Gastrointestinal Pathology
Esophagus and Stomach

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2nd Department of Pathology

2021 February, Budapest

250 years of EXCELLENCE in medical education, research & innovation and healthcare

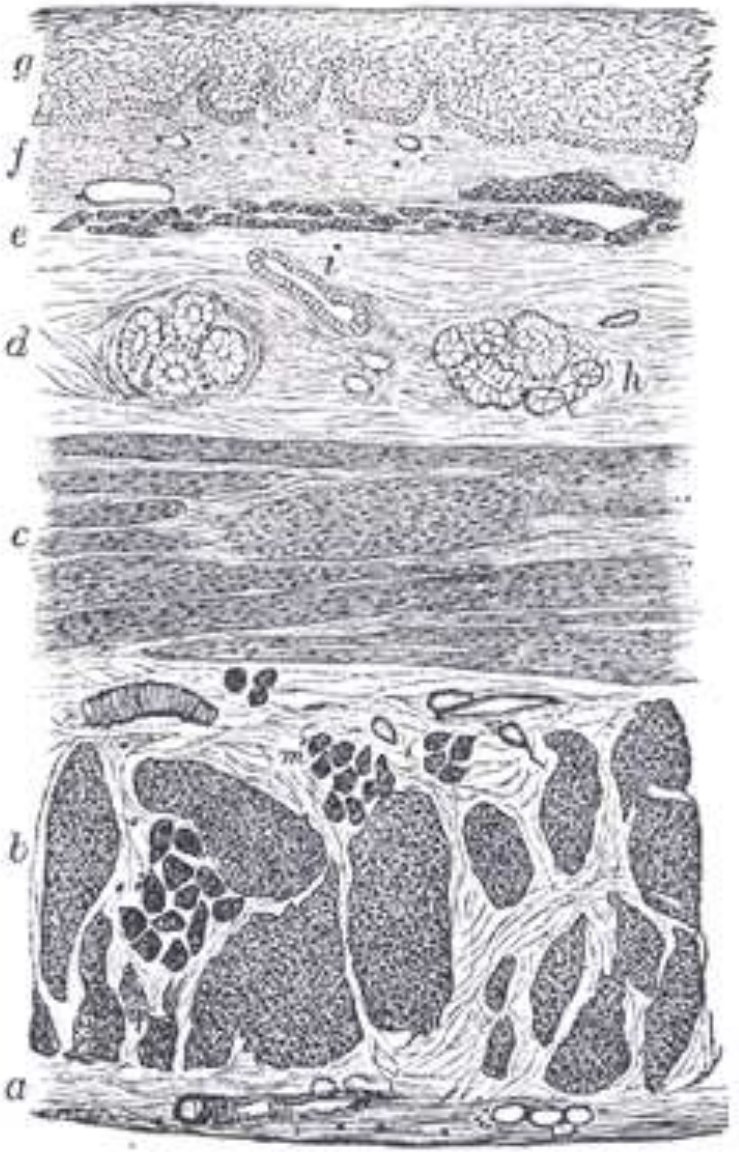

VERY FEW KIDS DREAM OF
BEING A GASTRONAUT.

Esophagus

- Anatomy
- Congenital anomalies
- Motor dysfunction
- Esophageal varices
- Inflammatory conditions
- Neoplasms

Anatomy

- between C6 and Th11-12
- Length:
  - 10 cm in the newborn
  - 25 cm in adults
  - by endoscopy: between 15 and 40 cm from the incisor teeth
- Areas of luminal narrowing
  - at the cricoid cartilage
  - at the anterior crossing of the left main bronchus and left atrium
  - at the diaphragm



Anatomy

Mucosa

squamous epithelium

Submucosa

glands, vessels, lymphatic vessels and follicules , venes!!!

Tunica musc propria

Adventitia

(No serosa)

ESOPHAGUS

Esophagusatresia: not or only paritally developed esophagus

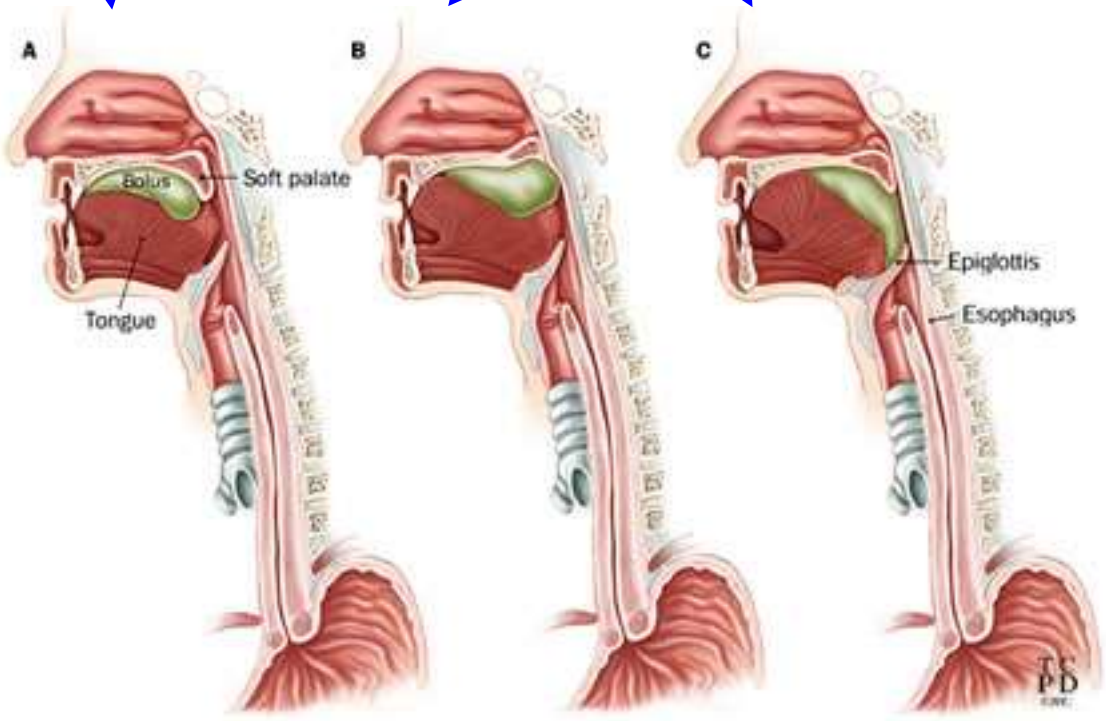
in 90 % of cases simultaneous ösophagotracheale Fistule
complication: Polyhydramnion
(because of intrauterine defect of swallowing)

Dysphagia lusoria: abnormally positioned aortic arch or arteria lusoria (atypical a. subcl.)
Compression: stenosis of the esophagus
Dysphagia: disturbed act of swallowing

Physiology of swallowing

Oral Phase

Pharyngeal Phase



Physiology of swallowing

Pharyngeal and esophageale phase:

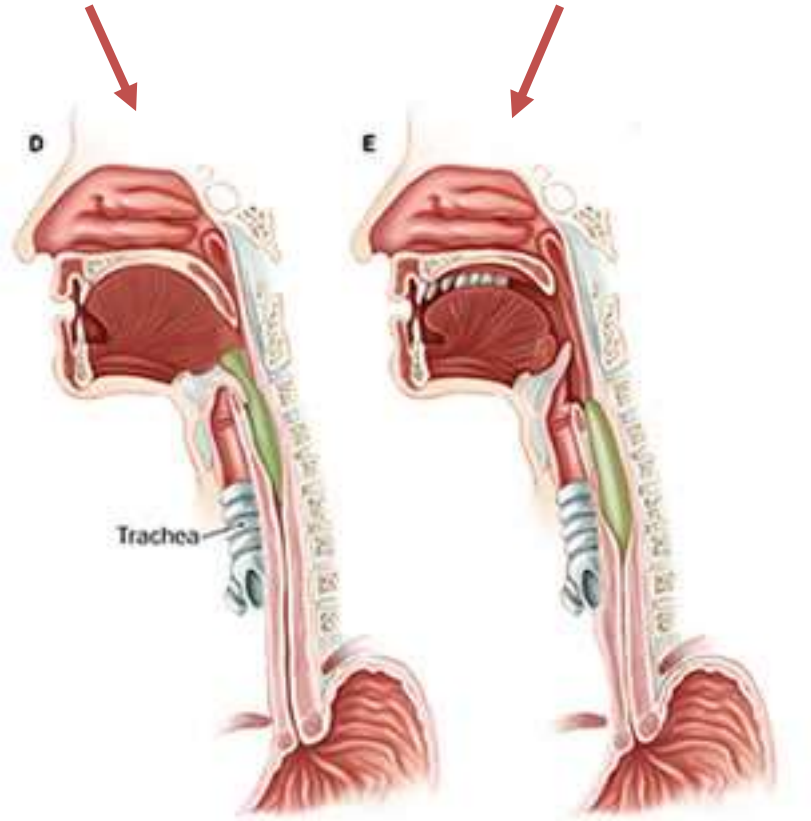
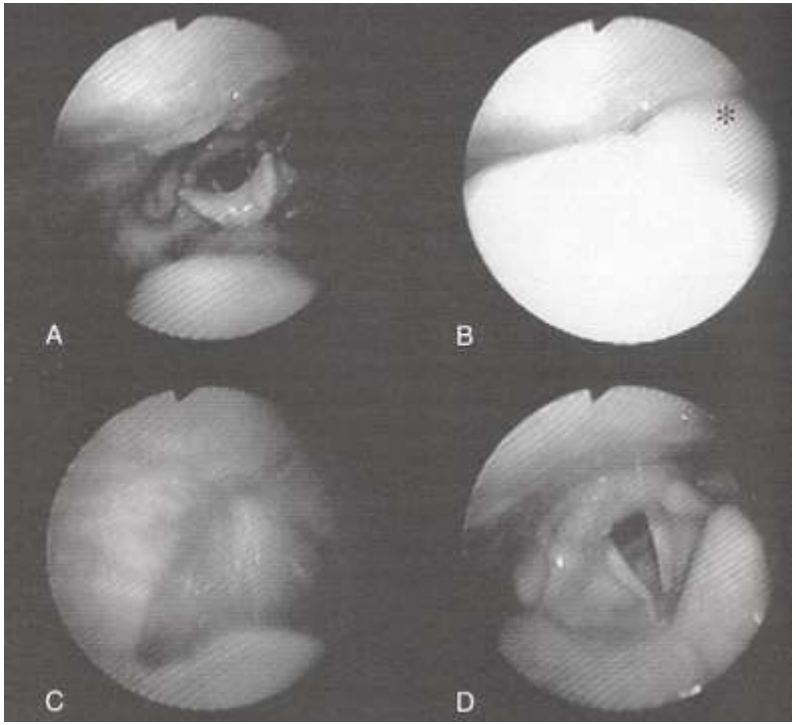
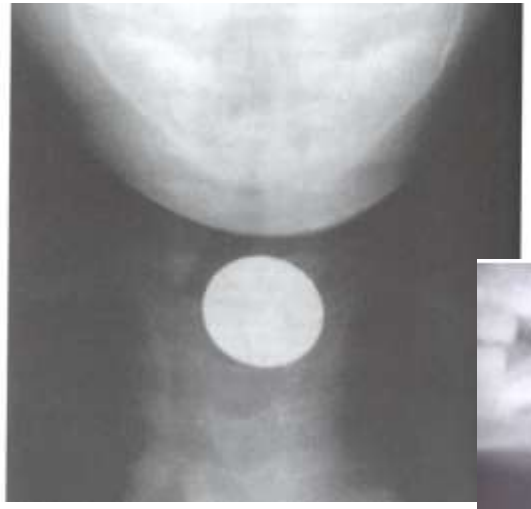


Figure 4 A-E. Pharyngeal phase of swallowing.

Fiberoptical endoscopy investigation of disturbed act of swallowing

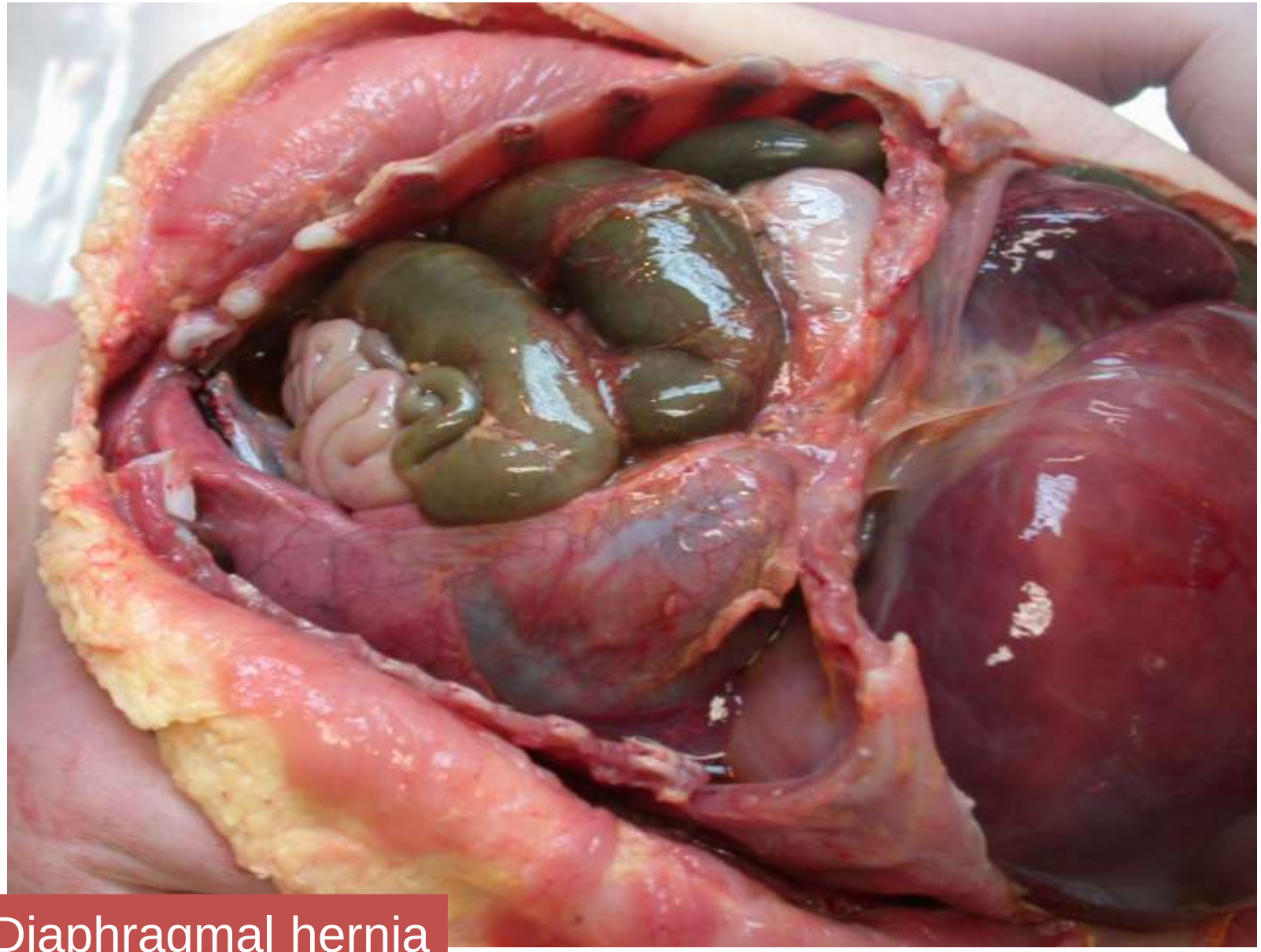


Foreign body



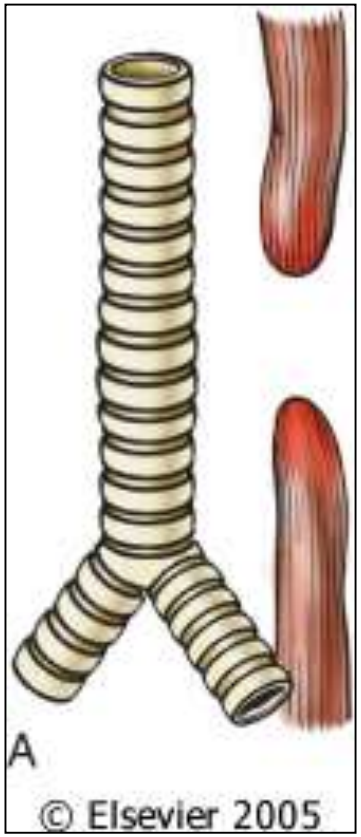
Congenital anomalies

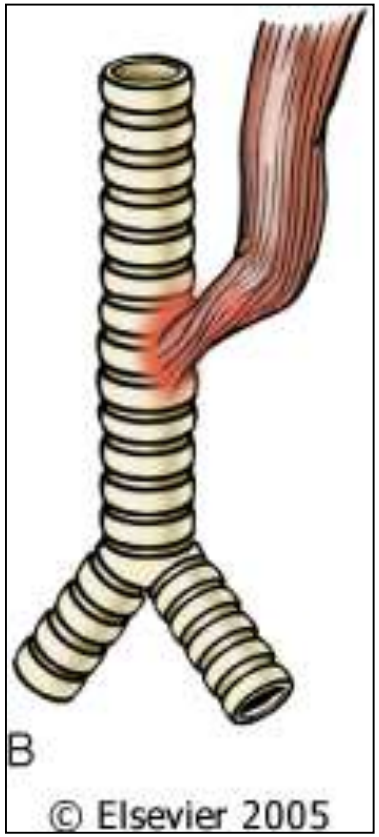
- **Ectopic tissues**: gastric, pancreatic
- **Congenital cysts**:
  - duplication cysts in the lower esophagus
- **Diaphragmal hernia**:
  - abdominal viscera in the thorax (not to confuse with hiatal hernia → see later)
- **Atresia**:
  - a segment of the esophagus is a thin cord, the proximal part communicates generally with the upper respiratory tract by a **fistula**- the distal pouch may also be connected to the trachea
- **Mucosal webs**:
  - semicircumferential protrusion of the mucosa into the lumen of the upper esophagus
- **Mucosal rings**:
  - mucosa, submucosa and sometimes hypertrophied muscle protruding into the lumen of the lower esophagus in a concentric fashion (A ring, B or Schatzki ring)

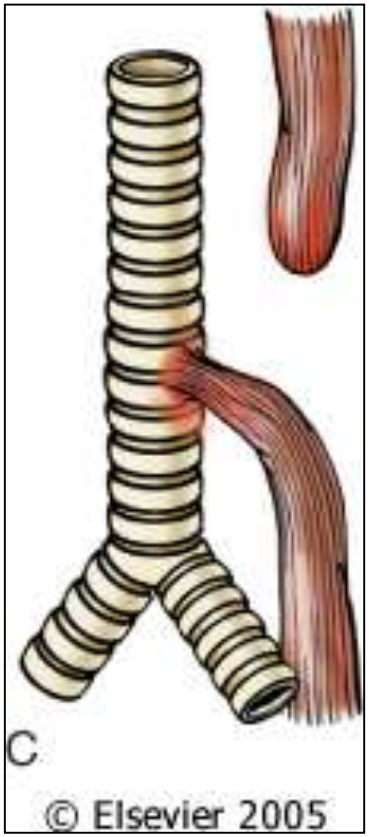


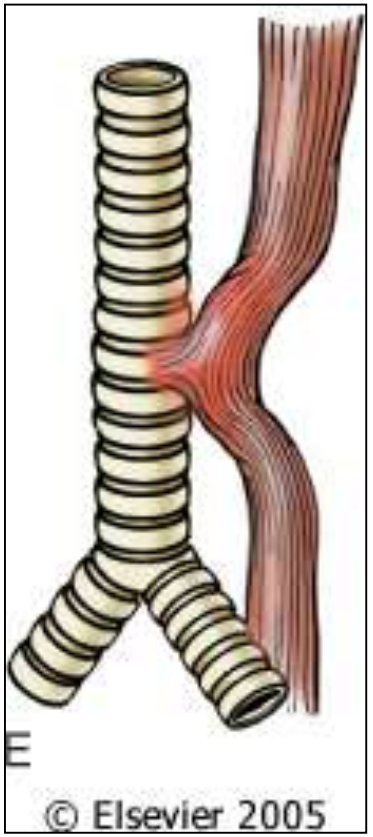
Diaphragmal hernia

Esophageal atresia and tracheoesophageal fistula









Changes of the Lumen

Achalasia: missing opening of the cardiasphincter

Retention of the food, Megaesophagus , "Wineglassform"

Metaplasia, Carcinoma, Perforation

in South-Amerika: Achalasie might be the complication of Chagas diseaes (Myositis - Trypanosoma cruzi).

Stenosis: inherited or caused by Tumors, Struma, Sklerodermia. It might come to Megaesophagus

Diverticles:

Varices:

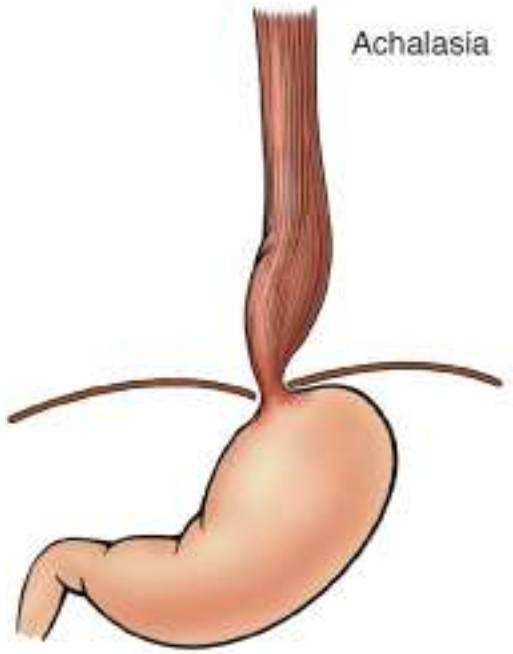
Lower esophagus sphincter

- Normally blocks the regurgitaion of the gastgric juice
- it relaxes by swallowing
- pressure: 15-30 mmHg auf.

Untere osophageale Sphincter
http://www.becomehealthynow.com/article/bodydigestive/727/

Motor dysfunction associated lesions I.

- **Achalasia:** failure to relax
  - →degenerative changes in motor innervation
  - →Chagas disease (Treponema cruzi infection)
  - →polio, surgery, malignancies, amyloidosis
  - →mostly of unknown etiology
  - →aperistalsis
  - →incomplete relaxation of the LES
    - » (lower esophageal sphincter)
  - →increased tone of the LES



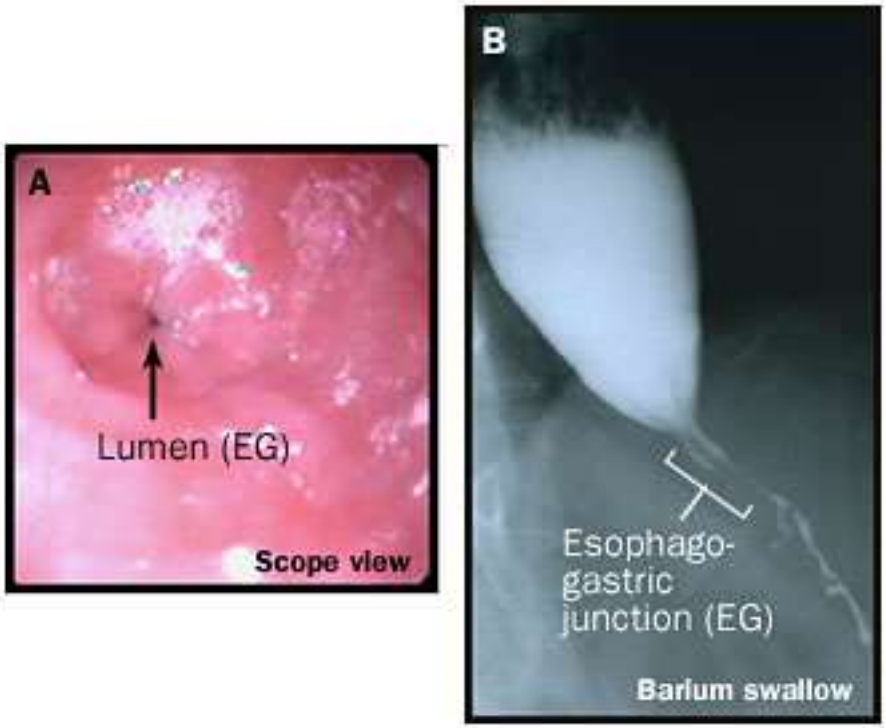
Gross: progressive dilation of the esophagus above LES

Micr: inflammation, ulceration, fibrosis

Sy: dysphagia in young adult or childhood, nocturnal regurgitation

Complications: aspiration, candidiasis

Achalasia



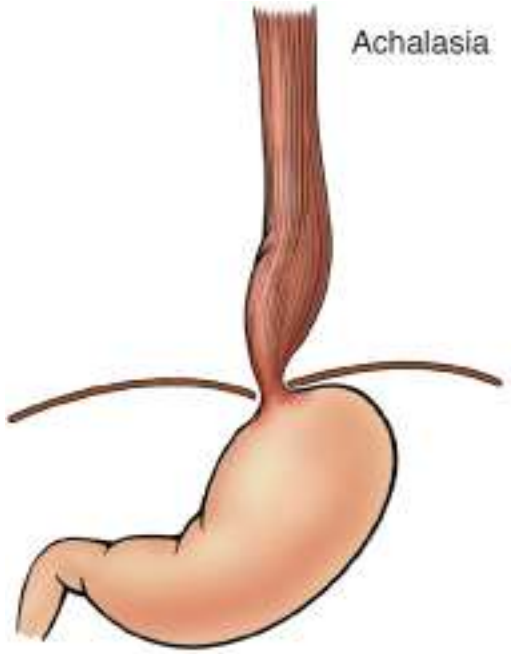


Figure 23. Endoscopic **(A)** and radiographic **(B)** findings in achalasia.

Cricopharyngeal Achalasia

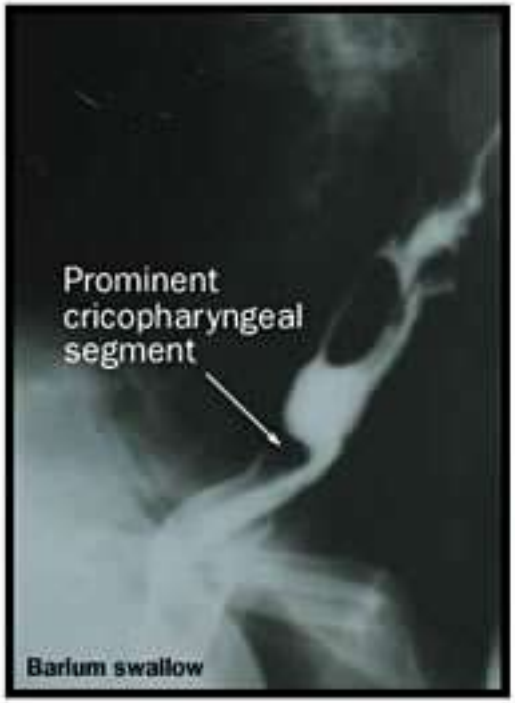


Figure 16. Radiographic image of cricopharyngeal dysfunction.

Striktures / Caustic Ingestion

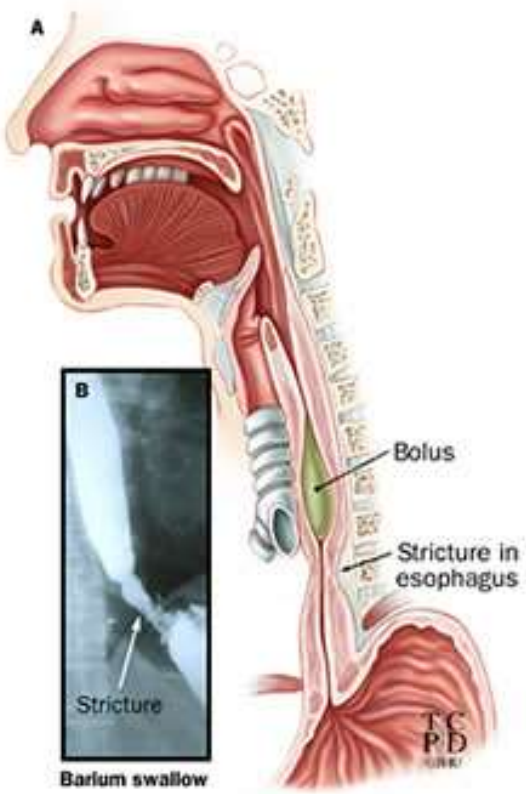


Figure 11. Esophageal stricture showing obstruction of food bolus with corresponding barium swallow.

Diffuse spasm of the esophagus

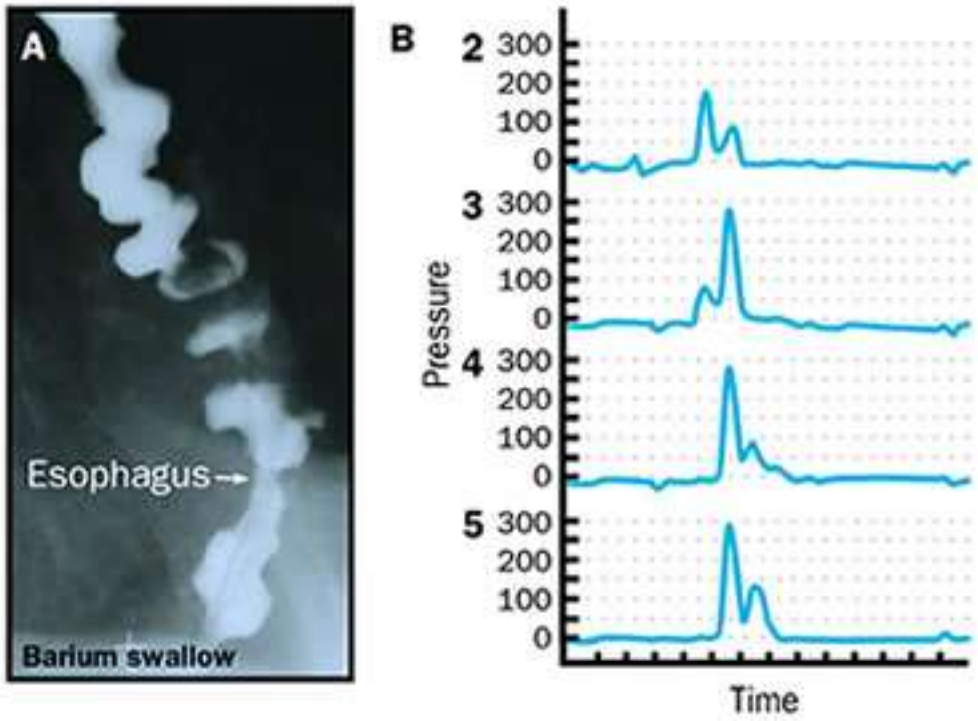


Figure 22. Barium swallow x-ray showing diffuse esophageal stricture **(A)** with corresponding manometric tracing **(B)**.

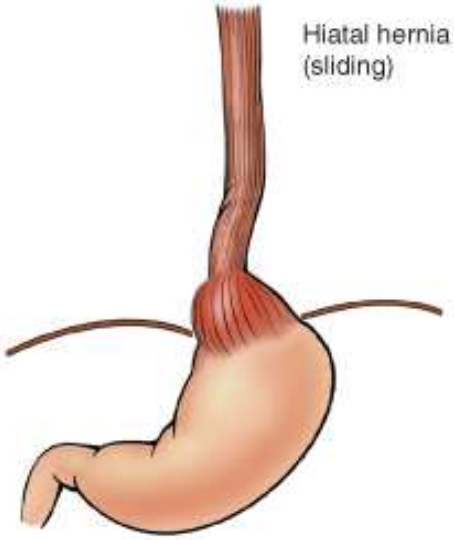
Motoric Dysfunction associated lesions

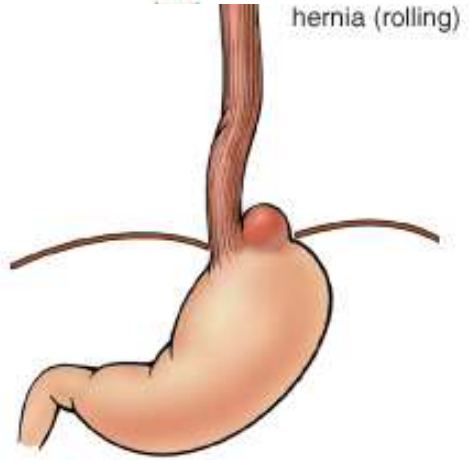
- **Hiatus Hernia**
  - **Axial or sliding** herniation: 95% of cases
    - Protrusion of the stomach across diaphragm
  - **Paraesophageal or „rolling”** herniation: part of the stomach along major curvature enters thorax.

SYMPTOMS: heartburn, Regurgitation

Complications: associated reflux esophagitis, Ulceration, bleeding,

Perforation: Mediastinitis





ÖSOPHAGUS

Hiatus herniation: content of the abdominal cavitiy is positioned in the thorax

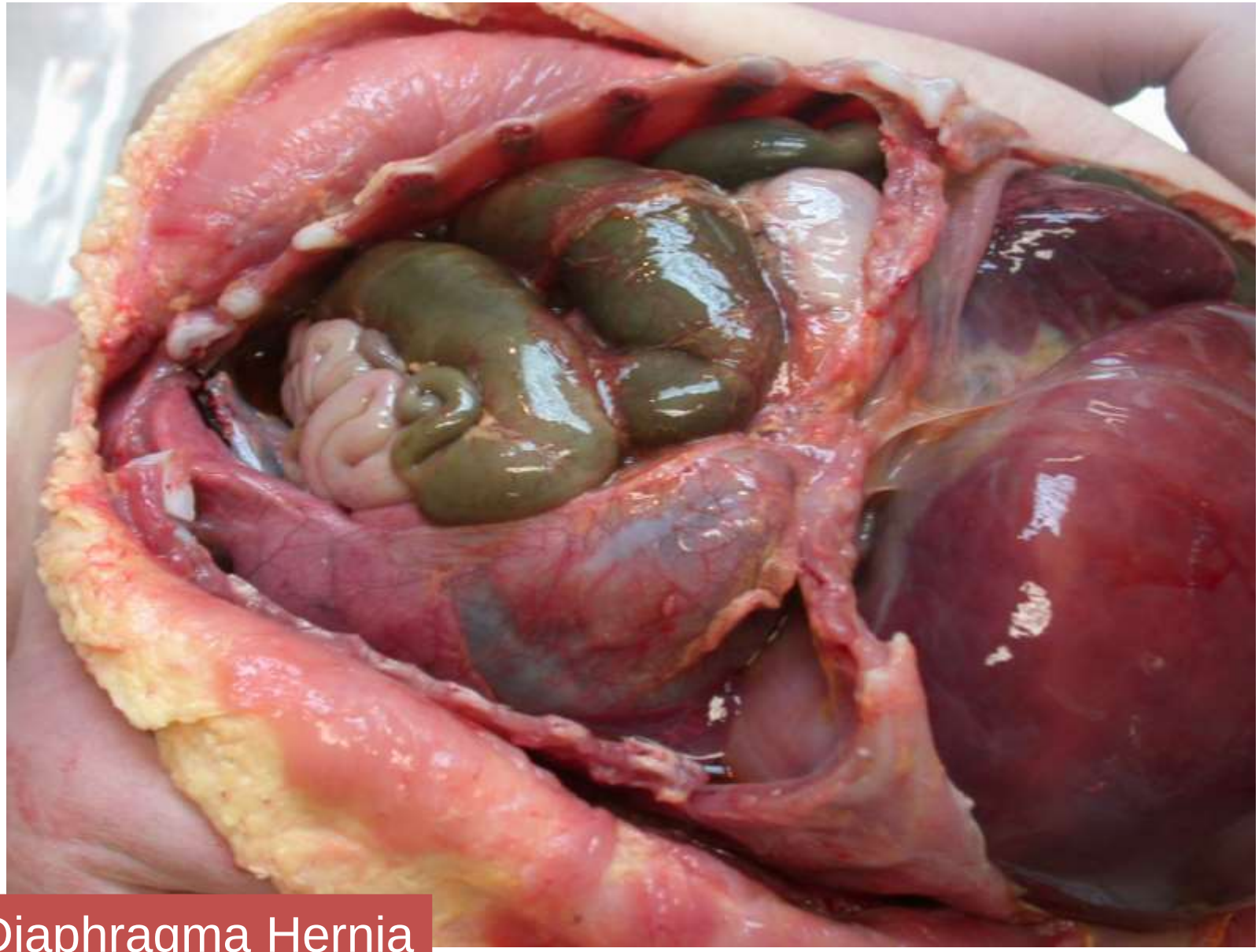
Diaphragma Hernia

sliding herniation: Cardia and Fundus, mostly asymptomatic

Paraesophageale Herniation: the position of the Cardia is normal, Regurgitation, Heartburns

Varices:

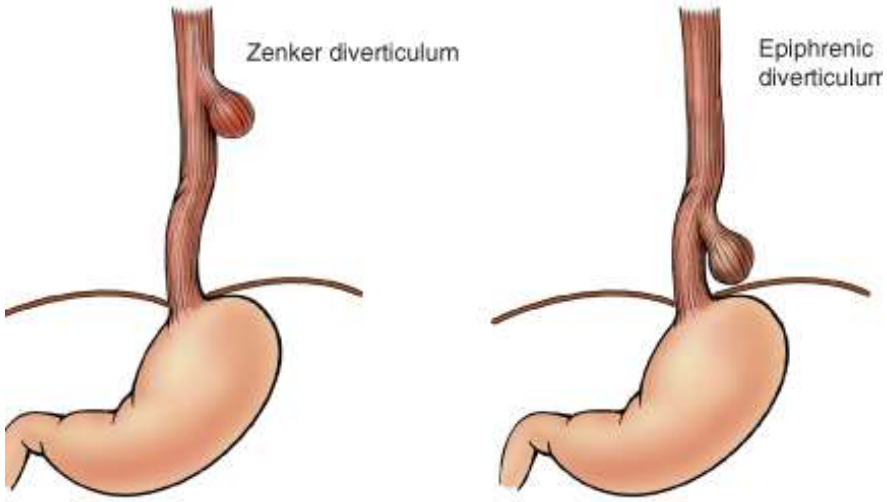
- inherited: weakness of the wall
- acquired: by portal Hypertension, mostly in the lower third of th esophagus



Diaphragma Hernia

Motor dysfunction associated lesions III. (congenital disorder)

- **Diverticula:** dilatation
  - true- contains all layers of the esophagus
    - Zenker diverticulum (pulsion-craniopharingeal),
    - Traction
    - Epiphrenic



  - false - mucosa/submucosa (no muscular layer)
  - Complications: diverticulitis, perforation, ulceration, mediastinitis, fistula

Motoric Dysfunction associated lesions

Diverticles: inherited or acquired outpouching

real (all layers of tghe wall)or Pseudodiverticle (mucosa only). Dysphagia, trigger for caughing, Regurgitation

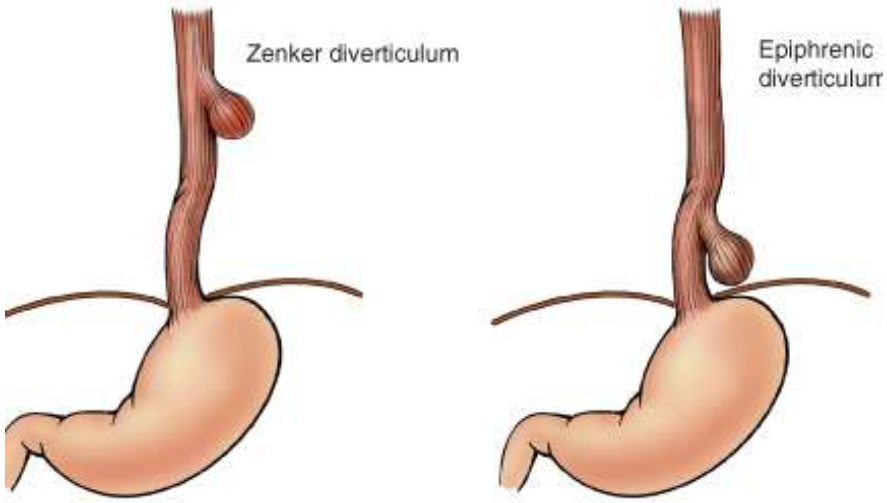
Pulsionsdiverticle: inherited or acquired weakness of the wall by increased intraluminar pressure

Zenker Diverticle: 70 % of all Esophagusdiverticles - Pseudodiverticle, in upper third

Epiphrenic Diverticle: 10 % , Pseudodiverticle
Tractionsdiverticle: 20 %, mostly asymptomatic, traction of parabronchial sacrs in the heights of bifurcation

Motoric Dysfunction associated lesions

- **Diverticle:** Dilation
  - **real**- **(all layers of the wall)** Zenker Divertikulum (Pulsion-craniopharingeal),
    - Traction
    - Epiphrenic



  - **false** - Mucosa/submucosa only: **Pseudodiverticle (the mucosa only, no muscle layer)**
  - Complications: Diverticulitis, Perforation, Ulceration, Mediastinitis, Fistule

Zenker's Diverticle

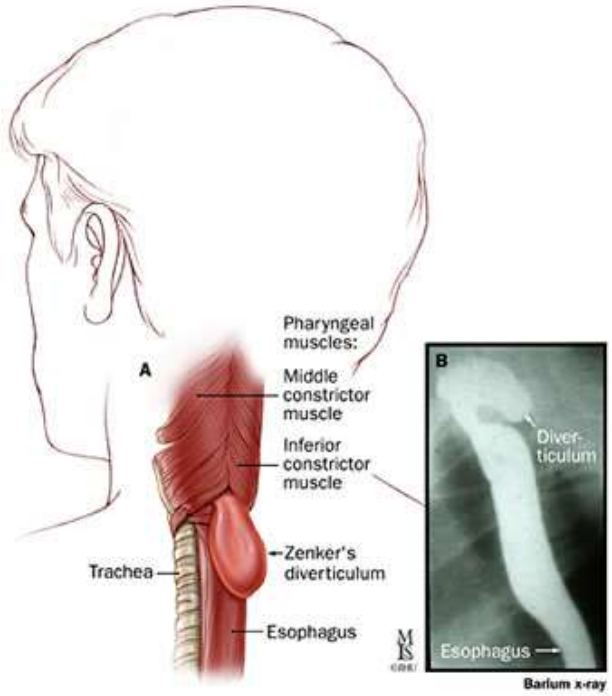
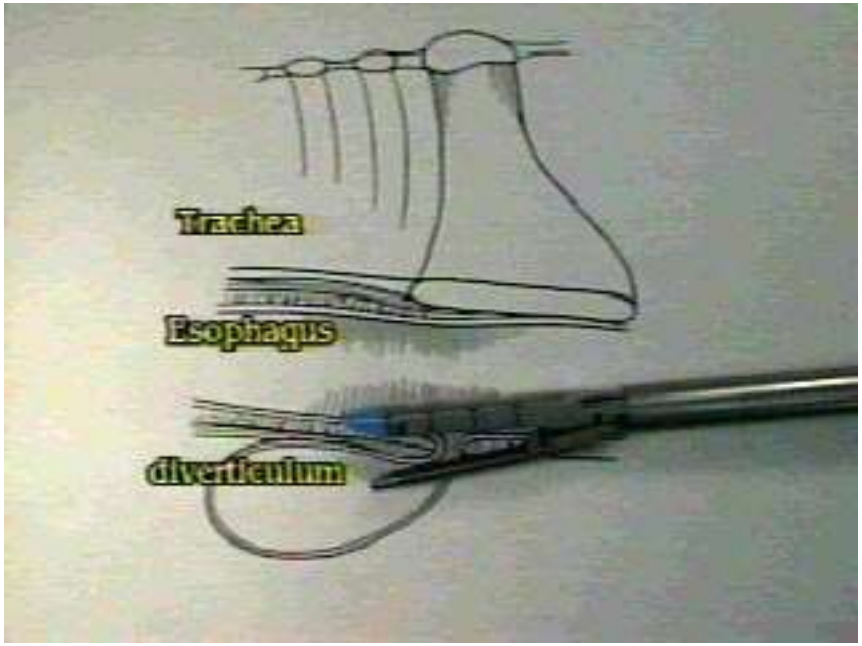
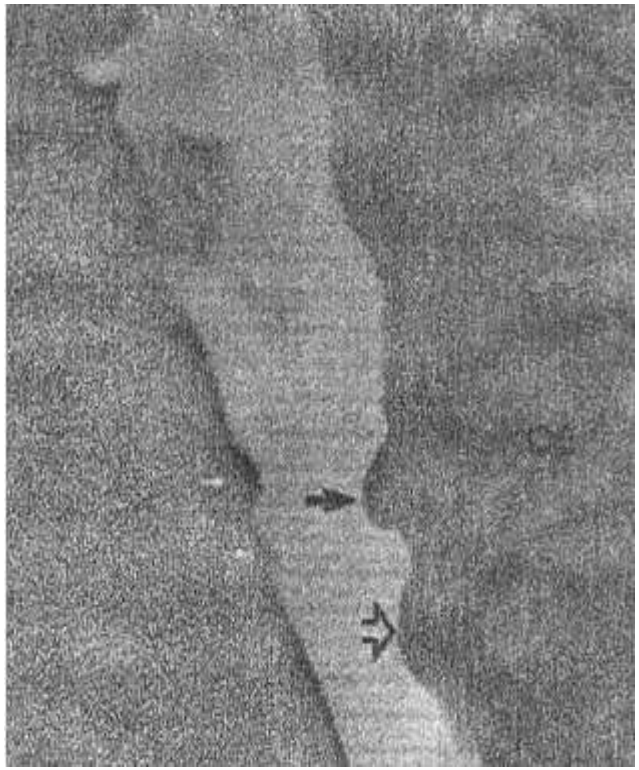


Figure 17. Zenker's diverticulum **(A)** with corresponding barium x-ray **(B)**.

Zenker's Diverticle



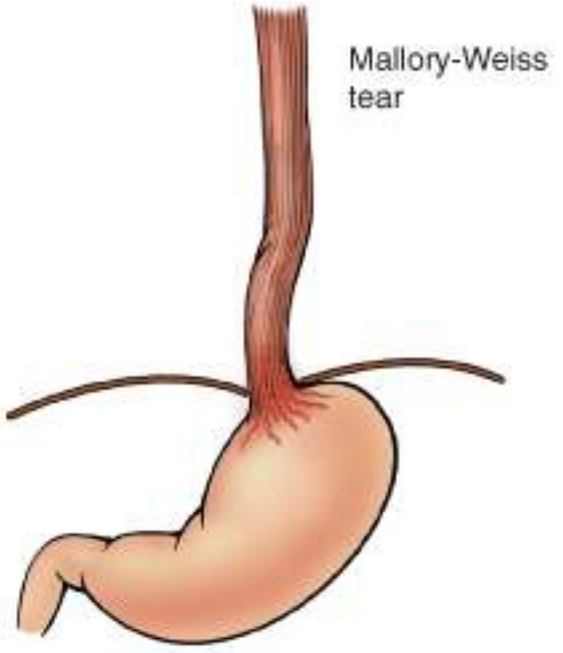
Cervical rib disease



Motor dysfunction associated lesions IV.
(Lacerations, perforations)

Mallory-Weiss syndrome

- **longitudinal lacerations and tears** at the **esophagogastric junction**
- Cause: alcoholism, chronic vomiting, hiatal hernia, reflux
- Symptoms: hemorrhage, hematemesis, melaena

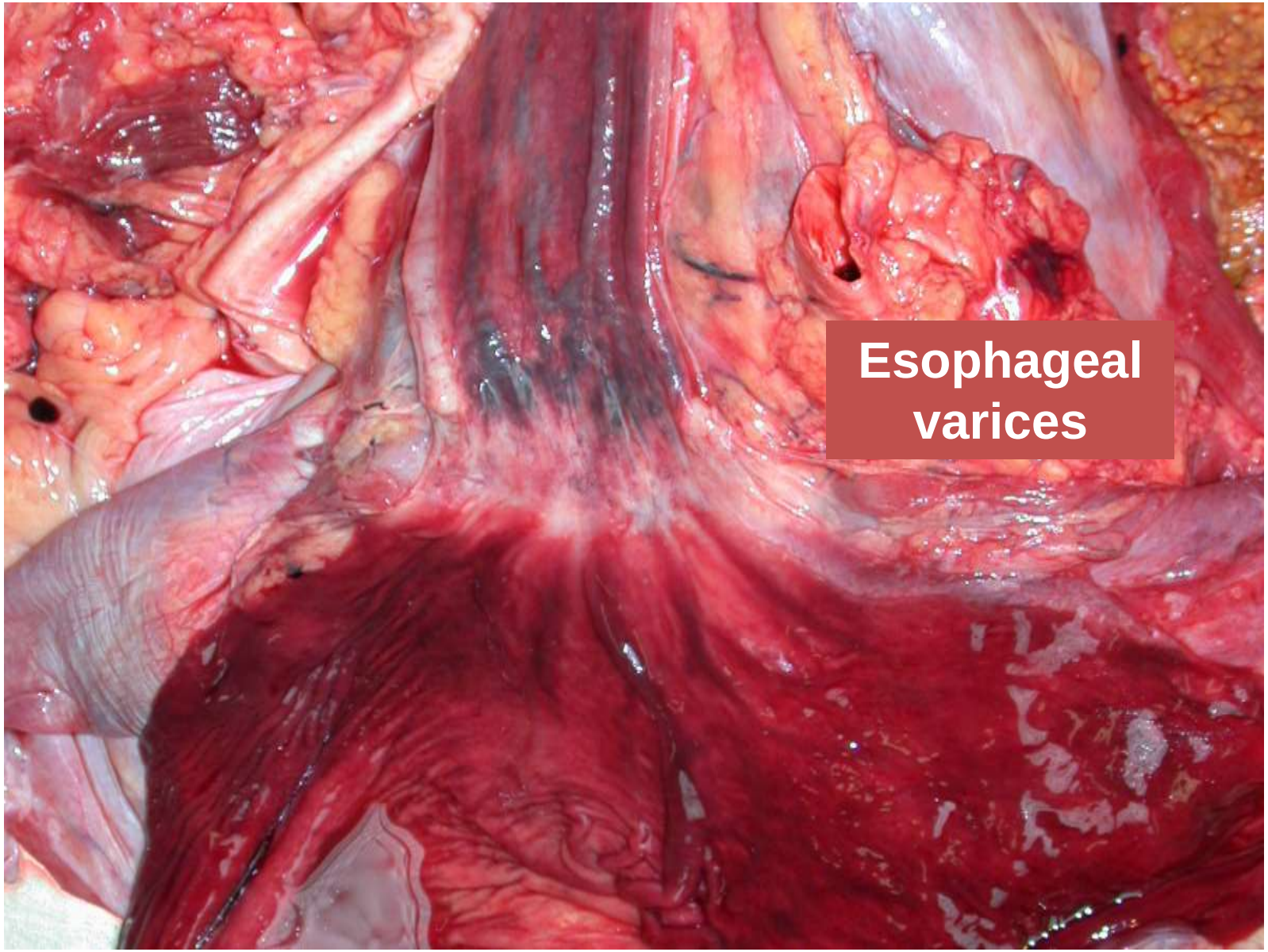


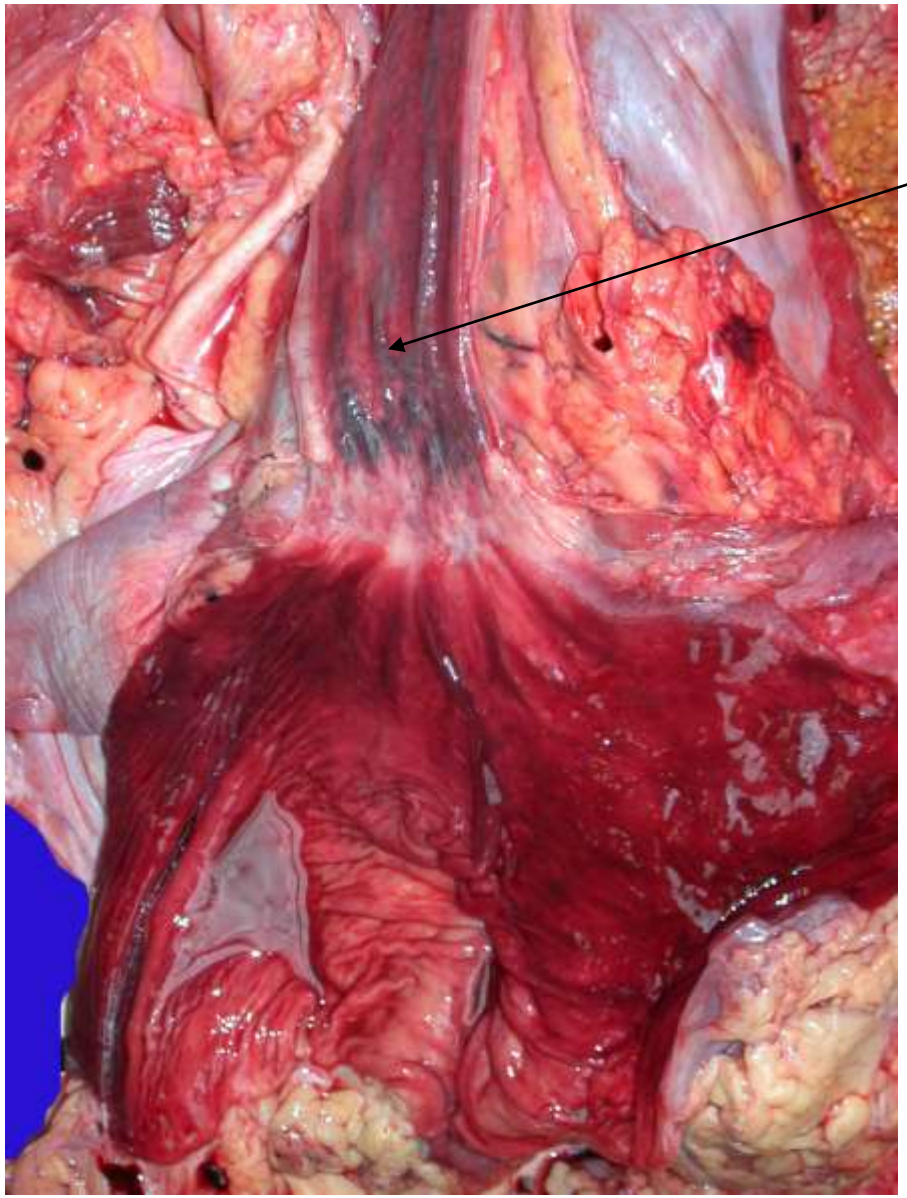
Boerhaave syndrome

- Esophageal **rupture** due to laceration and vomiting

Esophageal varicosity

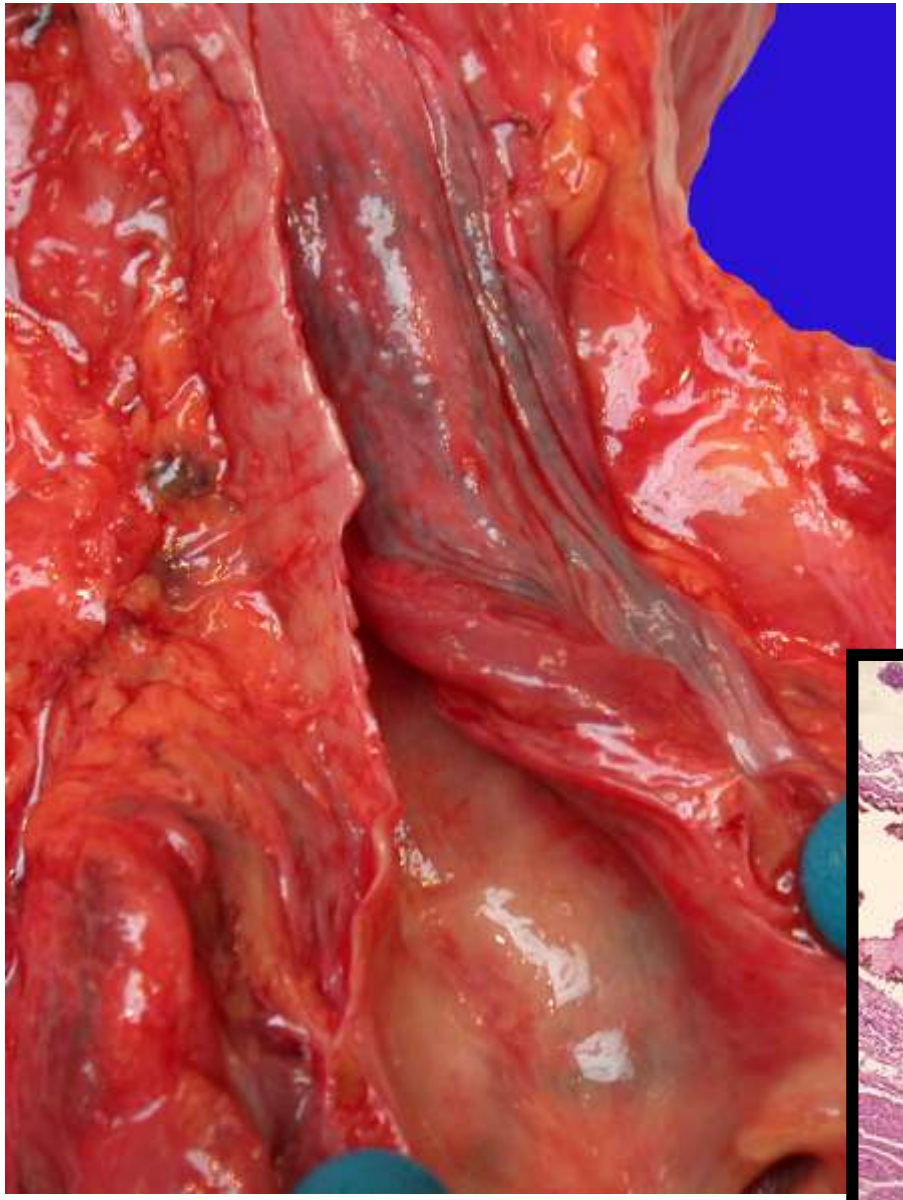
- in portal hypertension
- cirrhosis (alcoholism)
- ~ rupture⟶massive hematemesis
- therapy:
  - ⟶balloon tamponade and sclerotherapy

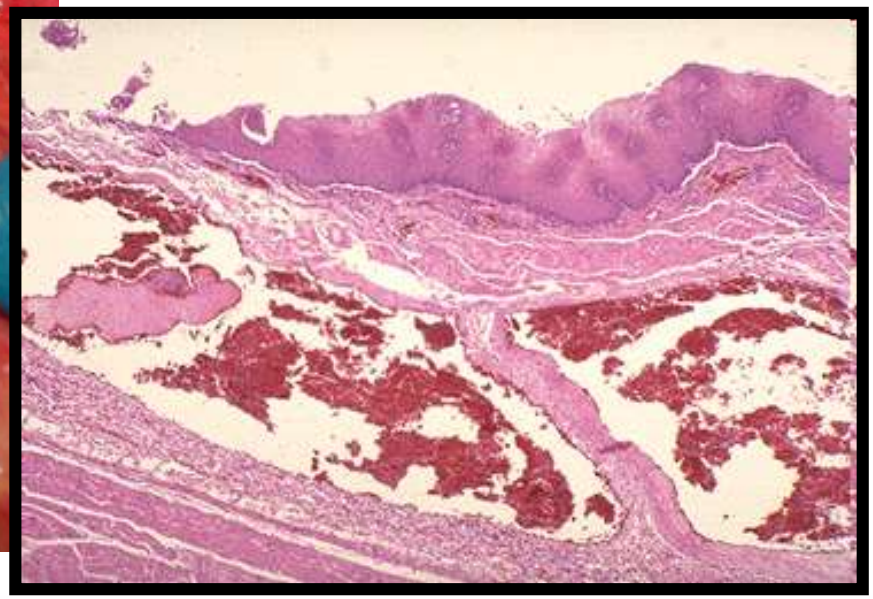

Esophageal varices



varices

bleeding





GERD

- Symptoms
  - Heartburn
  - Regurgitation
  - Nausea
- Reflux
  - LES is weak
  - Missing closing ability – it remains open

LES mit GERD

(Jackson Gastroenterology, 2004)

Esophagitis

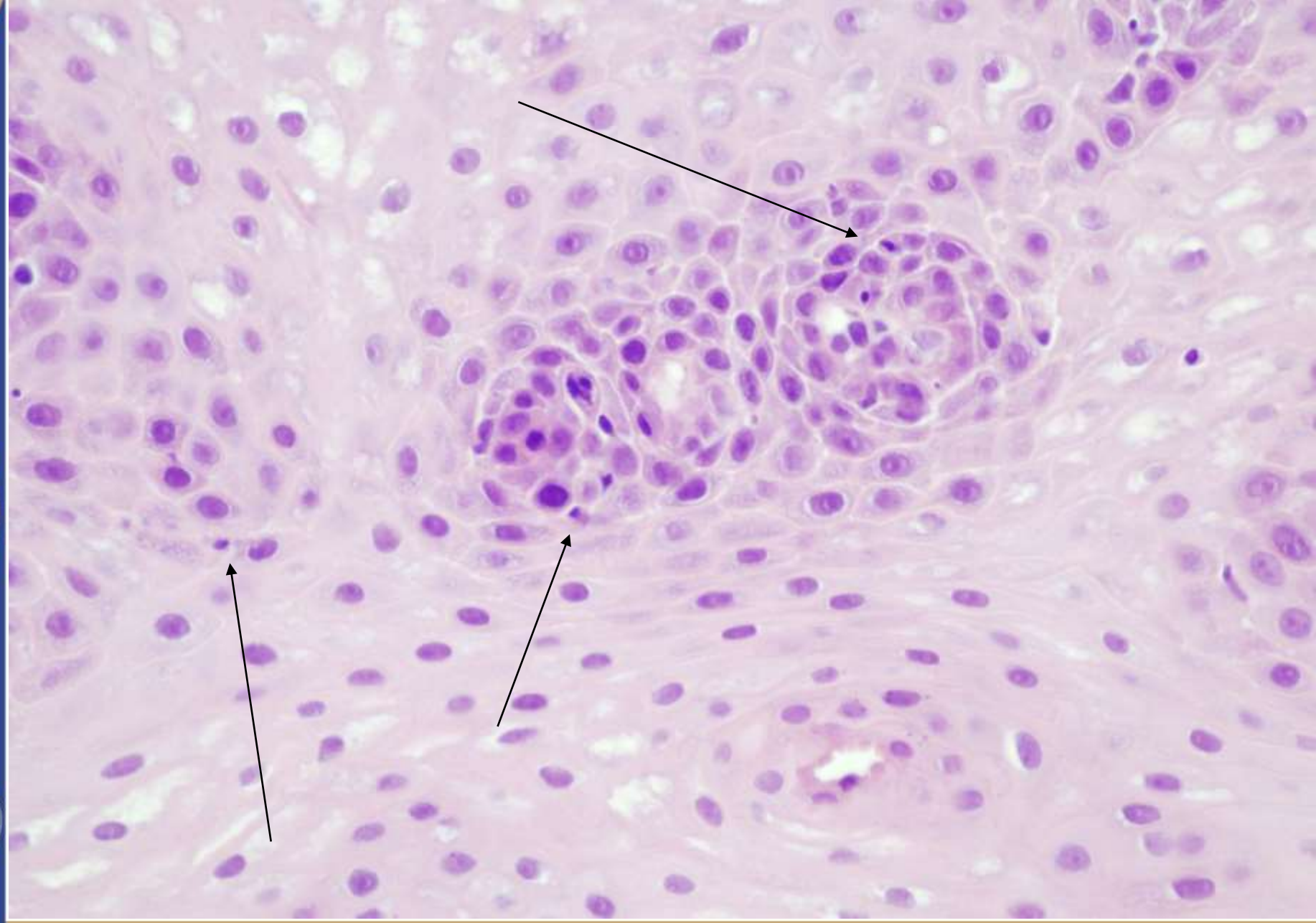
- **Reflux esophagitis** (Gastroesophageal reflux disease-GERD)
  - →due to a sliding hernia
  - →delayed gastric emptying

SY: dysphagia, heartburn, severe chest pain (mimicking AMI)

Gross: hyperemia, ulcerations, stricture (depending on severity)

Micr: inflammatory cells within squamous epithelium, elongation of lamina propria connective tissue papillae with dilated vessels showing marked congestion

Reflux esophagitis



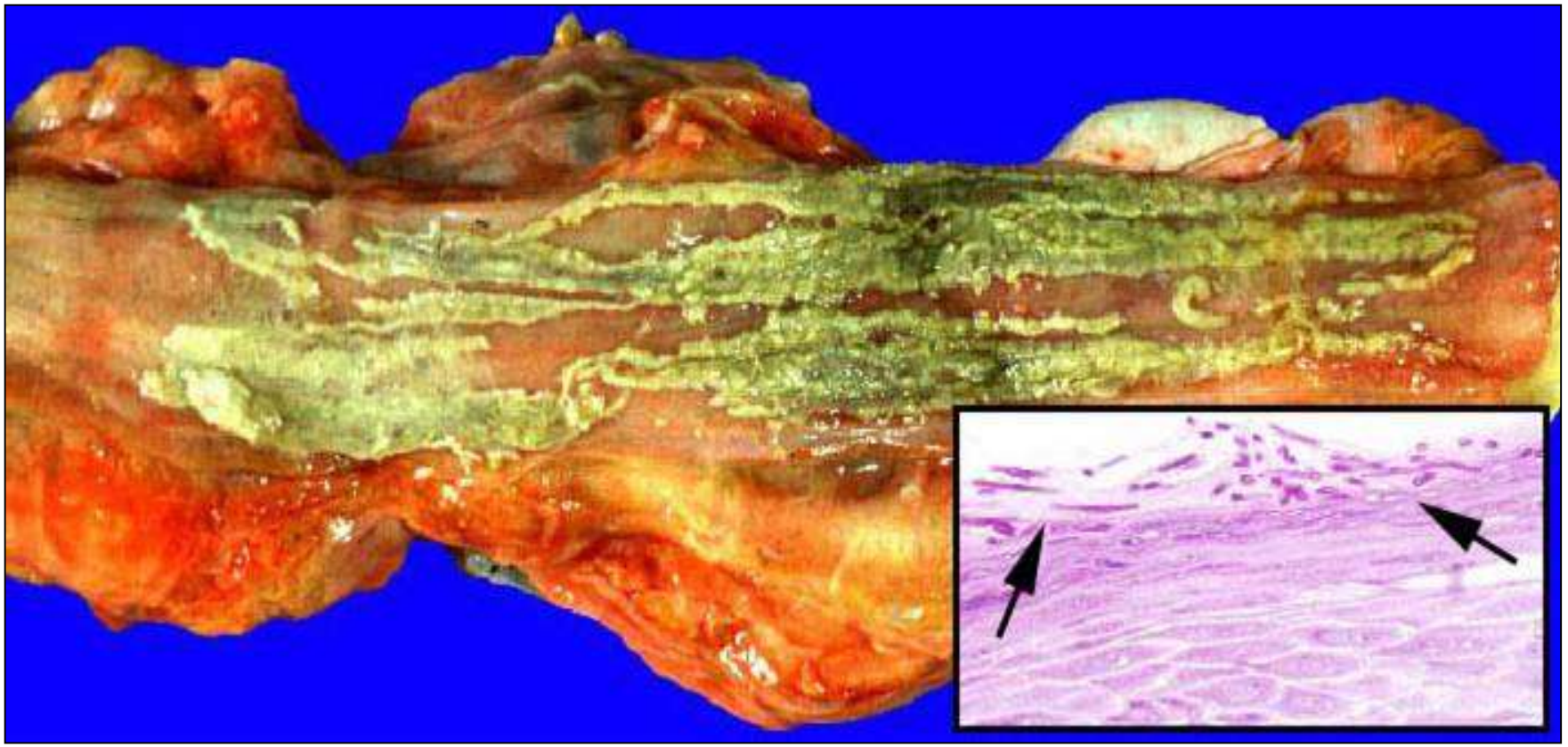
Chemical and physical injury induced esophagitis

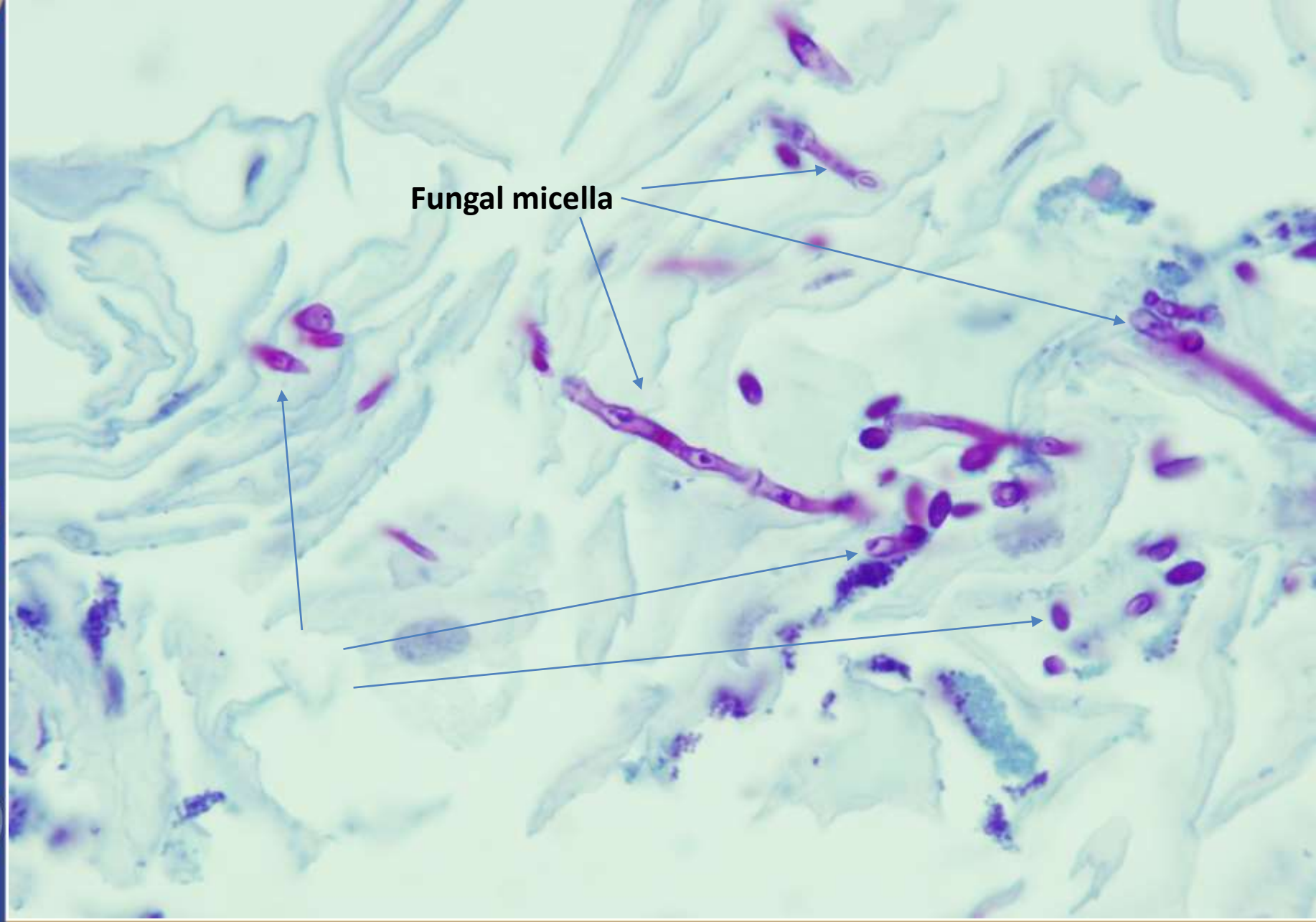
- Causes
  - alcohol
  - acids, alkalia (suicide)
  - cytotoxic therapy
  - irradiation

Complications: ulceration, mucosal necrosis, necrosis of the wall, stricture, fibrosis

Esophagitis of infectious origin

- Candidiasis: in antibiotic therapy, immunosuppression
- Herpes simplex and CMV: ulcerations with intranuclear inclusions




Fungal micella

Barrett esophagus

→with longstanding reflux

→salmon coloured mucosa above the gastroesophageal junction

→presence of metaplastic columnar epithelium (goblet cells)-diagnostic criterium (?)

→long segment Barrett esophagus (≥ 3 cm) or short segment Barrett esophagus (<3 cm)

→~⟶dysplastic epithelium⟶⟶**adenocarcinoma**

SURGERY

Vol. 41 **JUNE, 1956** **No. 6**

THE LOWER ESOPHAGUS LINED BY COLUMNAR EPITHELIUM

N. R. BARRETT, LONDON, ENGLAND

...esophagus lined with **columnar epithelium** (rather than the usual squamous epithelium) due to a congenitally shortened esophagus leading to a tubular portion of stomach being trapped in the chest.

Barrett NR – Br J Surg 1950; Barrett NR – Surgery 1956

3. Definition of Barrett's esophagus (BE)

In Japan (Japan Esophageal Society) and the UK (British Society of Gastroenterology), BE is defined simply as metaplastic CLE that is recognizable macroscopically.

i.e.; Barrett's esophagus = CLE

In Germany and the USA, however, BE is defined as the metaplastic replacement of any length of the esophageal epithelium that can be recognized at endoscopy and that is confirmed by biopsy of the tubular esophagus to show intestinal metaplasia, excluding intestinal metaplasia of the gastric cardia.

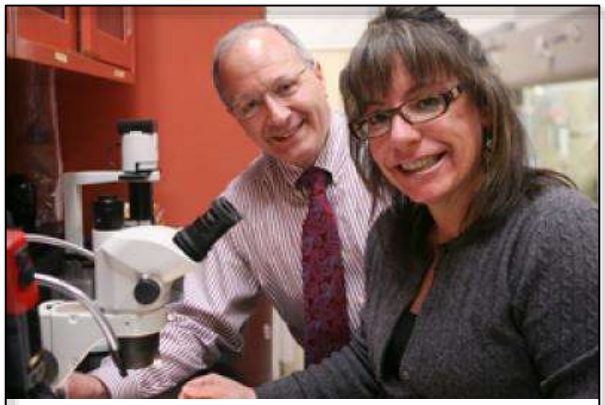
i.e.; Barrett's esophagus = CLE + goblet cells

Playford RJ: New British Society of Gastroenterology guidelines for the diagnosis and management of Barrett's oesophagus. Gut 2006;55:442

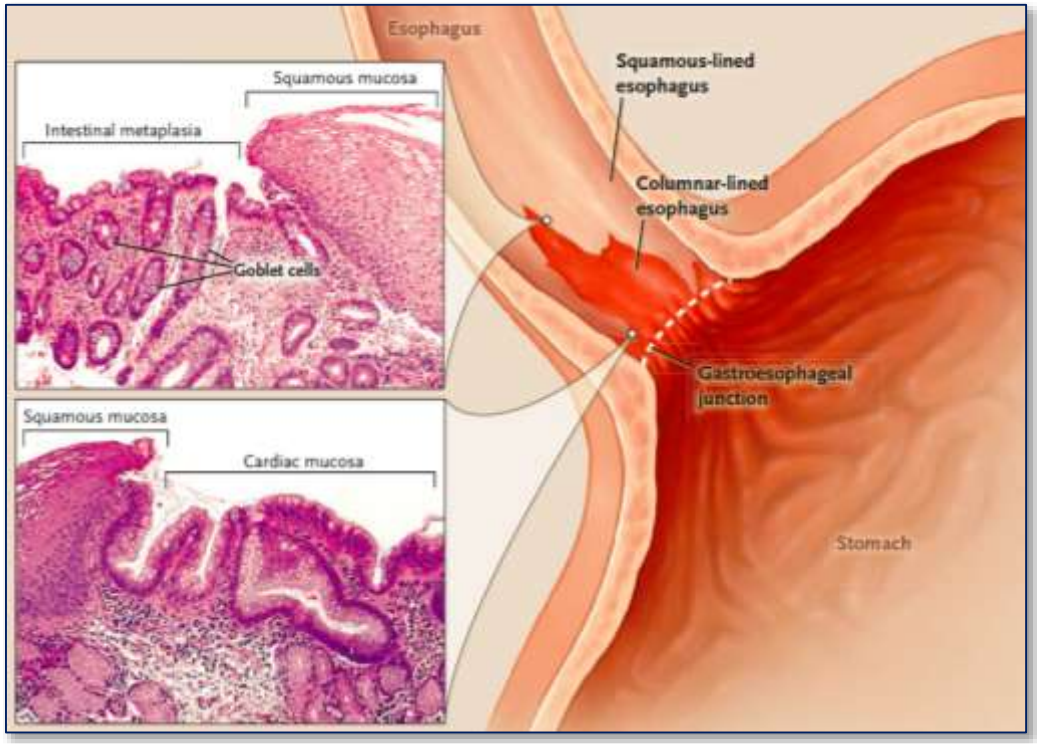
The Practice Parameter Committee of the American Colleges of Gastroenterology: Sampliner RE, et al. Am J Gastroenterol 2002;97:1998

Official Journal of the Japan Esophageal Society

BE diagnosis



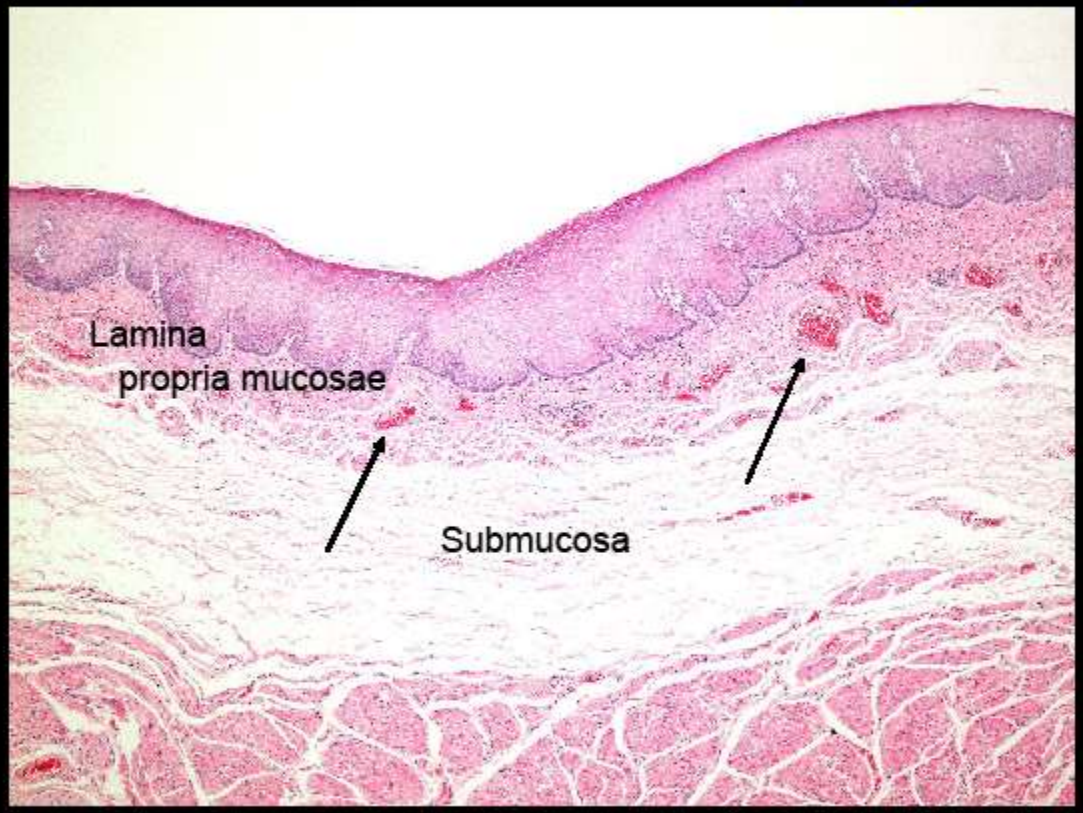
Spechler SJ & Souza RF
NEJM 2014



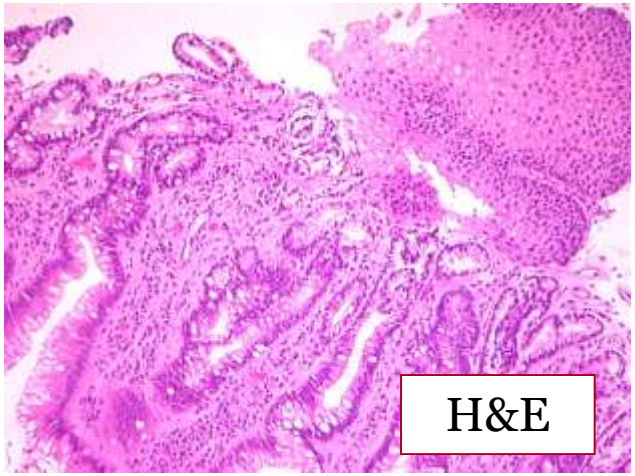
The ingredients for a correct diagnosis of BE

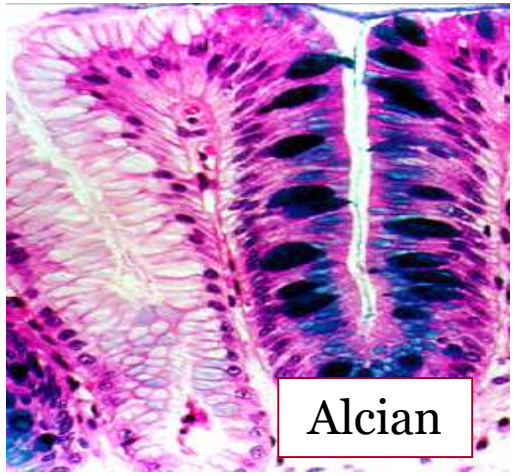
1. **endoscopic** evidence that columnar mucosa extends above the gastroesophageal junction and lines the distal esophagus
2. **biopsy** confirming the presence of columnar intestinal metaplasia

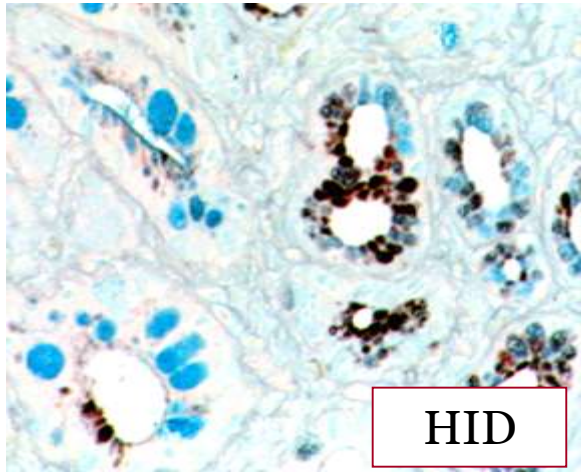
Transverse section of the lower esophagus



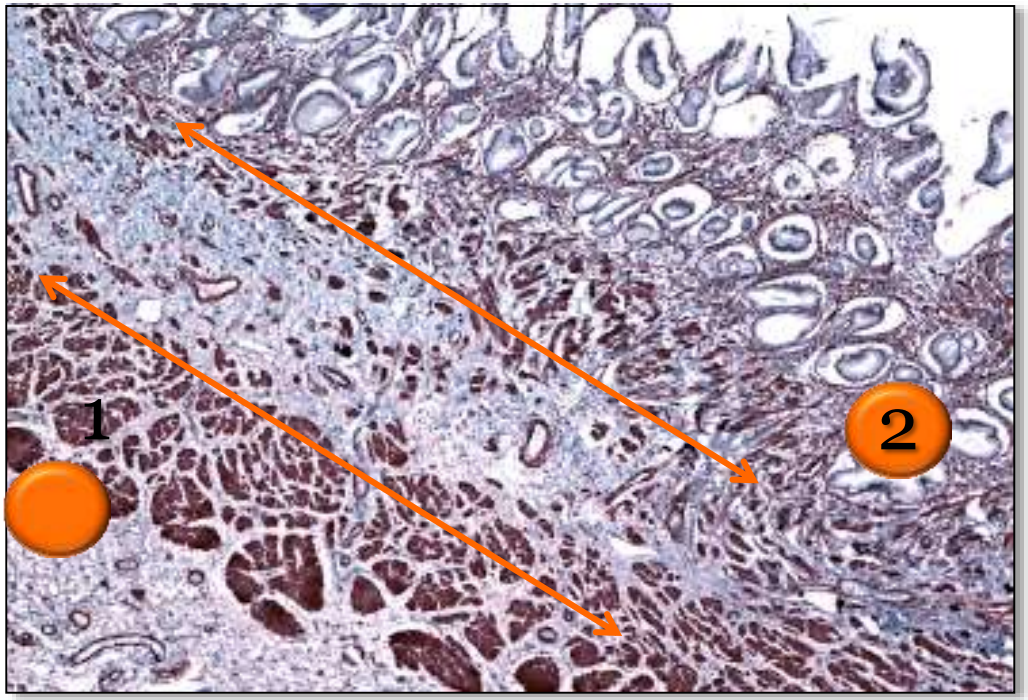
Large vessels (100-200 µm in this tissue section, arrows) are evident more frequently in the lamina propria mucosae than in the submucosa in the esophageal lower sphincter.

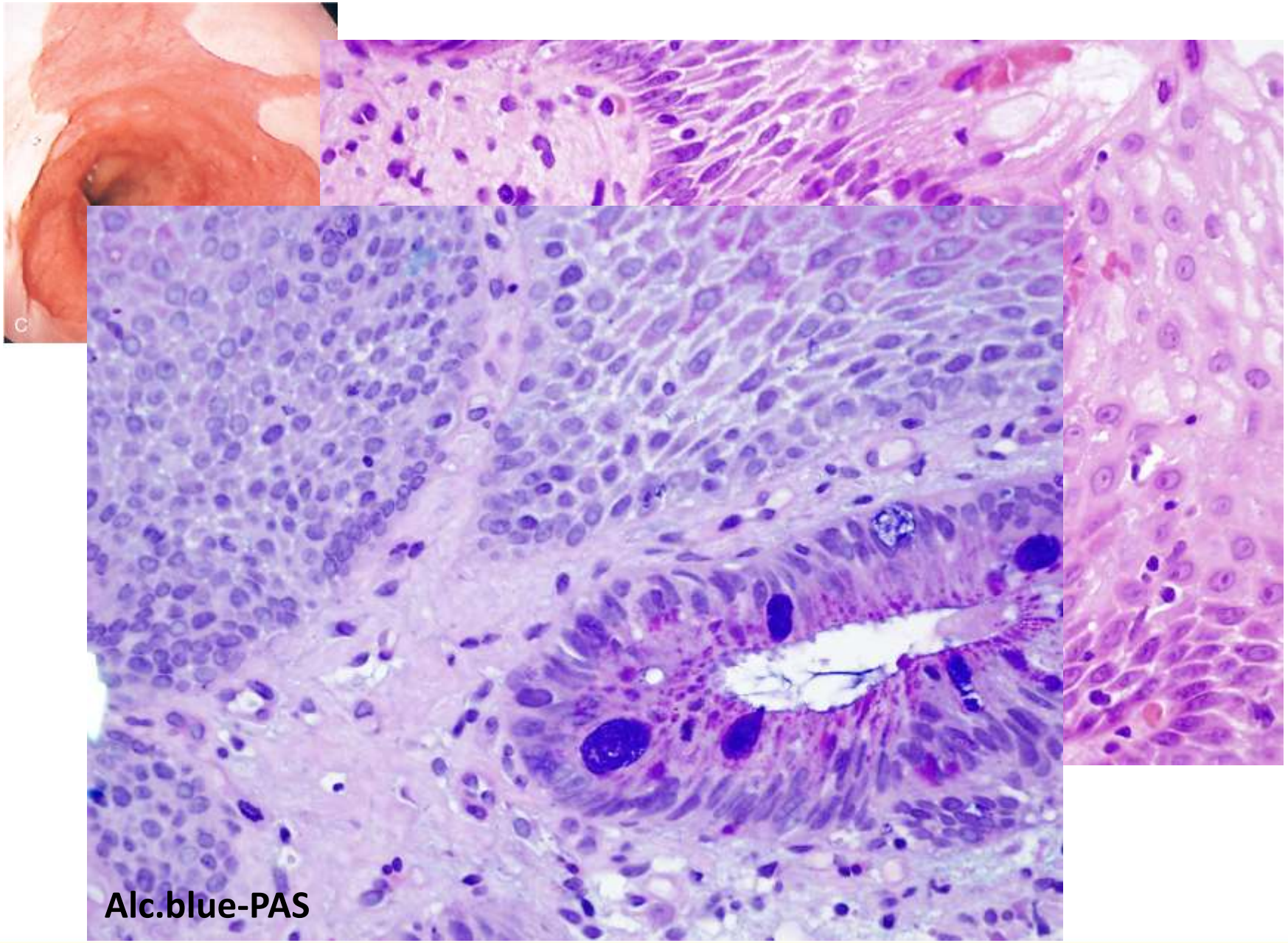


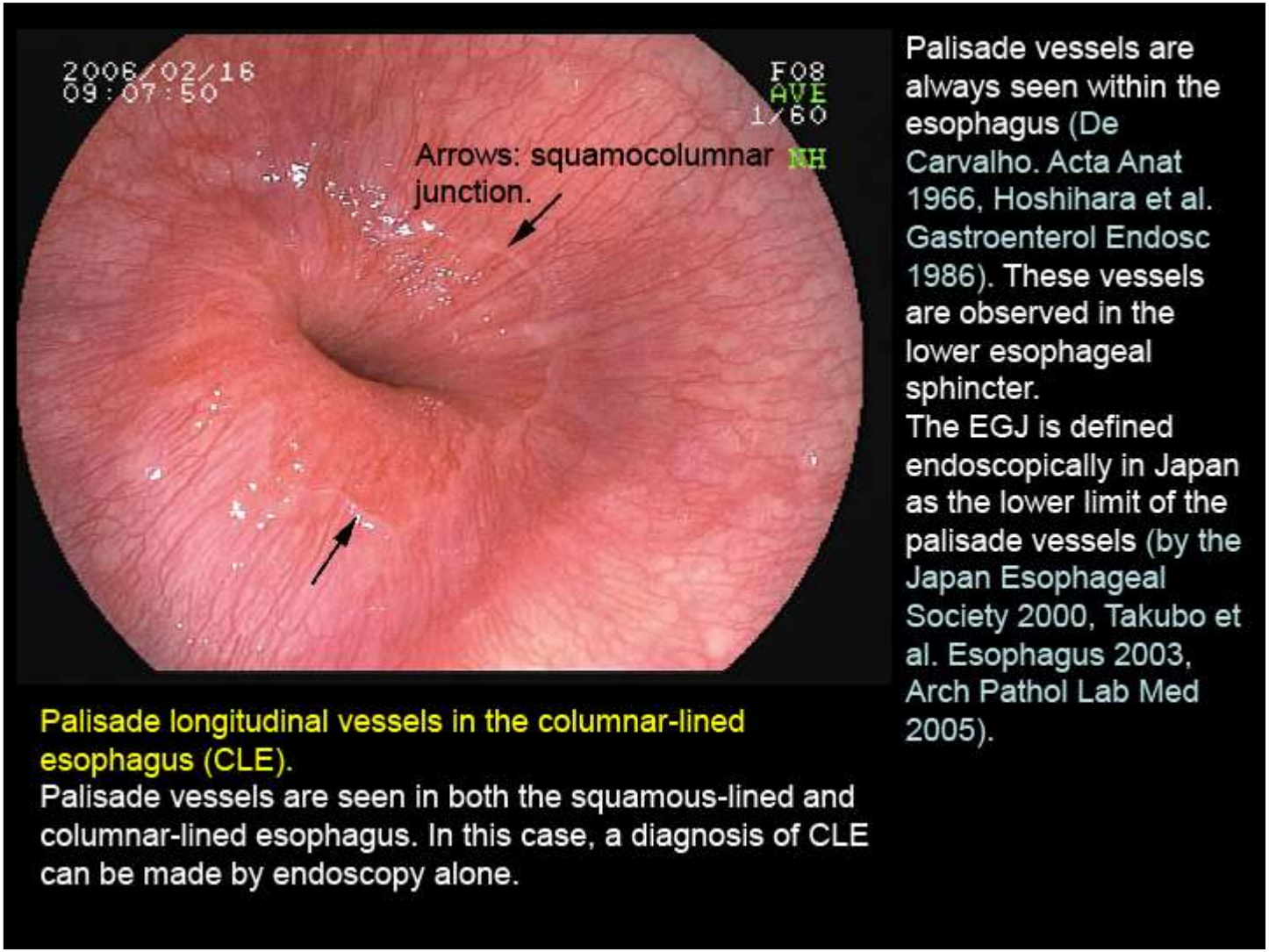




Velvet Mucosa On Endoscopy Becomes Barrett's Epithelium When Histology Features Columnar Metaplasia




C
Alc.blue-PAS

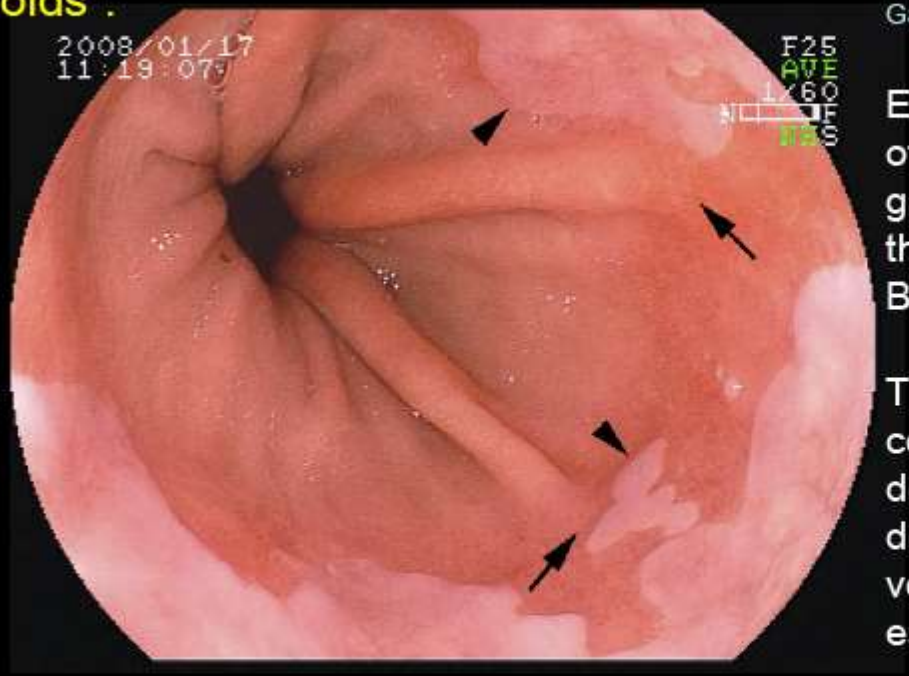


Palisade longitudinal vessels in the columnar-lined esophagus (CLE).
Palisade vessels are seen in both the squamous-lined and columnar-lined esophagus. In this case, a diagnosis of CLE can be made by endoscopy alone.

Palisade vessels are always seen within the esophagus (De Carvalho. Acta Anat 1966, Hoshihara et al. Gastroenterol Endosc 1986). These vessels are observed in the lower esophageal sphincter.
The EGJ is defined endoscopically in Japan as the lower limit of the palisade vessels (by the Japan Esophageal Society 2000, Takubo et al. Esophagus 2003, Arch Pathol Lab Med 2005).

Takubo et al 2009

In Western countries, the definition of the EGJ is the "upper limit of gastric folds".



When a small volume of air is present in the esophagus, the upper end of the mucosal folds (arrows) extends up to or beyond the level of the lower end (arrowheads) of the squamous epithelium.

Sharma P, Dent J, Armstrong D, Berman JJ, Gossner L, Hoshihara Y, Jankowski JA, Janghard O, Lundell T, Tytgat GN, Vieth M. Gastroenterology 2006;131: 1392-9

Endoscopic appearance of the upper end of the gastric mucosal folds at the EGJ in a patient with Barrett's esophagus.

The upper limit shows considerable up and down movement, depending on the volume of air in the esophagus.

Native epithelium

↓

Metaplastic changes

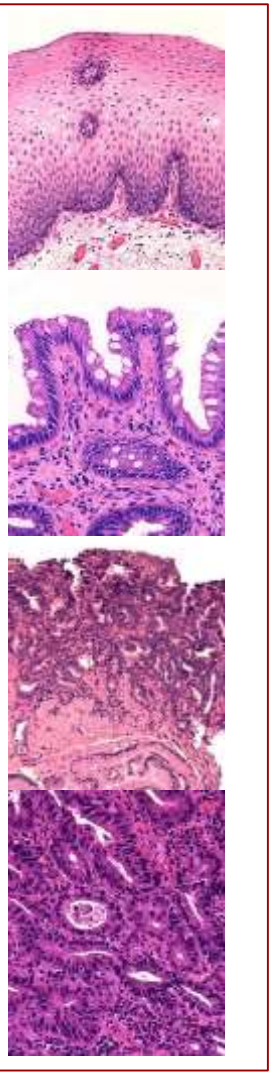
↓

Dysplastic lesions

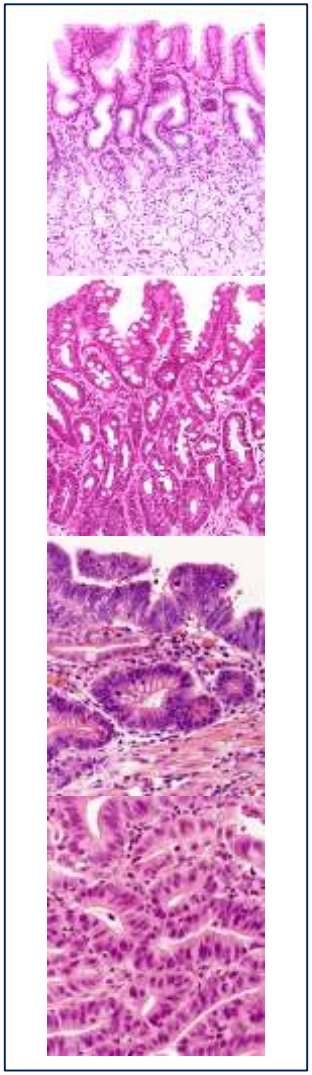
↓

Adenocarcinoma

Multi-step cascades

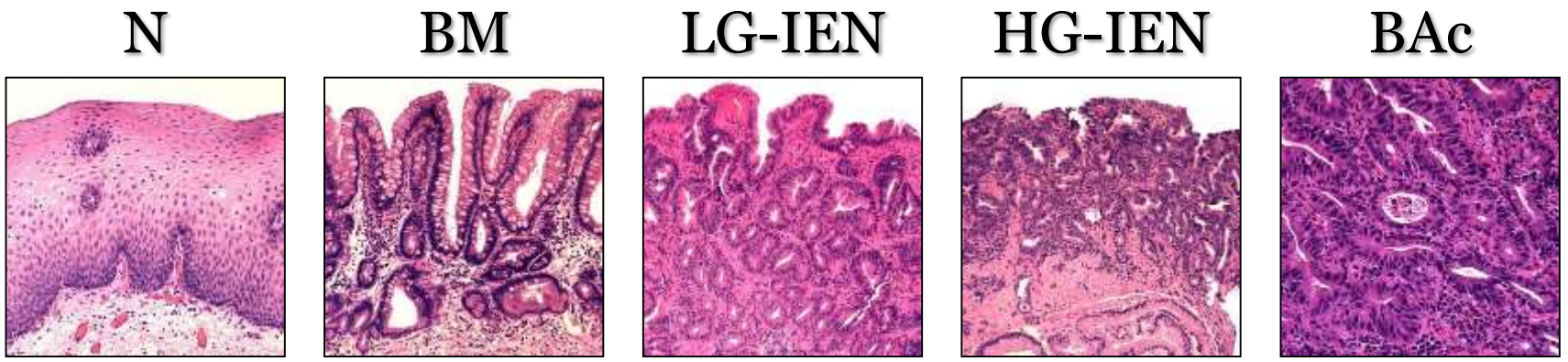


OESOPHAGUS
BARRETT



STOMACH
CORREA

BARRETT'S CASCADE



Gut

2015

Length of Barrett's esophagus and cancer risk: Implications from a population based study

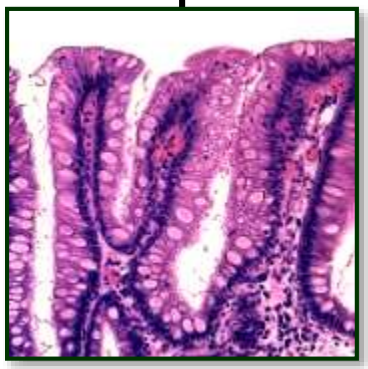
H Pohl, O Pech, H Arash, M Stolte, H Manner, A May, K Kraywinkel, A Sonnenberg, C Ell

AMONG PATIENTS WITH NEWLY DIAGNOSED **T1 BARRETT-CANCER**

%	BM Length at recruitment	Annual Transition to Barrett-Cancer	Patients who would need to undergo EGDS to find one BC/year
20%	ULTRA-SHORT	0.01 %	12,365
24%	SHORT	0.03 %	3,440
56%	LONG	0.22 %	450

Endoscopic surveillance

New diagnosis of Barrett's esophagus

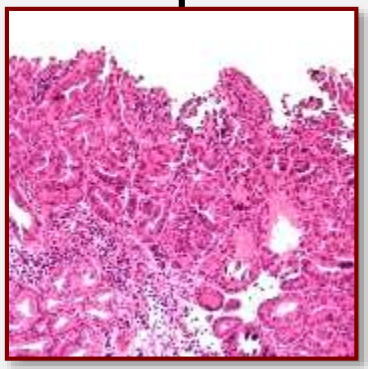


No dysplasia

Endoscopy every 3-5 yr

LGD

Endoscopy every 6-12 mo or eradication therapy



HGD

Endoscopic eradication therapy

...the clinical impact

Spechler SJ & Souza RF - NEJM 2014; De Jonge PJF, et al – Gut 2014

Gut

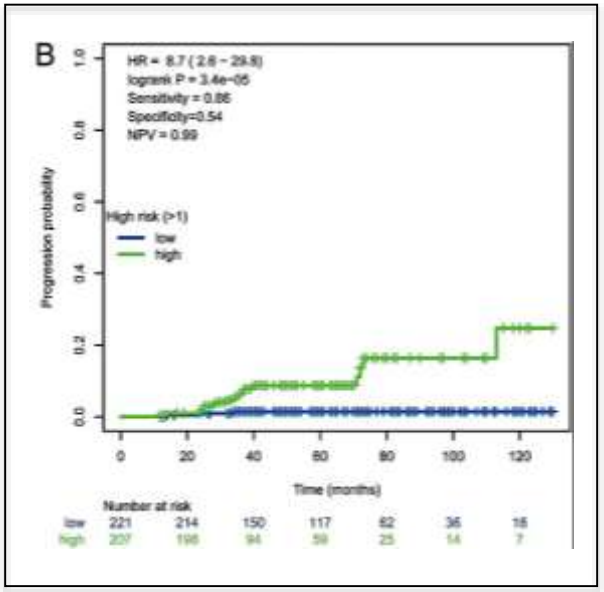
2015

Derivation of genetic biomarkers for cancer risk stratification in Barrett's oesophagus: a prospective cohort study

Margriet R Timmer, *et al*

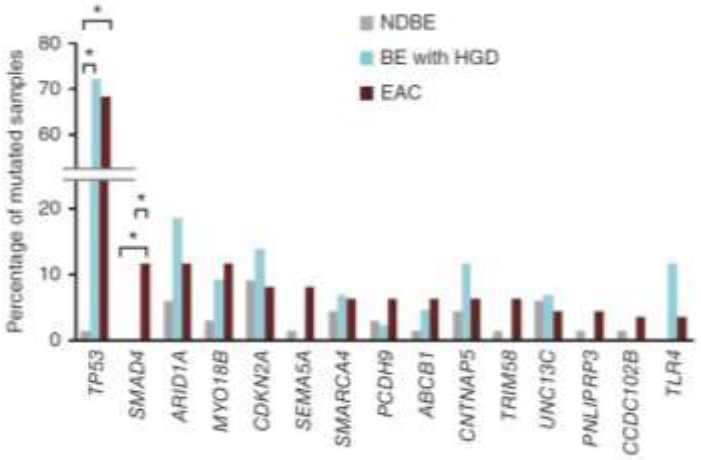
P16 loss, *MYC* gain, and aneuploidy (centromeric probes for CEP7/CEP17, as surrogate marker for DNA ploidy change) measured by FISH (brush cytology) is an independent predictor of progression in non-dysplastic BE.

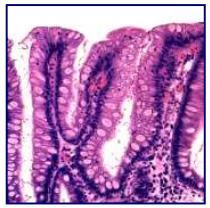
A prediction model including this Abnormal Marker Count is advantageous over a clinical model using only age and BE length for long-term risk stratification.



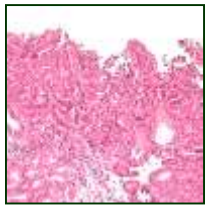
nature genetics

Ordering of mutations in preinvasive disease stages of esophageal carcinogenesis.

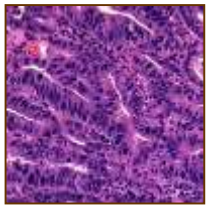




n= 66

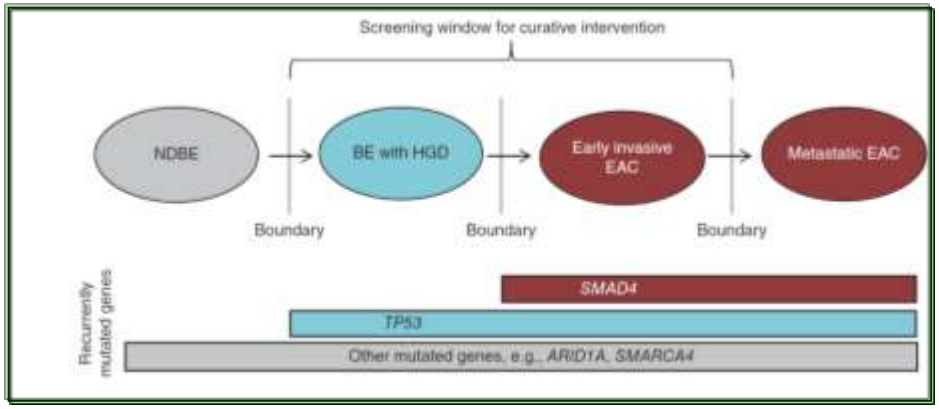


n= 43



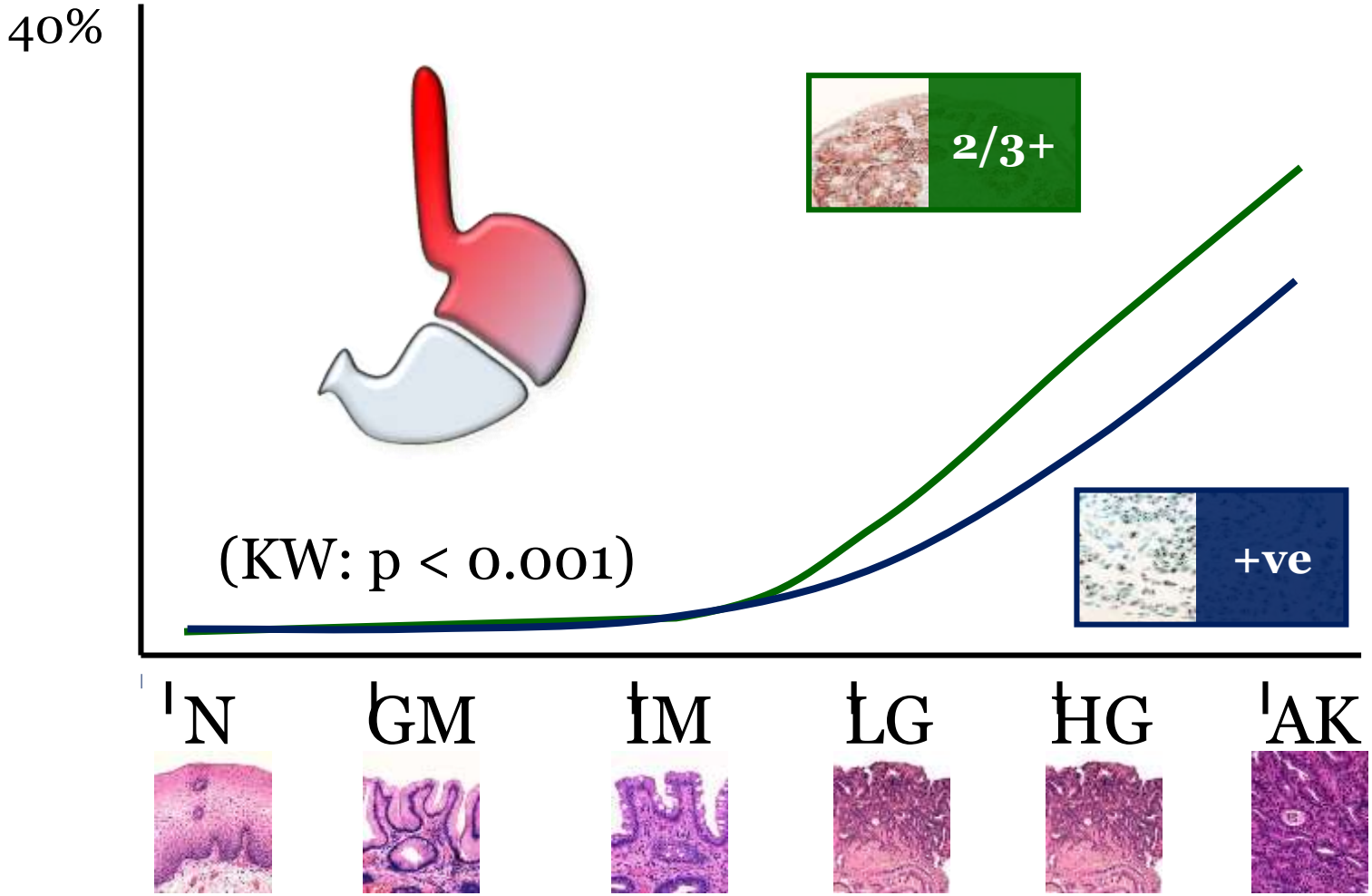
n= 112

Only *TP53* and *SMAD4* mutations occurred in a stage-specific manner, confined to HGD and EAC, respectively.

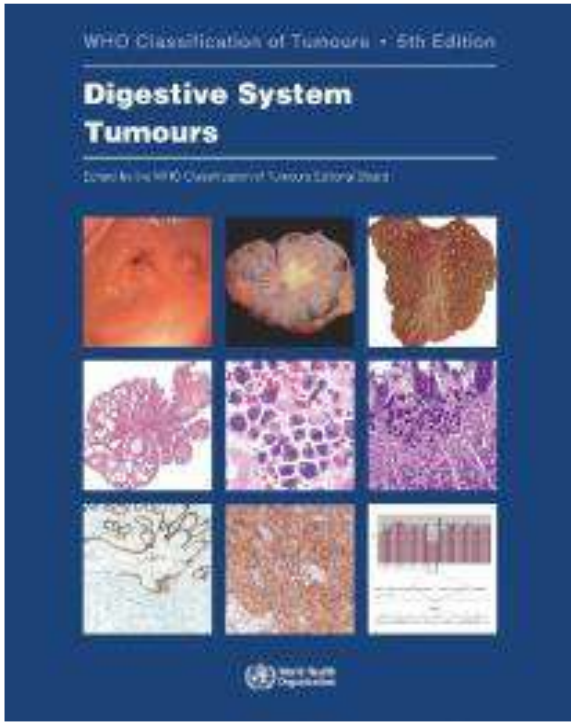


Weaver JM, et al – Nat Genet 2014

HER2 is overexpressed and amplified in dysplastic lesions



Fassan M, Rugge M et al - *Histopathology* 2012


WHO Classification of Tumours • 5th Edition
Digestive System Tumours

Neoplasms

- Benign tumors:
  - leiomyomas
  - lipomas
  - fibromas
  - squamous papillomas
  - condyloma
  - inflammatory polyp

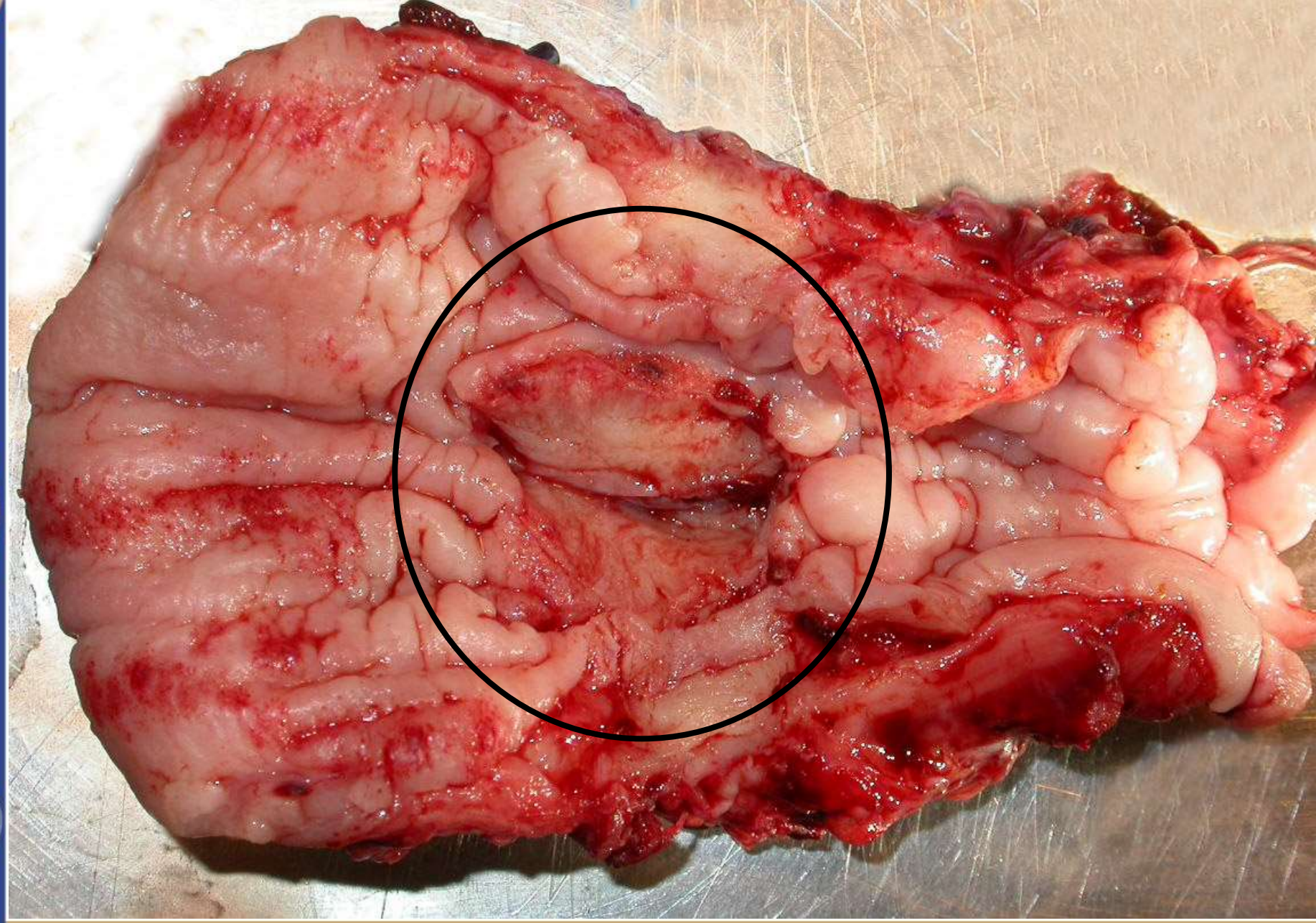
Malignant tumors

- Squamous cell carcinoma
- Adenocarcinoma

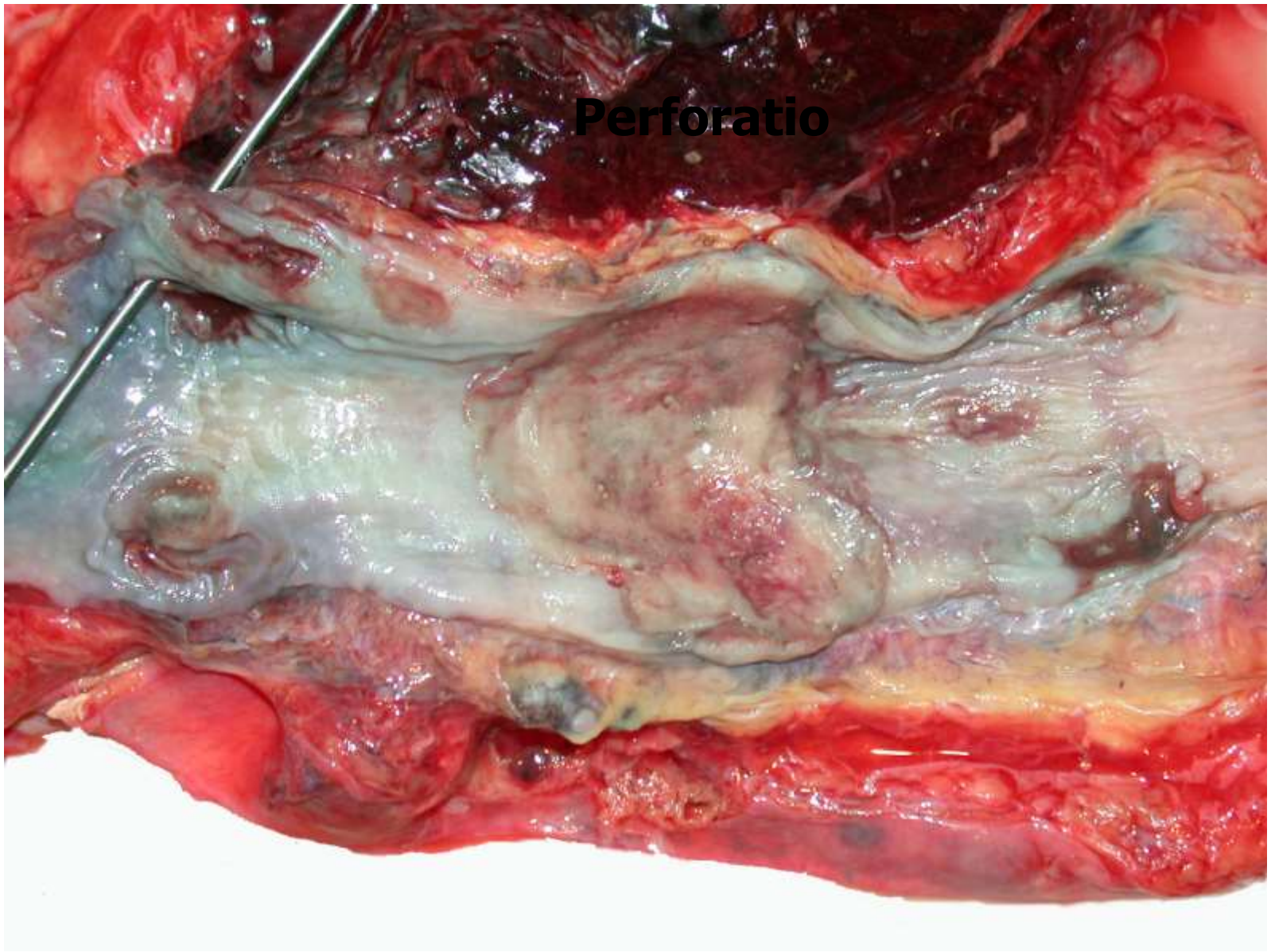
Squamous cell carcinoma

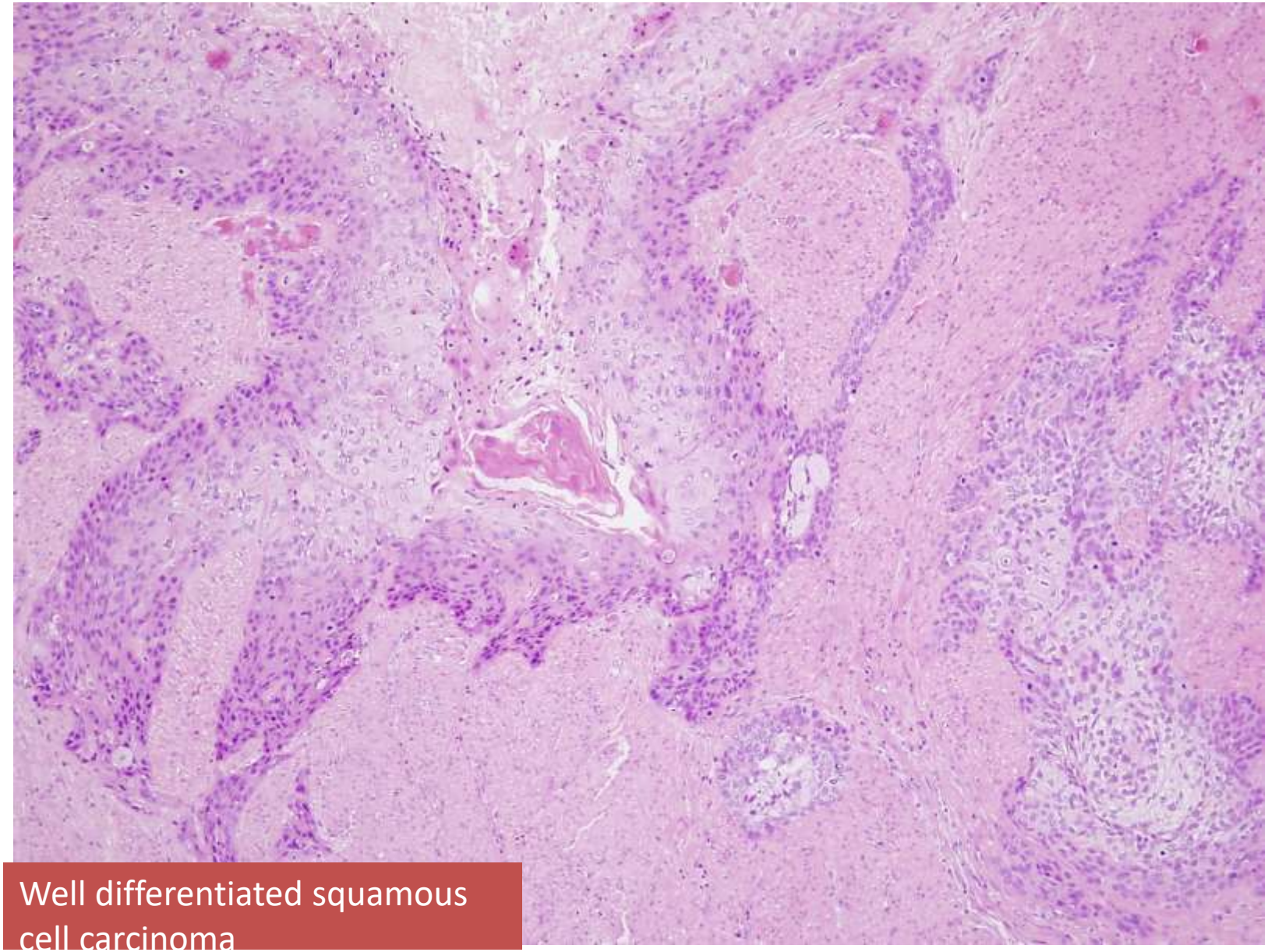
- over 50 yr, geographical differences
- male predominance
- Etiology:
  - **dietary** (deficiency of vitamin A, C, high nitrite content of water, fungal contaminated food)
  - **lifestyle** (hot beverages and food, alcohol, smoking)
  - **esophageal disease** (long standing esophagitis, achalasia),
  - **genetic predisposition**

- 20% upper third, 50% midportion, 30% lower third
- Macr:
  - →exophytic
  - →excavated (ulcerated)
  - →flat
- Micr: mostly moderately and well differentiated
  - →variants: verrucous, basaloid, spindle cell
- Metastases:
  - → to regional lymph nodes
- Local extension:
  - ➥ to mediastinum, respiratory tree, aortic wall

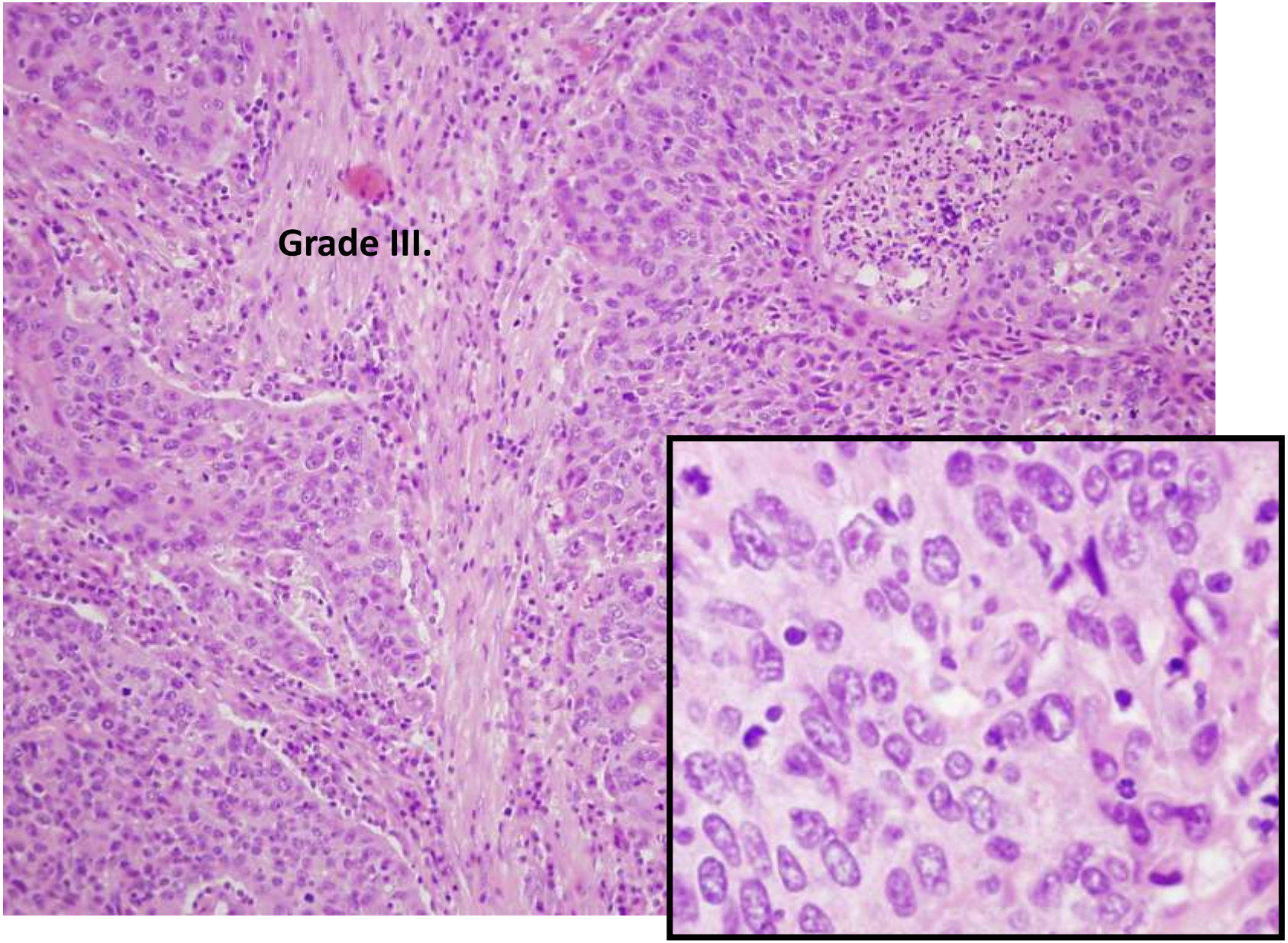


Perforation


Perforatio

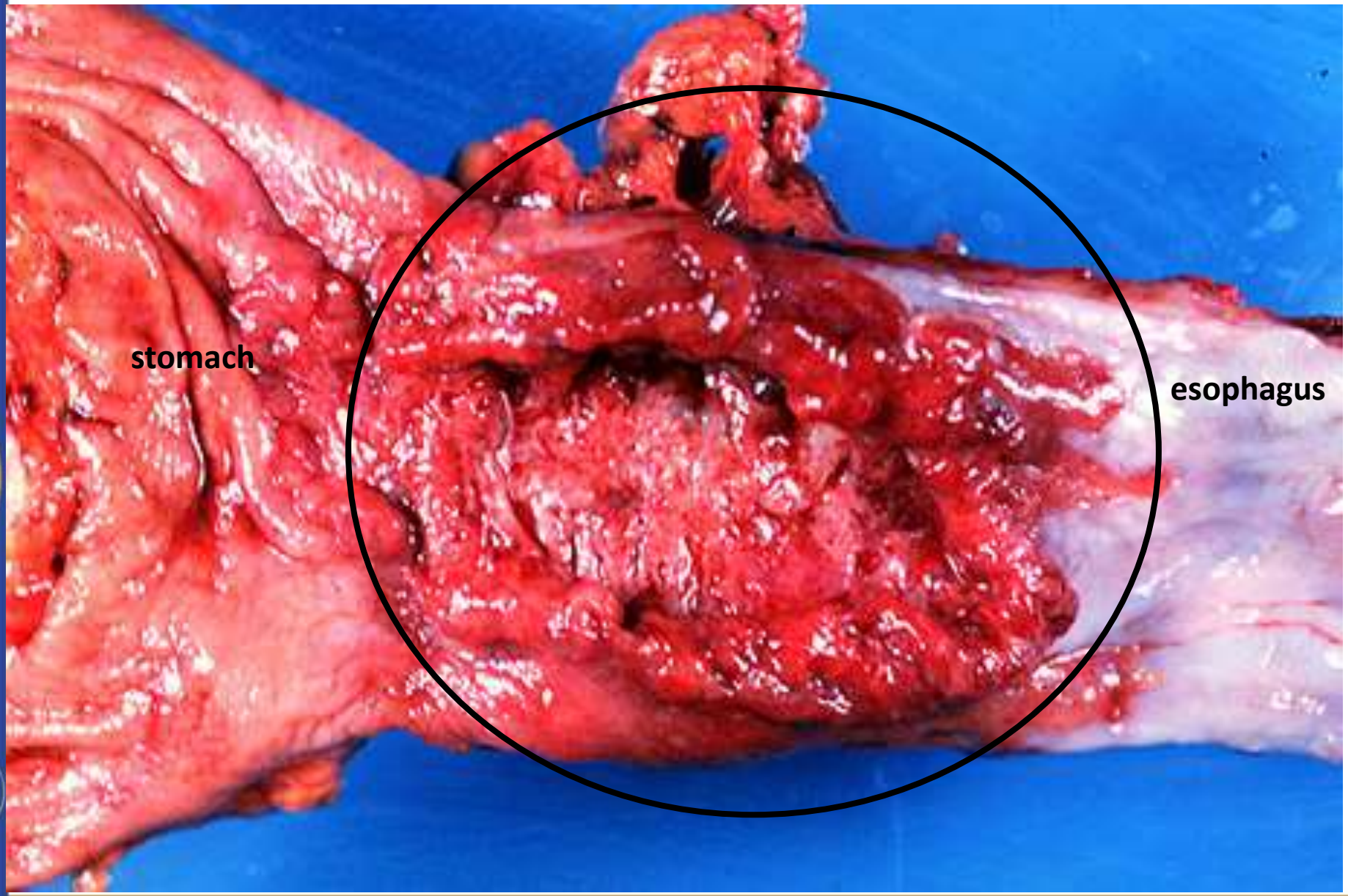


Well differentiated squamous cell carcinoma

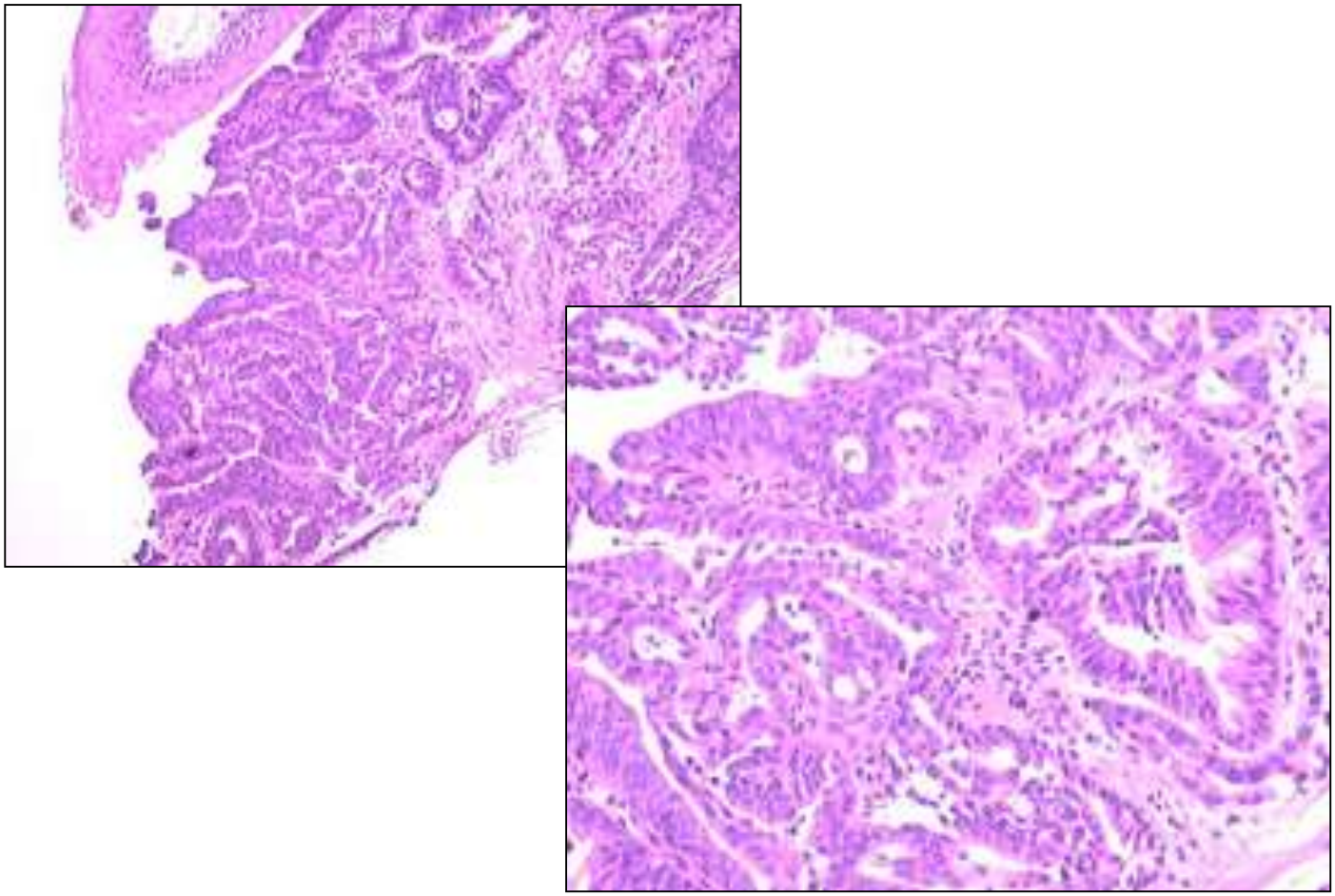

Grade III.

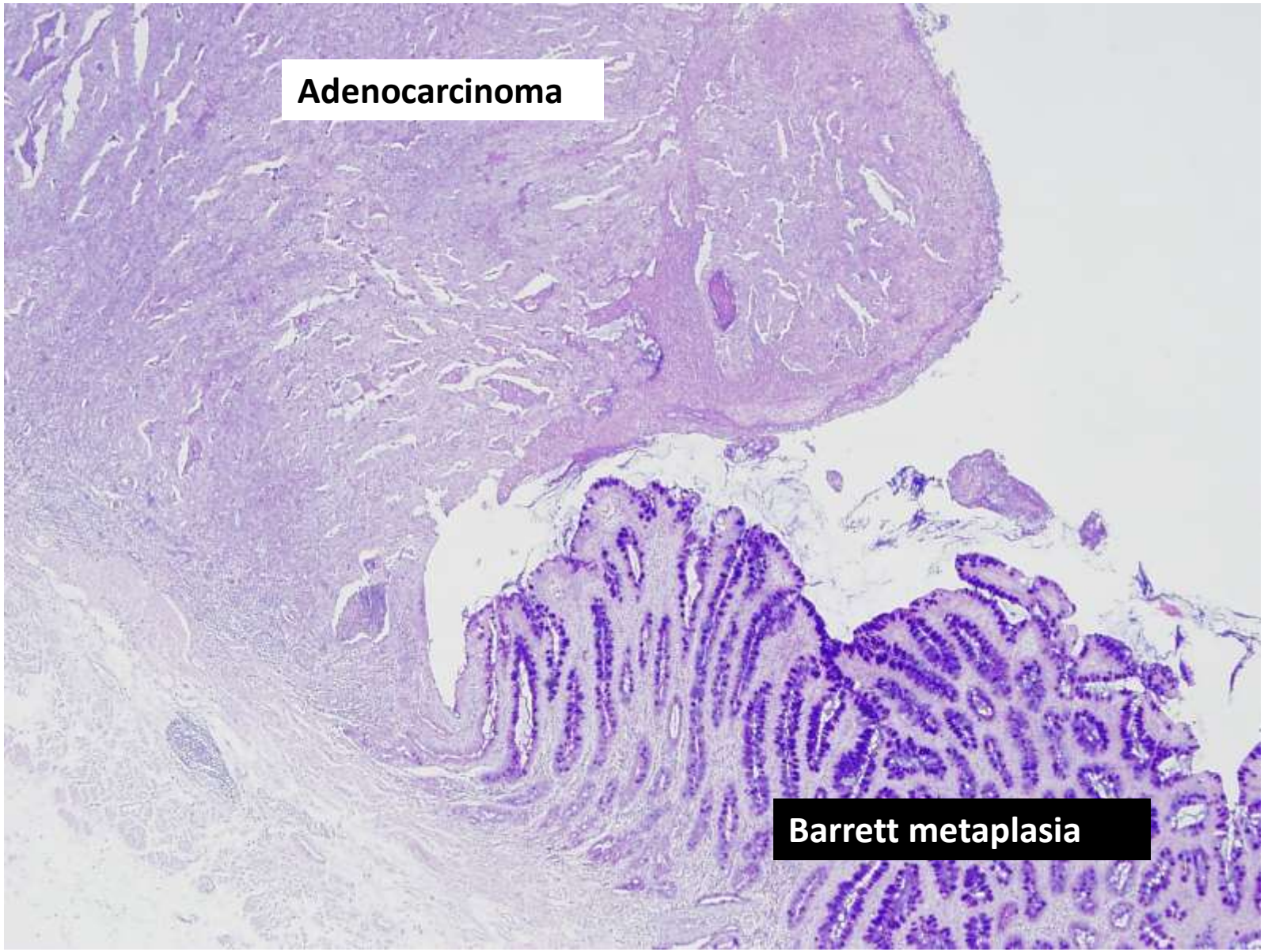
Adenocarcinoma

- Barrett mucosa associated
- distal esophagus, male predominance
- Symptomes:
  - difficulty in swallowing, bleeding, vomiting, pain, weight loss
- Gross:
  - flat patches, sometimes large nodular masses
- Micr:
  - intestinal type adenocarcinoma (in majority)
- poor prognosis


stomach
esophagus

Adenocarcinoma




Adenocarcinoma
Barrett metaplasia

TNM

Primary tumor (T)
TX: Primary tumor cannot be assessed
T0: No evidence of primary tumor
Tis: Carcinoma in situ
T1: Tumor invades lamina propria or submucosa
T2: Tumor invades muscularis propria
T3: Tumor invades adventitia
T4: Tumor invades adjacent structures
Regional lymph nodes (N)
NX: Regional lymph nodes cannot be assessed
N0: No regional lymph node metastasis
N1: Regional lymph node metastasis
Distant metastasis (M)
MX: Distant metastasis cannot be assessed
M0: No distant metastasis
M1: Distant metastasis
Tumors of the lower thoracic esophagus:
M1a: Metastasis in celiac lymph nodes
M1b: Other distant metastasis
Tumors of the midthoracic esophagus:
M1a: Not applicable
M1b: Nonregional lymph nodes and/or other distant metastasis
Tumors of the upper thoracic esophagus:
M1a: Metastasis in cervical nodes
M1b: Other distant metastasis

Surg Clin North Am 2005;85:555-567

AJCC stage groupings

Stage 0
Tis, N0, M0

Stage I
T1, N0, M0

Stage IIA
T2, N0, M0
T3, N0, M0

Stage IIB
T1, N1, M0
T2, N1, M0

Stage III
T3, N1, M0
T4, any N, M0

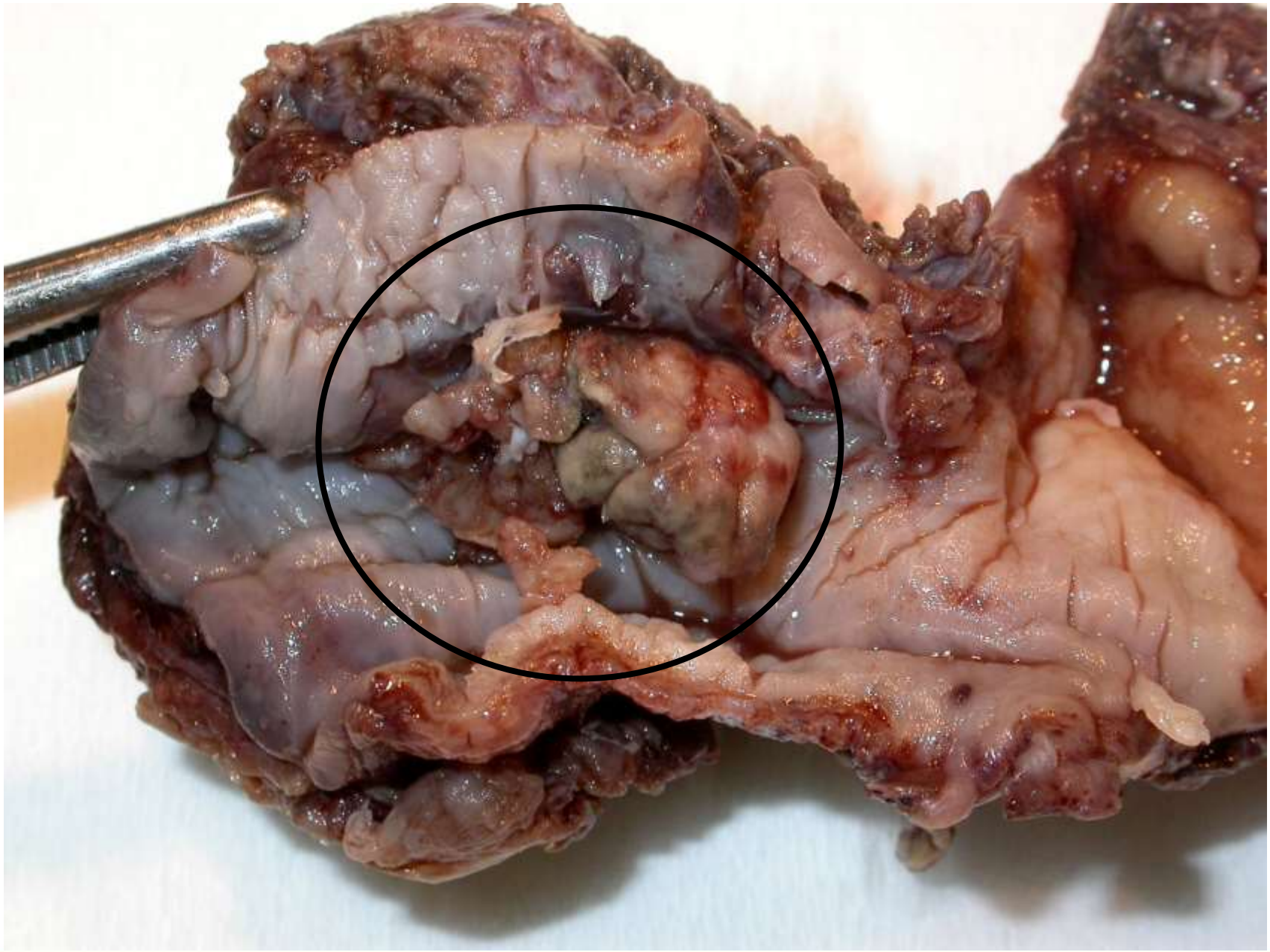
Stage IV
Any T, any N, M1

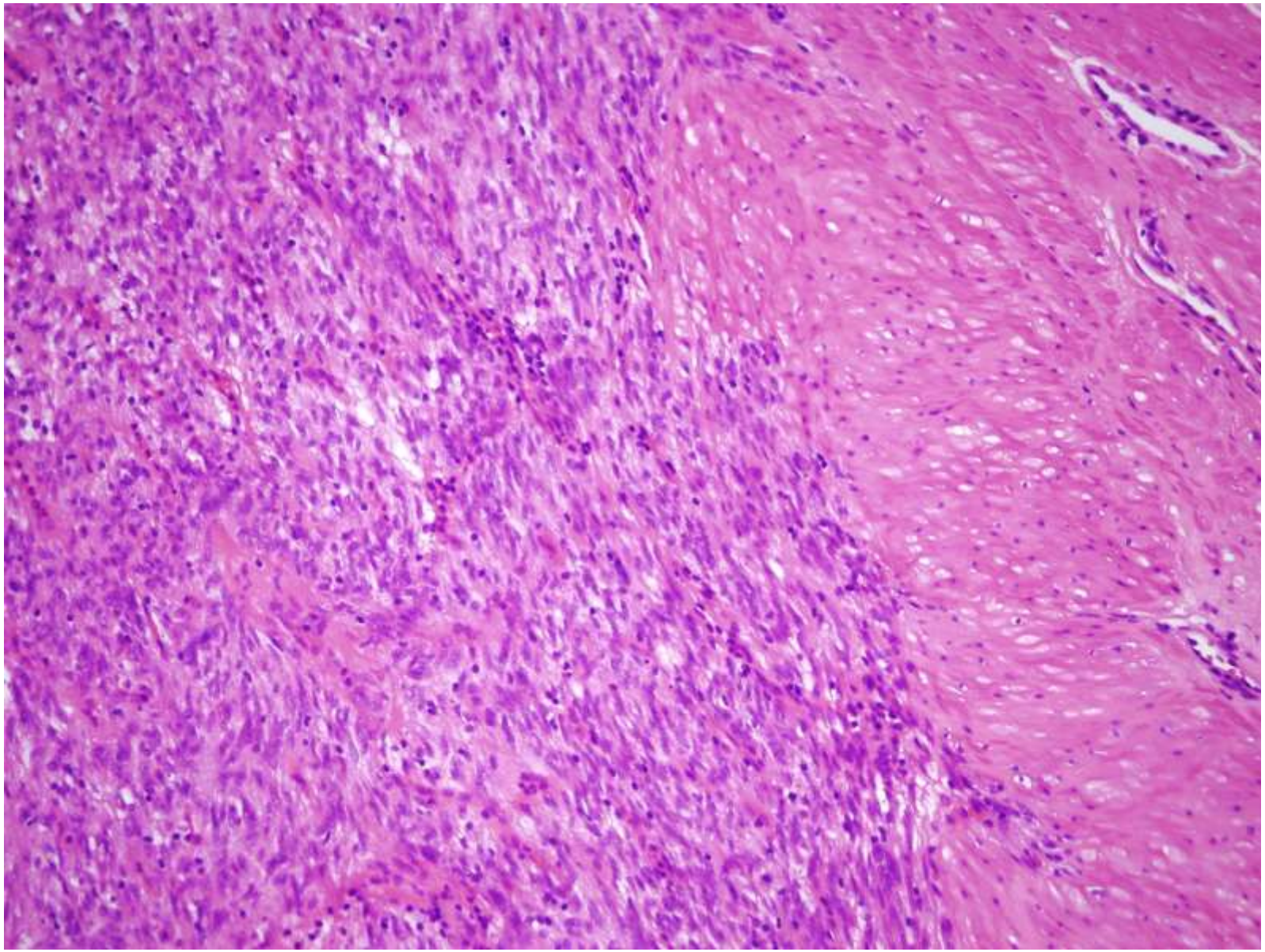
Stage IVA
Any T, any N, M1a

Stage IVB
Any T, any N, M1b

GIST

Gastrointestinal stromal tumor





STOMACH

Histology I.

- **foveolar** compartment
  - uniform in the whole stomach
  - consists of foveolar epithelial cells lining the surface and pits
- **glandular** compartment
  - major differences in composition:
    - cardia glands
    - fundic glands (oxynthic or corpus)
    - Pyloric (antral and pyloric) glands

Histology II.

- **Mucous cells**
- **Parietal cells:**
  - →oxyphyl (mitochondria)
  - →proton pump (H^+,K^+-ATP-ase)
  - ➥ I ntrinsic factor
- **Chief cells:**
  - →basophilia (RER, prominent Golgi)
  - →Pepsinogen I and II production
- **Endocrine cells:**
  - →scattered triangular cells with brightly eo granules
  - →G cells, D cells, X cells

Gastric diseases

- Congenital anomalies
- Gastritis
- Peptic Ulcer Disease
- Miscellaneous Conditions
- Neoplasms

Congenital diseases

- **Heterotopia**: pancreas
- **Diaphragmatic hernia**
- **Congenital hypertrophic pyloric stenosis**
  - male predominance
  - Turner sy, trisomy 18, esophagus atresia
  - vomiting, regurgitation in 2-3 weeks old baby
  - palpable mass in the pyloric region
  - Th: surgery (muscle splitting)

GASTRITIS

ACUTE GASTRITIS

Etiology

- →NSAIDs
- →alcohol, smoking
- →cytotoxic drugs
- →stress (surgery, burns, trauma)
- →ischemia, shock
- →uremia
- →systemic bacterial or viral infection
- →distal gastrectomy

Acute gastritis

- Symptoms:
  - pain, vomiting, nausea, hematemesis, melaena, severe blood loss
- Gross: hyperemia
  - in **acute erosive hemorrhagic gastritis**
    - → **erosions** (mucosal defect not deeper than the muscularis mucosae)
- Micr: congestion and edema of the lamina propria
  - neutrophils within surface and glandular epithelium („activity”)
  - in acute erosive hemorrhagic gastritis- surface necrosis with hemorrhage

Acute hemorrhagic erosive gastritis

Chronic gastritis

- Signs of chronic inflammation
  - lymphocytes, plasma cells mainly within the gastric mucosa, potentially leading to atrophy and intestinal metaplasia
- Etiology:
  - **Helicobacter pylori**
  - **autoimmune** (with pernicious anemia)
  - **alcohol, smoking**
  - distal gastrectomy
  - uremia
  - Crohn disease
  - irradiation
  - motor dysfunction

H.pylori

- 1983. Campylobacter pylori
- Gram neg rods
- urease production- diagnostic testing!
- Associated with
  - →chr. gastritis,
  - →peptic ulcer disease,
  - →gastric carcinoma,
  - →gastric MALT lymphoma
- Antibiotic therapy and proton pump inhibitors

H.pylori

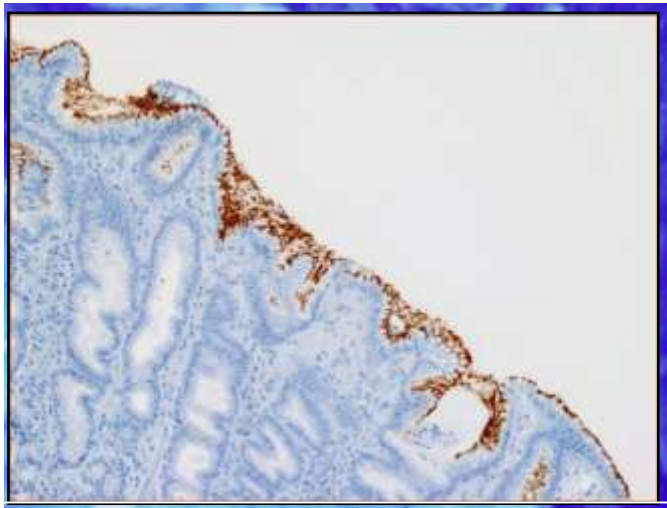
Photo: C. Northcott

Barry J. Marshall

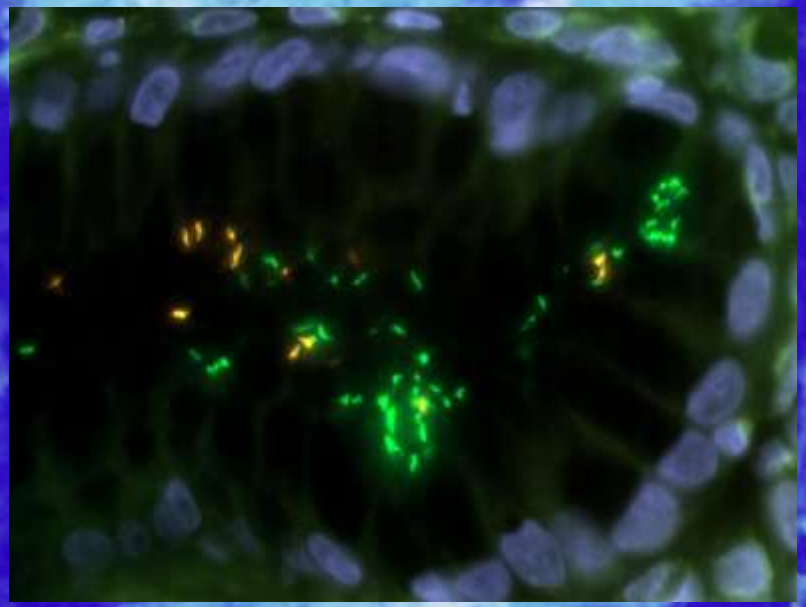
Photo: U. Montan

J. Robin Warren

The Nobel Prize in Physiology or Medicine 2005 was awarded jointly to Barry J. Marshall and J. Robin Warren *"for their discovery of the bacterium Helicobacter pylori and its role in gastritis and peptic ulcer disease"*



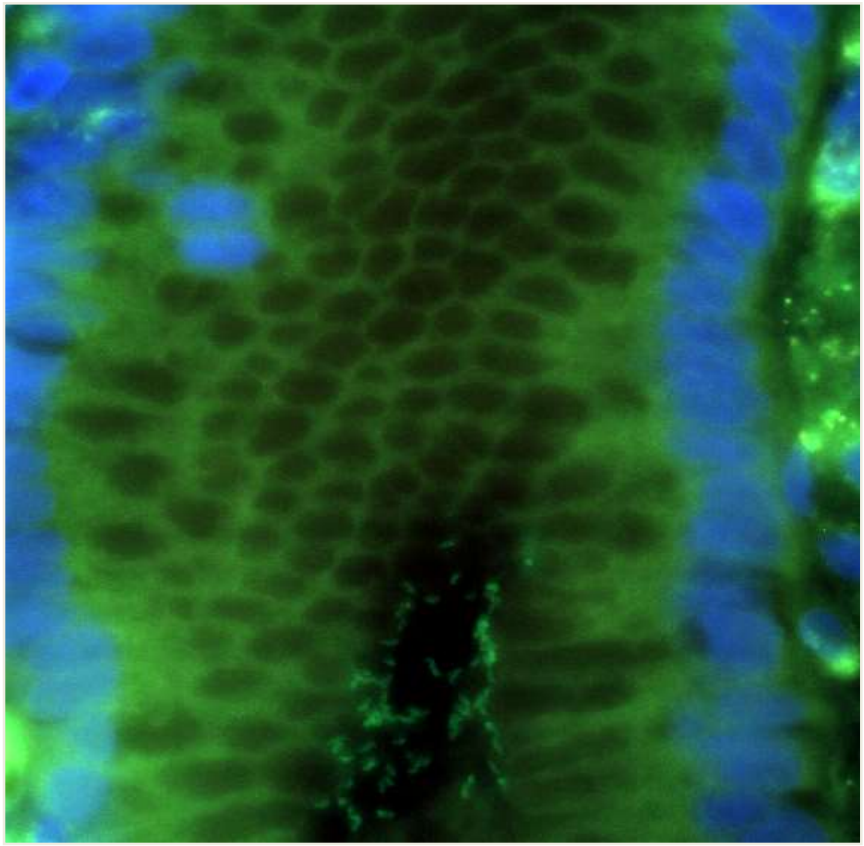
H. Pylori by immunohistochemistry



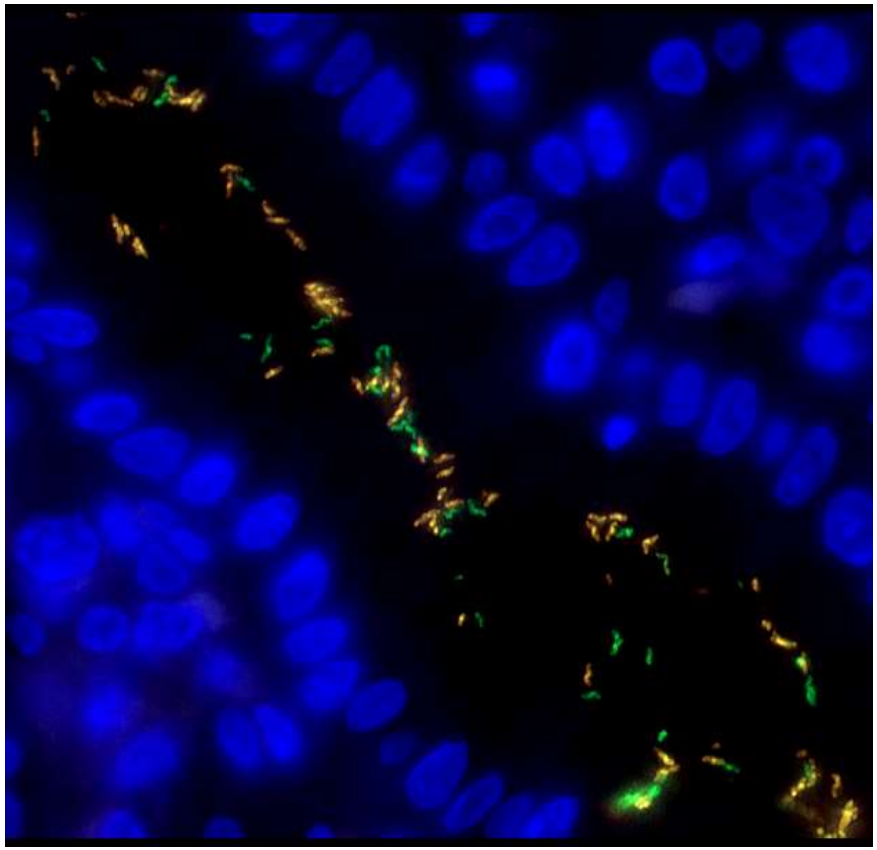
H.pylori-Giemsa

Importance of fixation–Helico FISH

Overfixed



Properly fixed



Autoimmune gastritis

- 10% of chronic gastritis
- diffuse damage of **corpus**, antral inflammation less pronounced
- autoantibodies to parietal cells⟶ gland destruction ⟶ mucosal atrophy ⟶ loss of acid production and intrinsic factor production leading to pernicious anemia
- associated with other autoimmune diseases (Hashimoto thyreoiditis, Addison disease, DM type I)
- increased risk of gastric cancer and carcinoid development

Special forms of gastritis

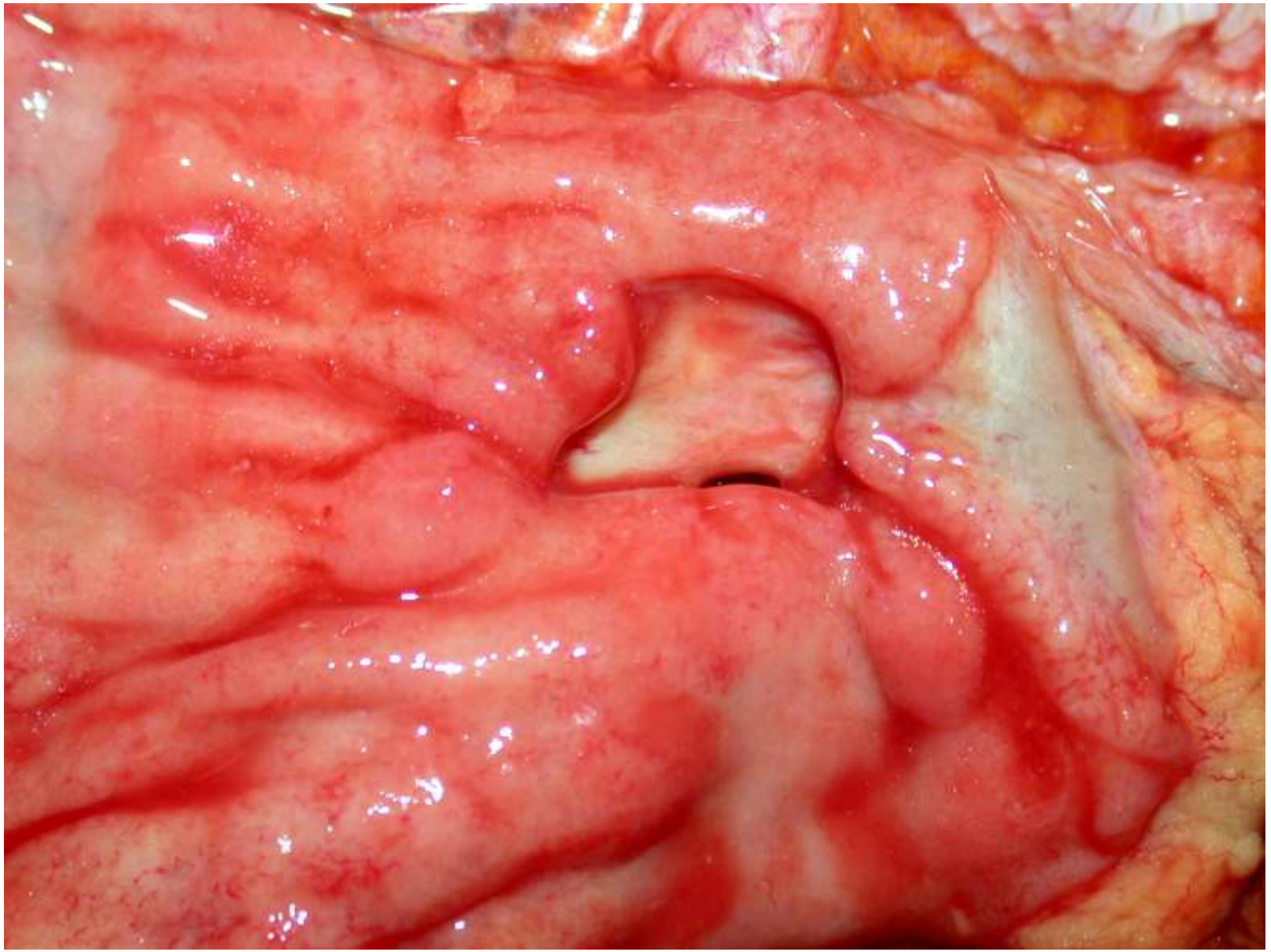
- Eosinophilic gastritis
- Allergic gastroenteropathy
- Lymphocytic gastritis
- Granulomatous gastritis
- Graft-versus-Host Disease
- Reactive gastropathy

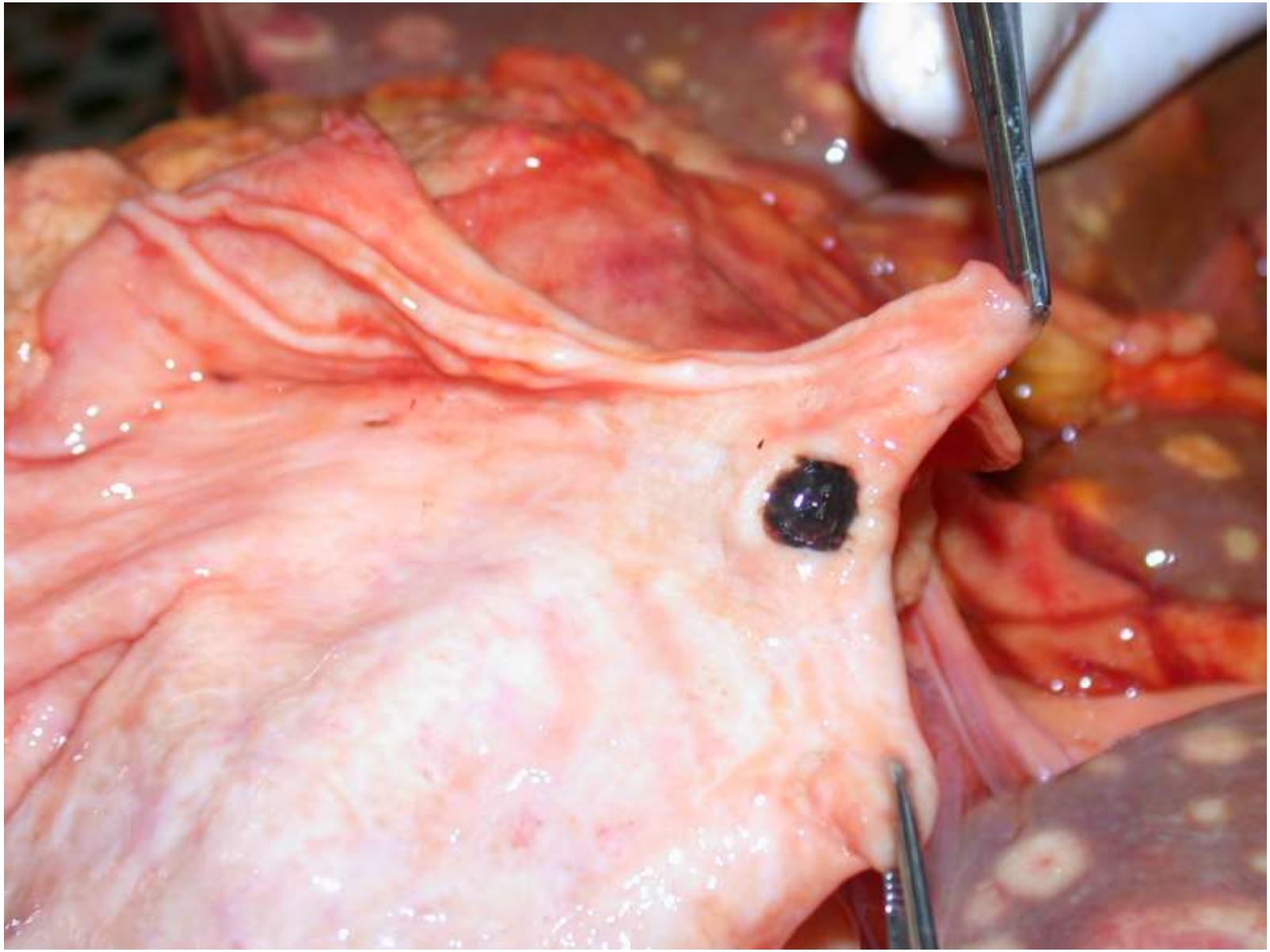
Peptic Ulcer Disease

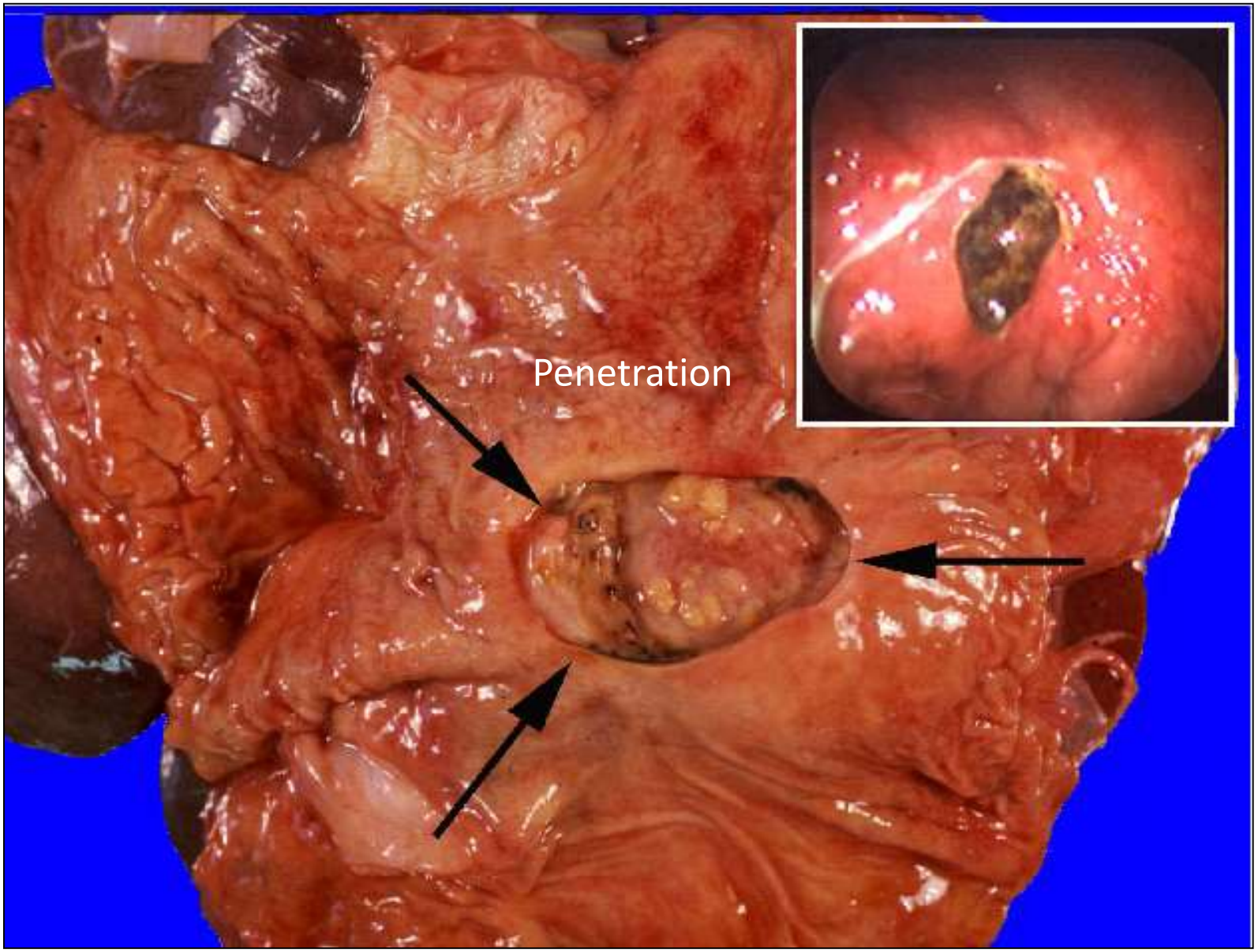
- **Ulcer**- mucosal defect deeper than the muscularis mucosae (↔ erosion)
- Location:
  - duodenum (98%)
  - antrum (lesser curvature)
  - gastroesophageal junction (GERD, Barrett)
  - in Meckel diverticulum (gastric heterotopia)
- male predominance
- due to an imbalance between mucosal defense and damaging injury

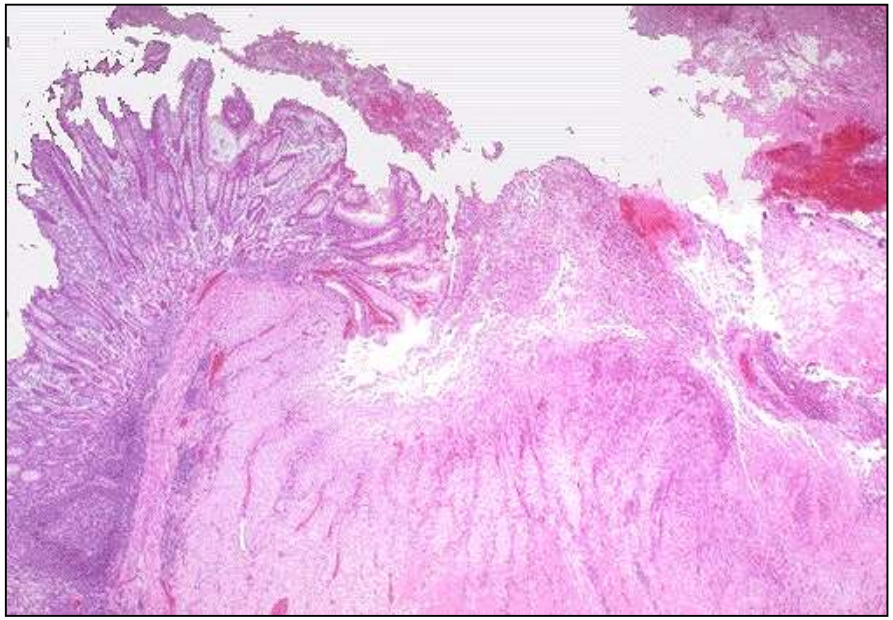
Ulcer- macroscopy

- punched-out defect
- margins level with the surrounding mucosa
  - (heaped up margins- sign of malignancy)
- Complications:
  - penetration to the pancreas, liver, omentum may occur
  - perforation to the abdominal cavity may occur
  - hemorrhage (hematemesis, melaena)
  - thrombosed blood vessel may be seen at the base

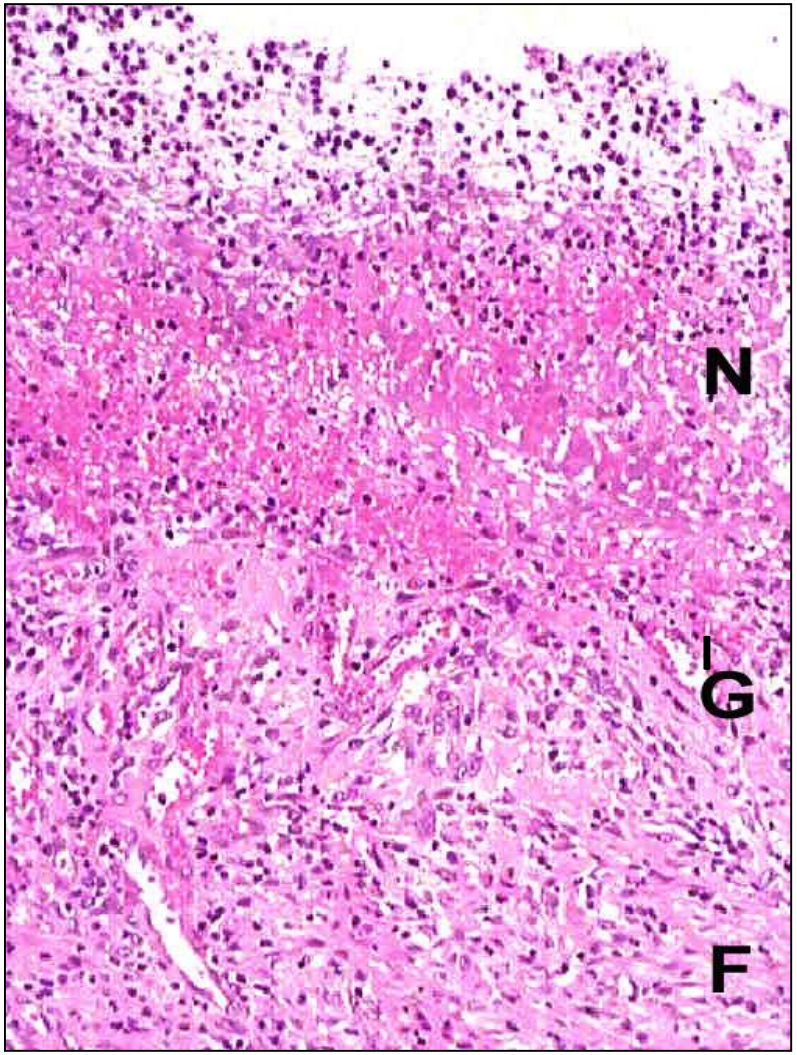





Penetration

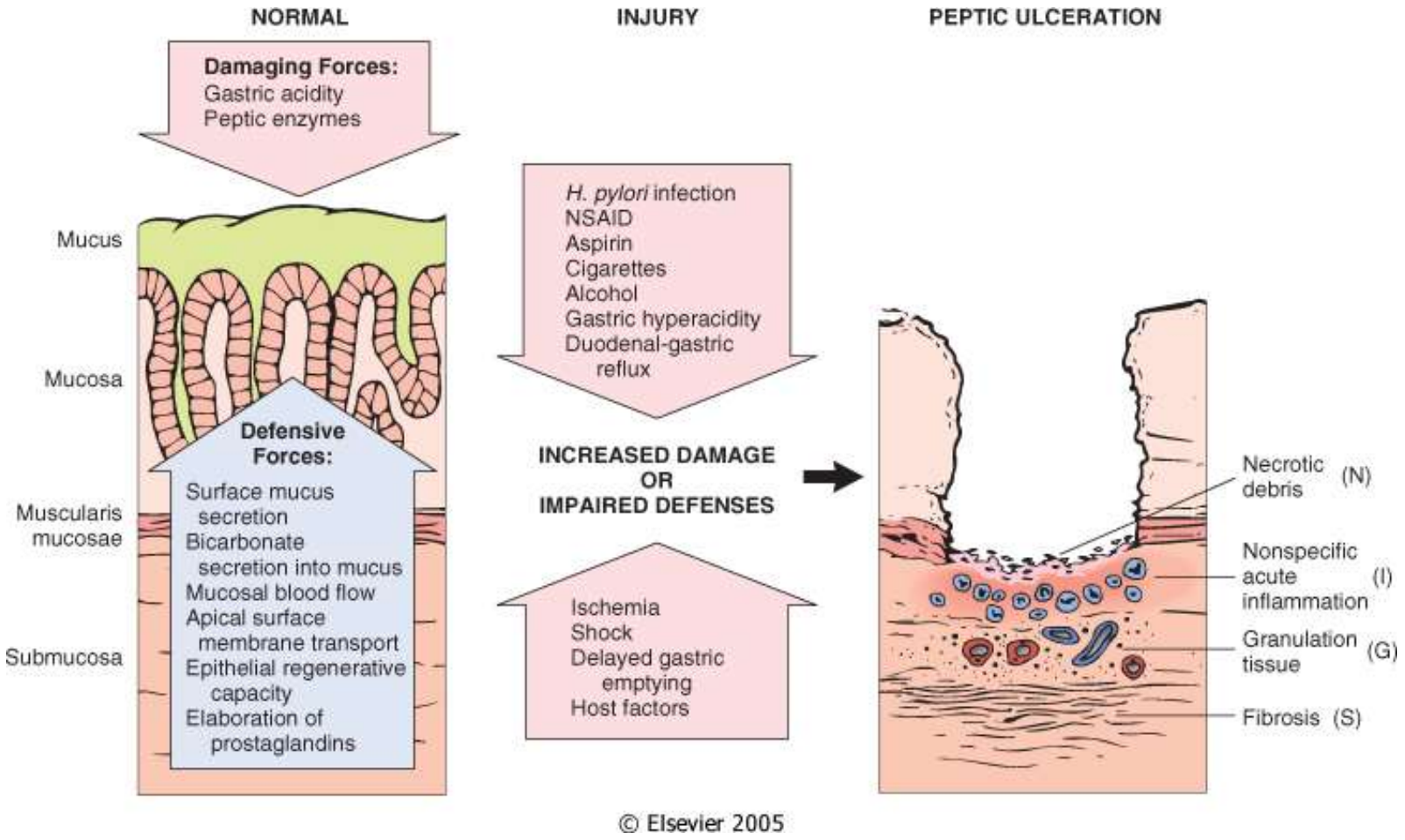


Peptic ulcer disease



- Necrosis (N)
- Non-specific inflammatory cells (I)
- Granulation tissue (G)
- Fibrosis (F)

PEPTIC ULCER DISEASE

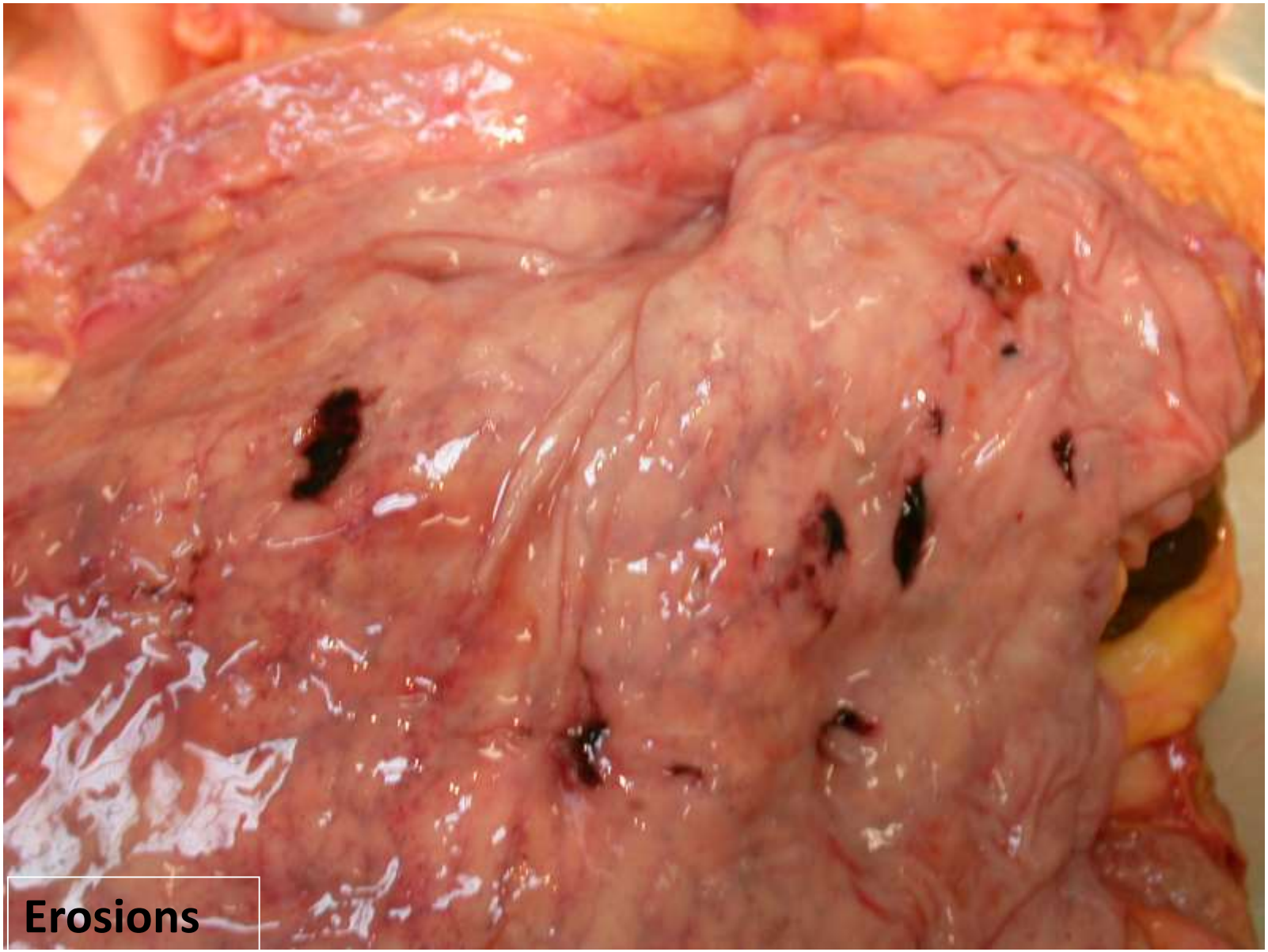


Ulcer- symptoms

- epigastric pain, nausea, vomiting
- iron-deficiency anemia
- gastrointestinal bleeding (hematemesis, melena)
- if perforation occurs- signs of muscular defense, shock, peritonitis
- if penetration occurs- may mimic AMI

Acute gastric ulceration

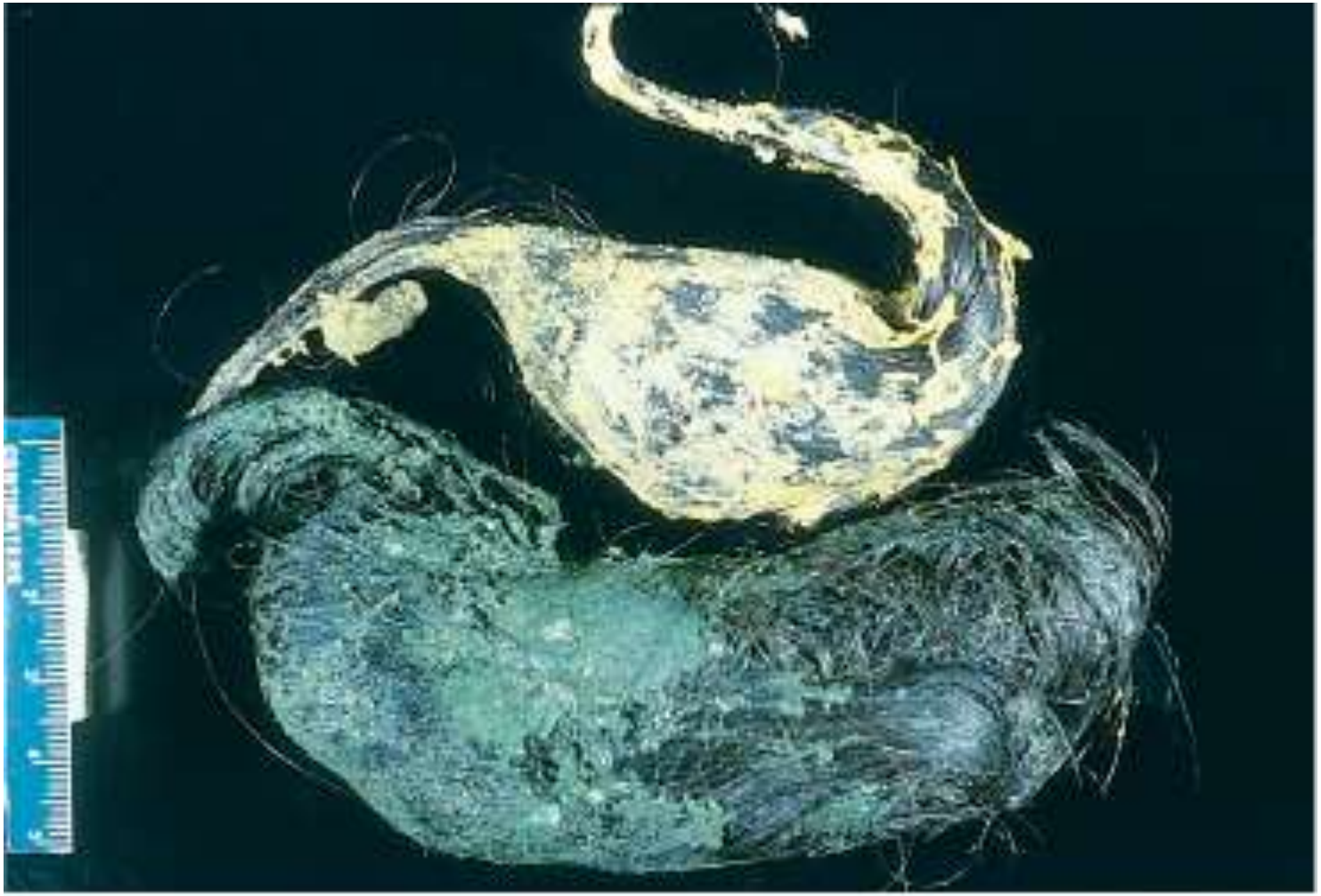
- **Stress erosions and ulcers**
  - in shock, with burns, sepsis, trauma, surgery, intracranial injury
    - →CURLING ulcers- with burns or trauma
    - →CUSHING ulcers- with intracranial injury



Erosions

Miscellaneous Conditions

- Gastric dilation
- Gastric rupture
- Phytobezoars
- Trichobezoars
- Hypertrophic gastropathy
  - Ménétrier disease
  - Hypertrophic - hypersecretory gastropathy
  - Zollinger- Ellison syndrome



trichobezoar

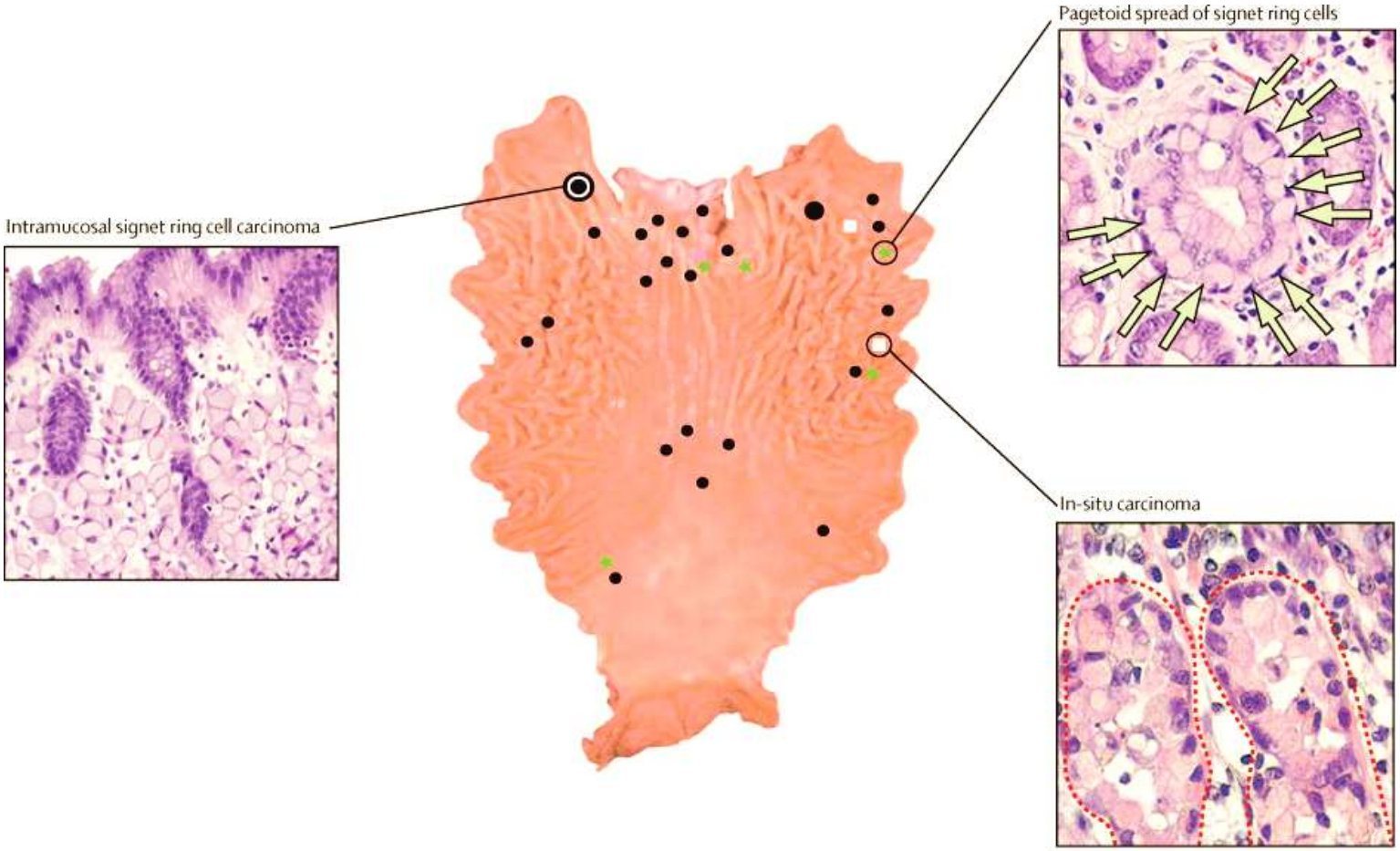
Thank you for Your Attention !!!

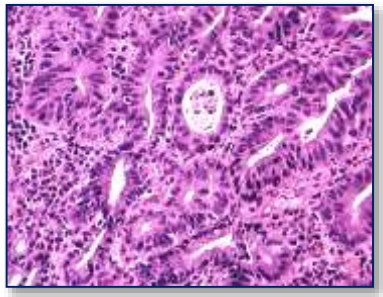
Pathology and Genetics of Familial Gastric Cancer

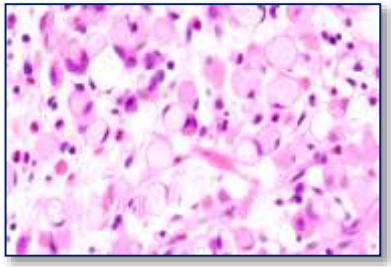
Fátima Carneiro, MD, PhD,[1,2] Carla Oliveira, PhD[1] and Raquel Seruca, MD, PhD[1]

About 10% of GCs show familial clustering and 1% to 3% are hereditary (HDGC). Due to *CDH1* germline mutations.

Early HDGC shows multiple foci of invasive (T1a) signet-ring cell cancer with no elective topography.







HEREDITARY **INTESTINAL**

SPORADIC **INTESTINAL**

MSI+ve

APC

p53

β-CATENIN

APC

MLH1-methyl

AG
(IM CDX2+)

IEN

ATROPHIC GASTRITIS

NATIVE MUCOSA

CHRONIC GASTRITIS

E-CADHERIN

p53

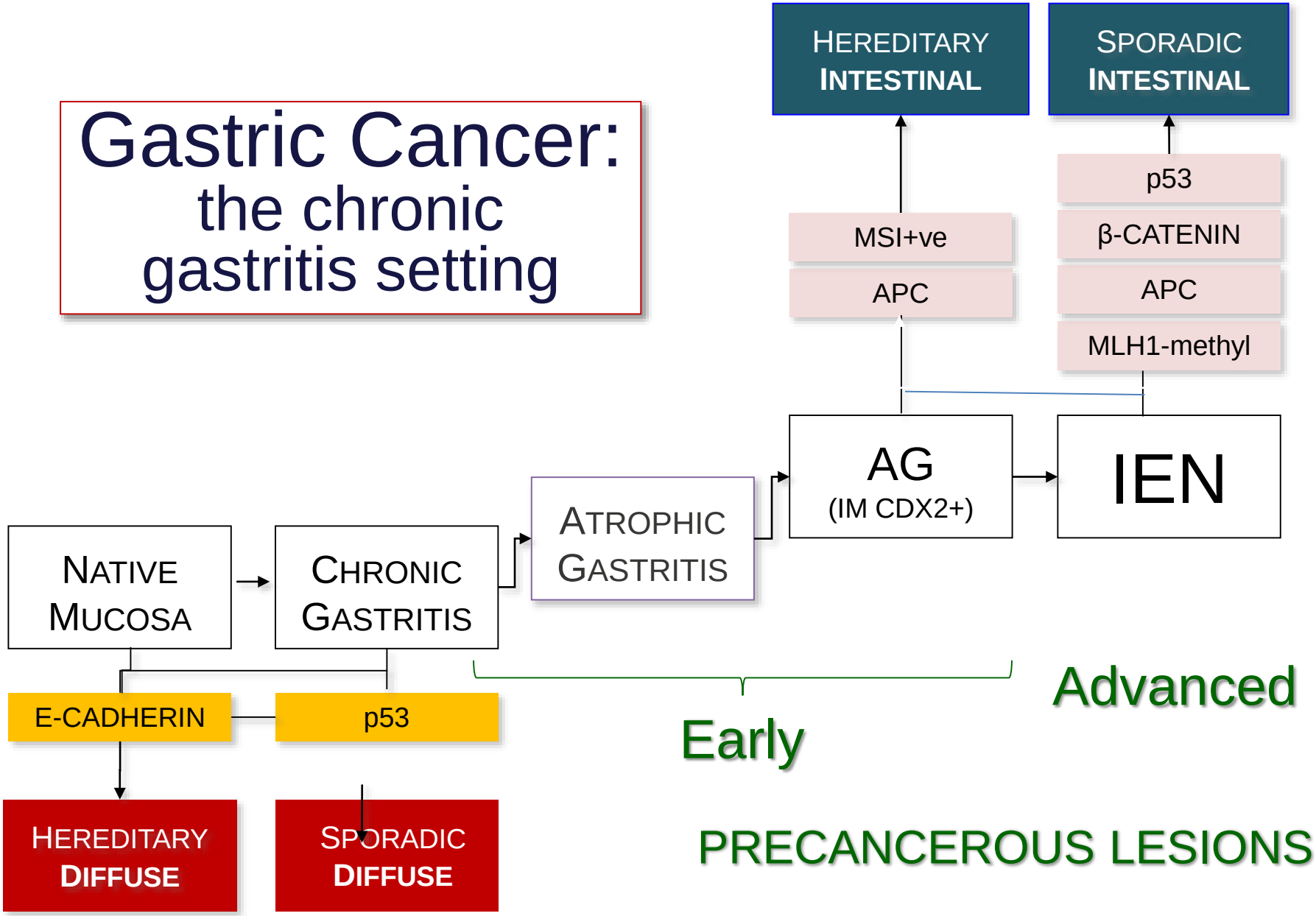
HEREDITARY **DIFFUSE**

SPORADIC **DIFFUSE**

Gastric Cancer

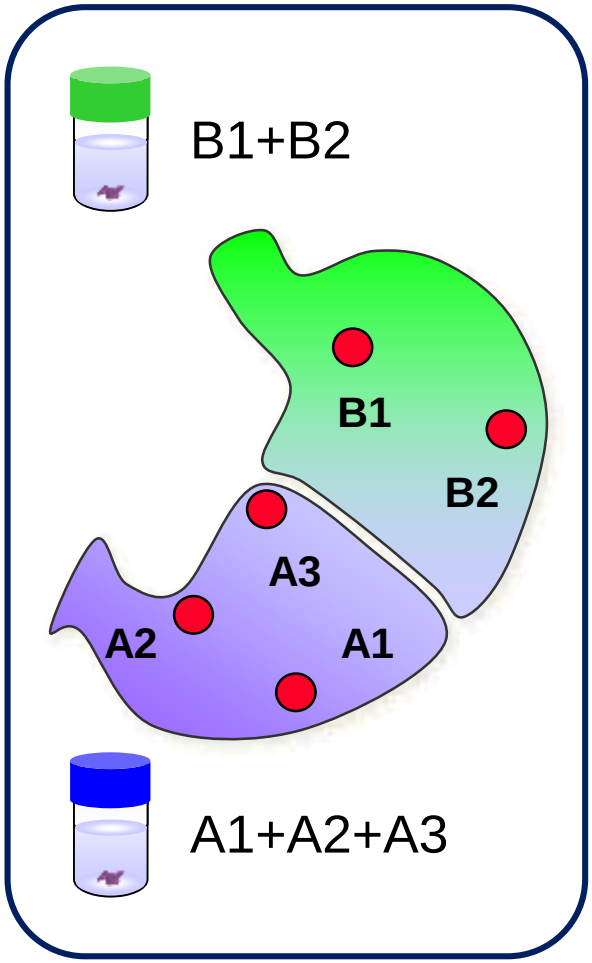
PHENOTIPIC & MOLECULAR PATHWAYS

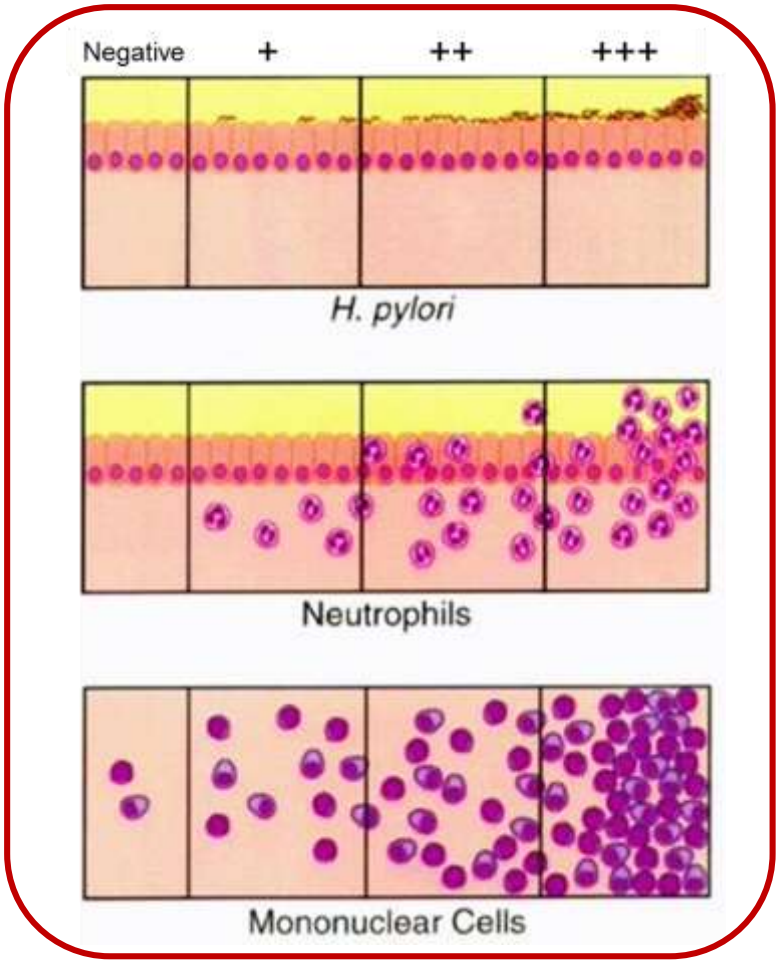
Gastric Cancer: the chronic gastritis setting



Combine **morphological**, **topographical** and **etiological** information to generate reproducible and clinically useful diagnoses

The Sydney revolution





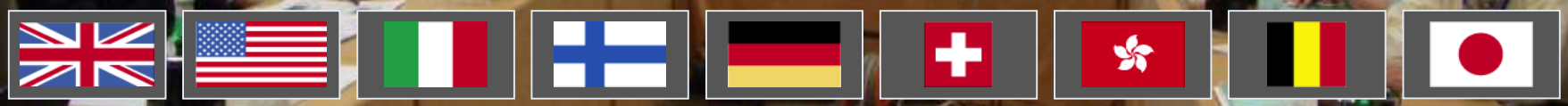
Sampling protocol

Visual analog scales

GASTRITIS STAGING: CONSENSUS CONFERENCE

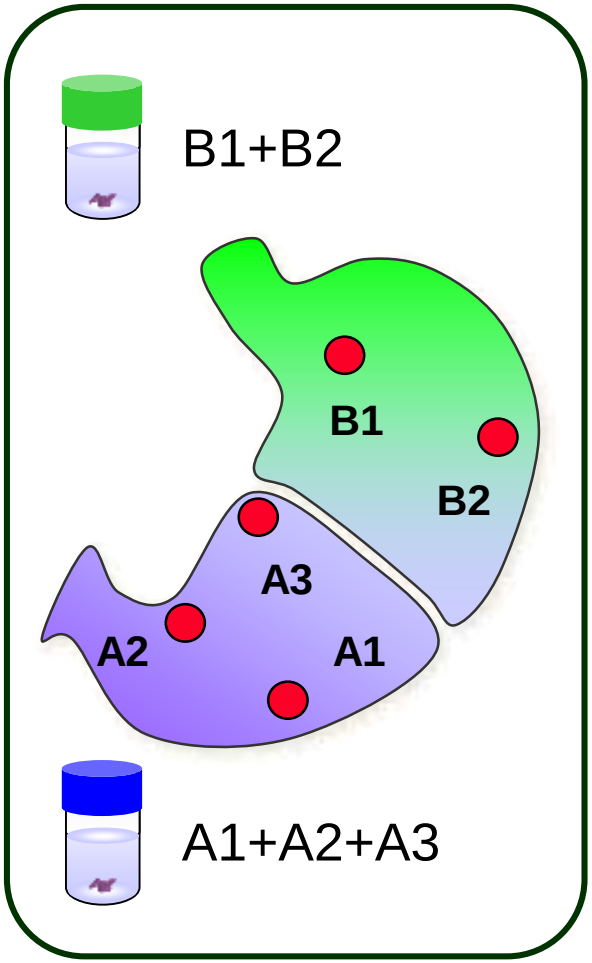
E El-Omar
DY Graham
P Correa
F Di Mario
P Sipponen
W Weinstein
M Vieth

M Rugge
RM Genta
R Fiocca
J Sung
K Geboes
T Hattori
P Malfertheiner

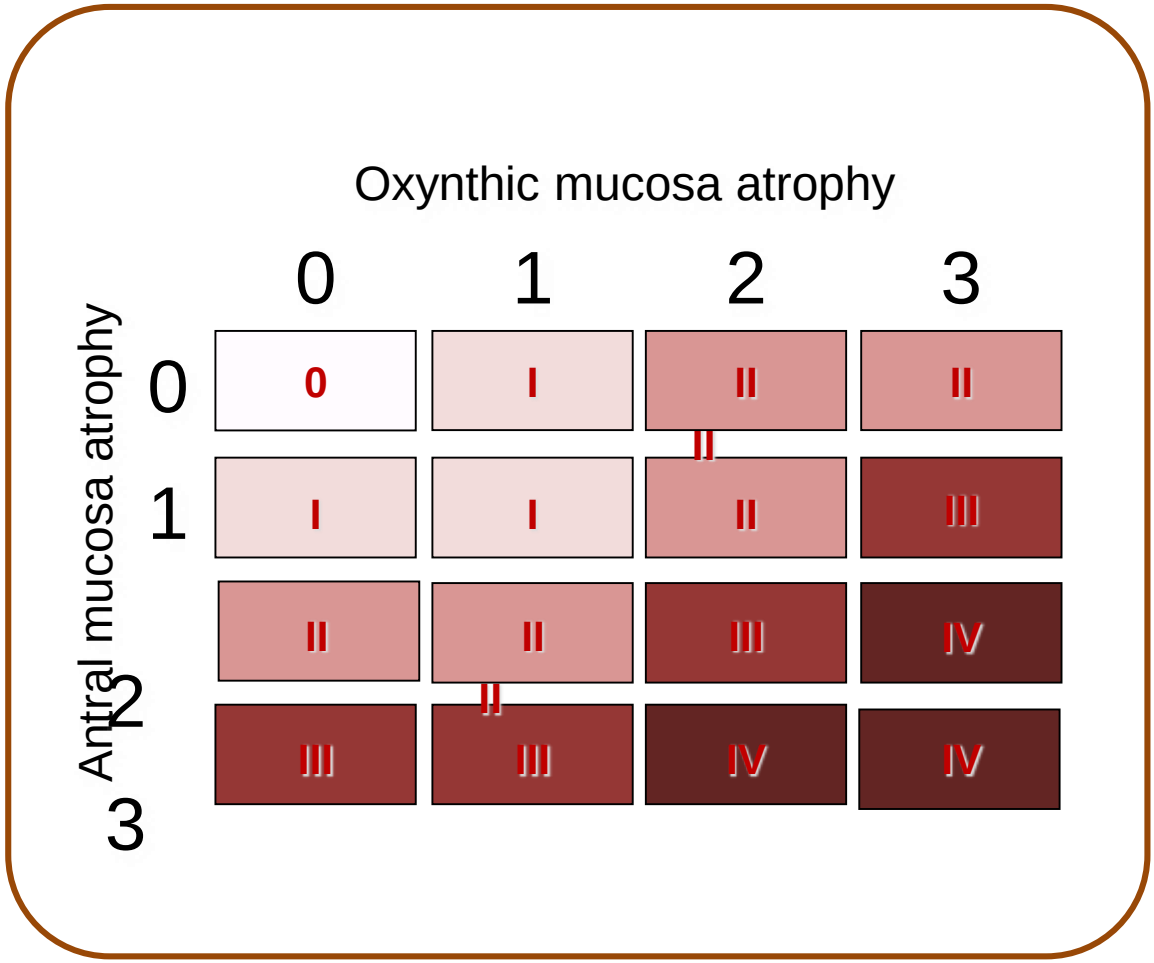


Operative Link on Gastritis Assessment - OLGA

The OLGA frame



Sydney system



Staging

OLGA Staging & cancer risk in Padua

8,416

Consecutive EGDS

5 BIOPSY SAMPLES