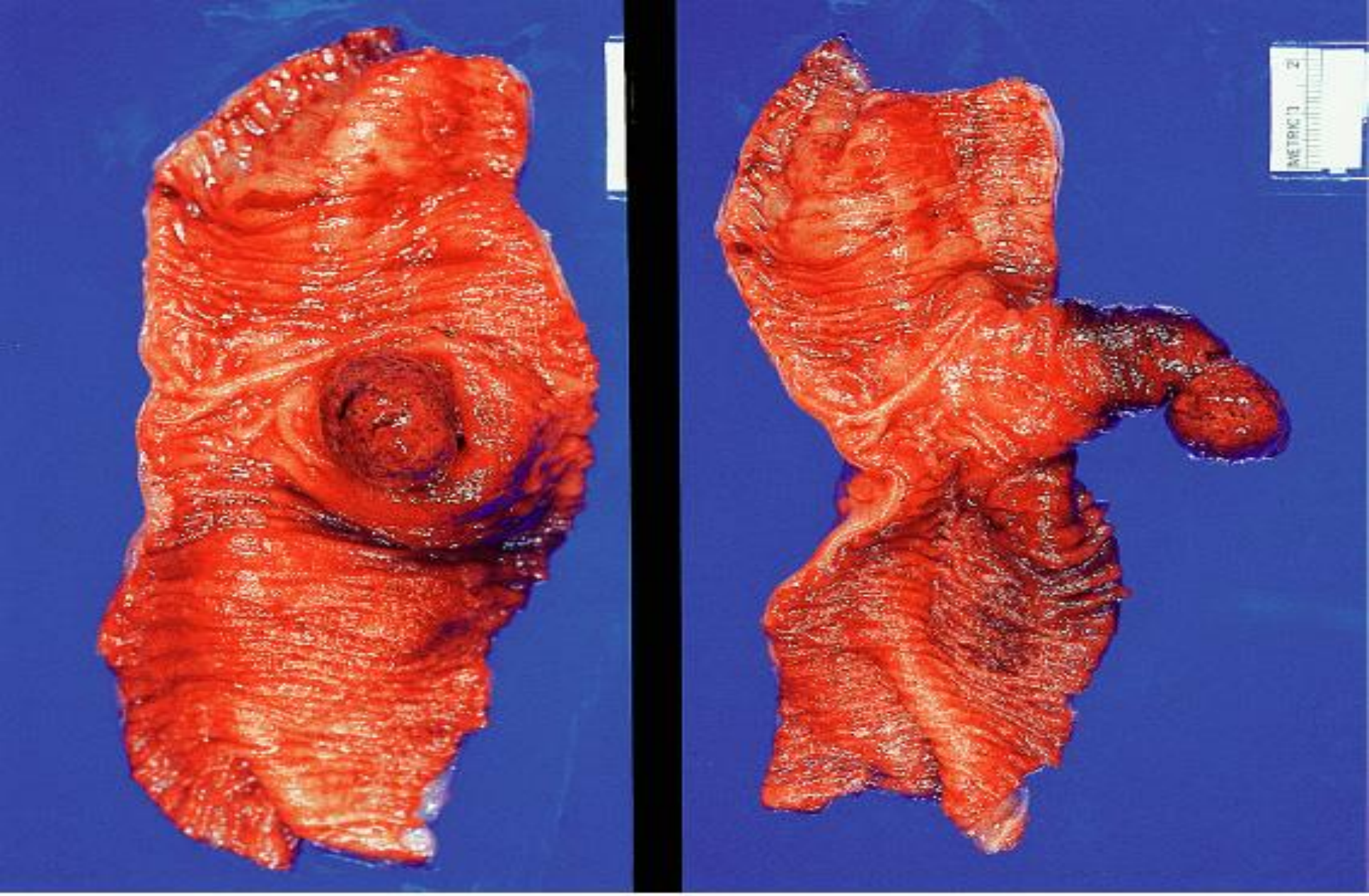




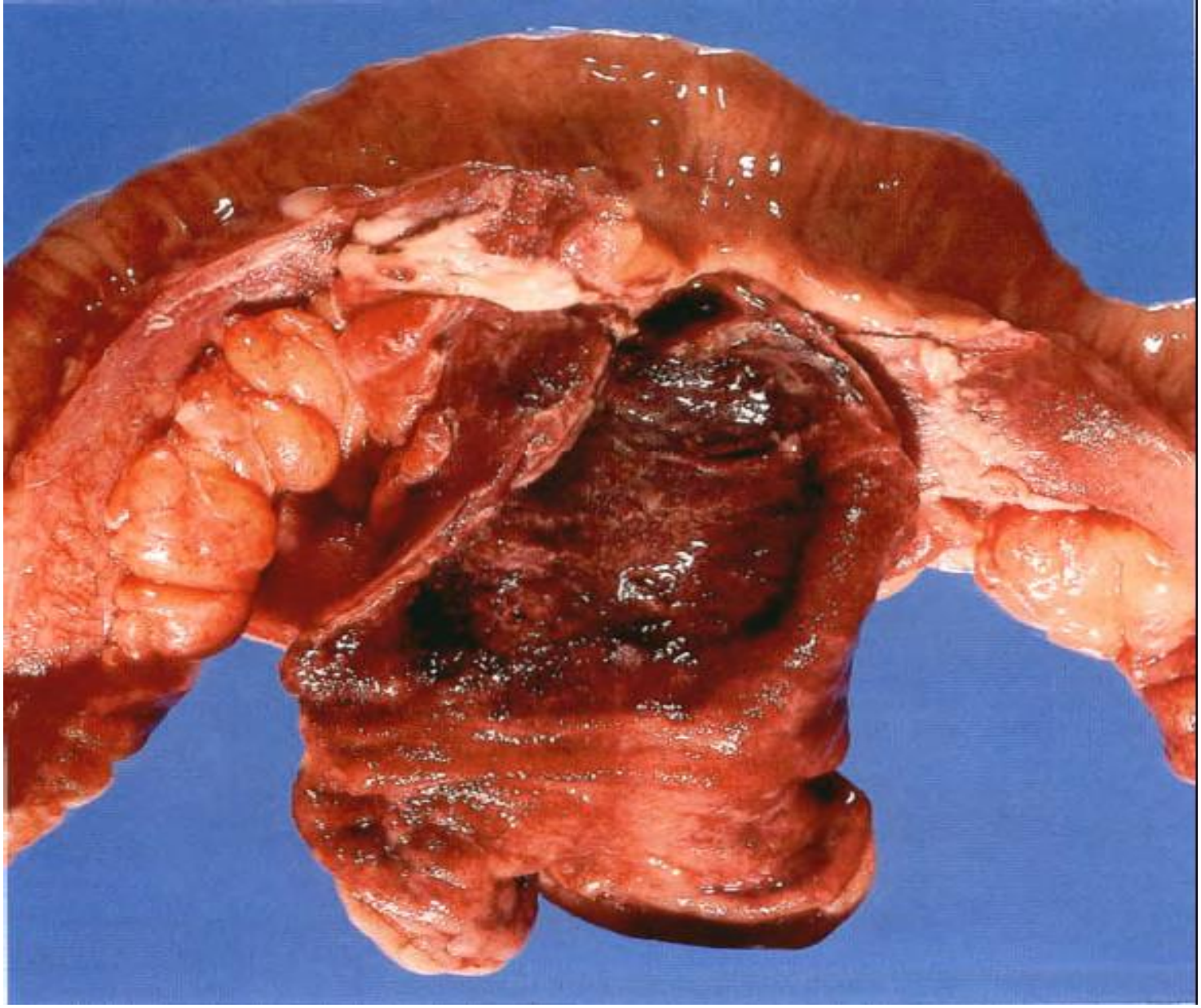


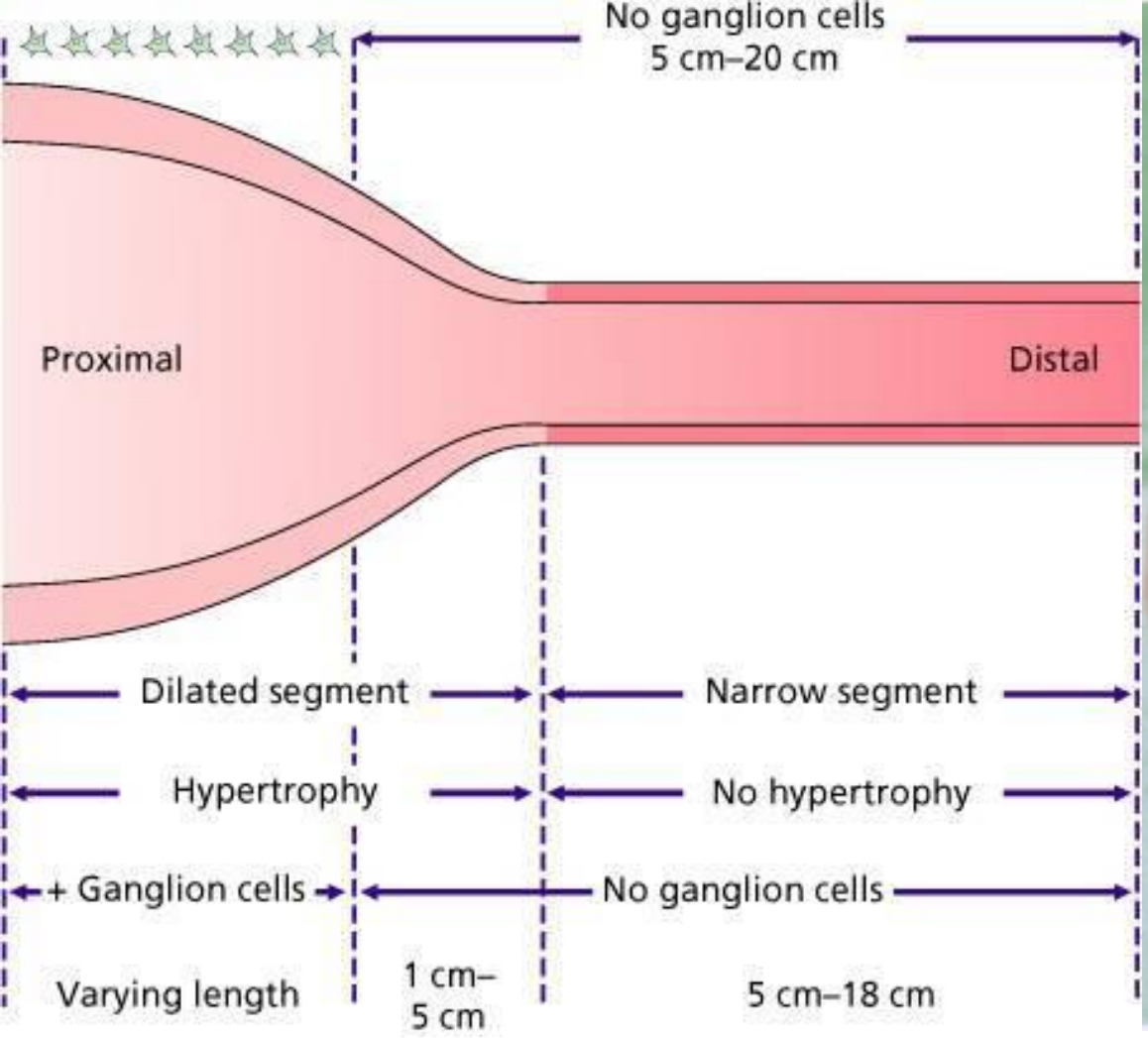


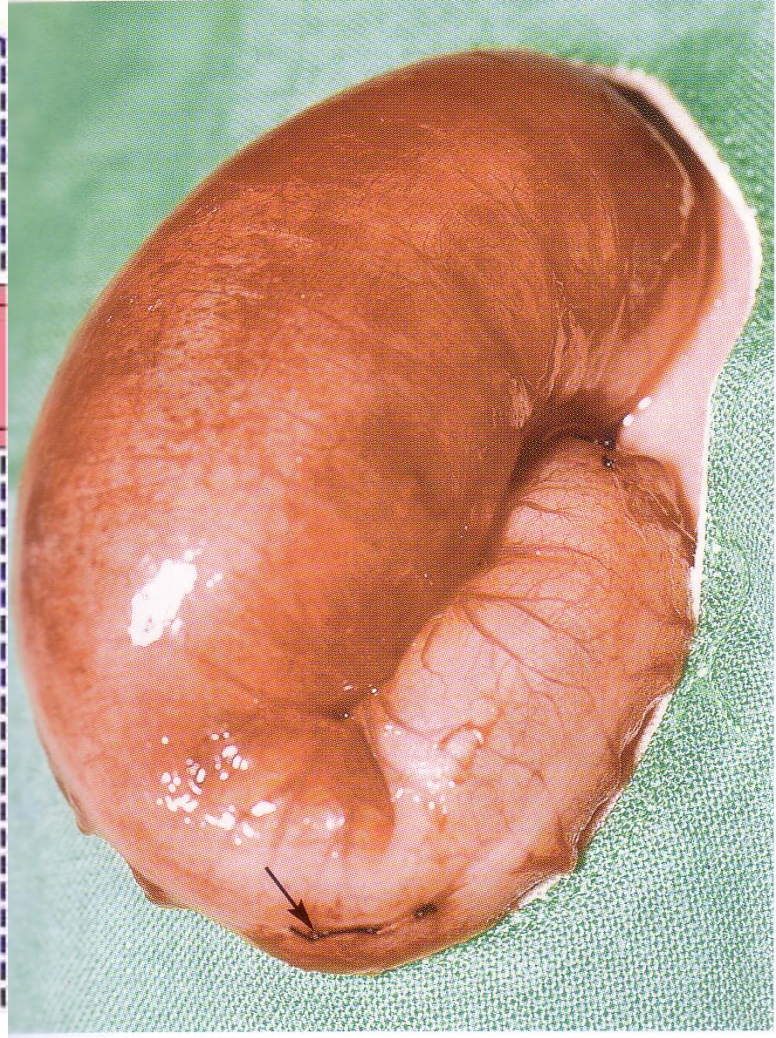

Ileocecal valve
Small bowel
Appendix (attached to the cecum and lying behind the small bowel)
Meckel's diverticulum (about 1.5-2 feet from ileocecal valve)



Intussusception of Meckel's diverticulum




No ganglion cells
5 cm–20 cm
Proximal
Distal
Dilated segment
Narrow segment
Hypertrophy
No hypertrophy
+ Ganglion cells
No ganglion cells
Varying length
1 cm–
5 cm
5 cm–18 cm



Enterocolitis

- *Inflammation of the mucus membrane of small and large intestine*
- **Infectious enterocolitis**
- **Drug related**
- **Radiation enterocolitis**
- **Ischemic bowel disease**
- **Lymphocytic colitis**

Infectious enterocolitis

- Viral enterocolitis
  - Rotavirus
  - Immunosuppressed: CMV and adenovirus

- Bacterial enterocolitis
  - Three mechanisms of disease
  1. Ingestion of a preformed toxin in food
     - Staph aureus, vibrios, clostridium perfringens
  2. Infection by non-invasive toxigenic organism
     - E.coli, Vibrios cholerae
  3. Infection by enteroinvasive organism
     - Shigella, salmonella, E.coli, campylobacter, yersinia

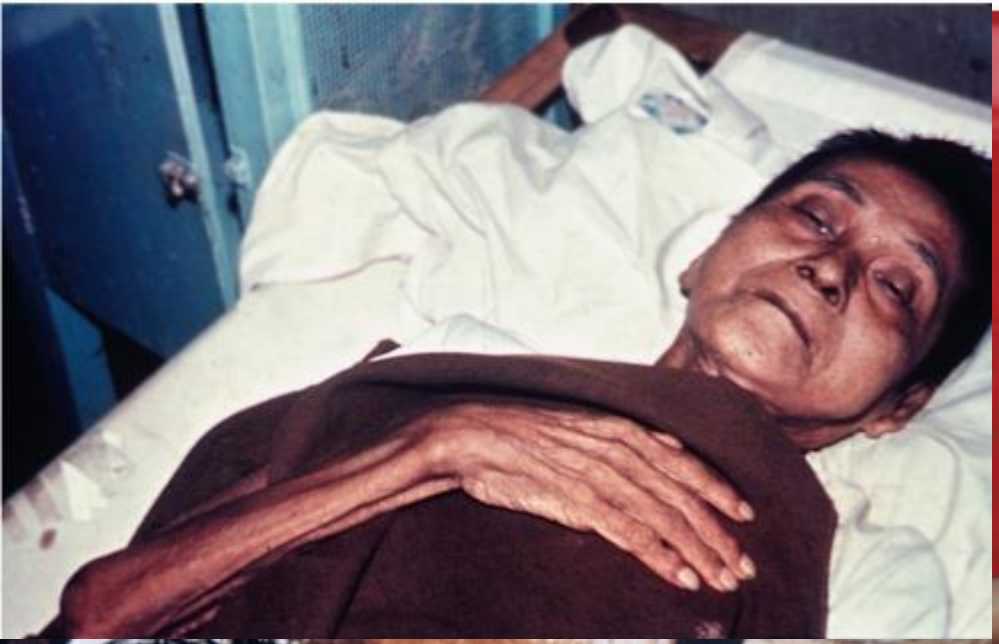
Cholera

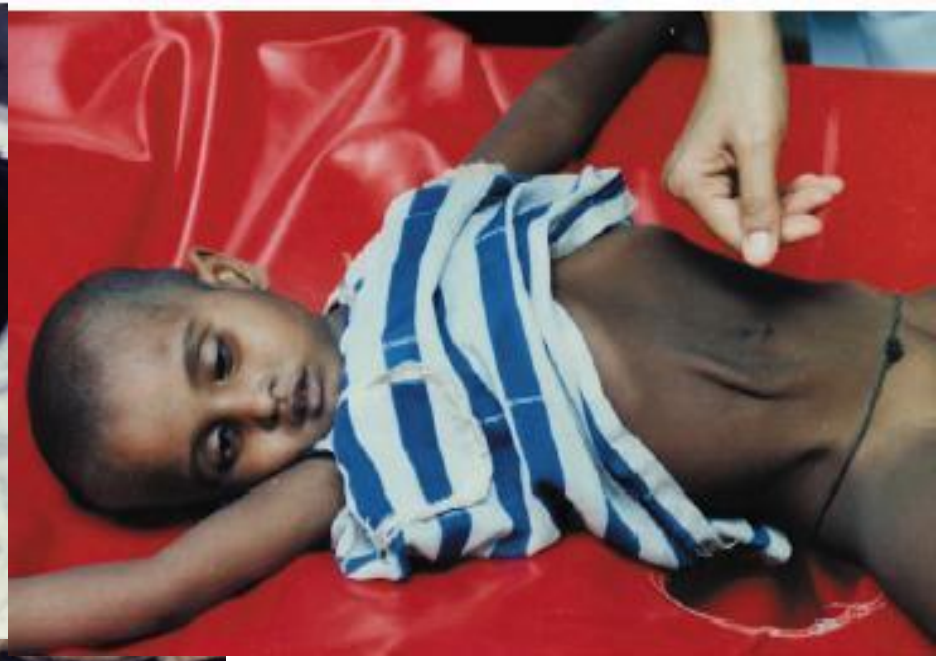
- **Vibrio cholerae (V. cholerae) is a gram-negative bacterium the causative agent of cholera. V. cholerae is transmitted primarily through contaminated water.**

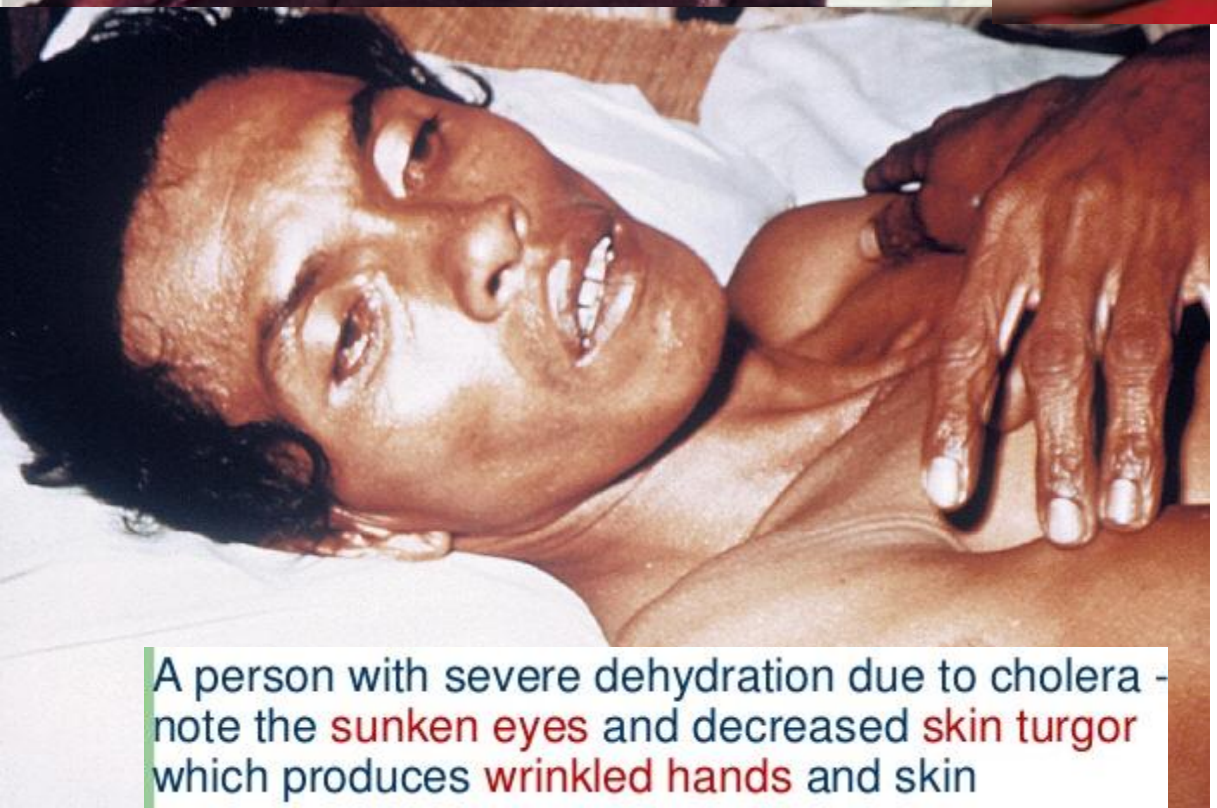
- **Despite severe diarrhea, V. cholerae are non-invasive microorganisms that live and multiply in the lumen of the intestine.**

- **Vibrio cholera toxin causes a disease. The infection is enteric and usually occurs when drinking infected water. The incubation period lasts 3-5 days. "Alkalophilous" vibrios find the optimal environment in the small intestine. Here they multiply and secrete exotoxin (cholerogen).**

- **Under the influence of exotoxin, the epithelium of the mucous membrane secretes a large amount of isotonic fluid. Abundant secretion of fluid occurs as a result of the interaction of cholerogen with the enzyme systems of the cell; at the same time, the blockade of the “sodium pump” of the cell is important, which violates the reverse absorption of fluid from the intestinal lumen. Profuse diarrhea is associated with abundant secretion of fluid and a violation of its reverse absorption.**







A person with severe dehydration due to cholera - note the sunken eyes and decreased skin turgor which produces wrinkled hands and skin

Cholera

Shigellosis

- **Shigella (Shigella spp.) Are gram-negative bacteria that were first isolated during the epidemic of red (bloody) diarrhea in Japan in 1897 (in Russia, the infection caused by shigella is called dysentery).**

- **Man is the only known reservoir of this microorganism. It has been established that 165 million new cases of shigellosis are recorded annually in the world.**

- **The infectious dose for Shigella is not more than a few hundred microorganisms, and 1 milliliter of feces in the acute period of the disease contains 10 millions microorganisms. As a result, shigella are quickly transmitted to humans by fecal-oral route or through contaminated water and products.**

Typhoid fever

- **Typhoid fever is an acute infectious disease from the intestinal group; typical anthroponosis. Caused by typhoid bacillus (Salmonella typhi).**

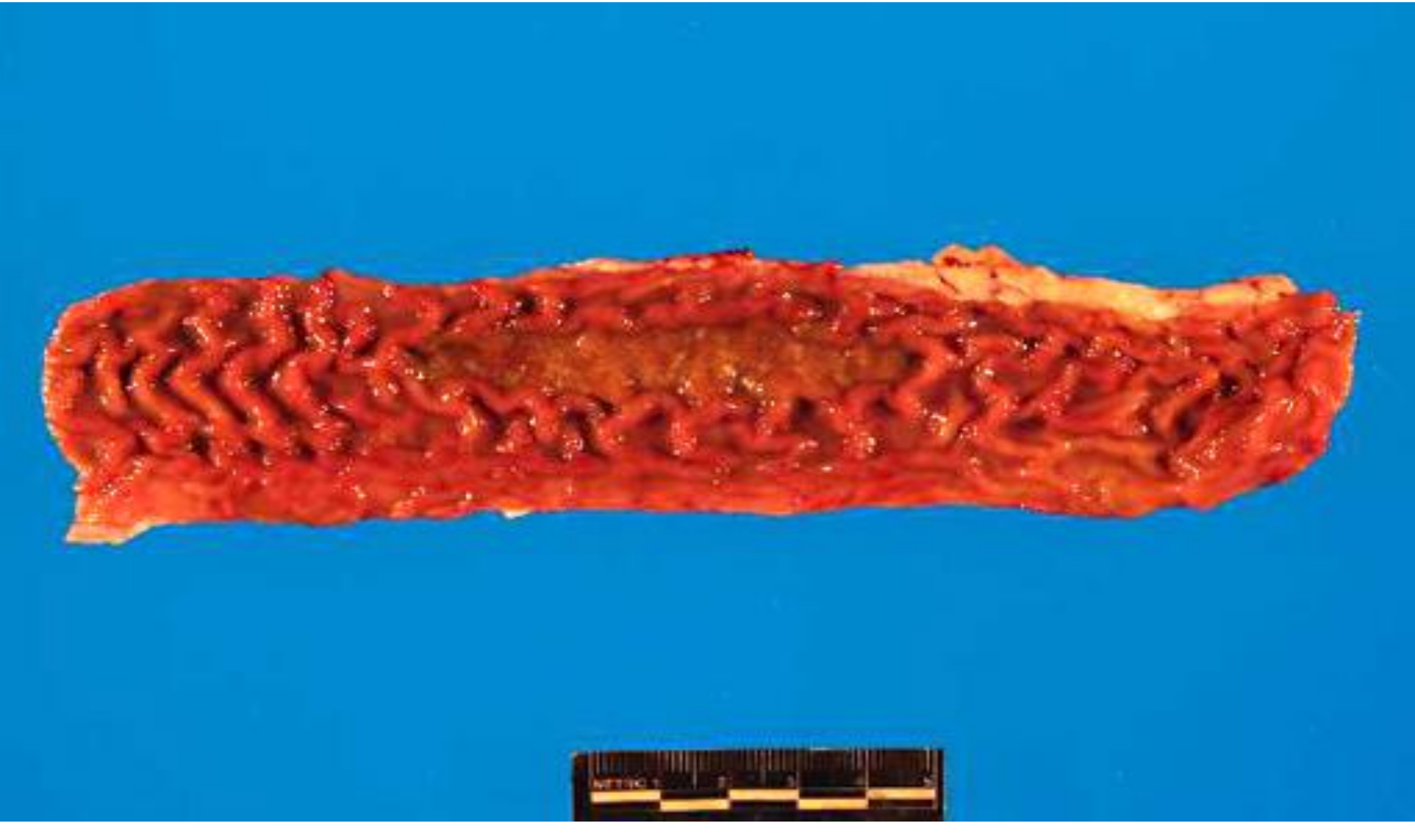
- **The source of infection is a sick person or a carrier, in the secretions of which (feces, urine, sweat) contains microbes. Infection occurs parenterally.**

- **The incubation period is 10-14 days. In the lower part of the small intestine, bacteria multiply, secrete endotoxins. From the intestine through the lymphatic paths, they enter the group lymphatic follicles (the so-called Peyer's patches) and solitary follicles, and then to the regional lymph nodes.**

- **Having overcome the lymphatic barrier, the pathogen enters the bloodstream. Bacteremia develops, which is especially pronounced during the 1st week of illness, when typhoid bacillus can be isolated from the blood (blood culture).**

Typhoid fever

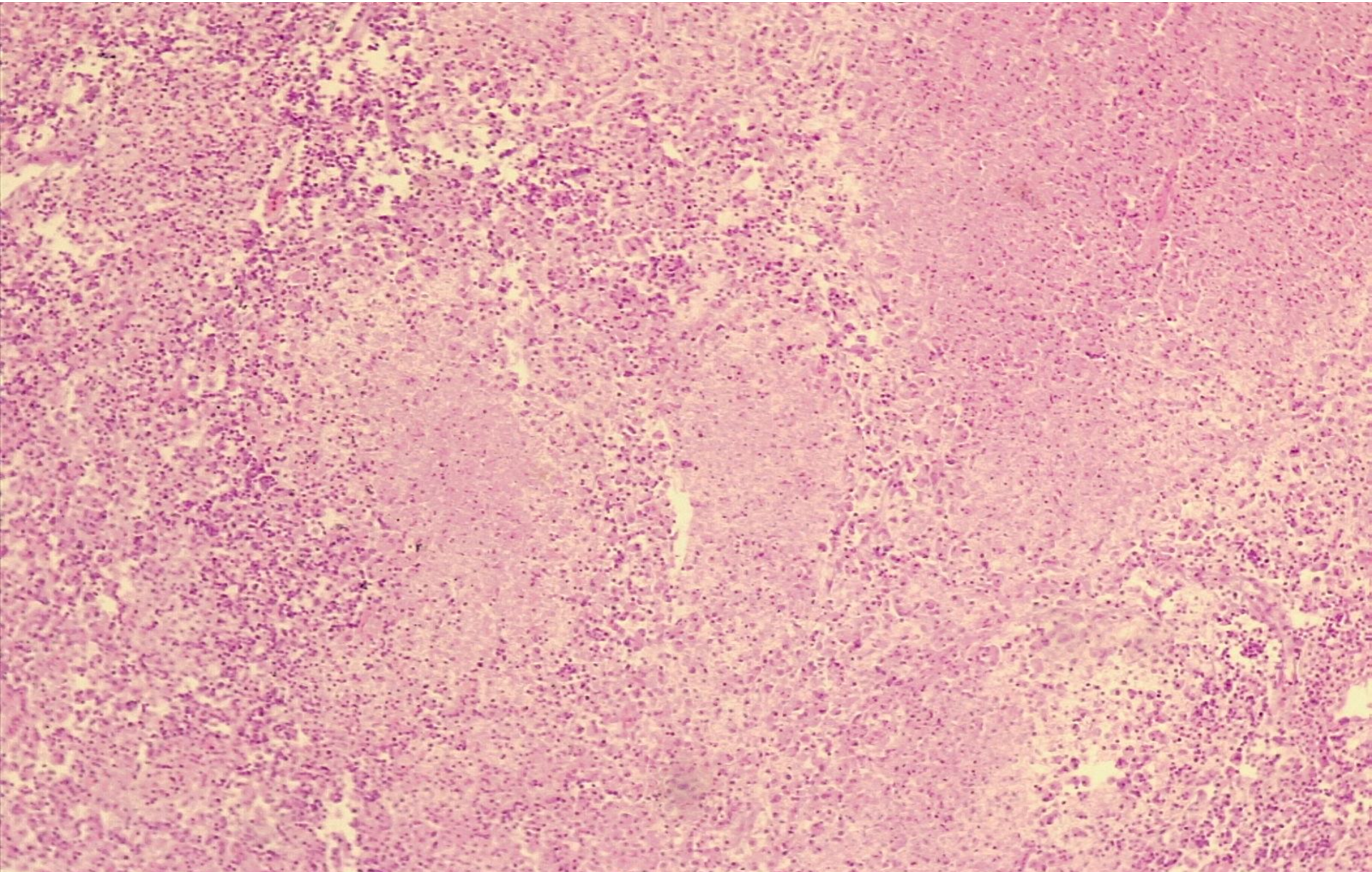
Typhoid fever



Typhoid fever

Typhoid fever

The stage of ulcer healing ends with the formation of tender scars in their place; the lymphoid tissue of the intestine is partially or completely restored, it becomes only slightly pigmented.

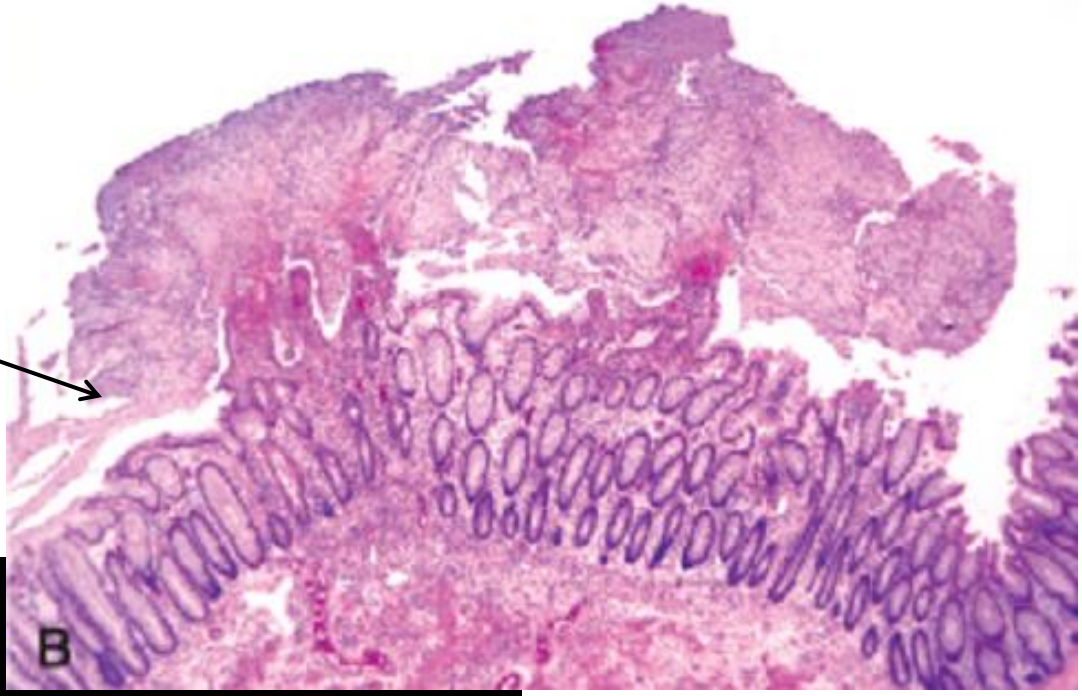


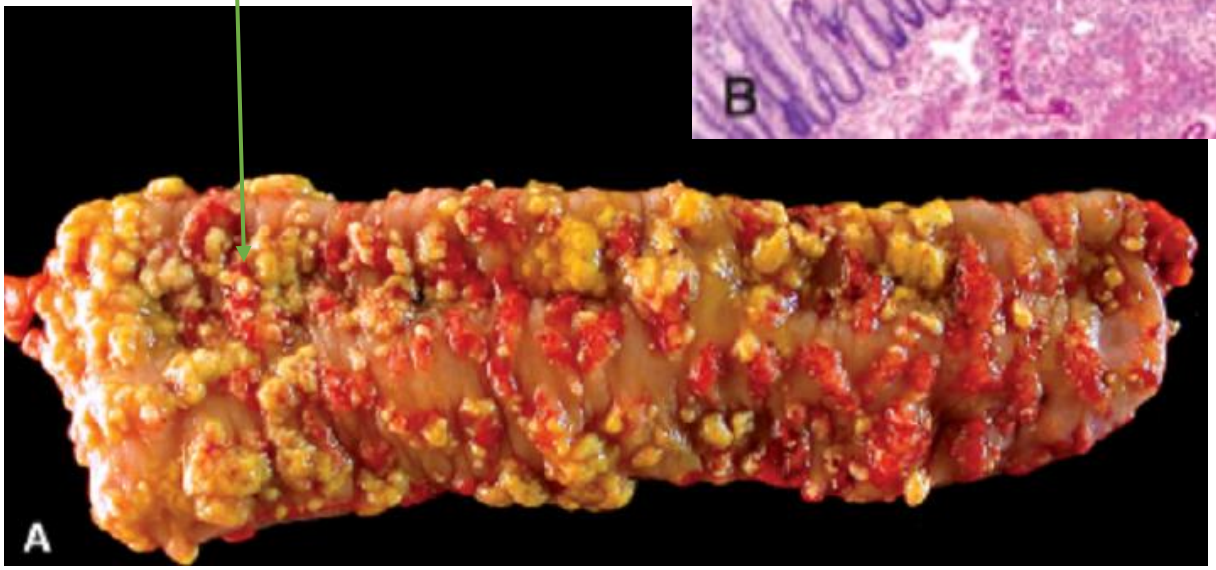
Infectious enterocolitis

- Pseudomembranous colitis
  - Exudative, fibrin-rich plaques (pseudomembrane) overlying sites of mucosal injury
  - Most associated antibiotic therapy
  - *Clostridium difficile*
    - Enterotoxin
    - Cytotoxin
  - Other - ischemia, anti-neoplastic drugs
- Parasitic
  - Amebiasis - *Entamoeba histolytic*
  - Giardiasis - *Giardia lamblia*
  - Cryptosporidiosis - *Cryptosporidium*
- Fungal
  - Candida

Pseudomembranous Colitis

Pseudomembrane





Non-infectious Colitis

- Drugs
- Radiation enterocolitis
- Ischemic Bowel Disease
  - Occlusive etiology
  - Non-Occlusive
- Collagenous colitis
- Lymphocytic colitis

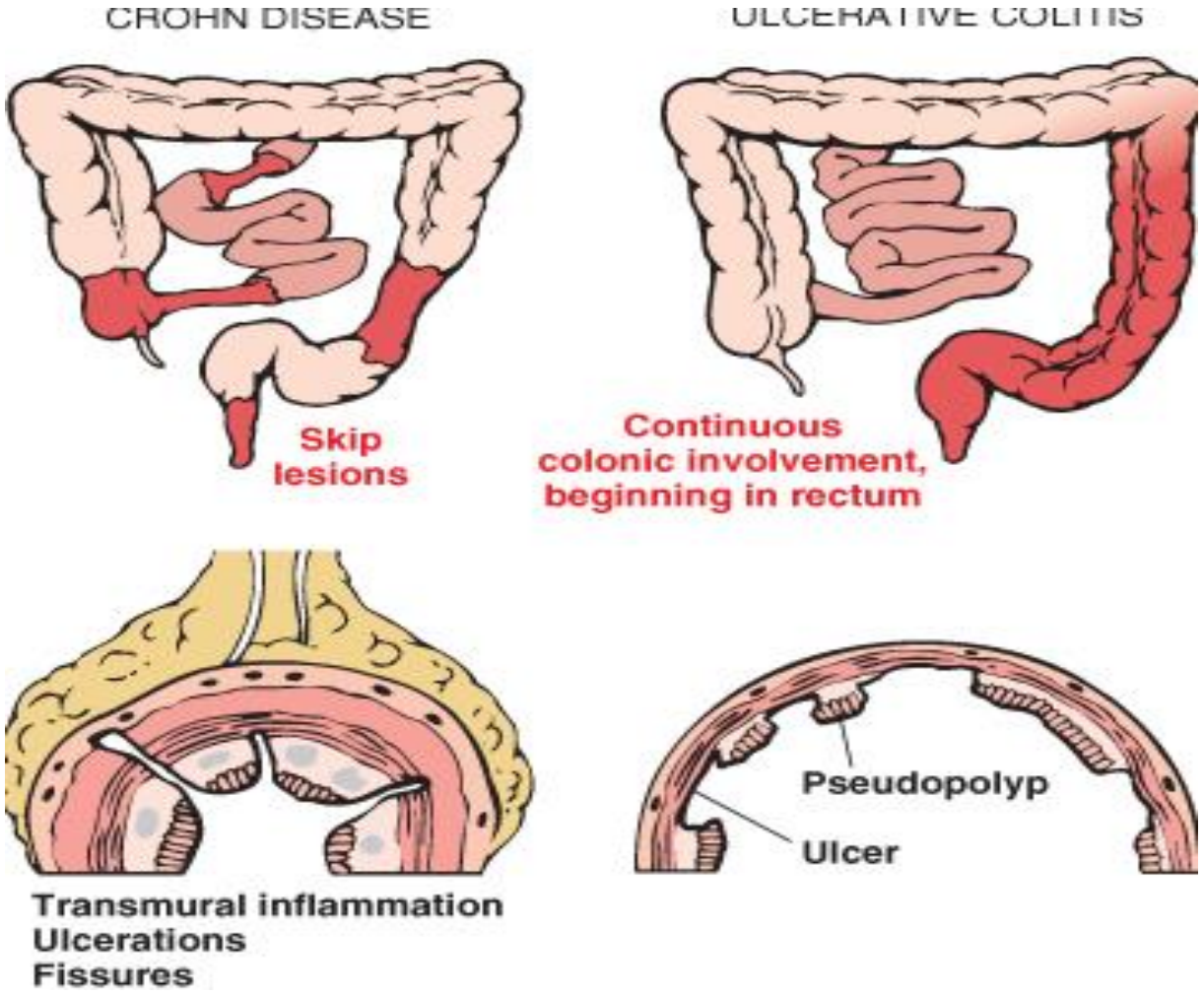
Idiopathic Inflammatory Bowel Disease (IBD)

- ***Chronic, relapsing inflammatory disorders of unknown origin***
- **Crohn's Disease**
- **Ulcerative colitis**

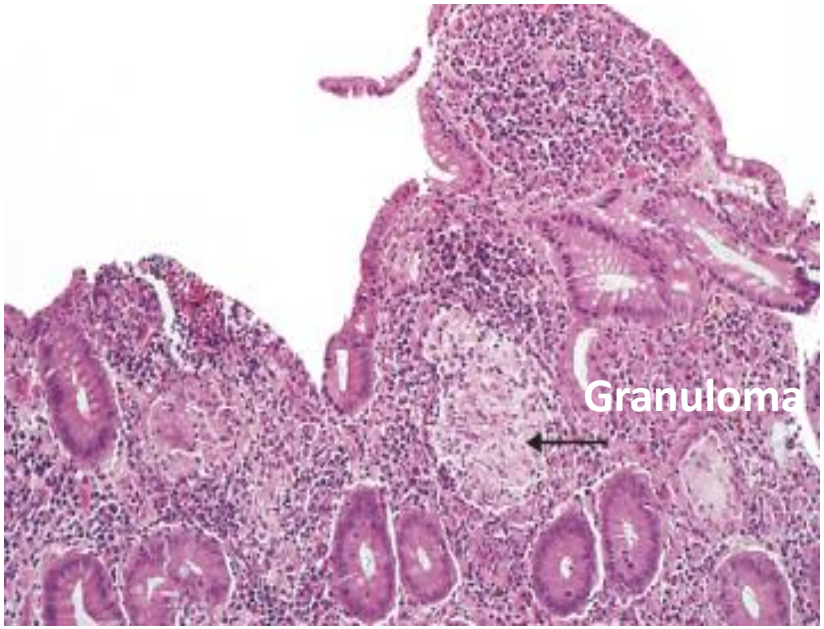
Crohn's vs UC

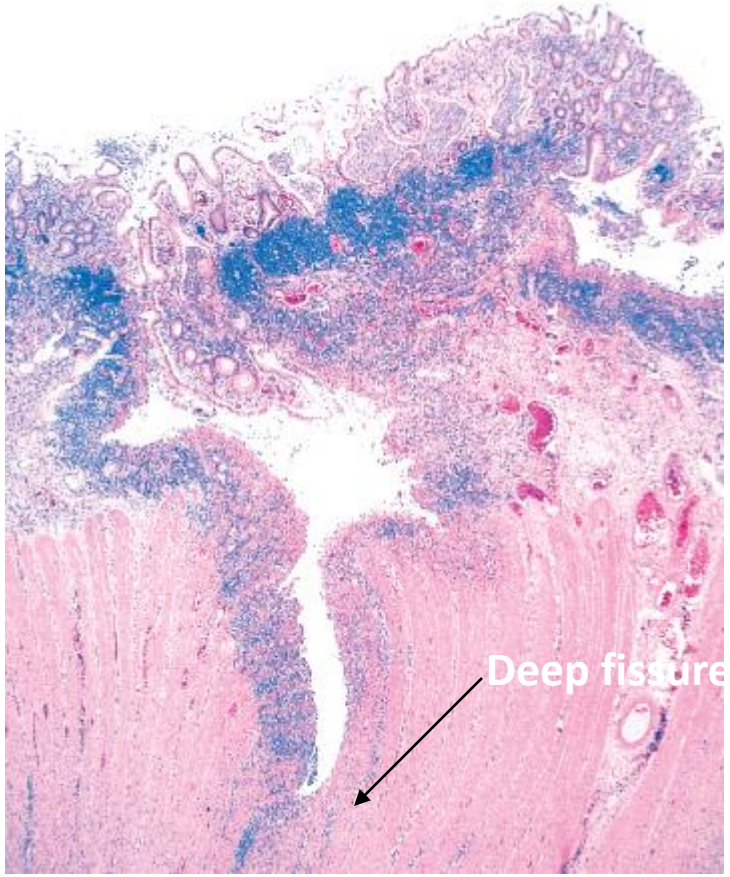
- **Crohn's Disease**
  - Any part of GI tract
  - Skip lesions
  - Rectum spared
  - Transmural inflammation
    - Fissures
    - Fistulas
  - Strictures
  - Granulomas
  - Small increased risk CA
  - Crypt abscess
- **Ulcerative Colitis**
  - Colon only
  - Continuous
  - Rectum always involved
  - Mucosal inflammation
    - No fissures
    - No fistulas
  - Strictures rare
  - No granulomas
  - > 10 % for 25 yr hx
  - Crypt abscess

Crohn's vs UC

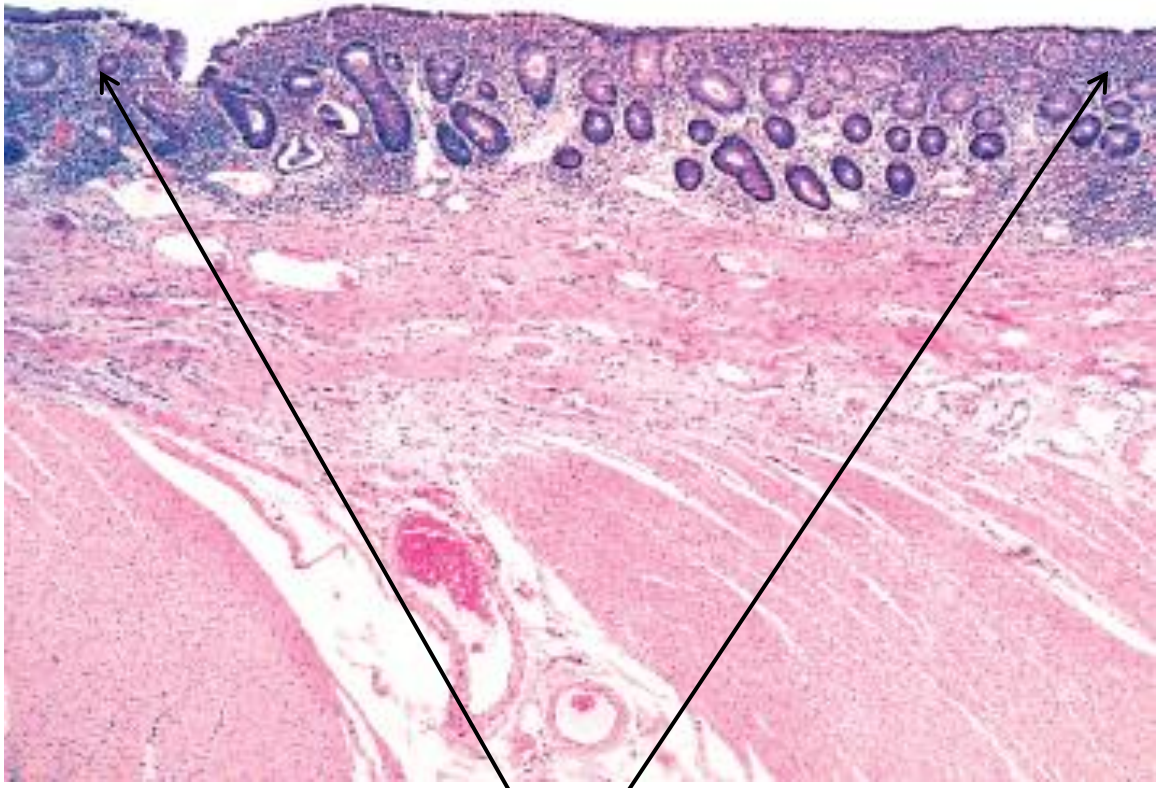

CROHN DISEASE
ULCERATIVE COLITIS
Skip lesions
Continuous colonic involvement, beginning in rectum
Transmural inflammation
Ulcerations
Fissures
Pseudopolyp
Ulcer

Crohn's Disease



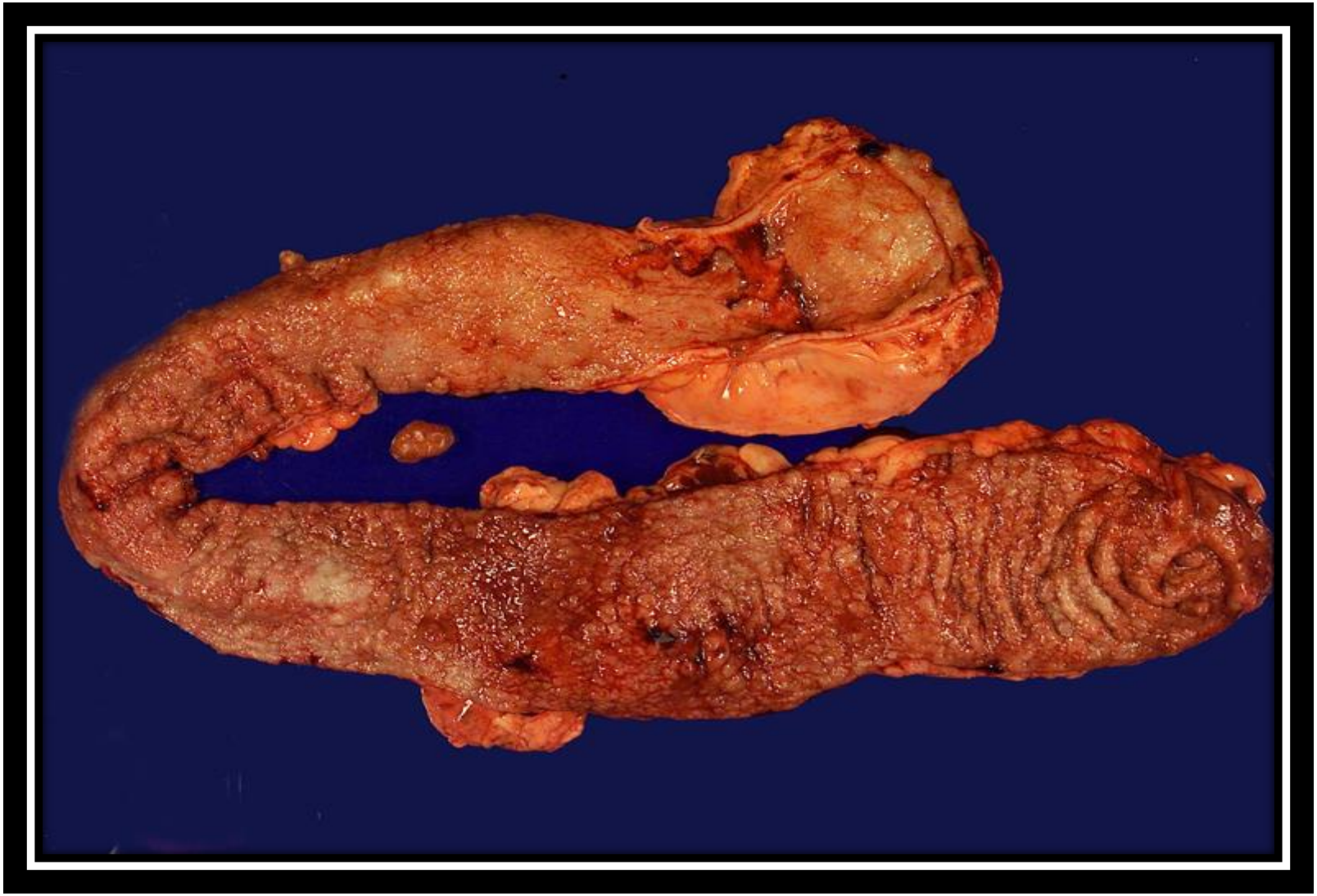


Ulcerative Colitis

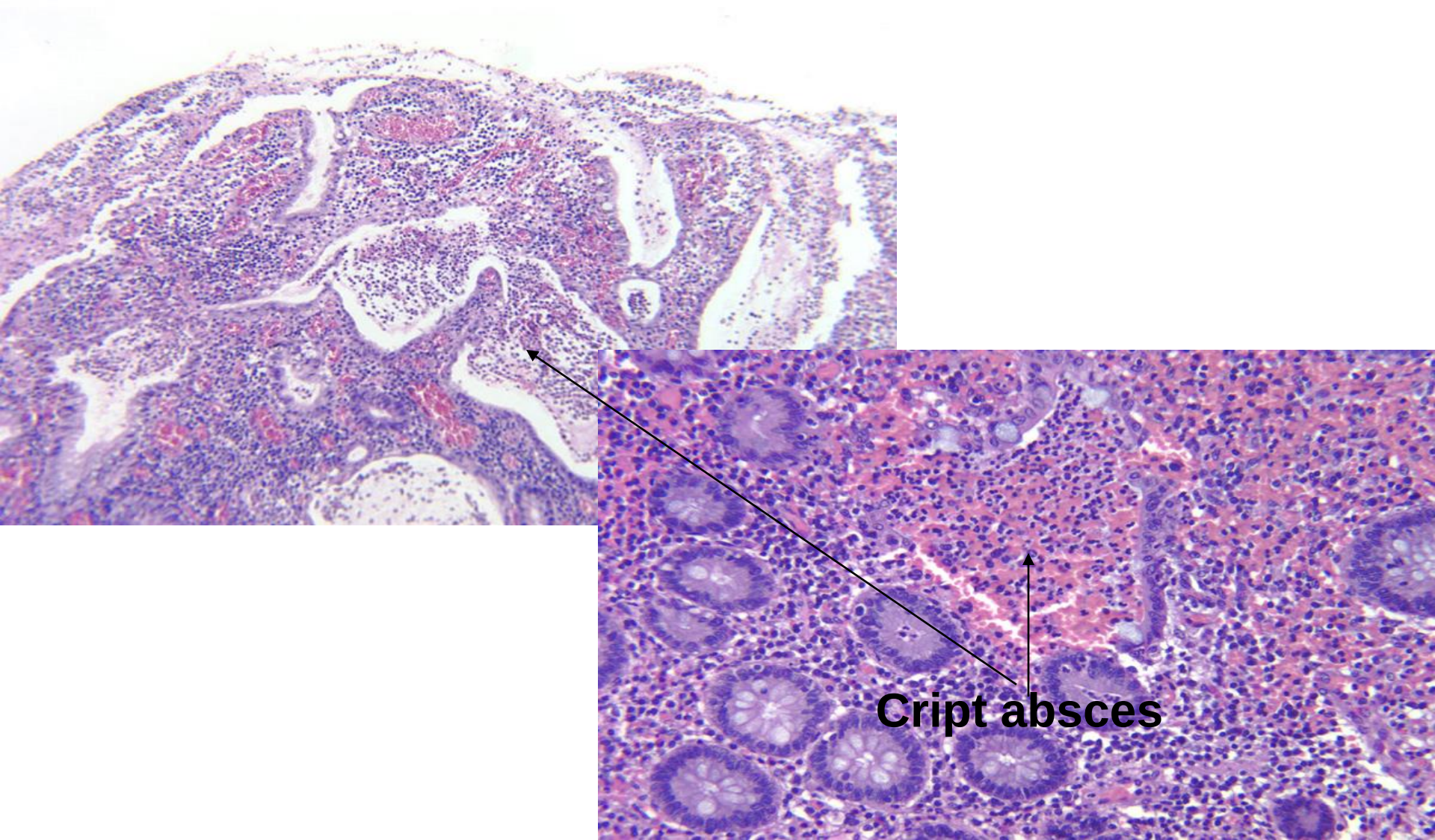


Isolated mucosal inflammation.

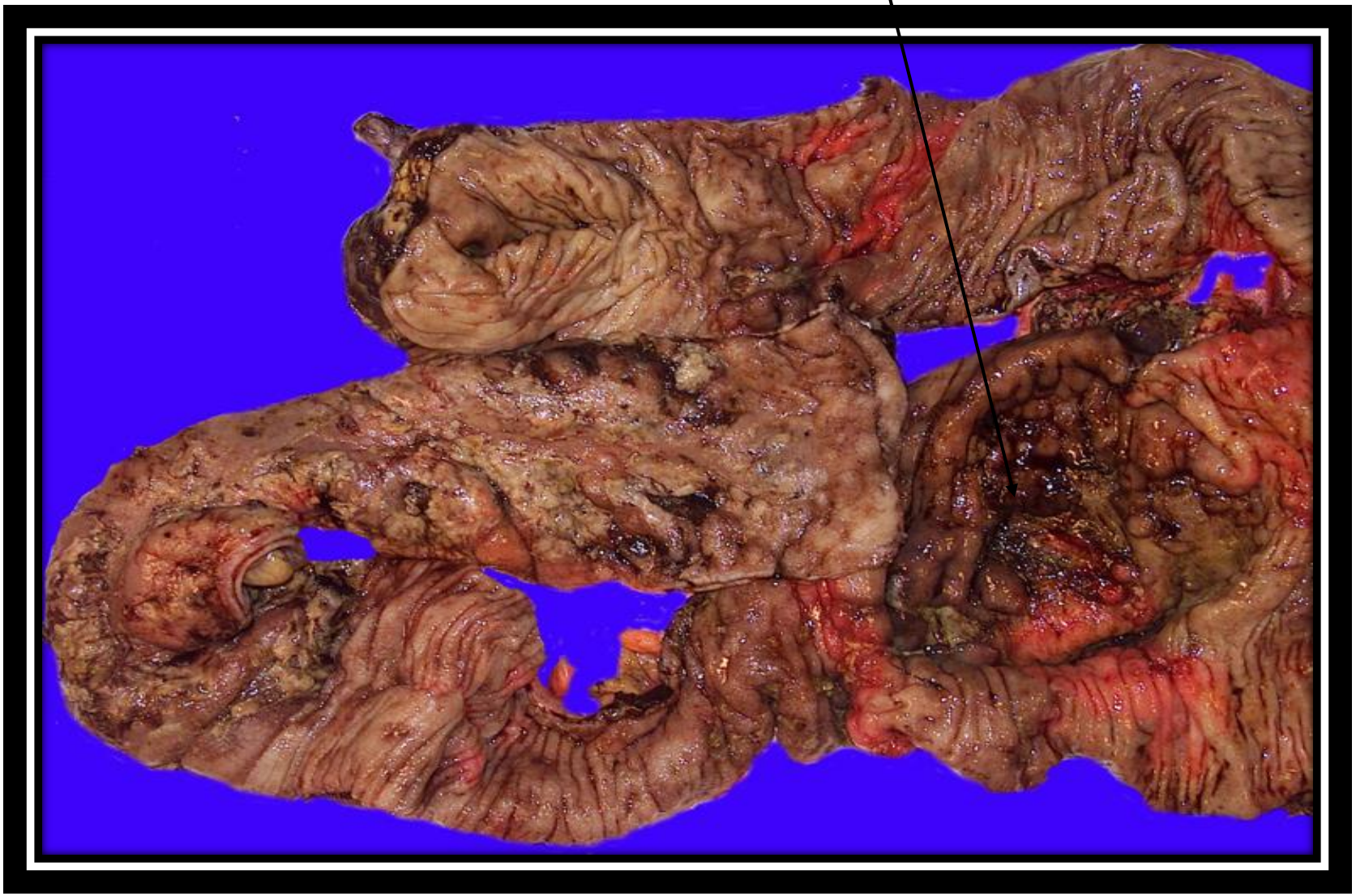
Ulcerative colitis. Continuous involvement starting at rectum and extending proximal.



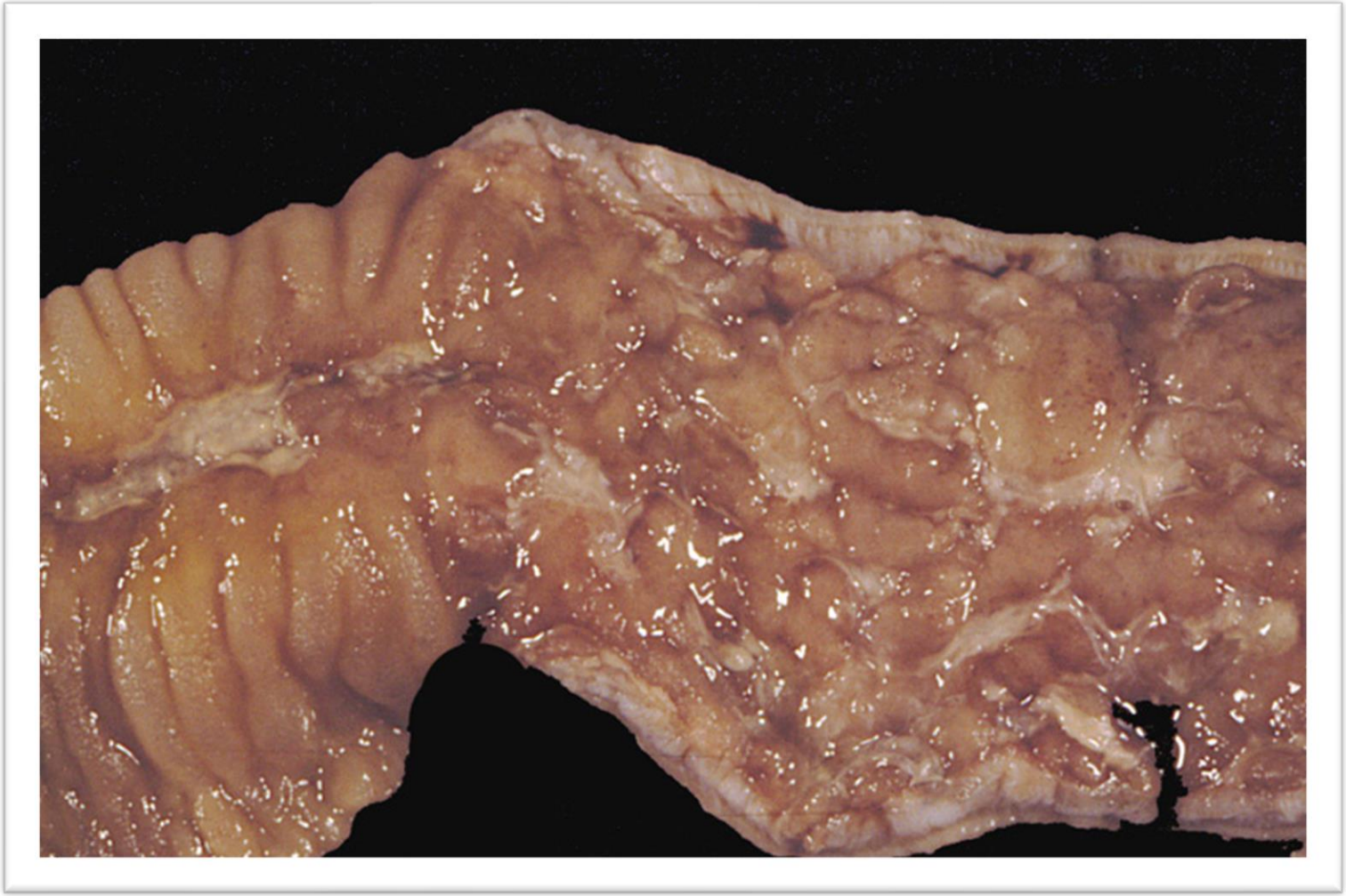
Crypt abscess



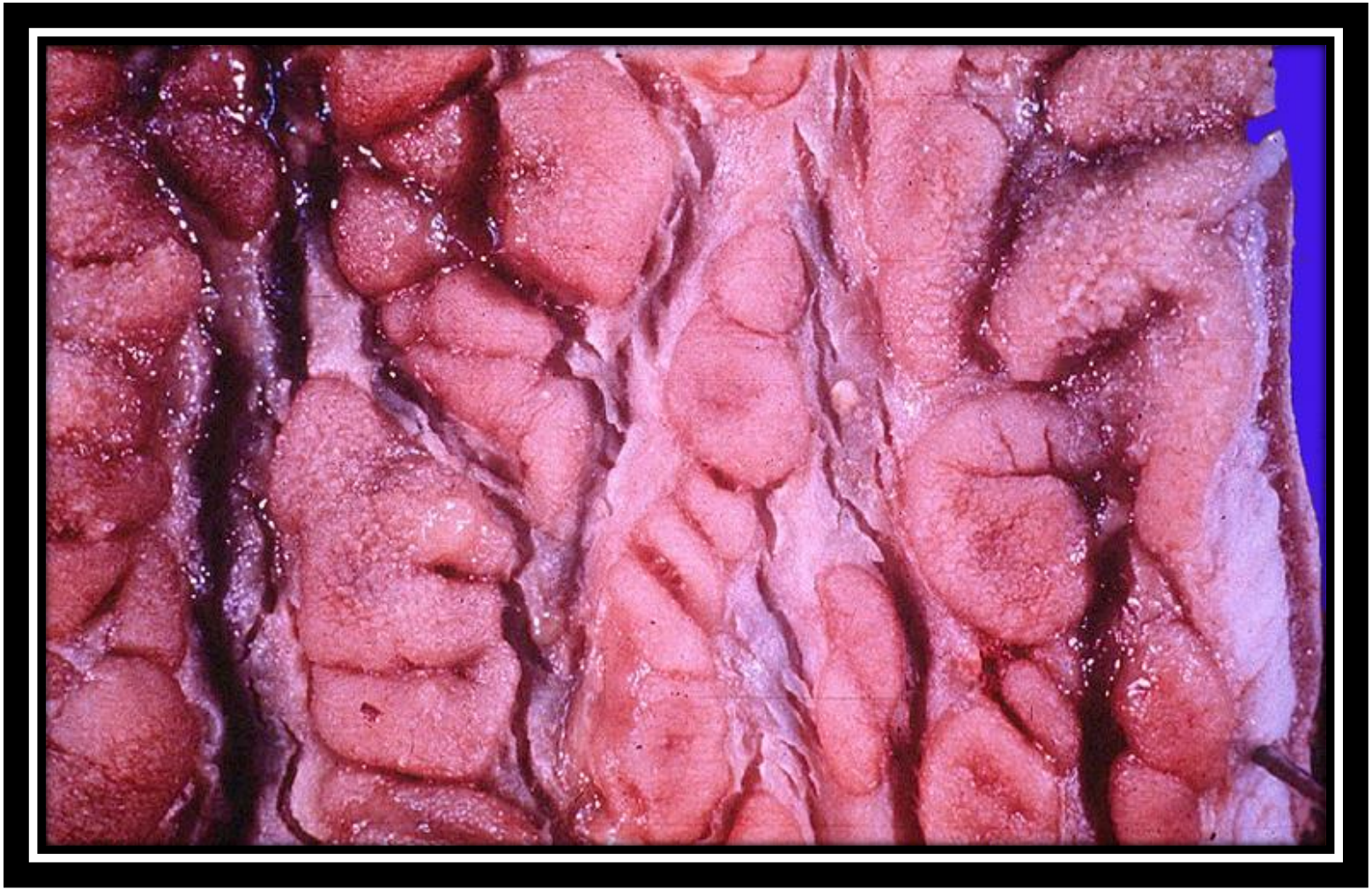
Toxic Megacolon



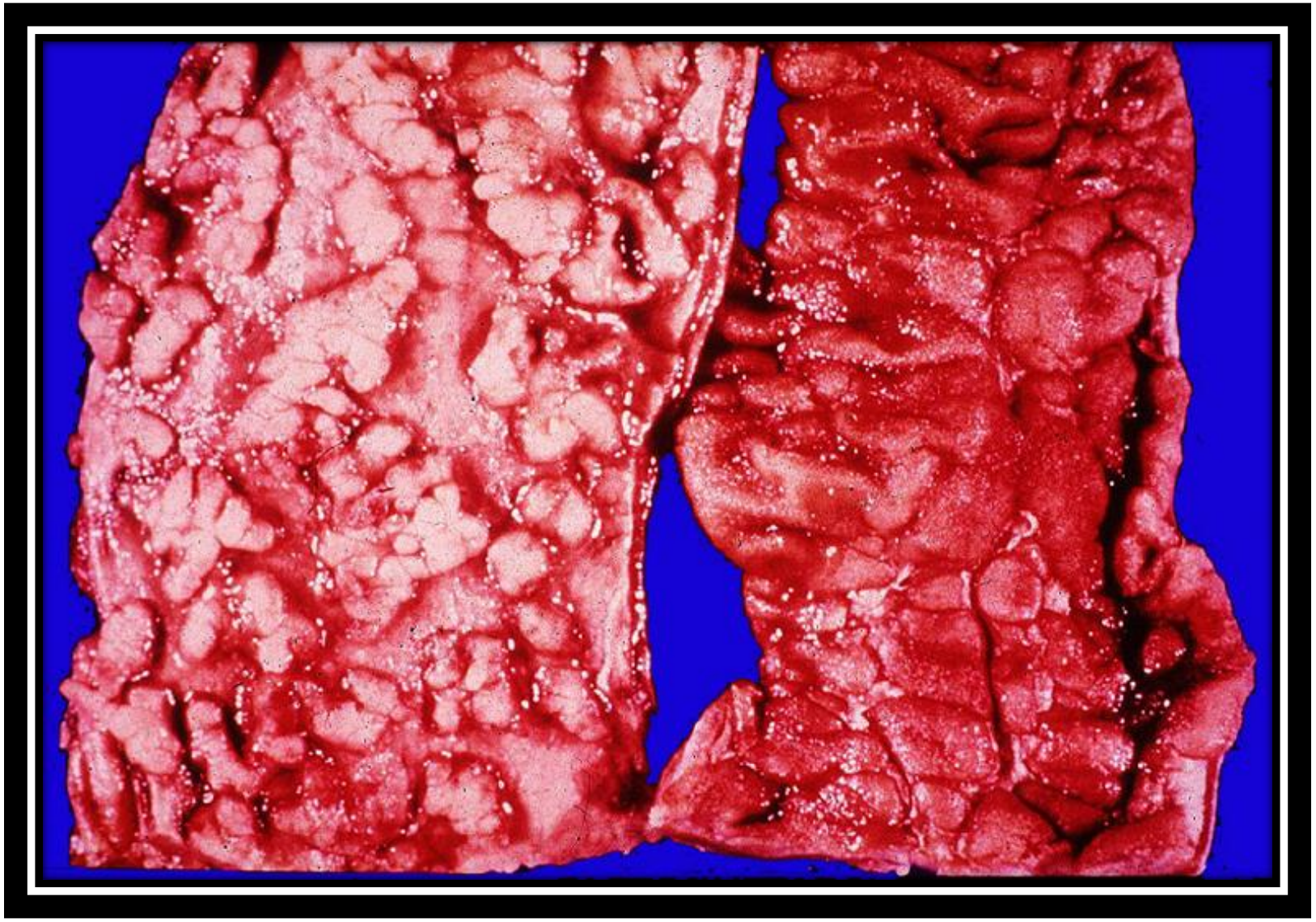
“Cobblestone”. Serpinginous linear ulcers surrounding normal mucosa



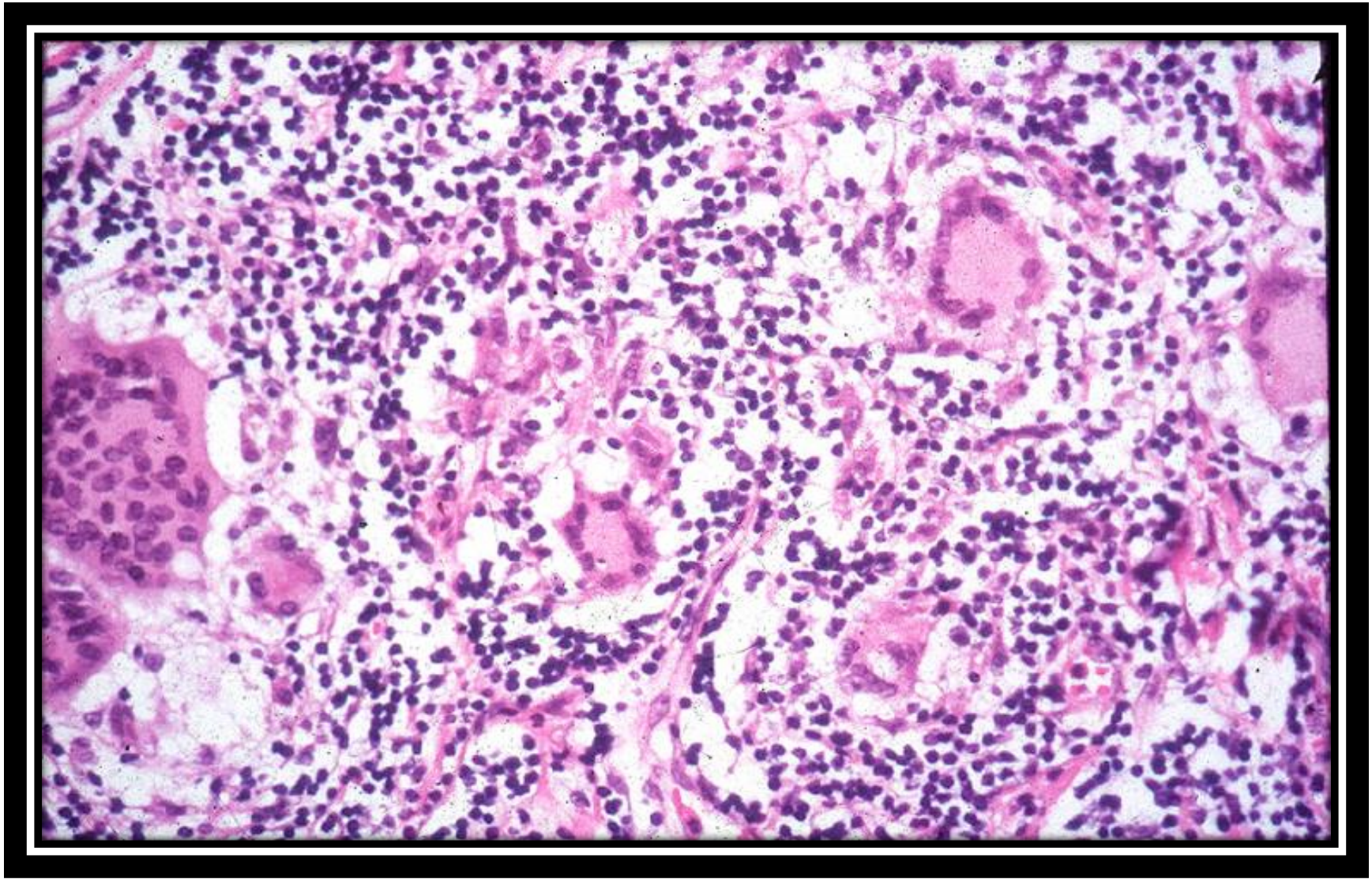
Serpinginous linear ulcers surrounding normal mucosa

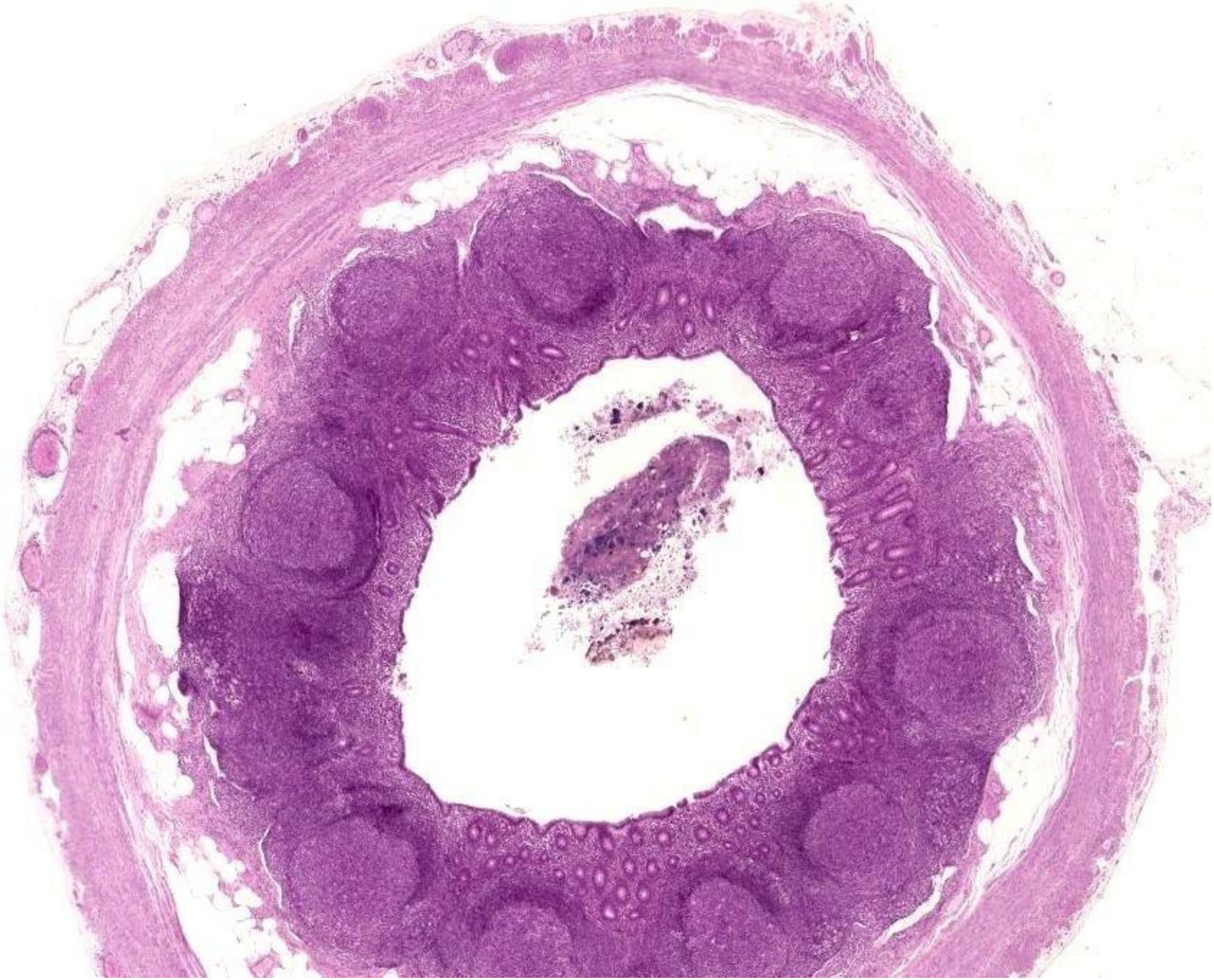


Ulcerative Colitis Crohn's Disease



Granulomas = Crohn's Disease



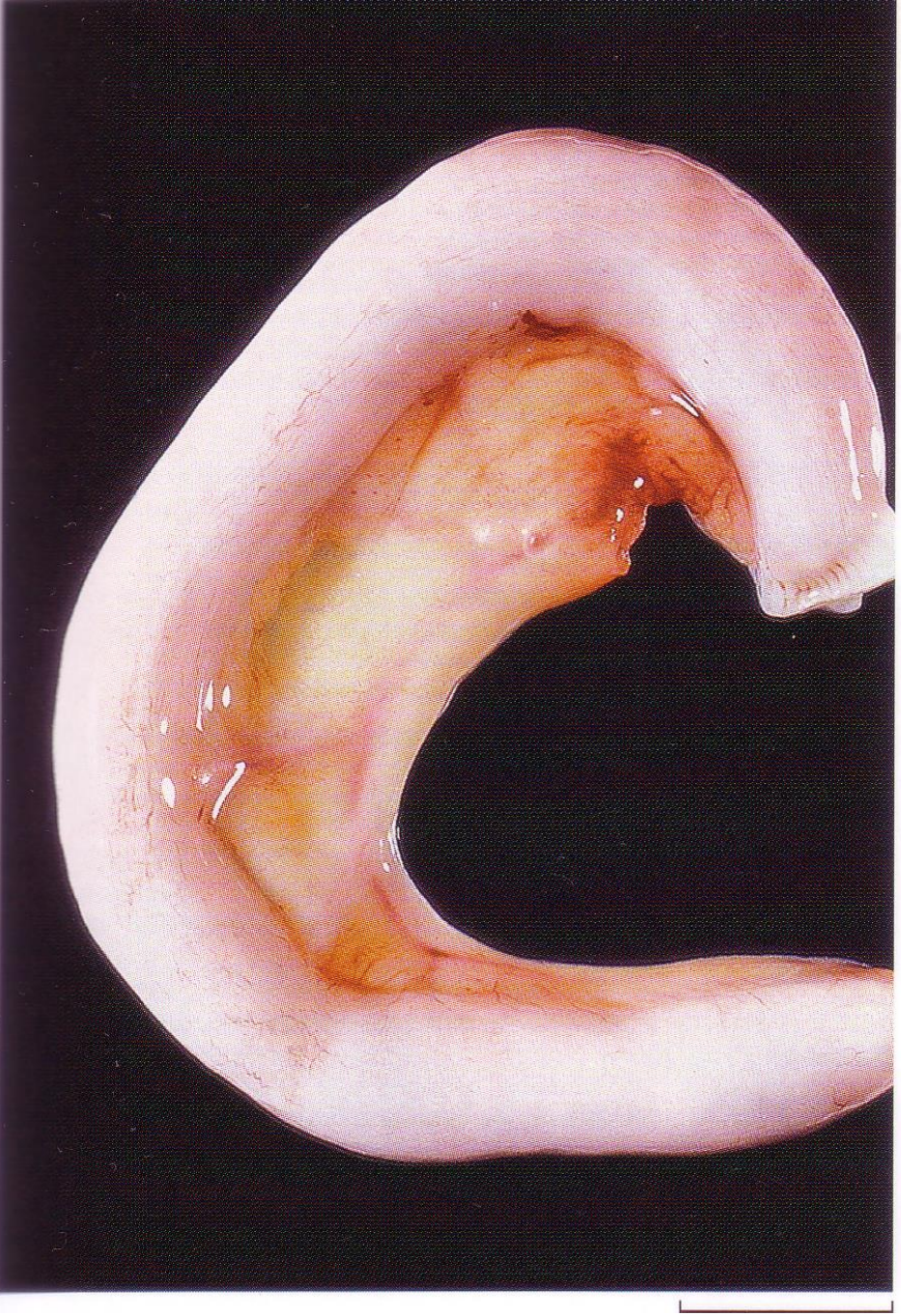


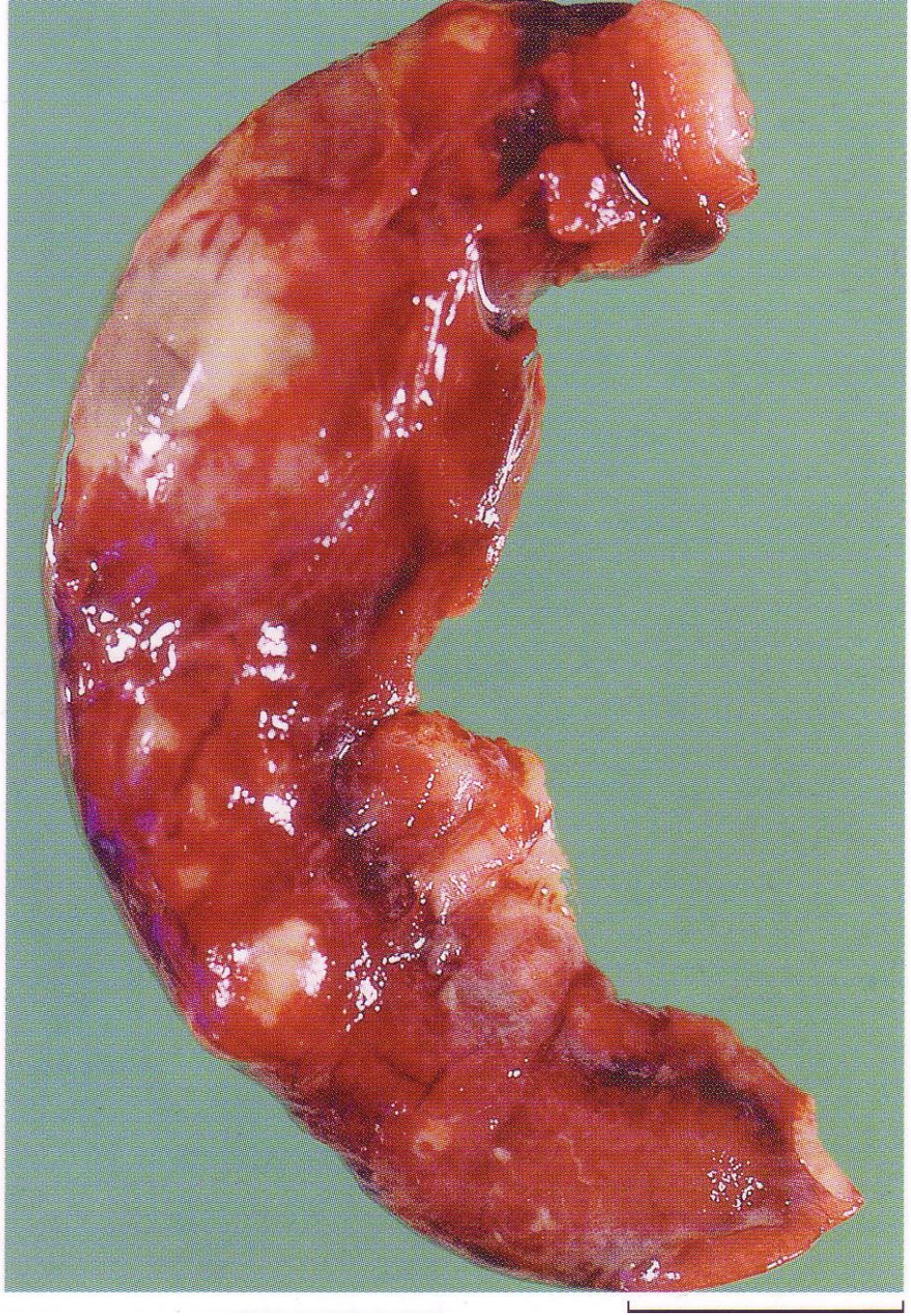
APPENDIX

ANATOMY

- **Junction of 3 tenia coli, variable in location**
- **All 4 layers, true serosa**
- **Thickest layer is submucosal lymphoid tissue**

- **APPENDICITIS (ACUTE)**
- **MUCOCELE**
- **MUCUS CYSTADENOMA**
- **MUCUS CYSTADENOCARCINOMA**





Аппендицит.

ACUTE APPENDICITIS

- **GENERALLY, a disease of YOUNGER people**
- **OBSTRUCTION by FECALITH the classic cause but fecaliths present only about half the time**
- **EARLY APPENDICITIS: NEUTROPHILS→Mucosa, submucosa**
- **NEED NEUTROPHILS in the MUSCULARIS to confirm the DIAGNOSIS**
- **25% normal rate, usually**
- **Perforation→peritonitis the rule, if no surgery**

Acute Appendicitis

Is the most common acute abdominal condition the surgeon is called on to treat.

- **Inflammation in the right lower quadrant**
- **Adolescents and young adults**

– Acute Simple Appendicitis

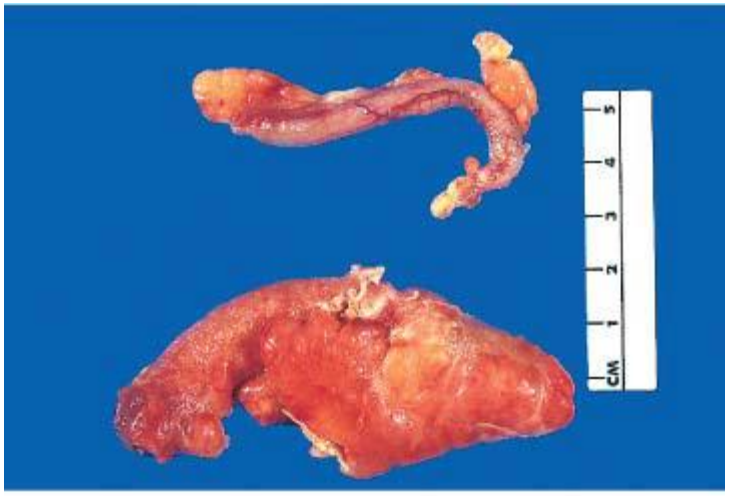
– Acute Suppurative Appendicitis

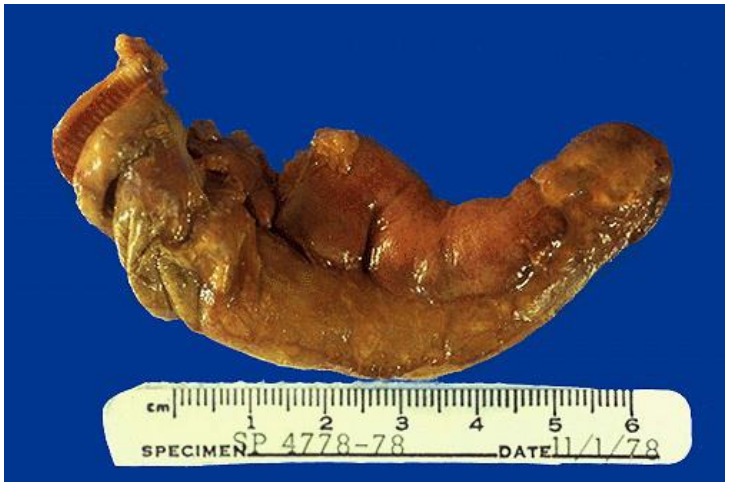
– Acute Gangrenous Appendicitis

Morphology

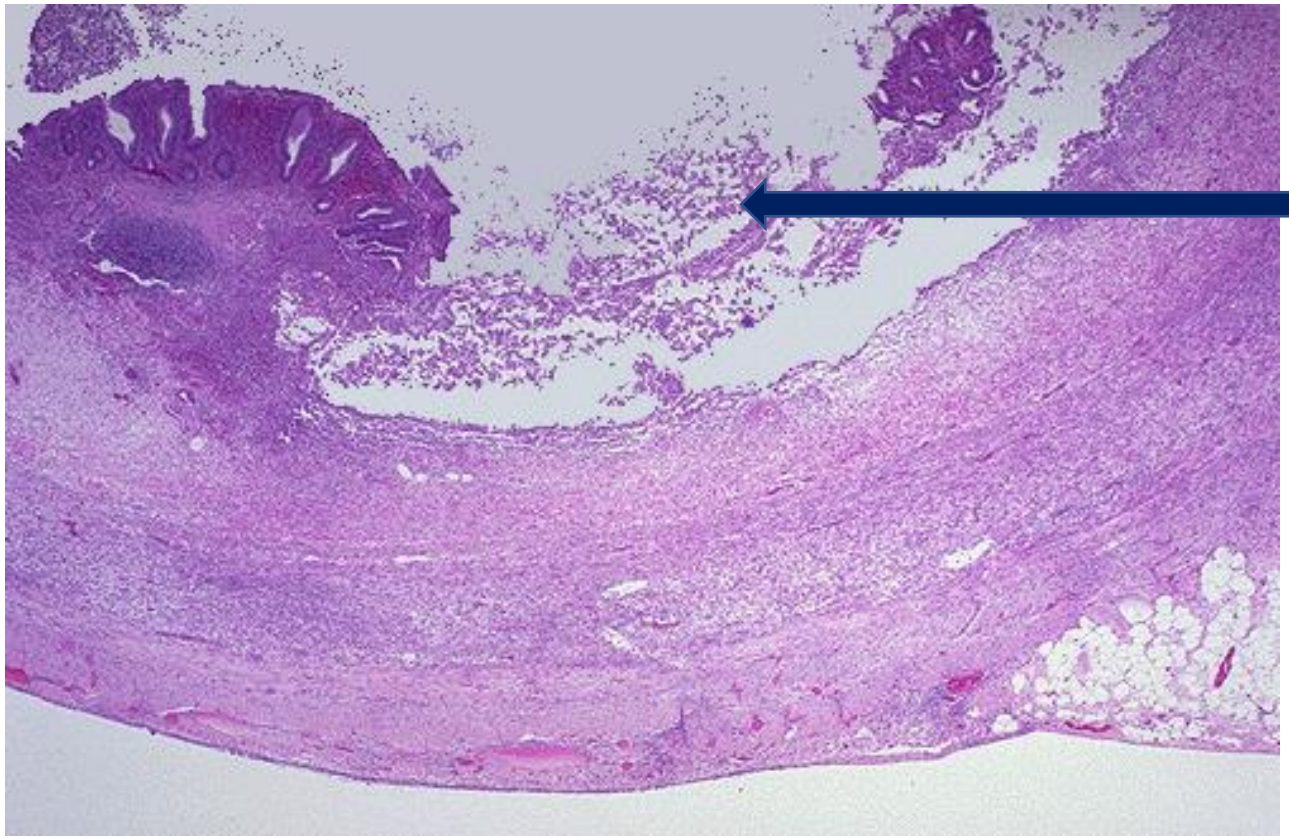
Scant neutrophilic exudate throughout the mucosa, submucosa, and muscularis propria

- Subserosal vessels are congested
- Fibrinopurulent reaction over the serosa
- Abscess formation within the wall, along with ulcerations and foci of suppurative necrosis in the mucosa
- Green-black gangrenous necrosis through the wall, extending to the serosa



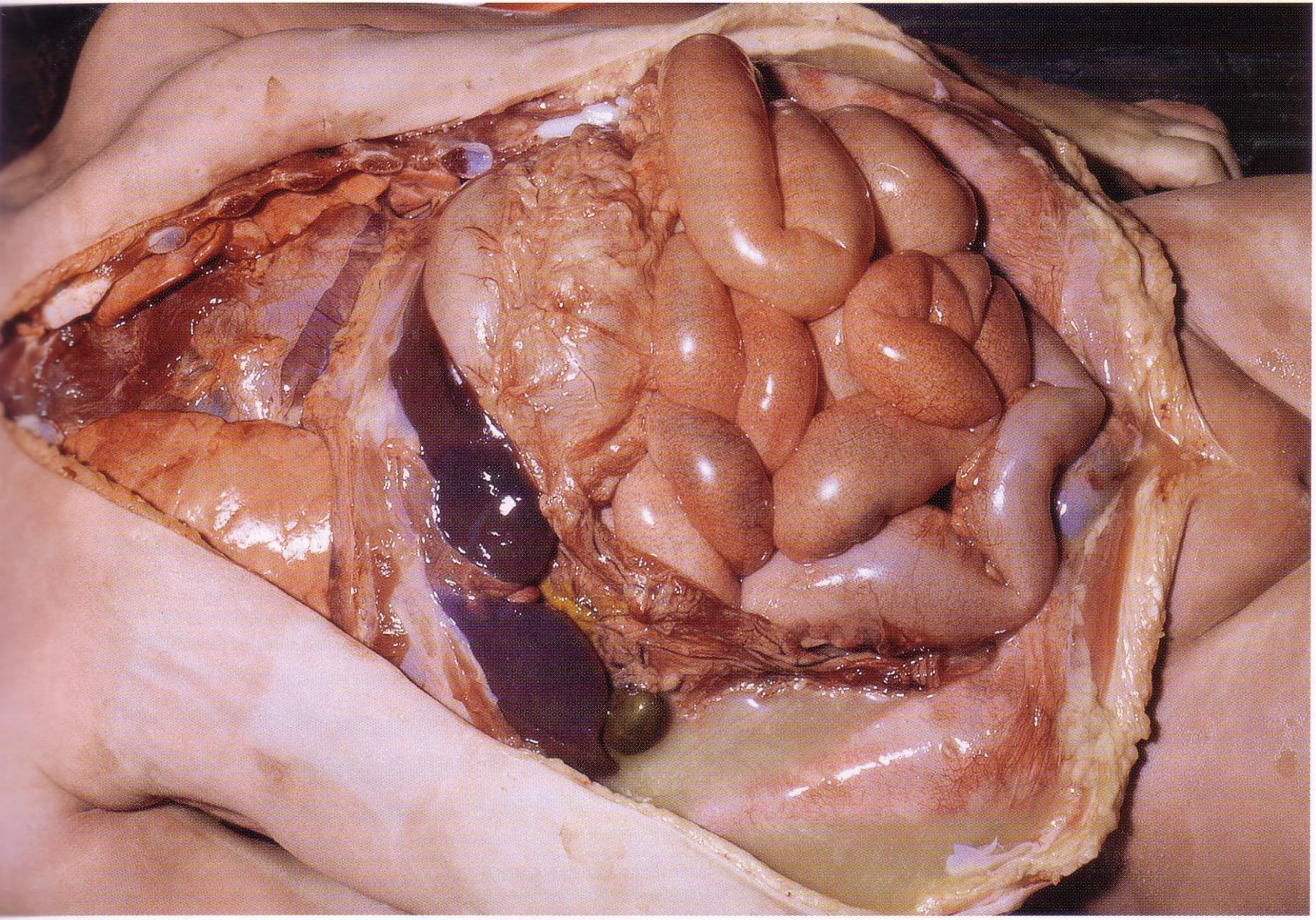


Microscopically, acute appendicitis is marked by mucosal inflammation and necrosis.

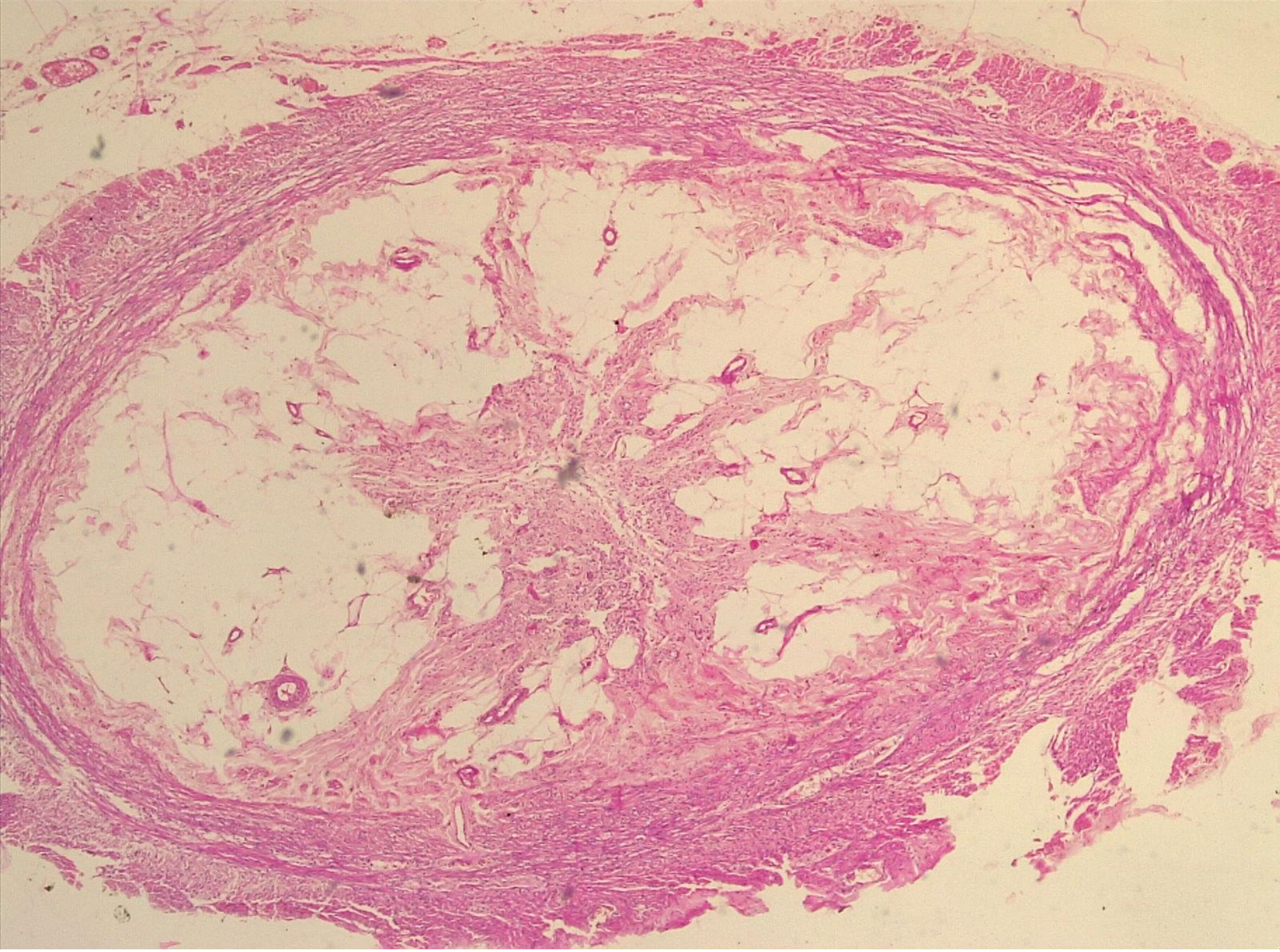


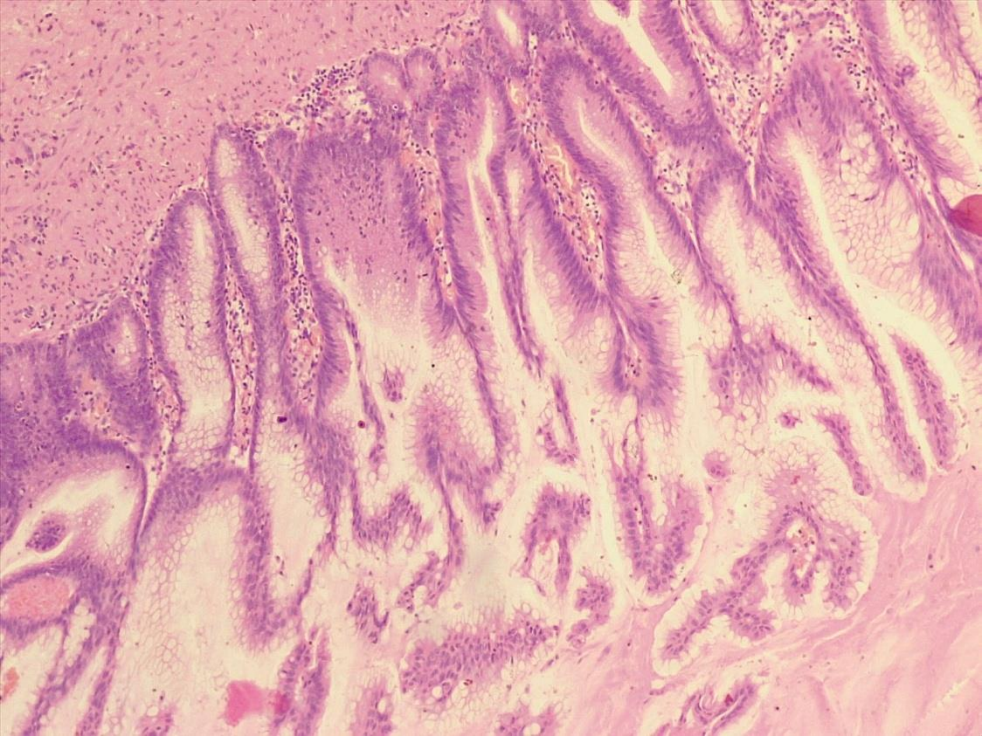
Complications

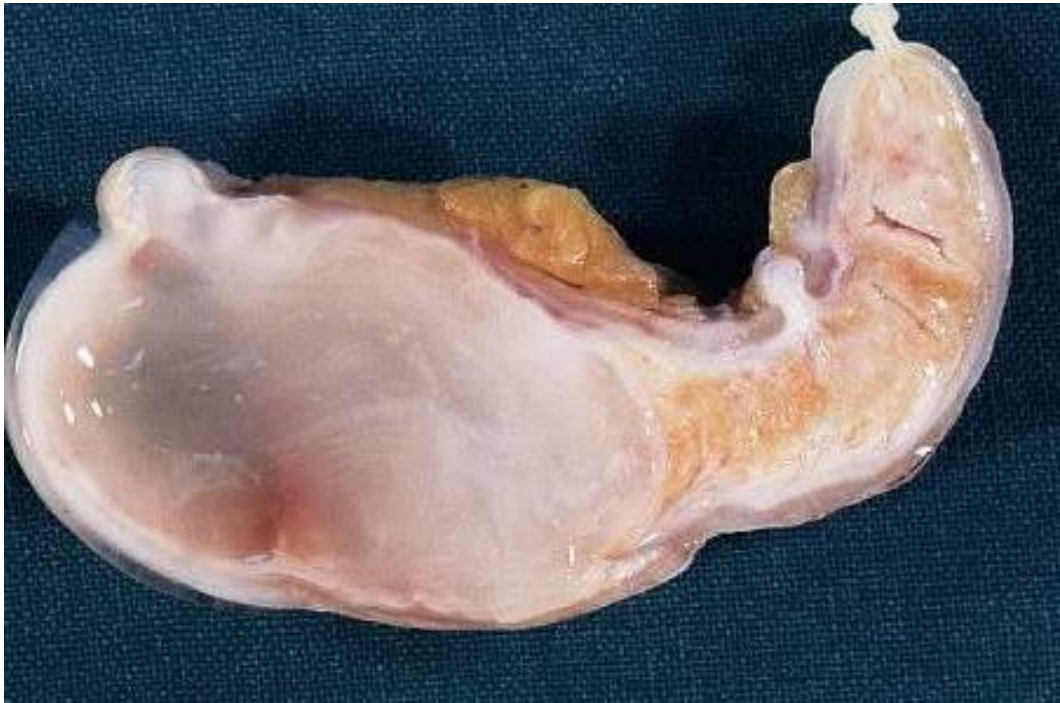
- Rupture
- Suppurative peritonitis
- Pyelophlebitis with thrombosis of the portal venous drainage
- Chronic inflammation of the appendix
- Cystic fibrosis



Гнойный перитонит - осложнение острого аппендицита.







Mucus “TUMORS”

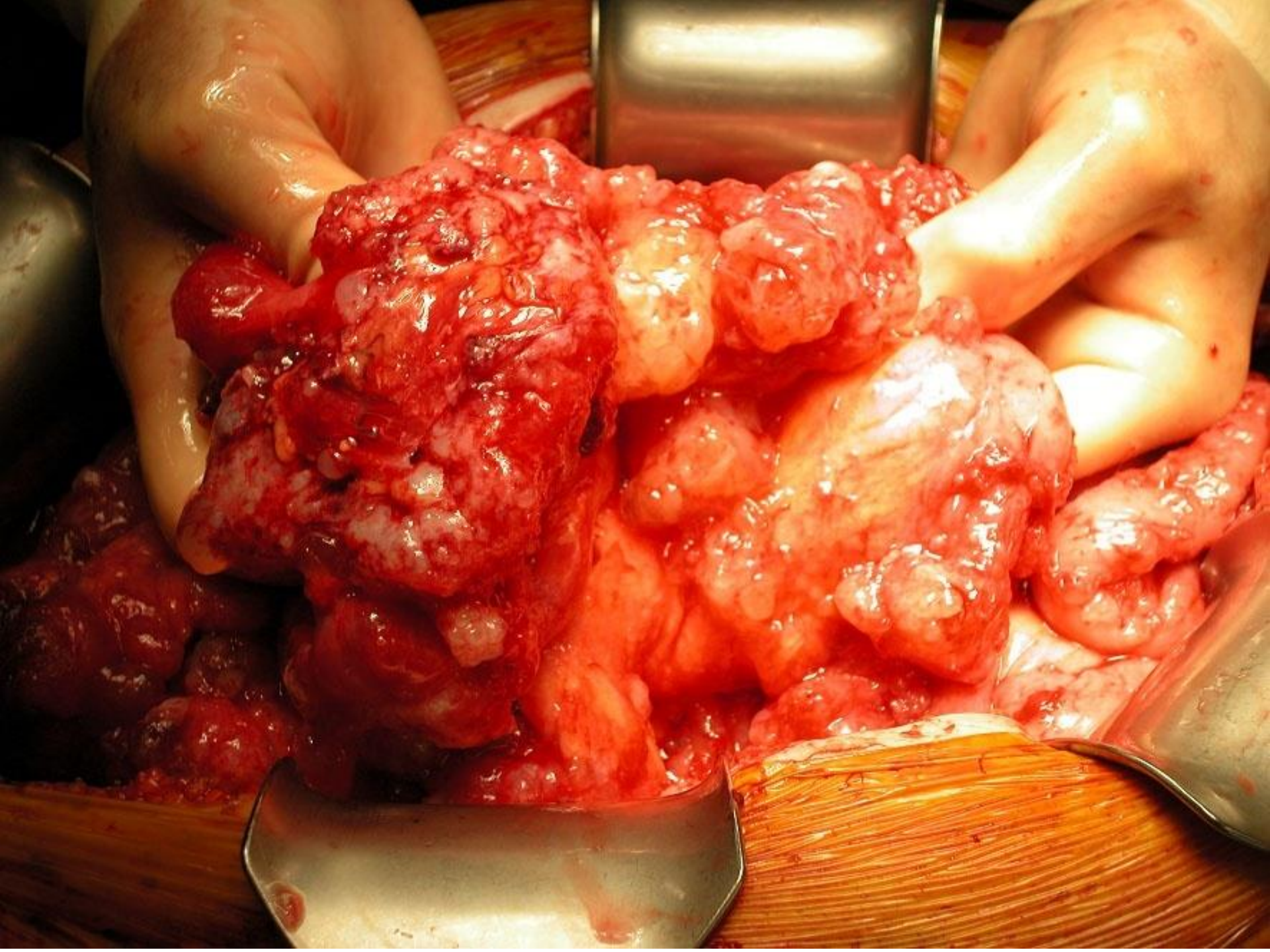
- **Mucocele (common)**
- **Mucinous Cystadenoma (rather rare)**
- **Mucinous Cystadenocarcinoma (rare)**

MUCOCELE

- **COMMON CYST on APPENDIX filled with MUCIN**
- **Can RUPTURE to become:**

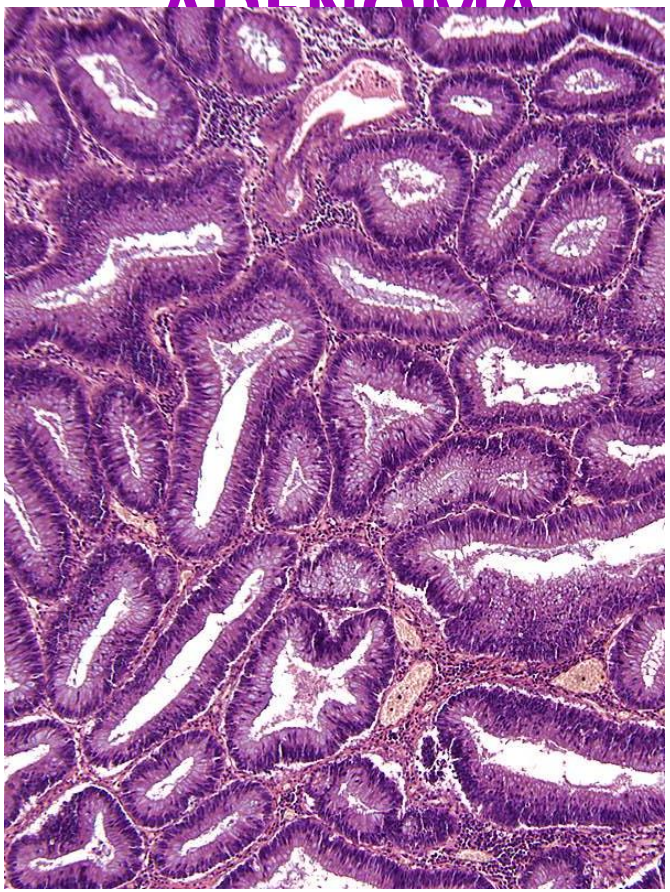
PSEUDOMYXOMA PERITONEII

(Jelly Belly)

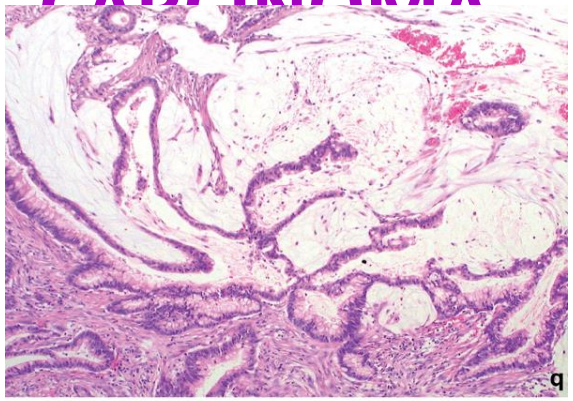


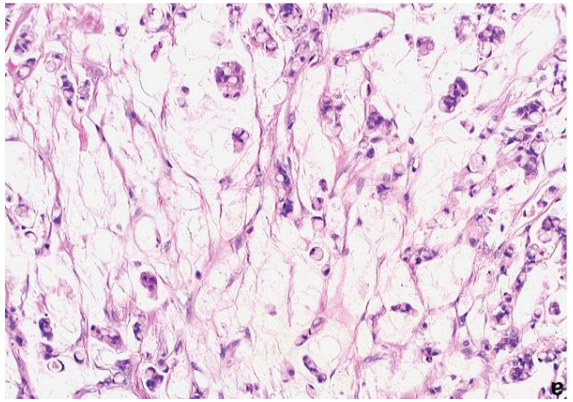
MUCINOUS CYSTADENO(CARCINO)MA

ADENOMA

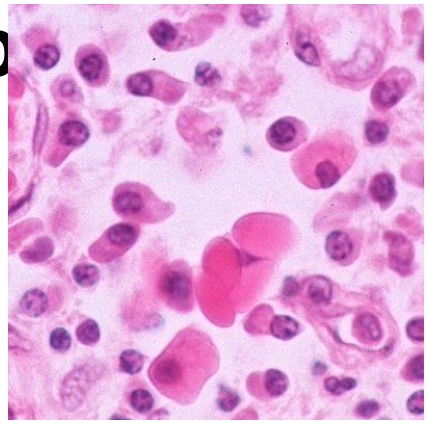


CARCINOMA





PERITONEUM



- Visceral, Parietal: all lined by meso
- Peritonitis, acute:
  - Appendicitis, local or with rupture
  - Peptic ulcer, local or ruptured
  - Cholecystitis, local or ruptured
  - Diverticulitis, local or with rupture
  - Salpingitis→ gonococcal or chlamydial, retrograde or perforated
  - Ruptured bowel due to any reason
  - Perforating abdominal wall injuries

PERITONITIS

- **E. coli**
- **STREP**
- **S. aureus**
- **ENTEROCOCCUS**

PERITONITIS, outcomes:

- **Complete RESOLUTION**
- **Walled off ABSCESS**
- **ADHESIONS**

Tumors of Small and Large Intestine

- Non-neoplastic polyps
- Hyperplastic
  - Hamartomatous polyps
    - Juvenile polyps
    - Peutz-Jeghers polyps
  - Inflammatory polyps
  - Lymphoid polyps
- Lymphoma

- Neoplastic epithelial tumors
  - Benign (Adenoma)
  - Malignant
    - Adenocarcinoma
    - Carcinoid
    - Squamous cell carcinoma (rectum)
- Mesenychymal lesions
  - GIST, lipoma

POLYPS

- **ANY mucosal bulging, blebbing, or bump**

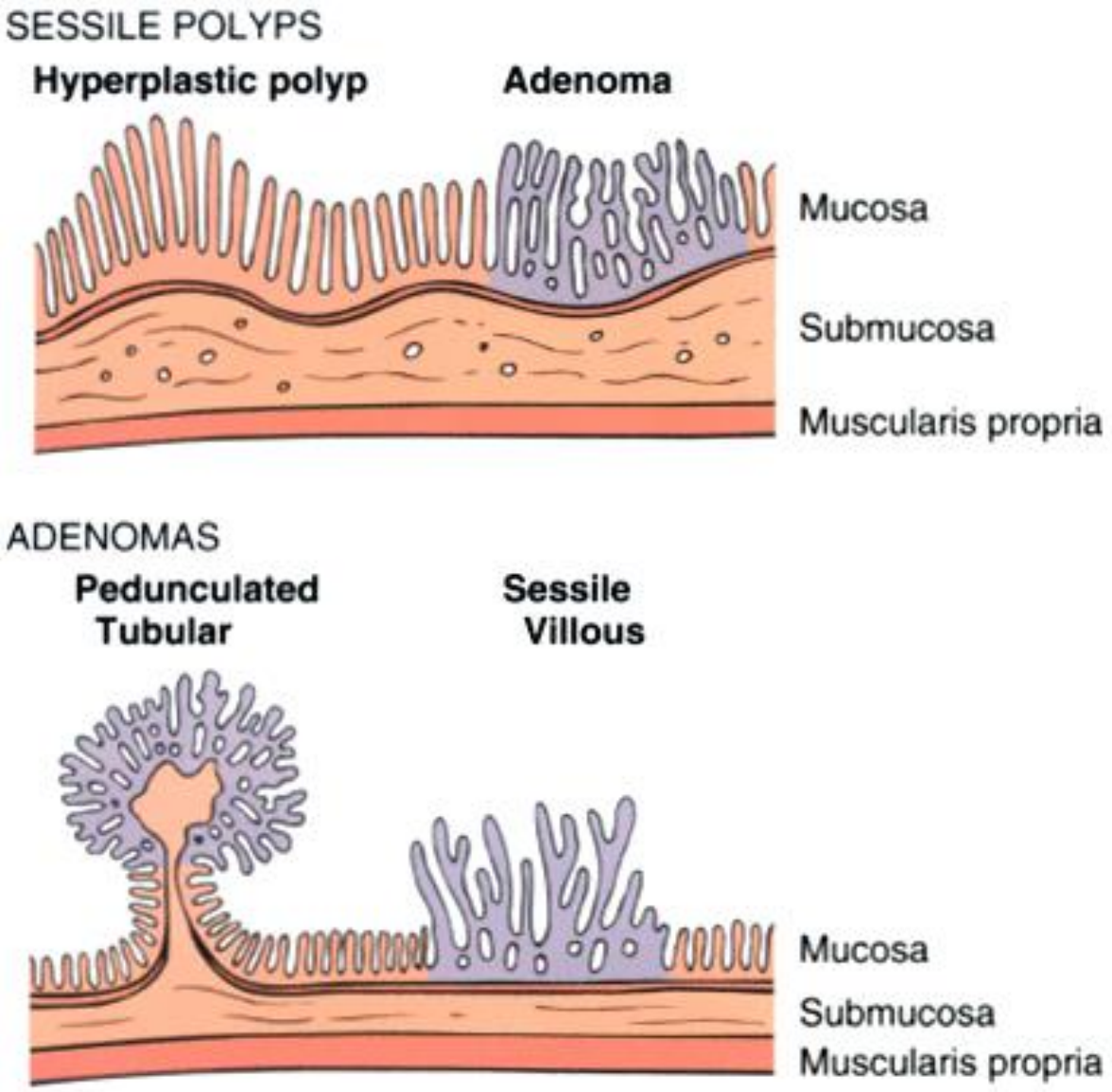
NON-NEOPLASTIC)

(NON-NEOPLASTIC)

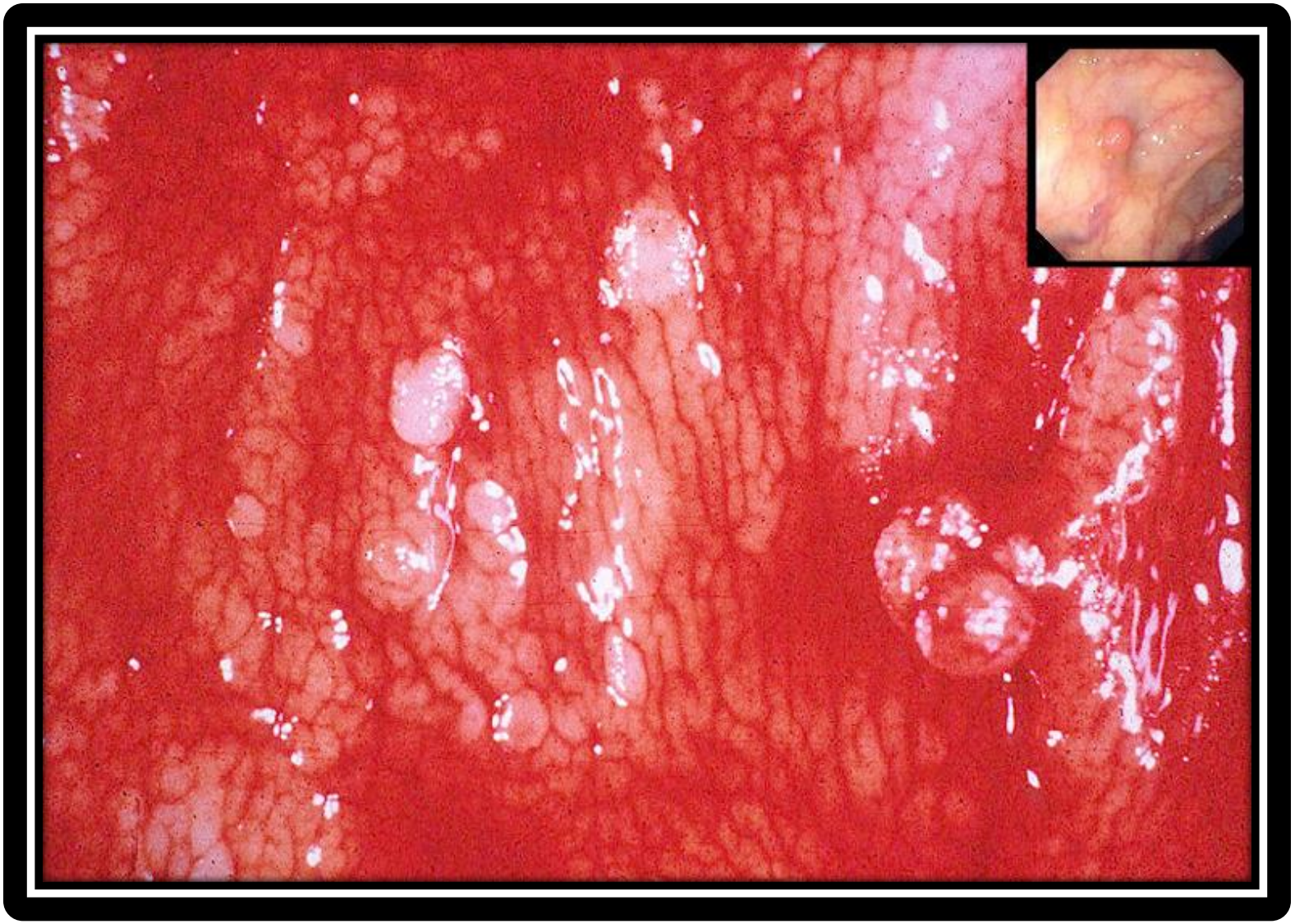
(TRUE NEOPLASM, and regarded by many as "potentially" PRE-MALIGNANT as well)

- **SESSILE vs. PEDUNCULATED**
- **TUBULAR vs. VILLOUS**

POLYPS


SESSILE POLYPS
Hyperplastic polyp
Adenoma
Mucosa
Submucosa
Muscularis propria
ADENOMAS
Pedunculated
Tubular
Sessile
Villous
Mucosa
Submucosa
Muscularis propria

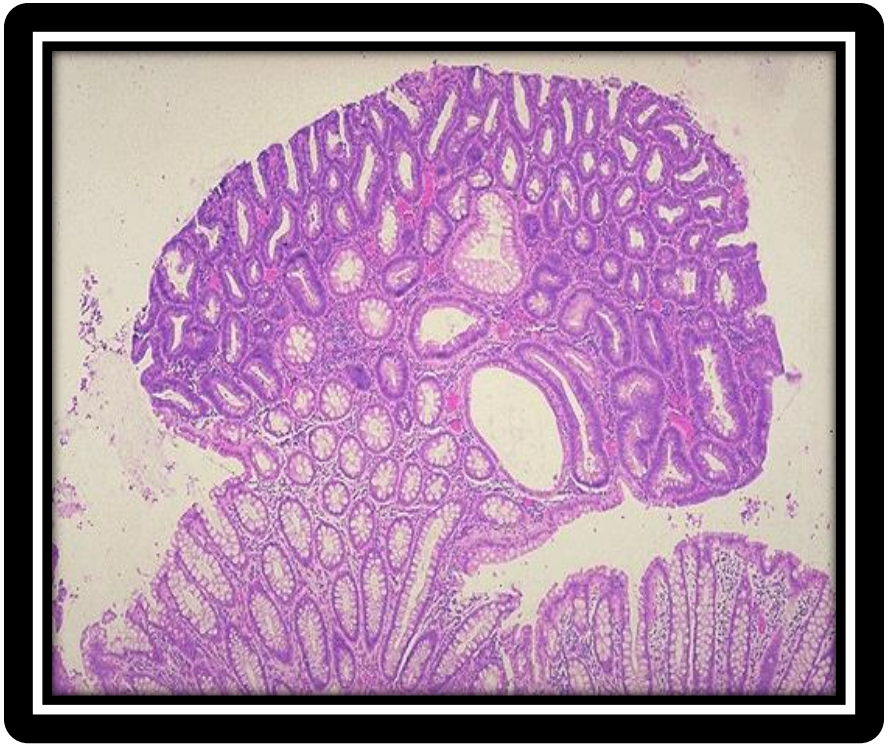
Hyperplastic Polyp



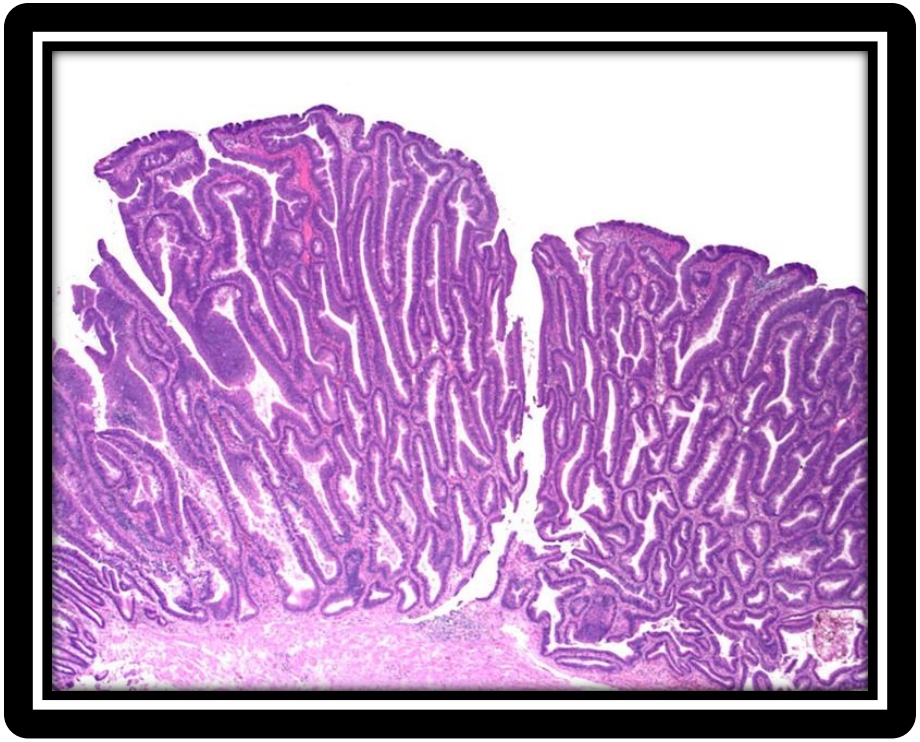
Pre-malignant Polyps

Adenomatous polyps

Tubular Adenoma



Villous Adenoma



Tubulovillous Adenoma

“FAMILIAL” NEOPLASMS

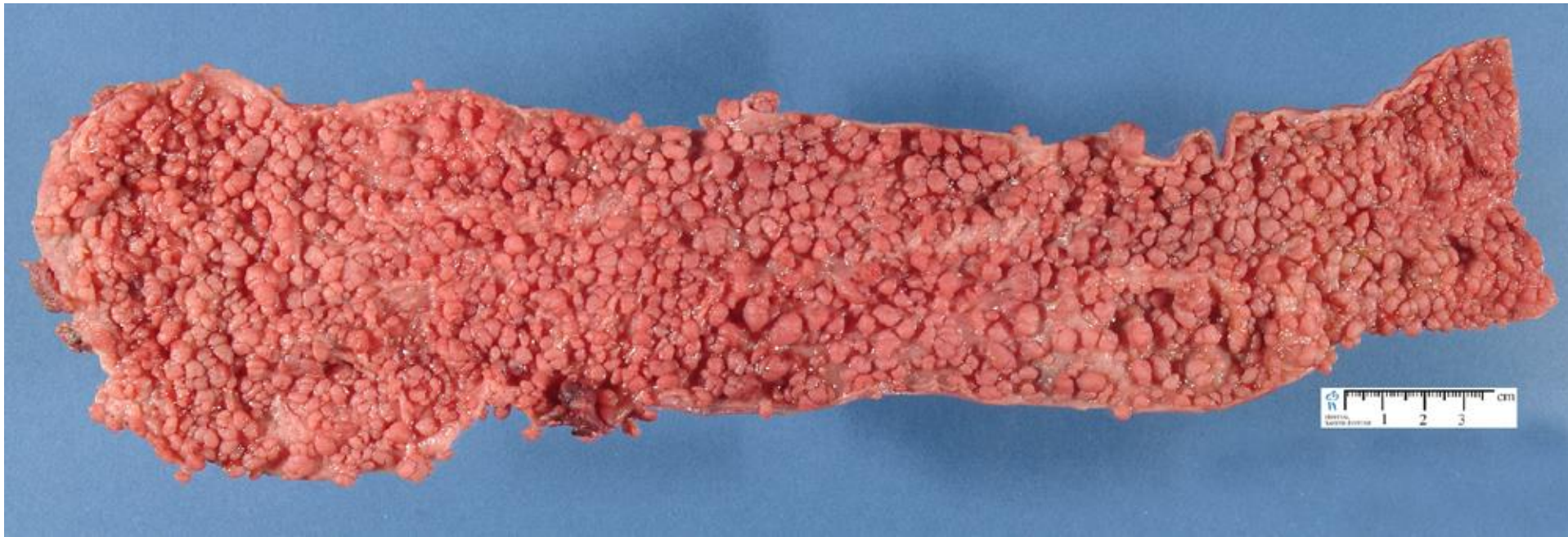
- **1) POLYPOSIS (NON-NEOPLASTIC, hamartomatous)**
- **2) POLYPOSIS (NEOPLASTIC, i.e., cancer risk). FAP.**
- **3) HNPCC: (Hereditary Non Polyposis Colorectal Cancer)**

Hereditary Syndromes.

Some syndromes are known, characterized by the presence of colon polyps and an increased incidence of colon cancer. These syndromes are based on clearly defined genetic disorders.

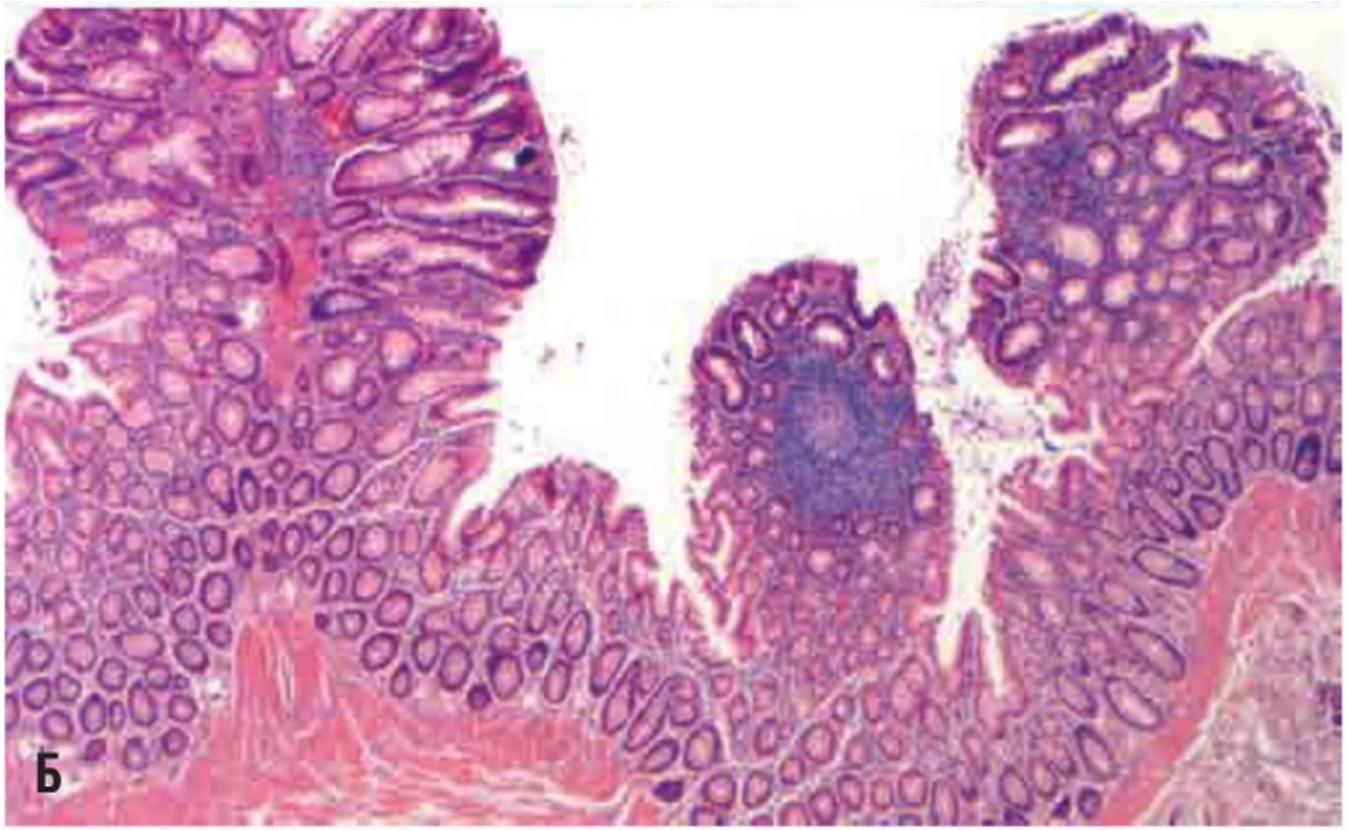
Familial adenomatous polyposis (FAP) is an autosomal dominant disease in which multiple colon adenomas develop in adolescents.

The diagnostic criterion for classic FAP is the presence in the colon of at least 100 polyps (their number can reach several thousand!)



Without treatment of FAP, colon adenocarcinomas develop in 100% of cases, often under the age of 30 years. That is why the standard treatment is prophylactic colon removal. This operation prevents the development of colon cancer, but the increased risk of developing neoplasms of other locations remains. For example, in areas adjacent to the ampulla of the Vater papilla, and adenomas may develop in the stomach.

It is important to note that in FAP flat adenomas predominate, and microadenomas consisting of only one or two dysplastic glands are often determined in areas of externally unchanged mucous membrane.



CANCER GENETICS

- **Loss of APC gene**
- **Mutation of K-RAS**
- **Loss of SMADs (regulate transcription)**
- **Loss of p53**
- **Activation of TELOMERASE**

CANCER RISK FACTORS

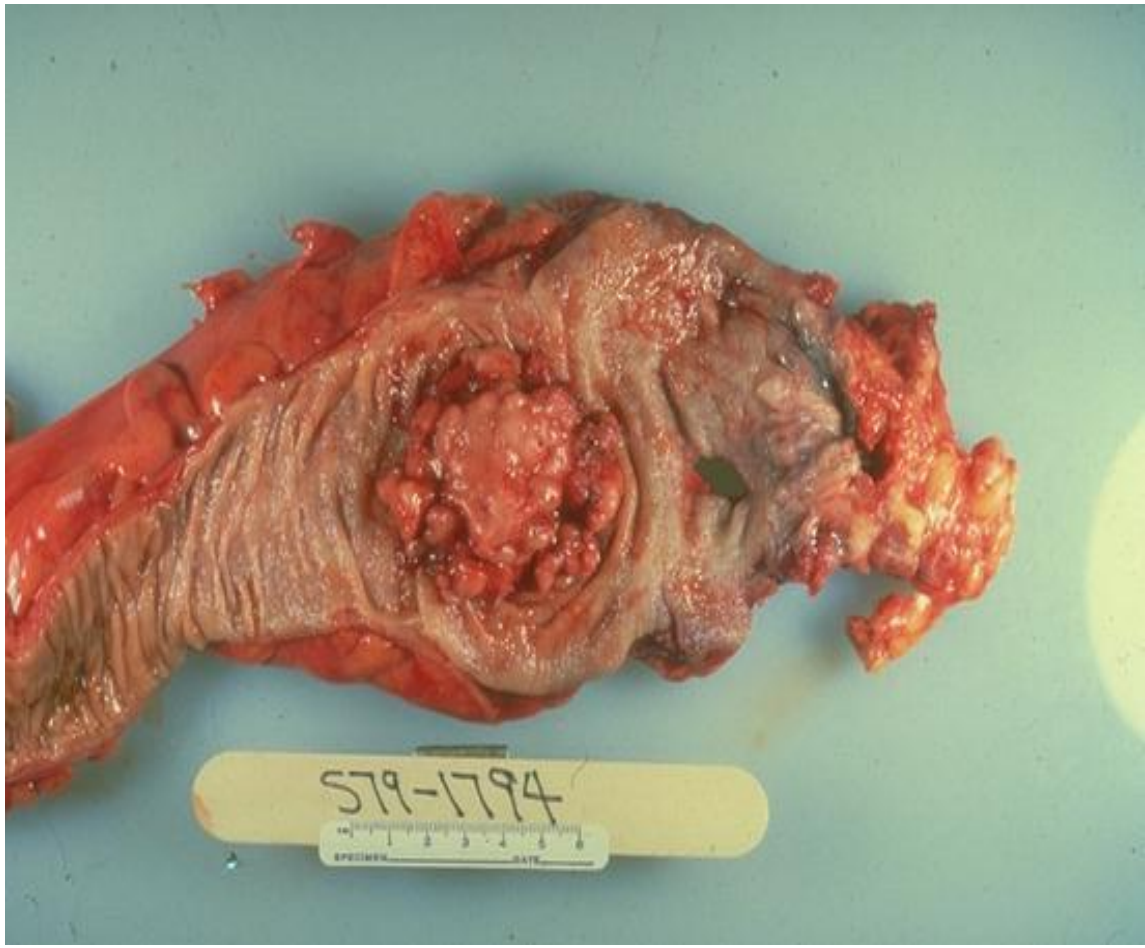
- **Family history**
- **Age (rare <50)**
- **LOW fiber, HIGH meat, LONG transit time, refined carbs**

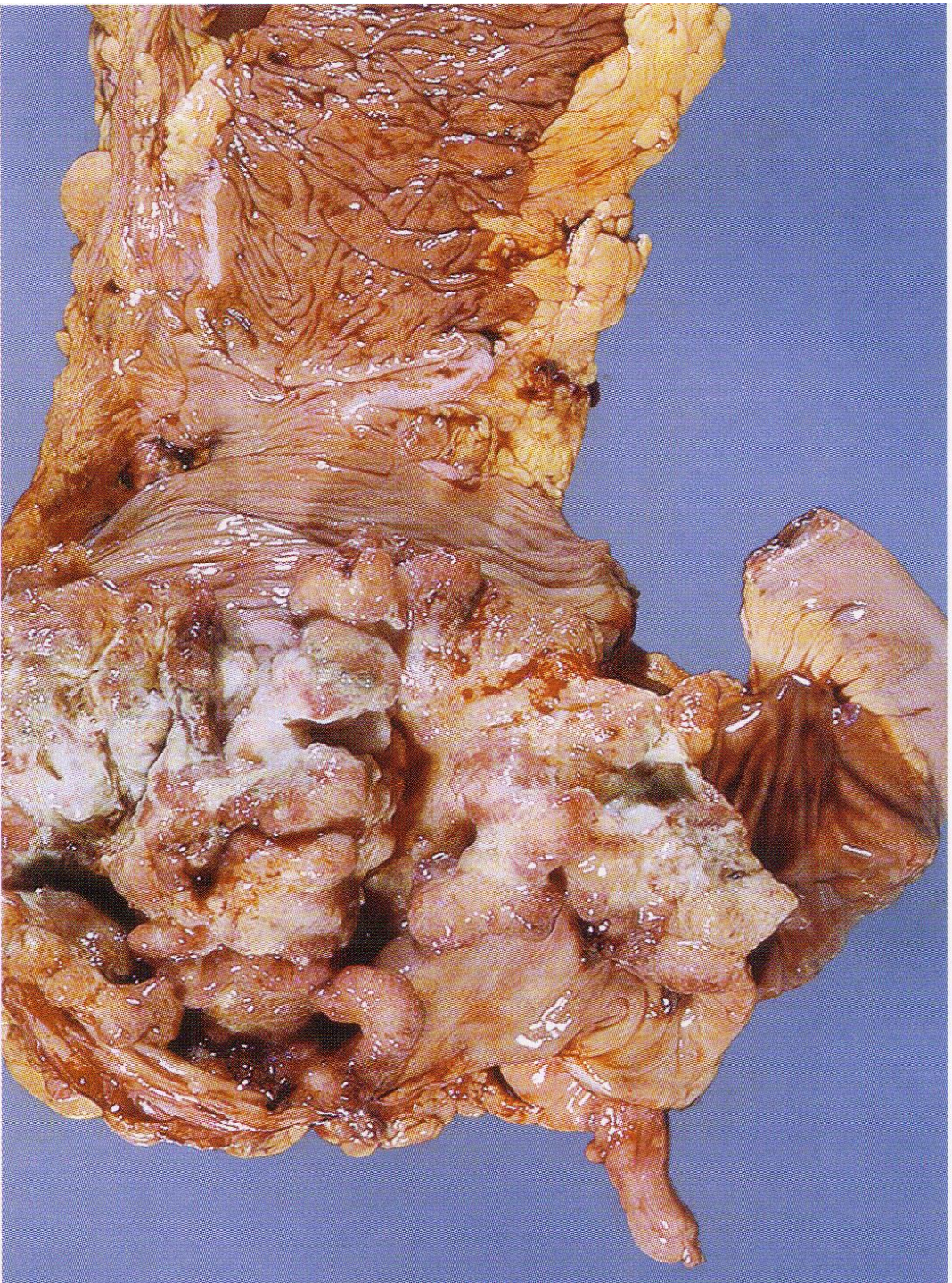
PATHOGENESIS

- **From existing ADENOMATOUS POLYPS**
- **DE-NOVO**

- **DYSPLASIA→INFILTRATION→ METASTASIS**

Features of the diet that affect the incidence of colon cancer are low fiber intake and high intake of refined carbohydrates and fats. It is assumed that a decrease in fiber intake decreases the rate of movement of feces and disrupts the composition of the intestinal microflora. These changes can lead to the accumulation of potentially toxic products of bacterial metabolism, which for a long time come into contact with the mucous membrane of the colon.



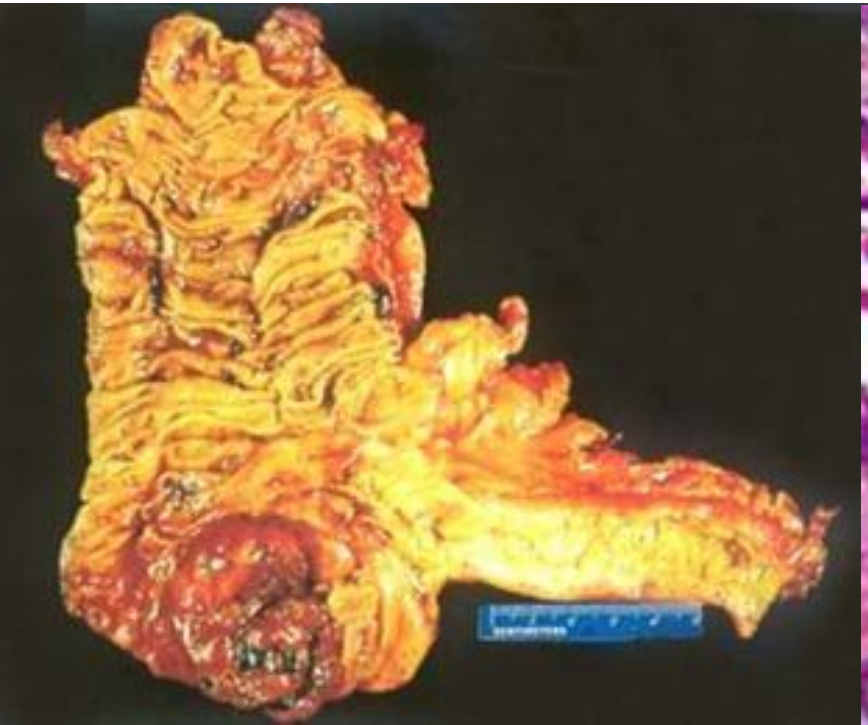


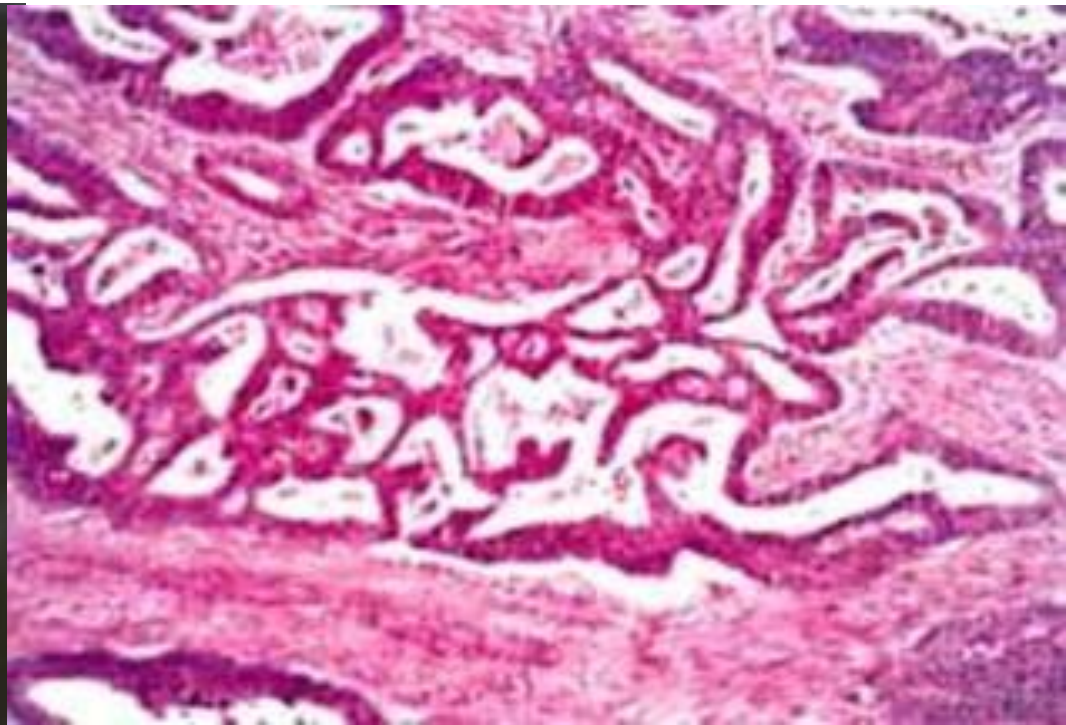
Adenocarcinoma

Colon adenocarcinoma is the most common malignant tumor of the gastrointestinal tract In contrast, in the small intestine, which accounts for 75% of the entire gastrointestinal tract, benign and malignant tumors are extremely rare.

GROWTH PATTERNS

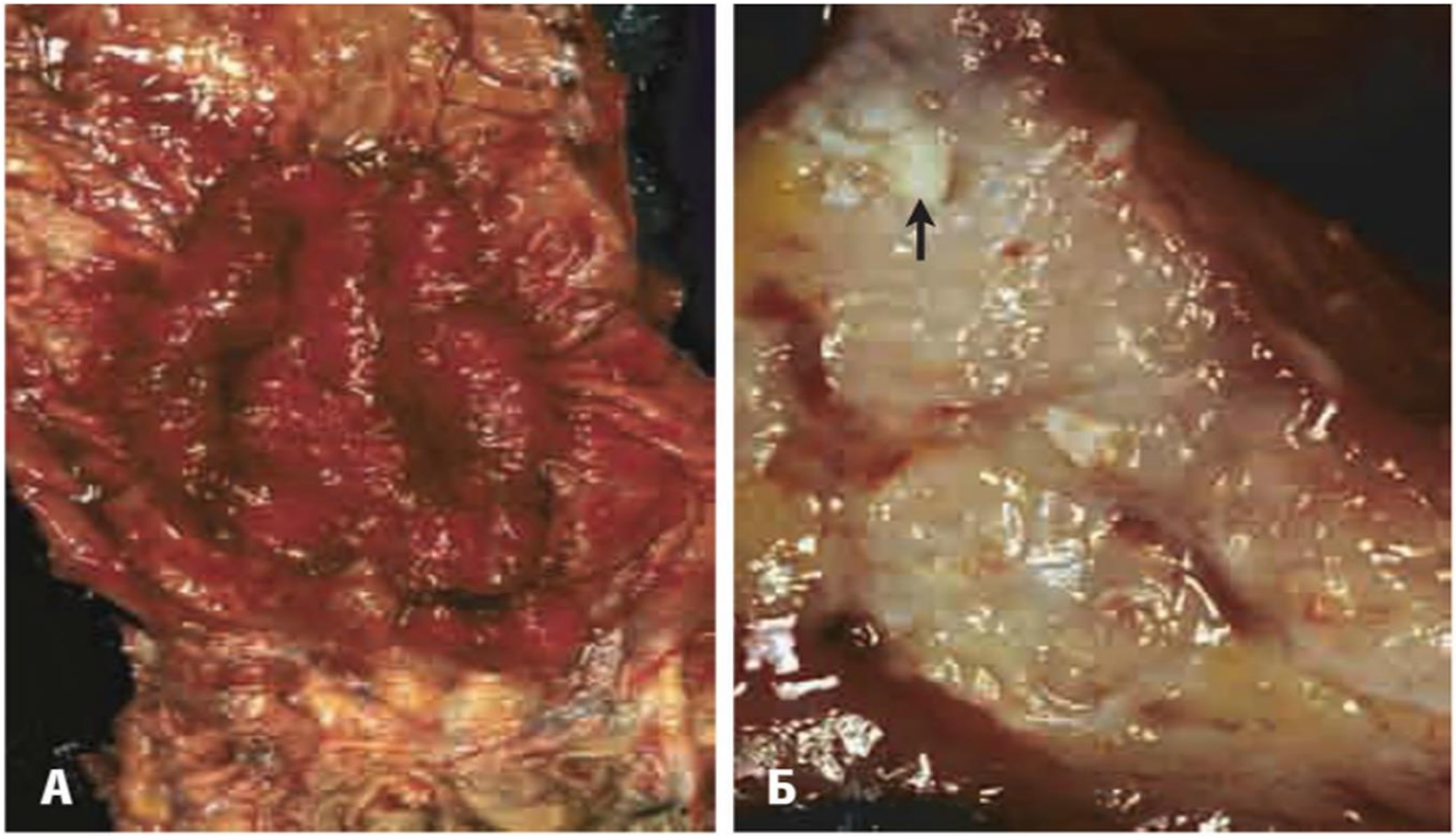
- **POLYPOID**
- **ANNULAR, CONSTRICTING**
- **DIFFUSE**

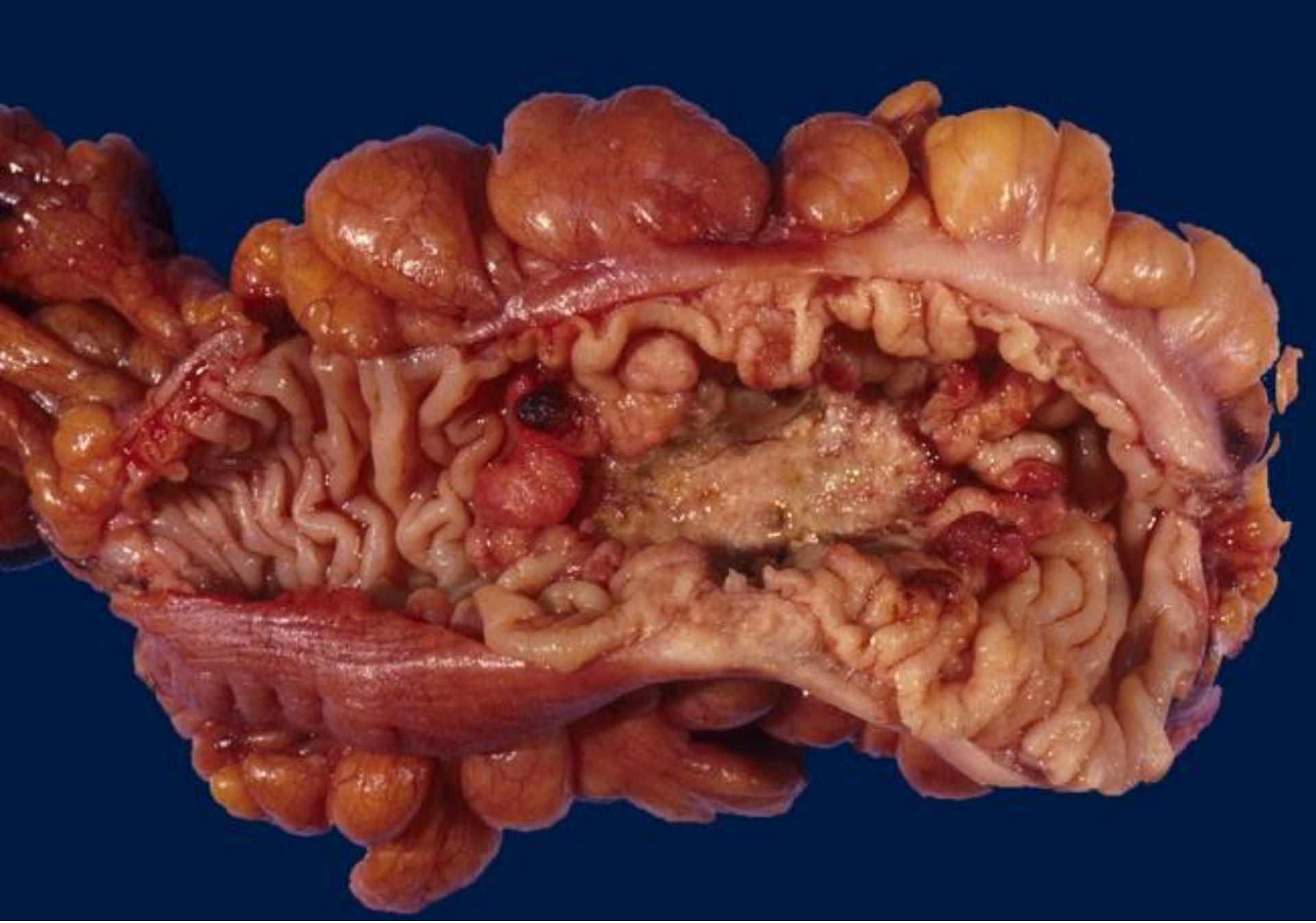


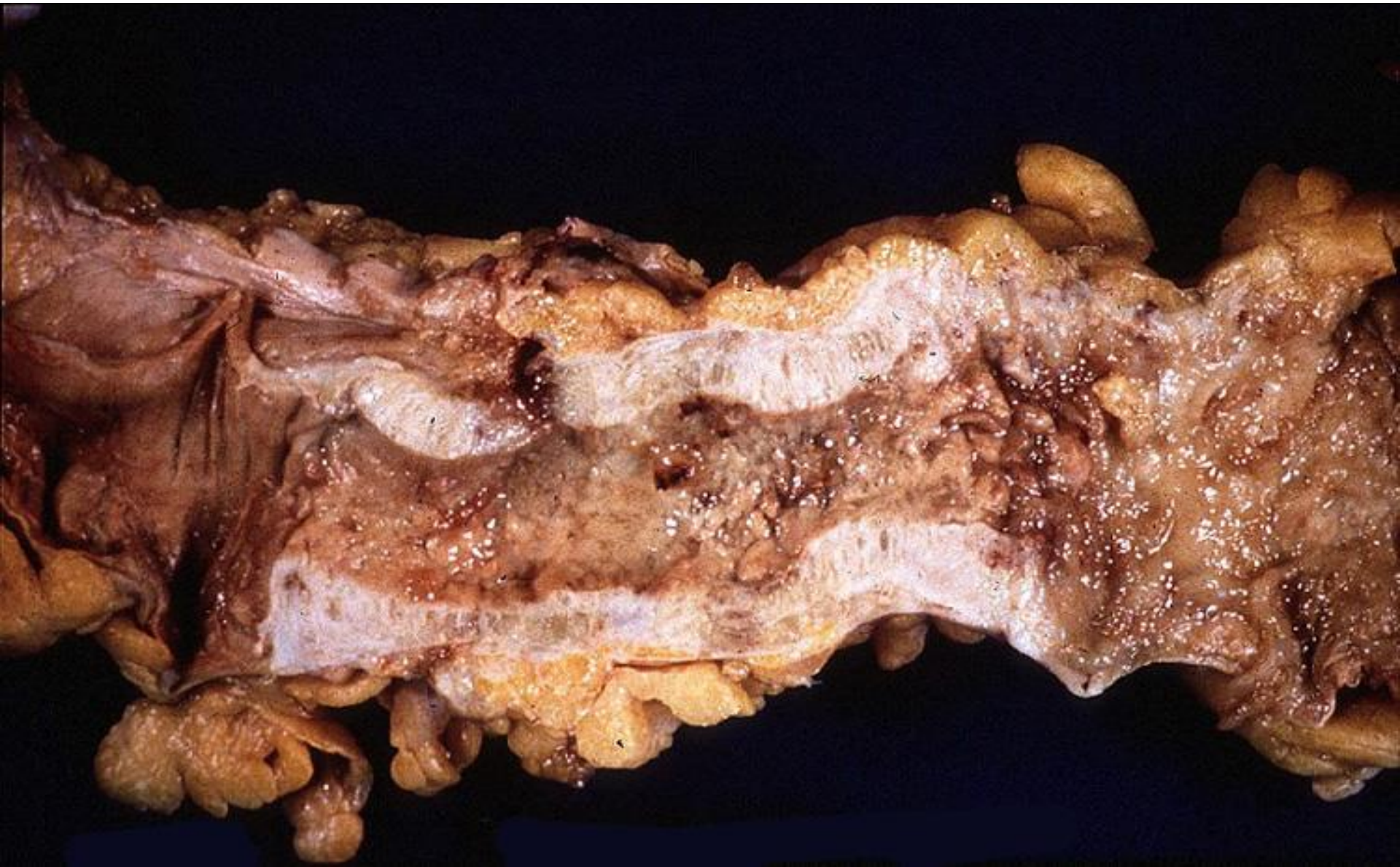


Morphology. Adenocarcinomas with almost the same frequency affect all parts of the colon. Tumors of the proximal colon usually grow in the form of polypoid exophytic masses spreading along one wall of the cecum or ascending colon. Such tumors rarely lead to intestinal obstruction.

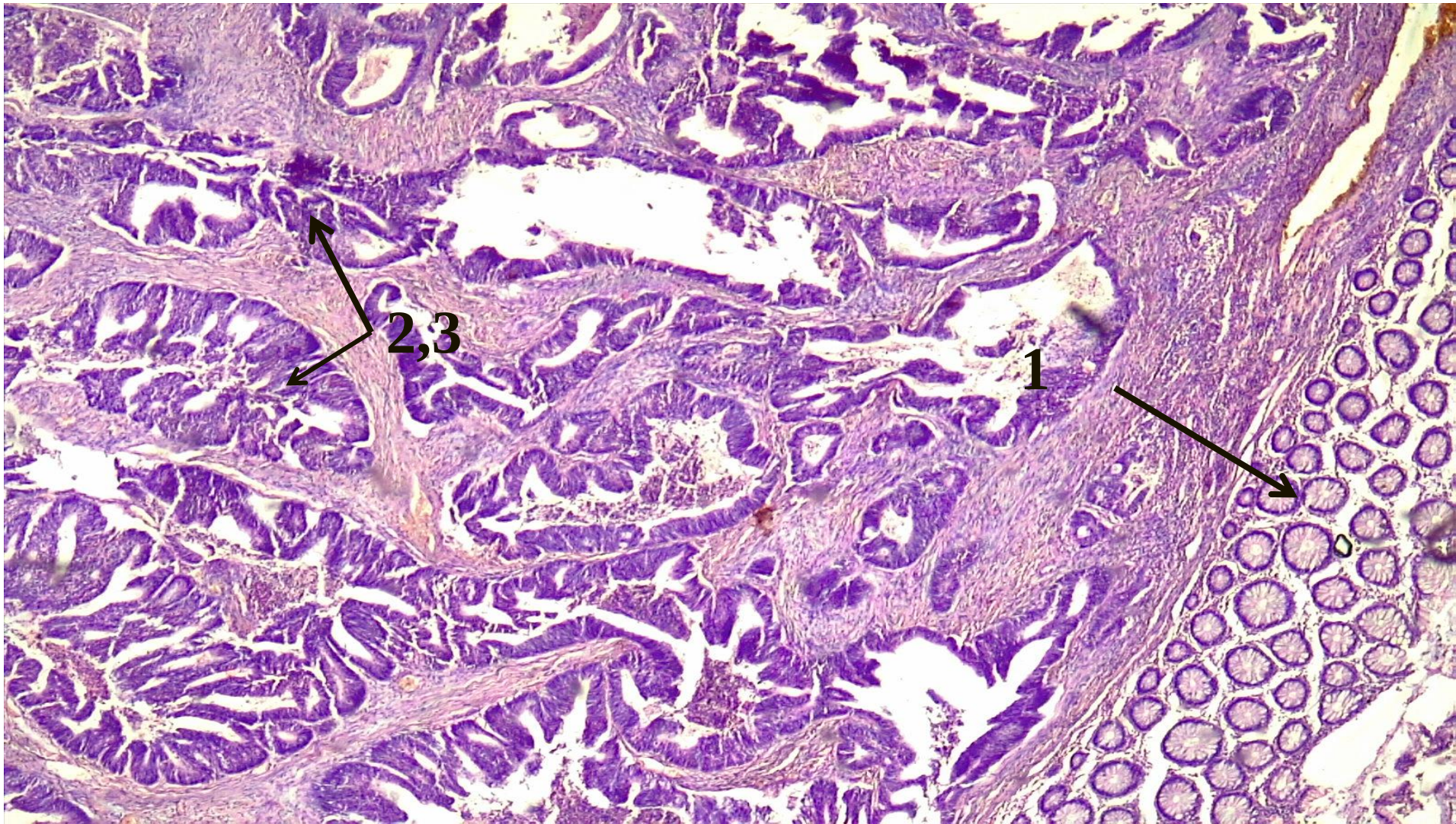
On the contrary, tumors of the distal colon usually have the form of ring-shaped formations and lead to a narrowing of the lumen of the intestine and sometimes to intestinal obstruction. In both cases, tumors grow over time into the wall of the colon and upon palpation are determined in the form of dense masses.



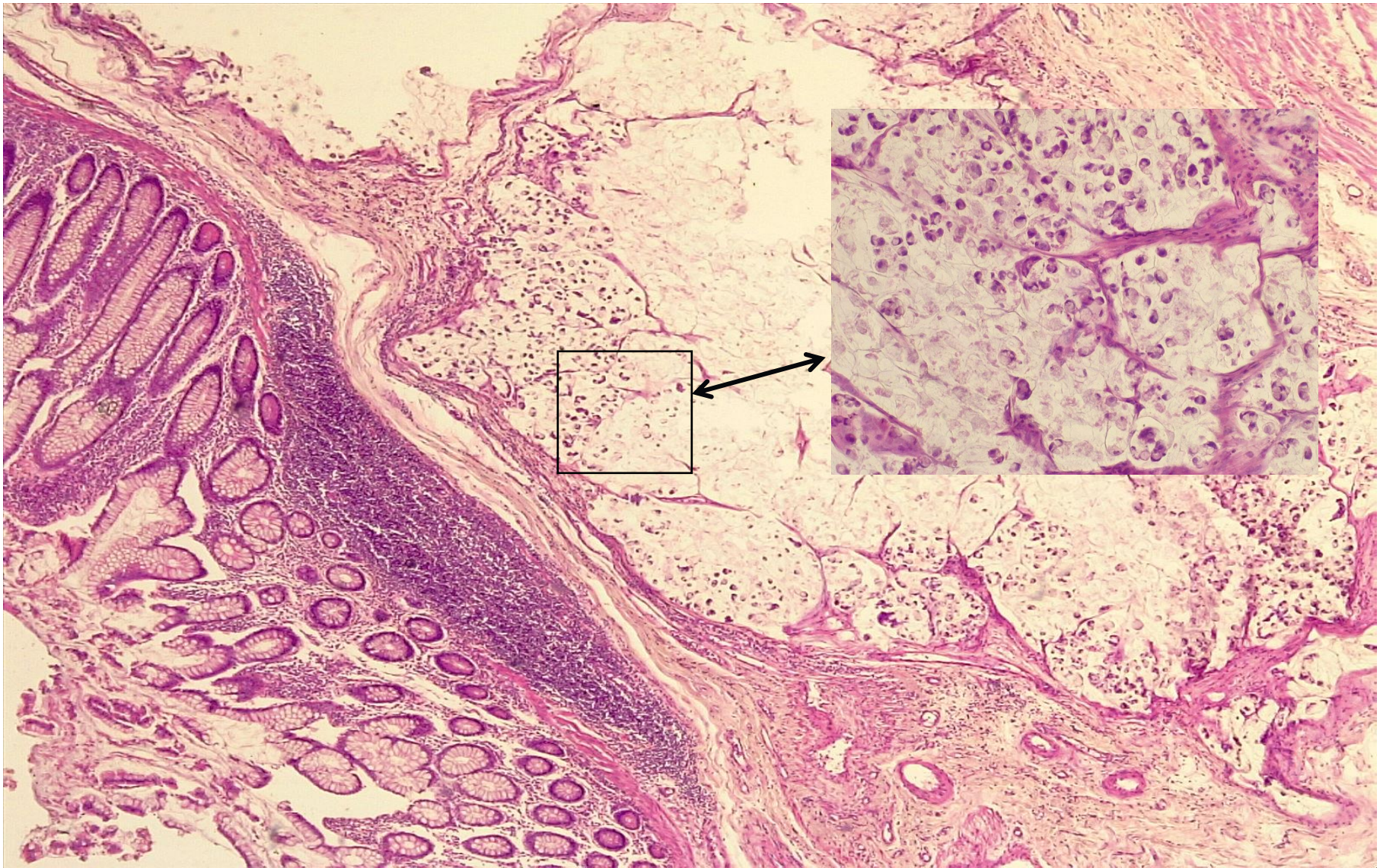




General histological characteristics of adenocarcinomas of the distal and proximal colon are similar. Most tumors consist of tall cylindrical cells resembling the dysplastic epithelium found in adenomas. The invasive component of these tumors causes a pronounced desmoplastic reaction of the stroma, which provides a characteristic dense consistency.



Some low-grade tumors form just a few glands, while others can produce mucus that builds up in the intestinal wall. Such adenocarcinomas have a poor prognosis. Tumors can also consist of signet ring cells, similar to those in similar tumors of the stomach.



Tumor Stage	Histologic Features of the Neoplasm
Tis	Carcinoma in situ (high-grade dysplasia) or intramucosal carcinoma (lamina propria invasion)
T1	Tumor breaches the musc. Muc. invades into submucosa
T2	Extending into the muscularis propria but not penetrating through it
T3	Penetrating through the muscularis propria into subserosa
T4	Tumor directly invades other organs or structures
Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in 1 to 3 lymph nodes
N2	Metastasis in 4 or more lymph nodes
Mx	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

OTHER TUMORS

- **CARCINOID, with or without syndrome**
- **LYMPHOMA (MALTOMAS, B-Cell)**
- **LEIOMYOMA/-SARCOMA**
- **LIPOMA/-SARCOMA**

ANAL CANAL CARCINOMAS

- **MORE LIKELY TO BE SQUAMOUS, or "basaloid"**
- **WORSE IN PROGNOSIS**
- **HPV RELATED**