EXAMINATIONS IN GASTROENTEROLOGY

Esophagus, Stomach, Small Intestine

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Outline

- Functional examination of
  - Esophagus
  - Stomach
  - Small Intestine

FUNCTION OF GIT

- Digestion and nutrient uptake
- Barrier function
  - pathogens
  - toxins

ESOPHAGUS

Motility disorders of esophagus

Motility disorders of esophagus

- **Gastroesophageal reflux disease (GERD)**
- Achalasia
- Diffuse esophageal spasm
- Hypertensive esophageal peristaltic contractions (nutcracker esophagus)
- Hypertensive and hypercontracting LES

GERD

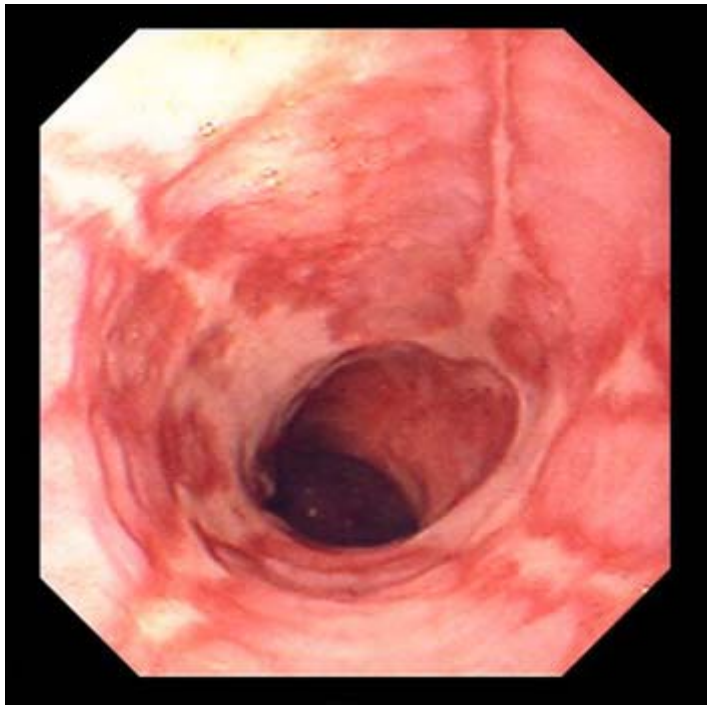
- Squamous mucosa of esophagus is more vulnerable to peptic digestion than columnar gastric epithelium

- Manifestation
  - heartburn (pyrosis)
- Consequences
  - inflammation of esophageal mucosa
  - Barrett's esophagus
  - Esophageal adenocarcinoma

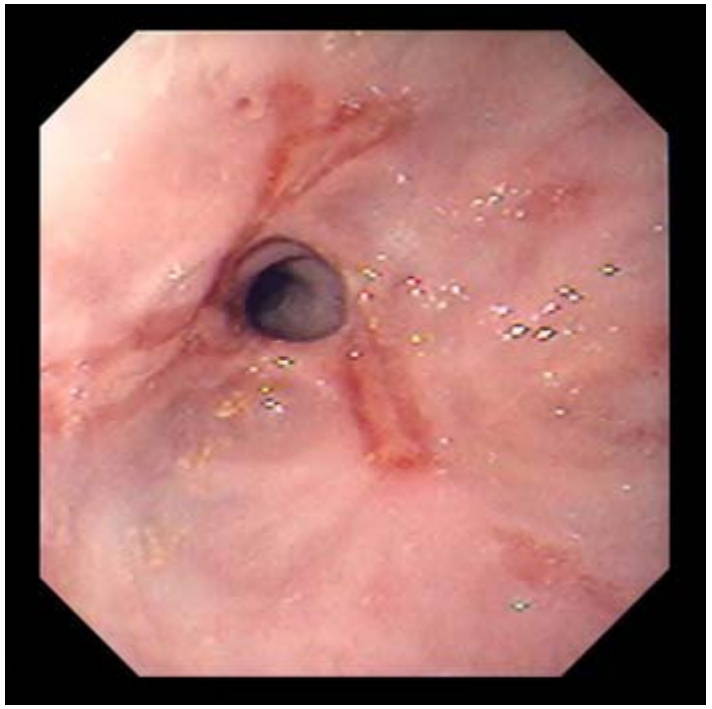
- 7% of the population experiences heartburn daily and 44% at least once a month

Complications of Gastroesophageal Reflux Disease (GERD)

Reflux esophagitis

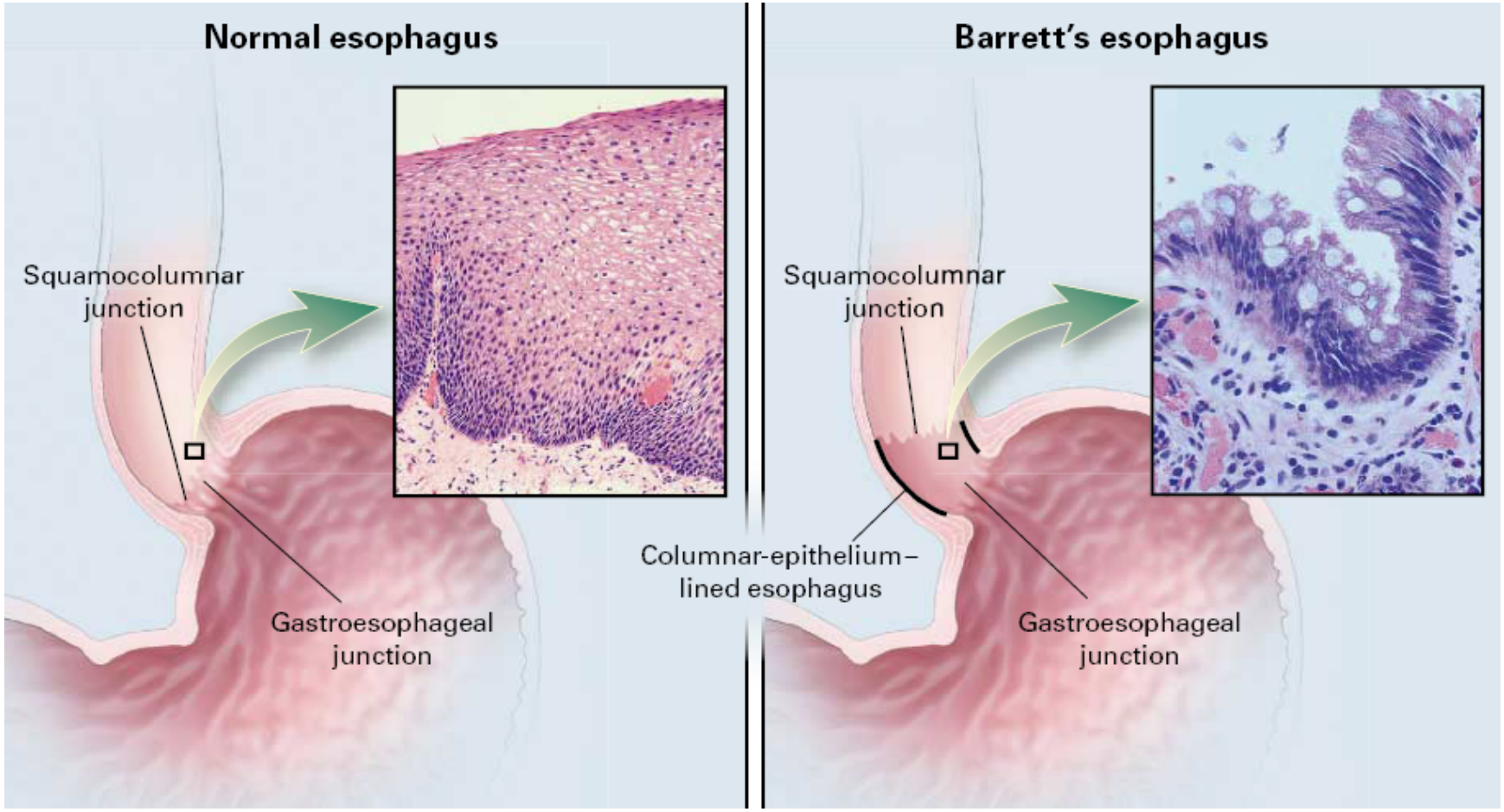


Peptic esofageal stricture



Reflux esophagitis
Barrett's Esophagus

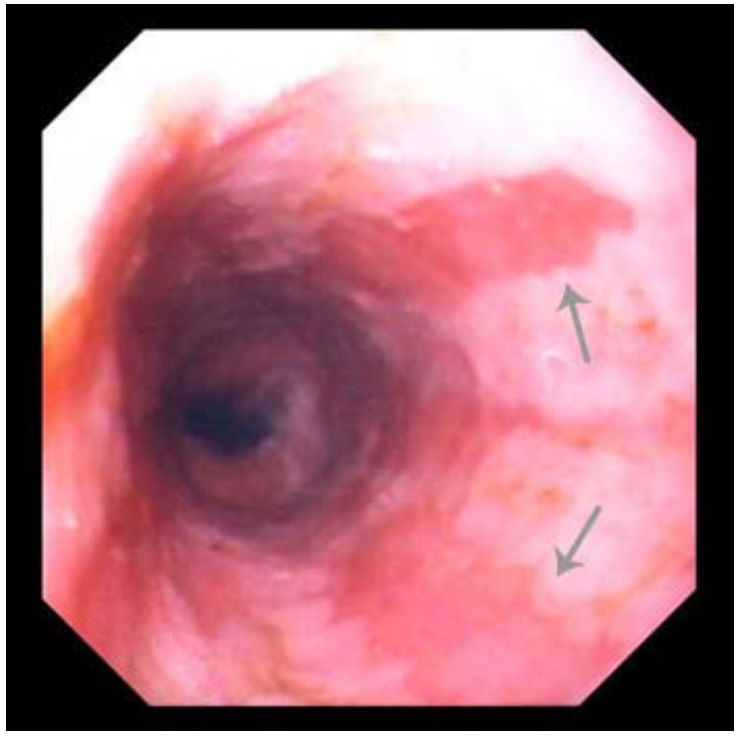
The squamocolumnar junction is proximal to the gastroesophageal junction

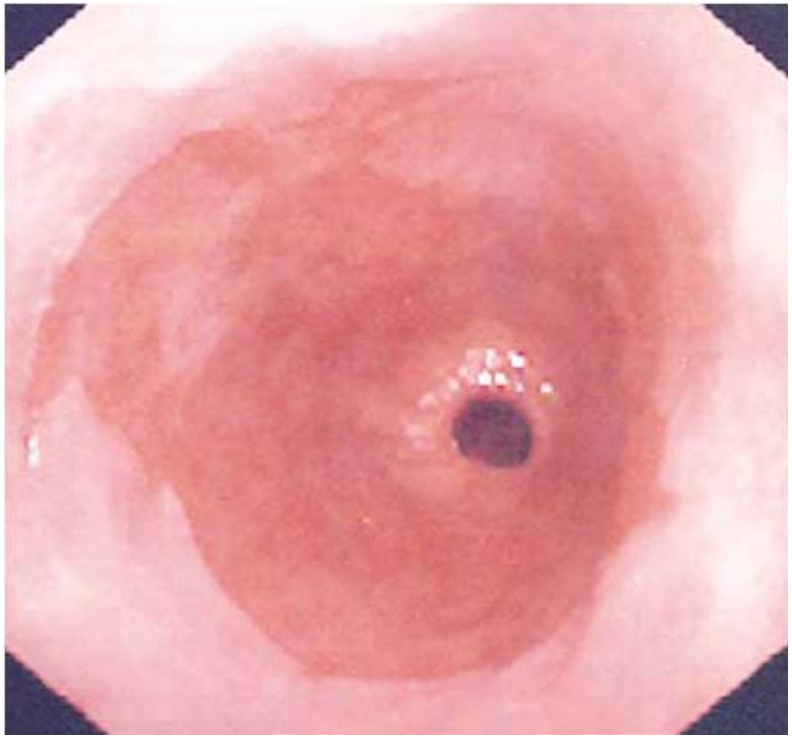


Esophagus: squamous epithelium; Stomach: columnar epithelium

Complications of Gastroesophageal Reflux Disease (GERD)

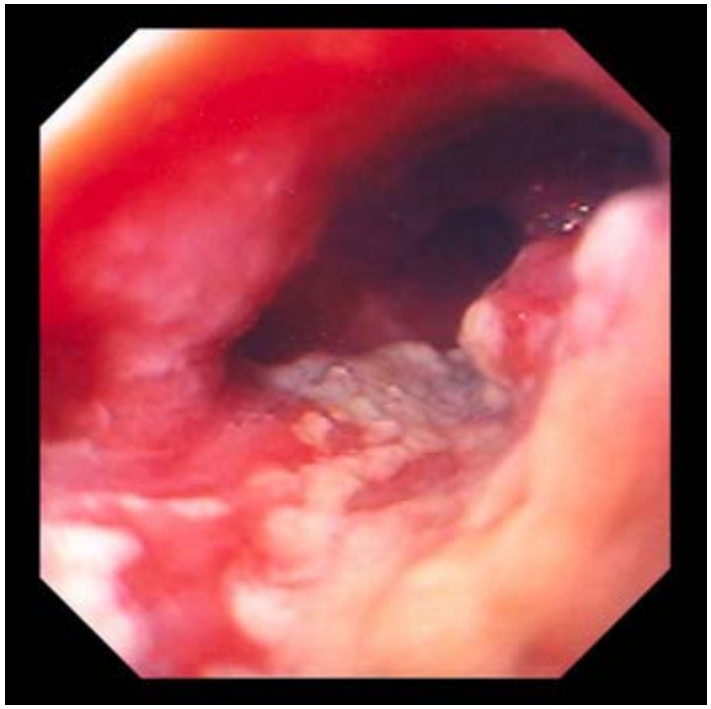
Barrett's esophagus



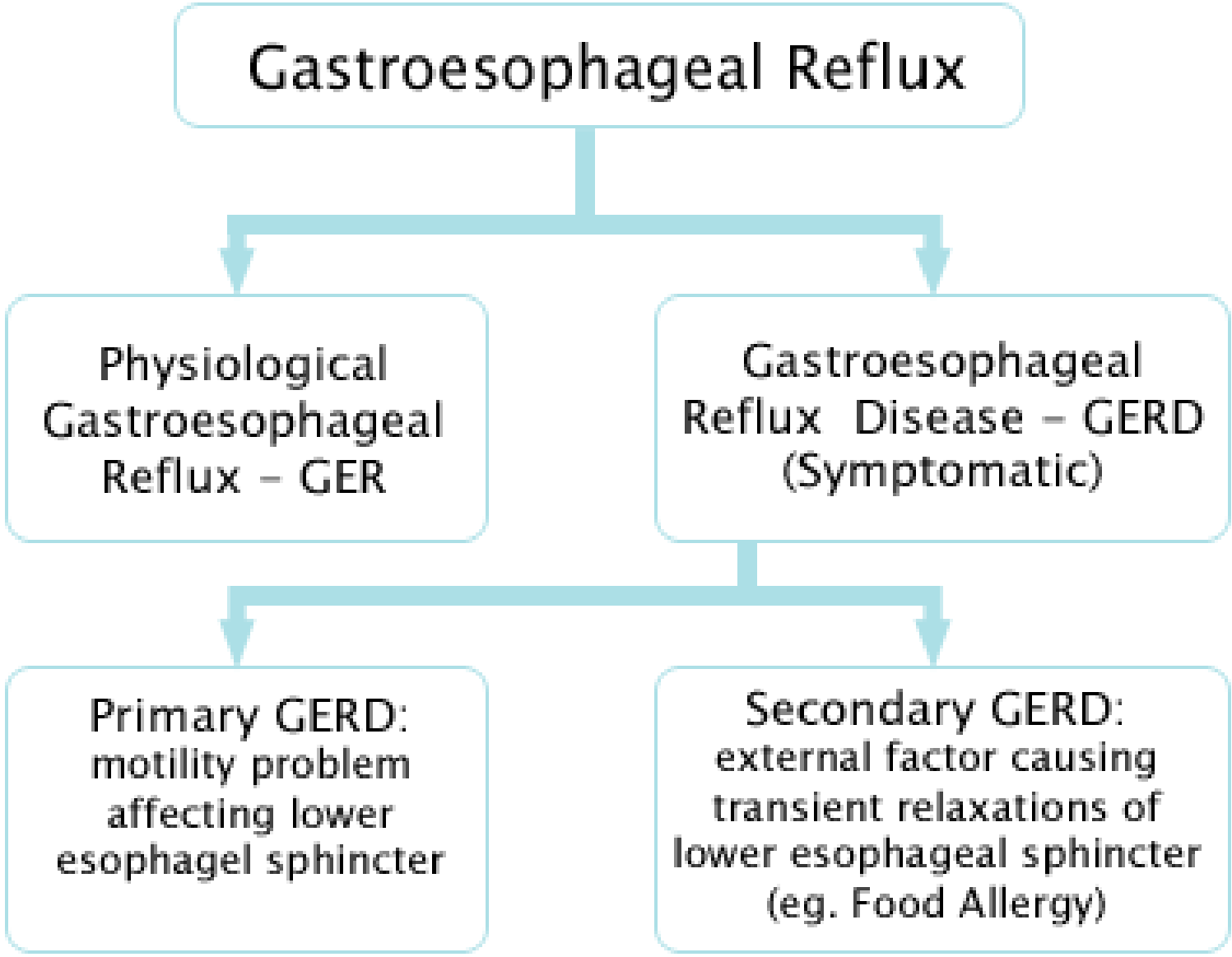


Presence of columnar epithelia in the lower esophagus, replacing the normal squamous cell epithelium = **METAPLASIA**

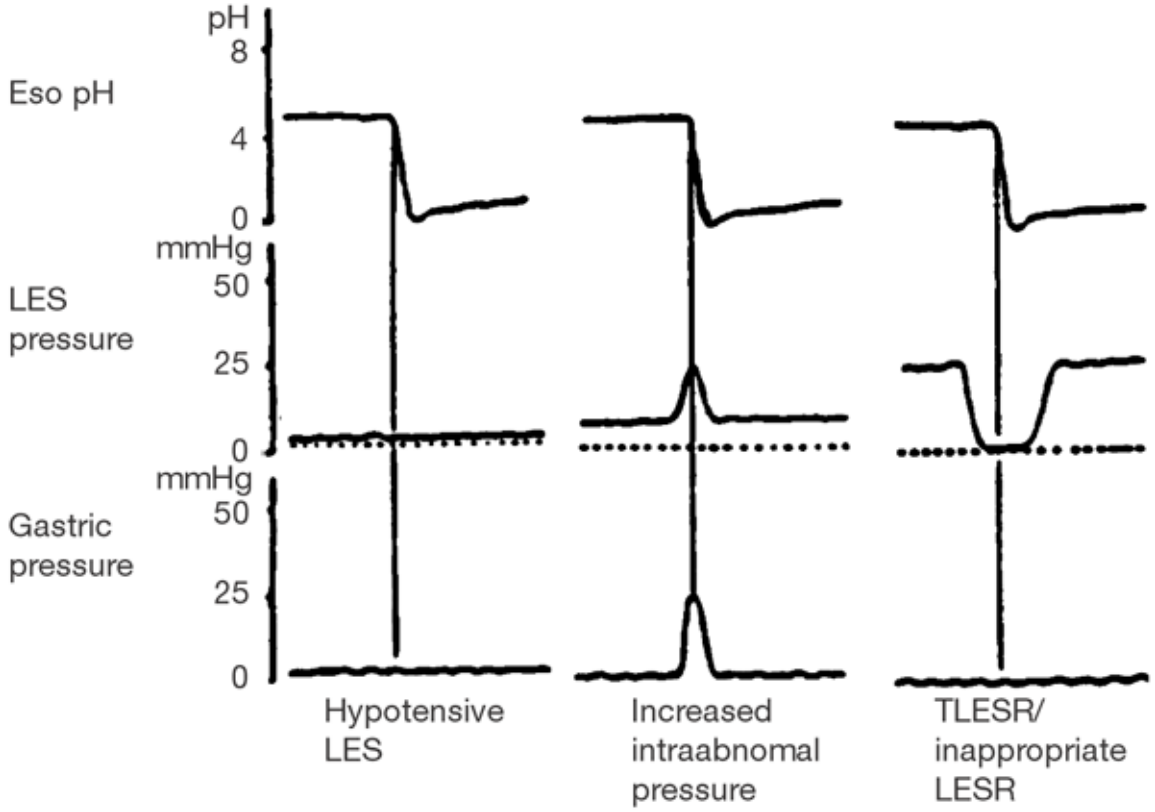
Adenocarcinoma of the esophagus



Nearly all patients with primary adenocarcinoma of the distal esophagus first have **Barrett's esophagus,** which results from chronic gastroesophageal reflux disease and reflux esophagitis.


Gastroesophageal Reflux
Physiological Gastroesophageal Reflux – GER
Gastroesophageal Reflux Disease – GERD (Symptomatic)
Primary GERD: motility problem affecting lower esophagel sphincter
Secondary GERD: external factor causing transient relaxations of lower esophageal sphincter (eg. Food Allergy)

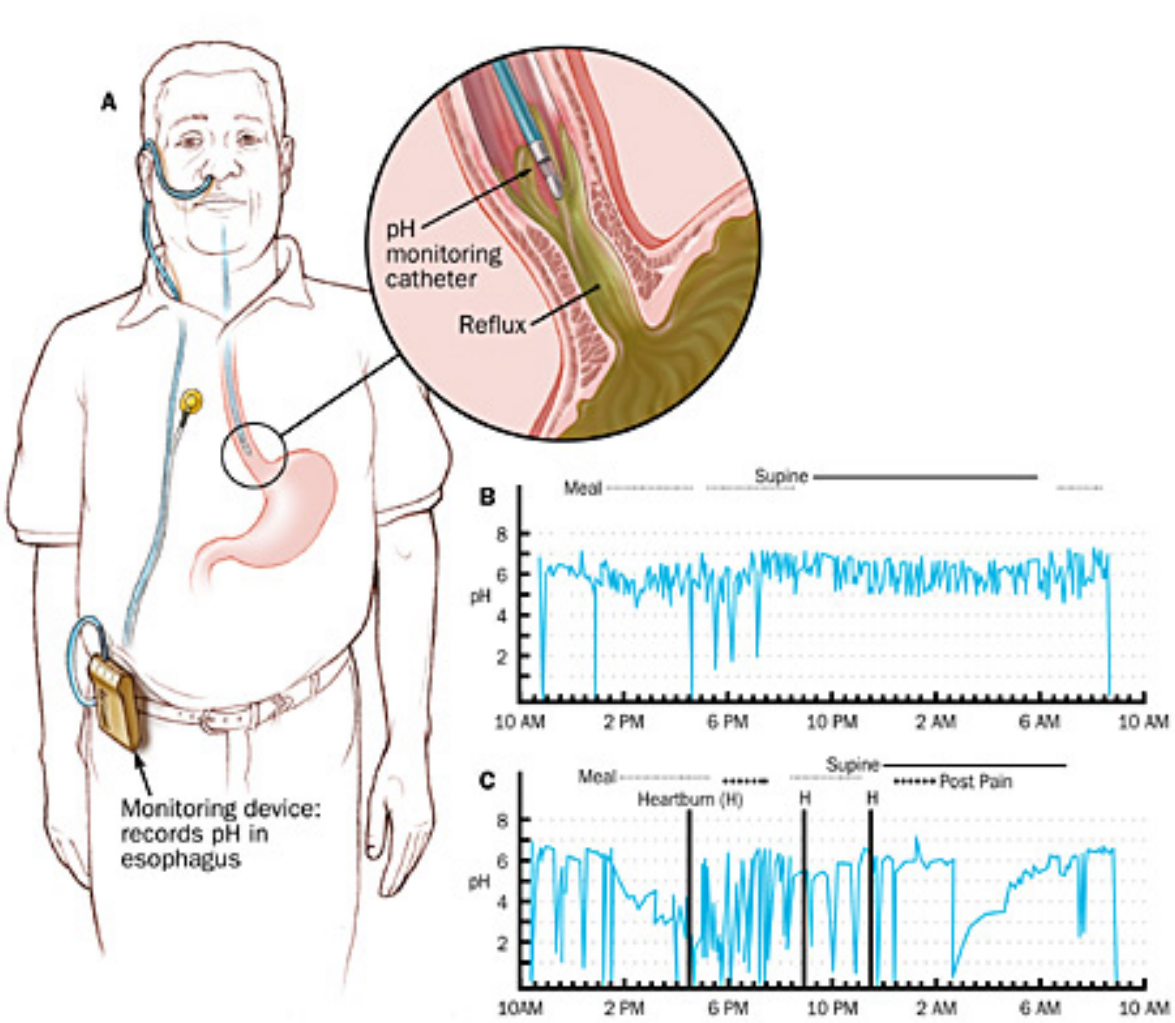
Mechanisms of LES incompetence in gastroesophageal reflux



- Hypotensive LES
- Increased intragastric pressure (e.g. obesity, pregnancy).
- LES may exhibit frequent reflex transient LES relaxation (TLESR) vagovagal inhibitory reflex

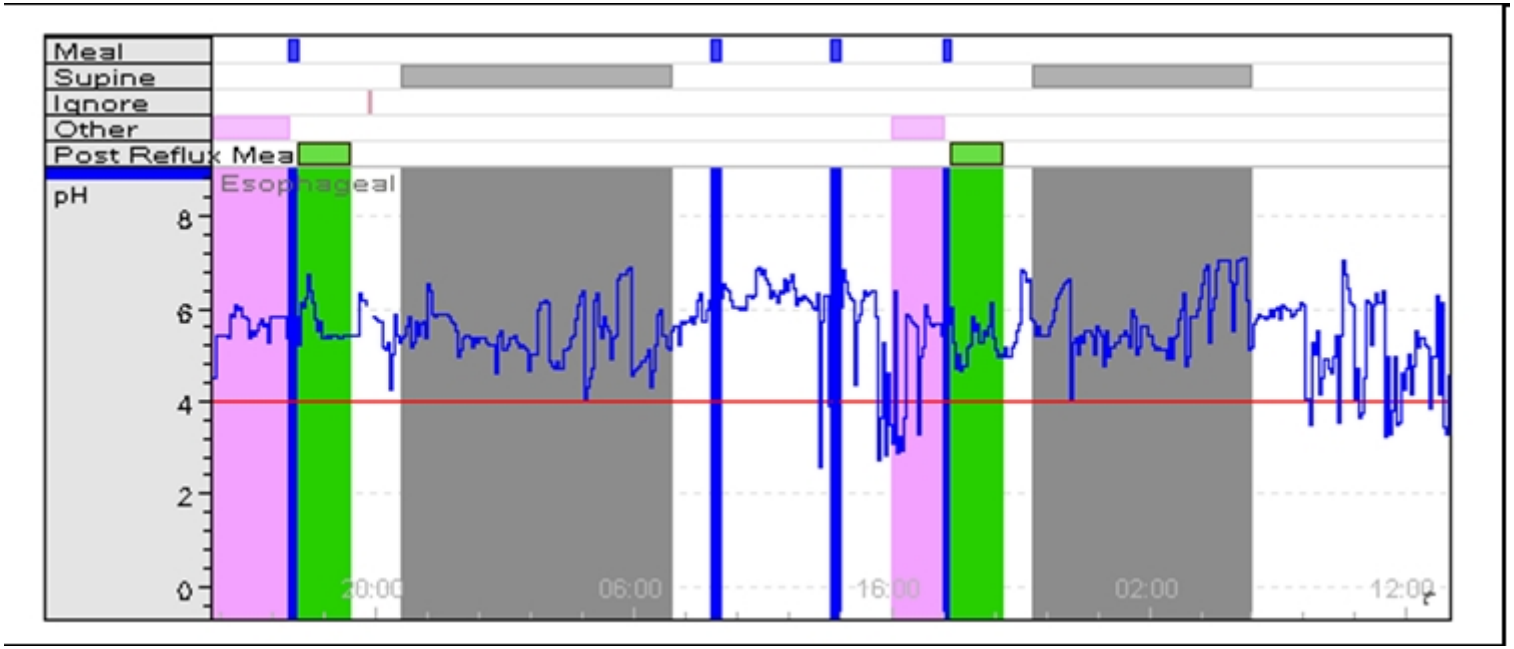
Dodds WJ et al. N. Engl J Med. 1982;307(25):1547–1552

Continuous (24 Hour) pH Monitor



Clinical Evaluation of G-E Reflux

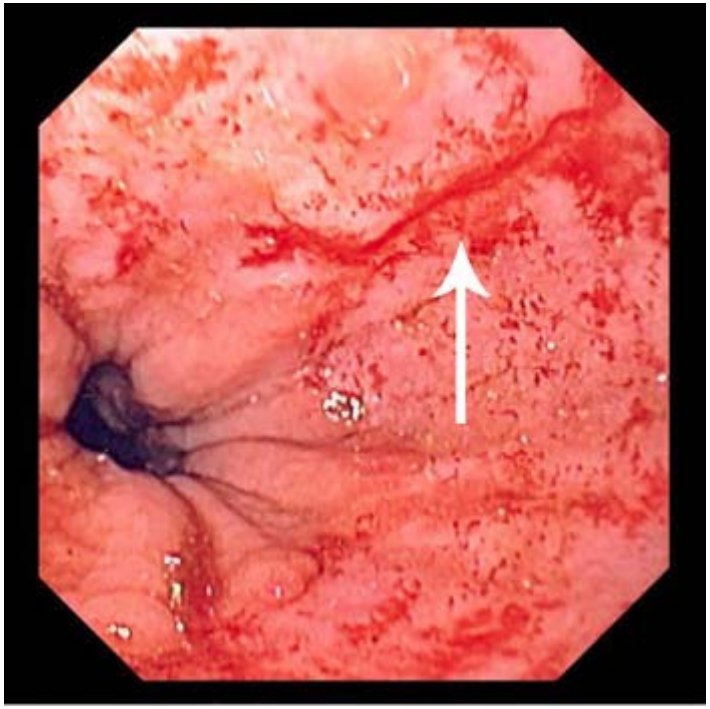
- **Gastro-esophageal reflux is physiological**
- pH monitoring (24h or 48h)
  - normal esophageal pH > 4
  - reflux index (percentage of the total time that the esophageal pH is <4)
    - Should be ~ < 6% of the total time in adults

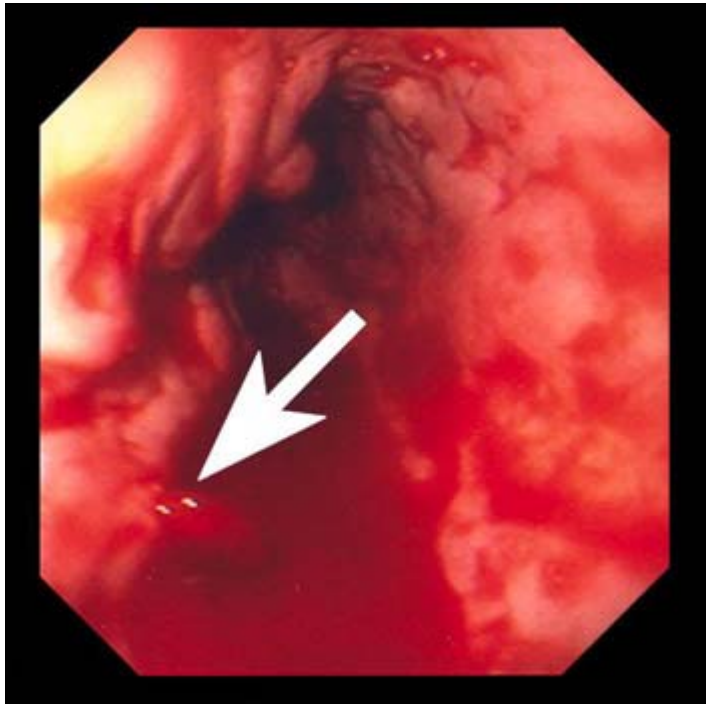


Clinical Evaluation of G-E Reflux

- Acid perfusion (Bernstein) test:
  - Whether the G-E acid reflux cause the pain (heartburn)
  - Perfusing the esophagus with alternating solutions of isotonic saline and 0.1 N hydrochloric acid through a nasogastric tube at a rate of 6-8 mL/min)

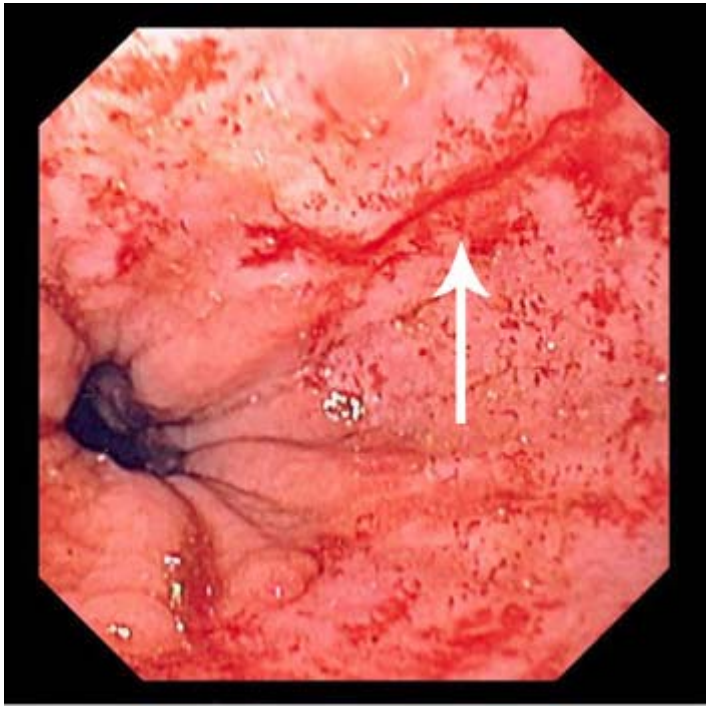
Esophageal bleeding





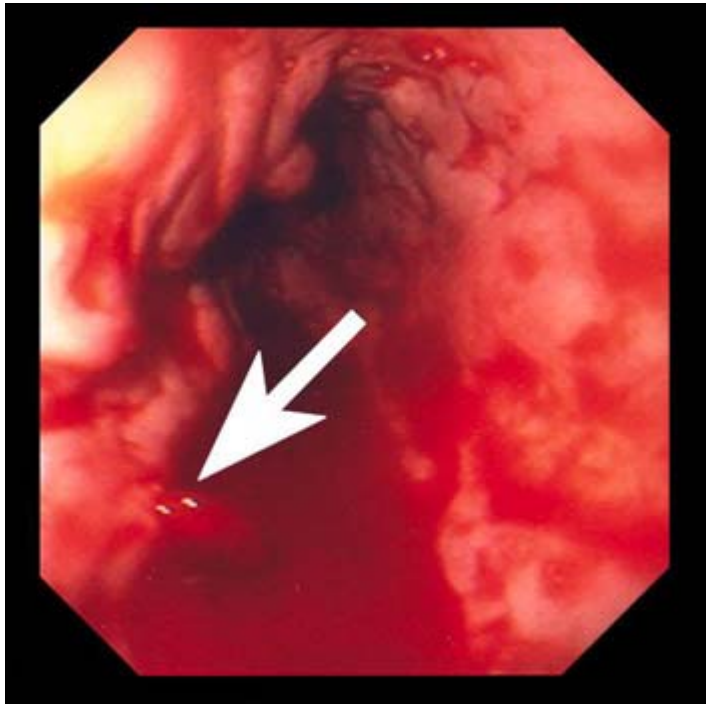
Esophageal bleeding

Mallory-Weiss tear



Laceration of the distal esophagus and proximal stomach during vomiting, retching, or hiccuping

Acute varicose hemorrhage



STOMACH AND DUODENUM

Peptic ulcer disease (PUD)

- 5-10% of population (50% relapses within 5 years after the treatment)
- Pathophysiology of peptic ulcer:
  - Ulcer:
    - mucosal defect reaching under the lamina muscularis mucosae
  - Localization:
    - stomach (malignant in about 5% of cases)
    - duodenum (usually non-malignant)
    - other:
      - esophagus
      - small intestine (gastro-enteroanastomosis or ectopic gastric mucosa in Meckel's diverticle)

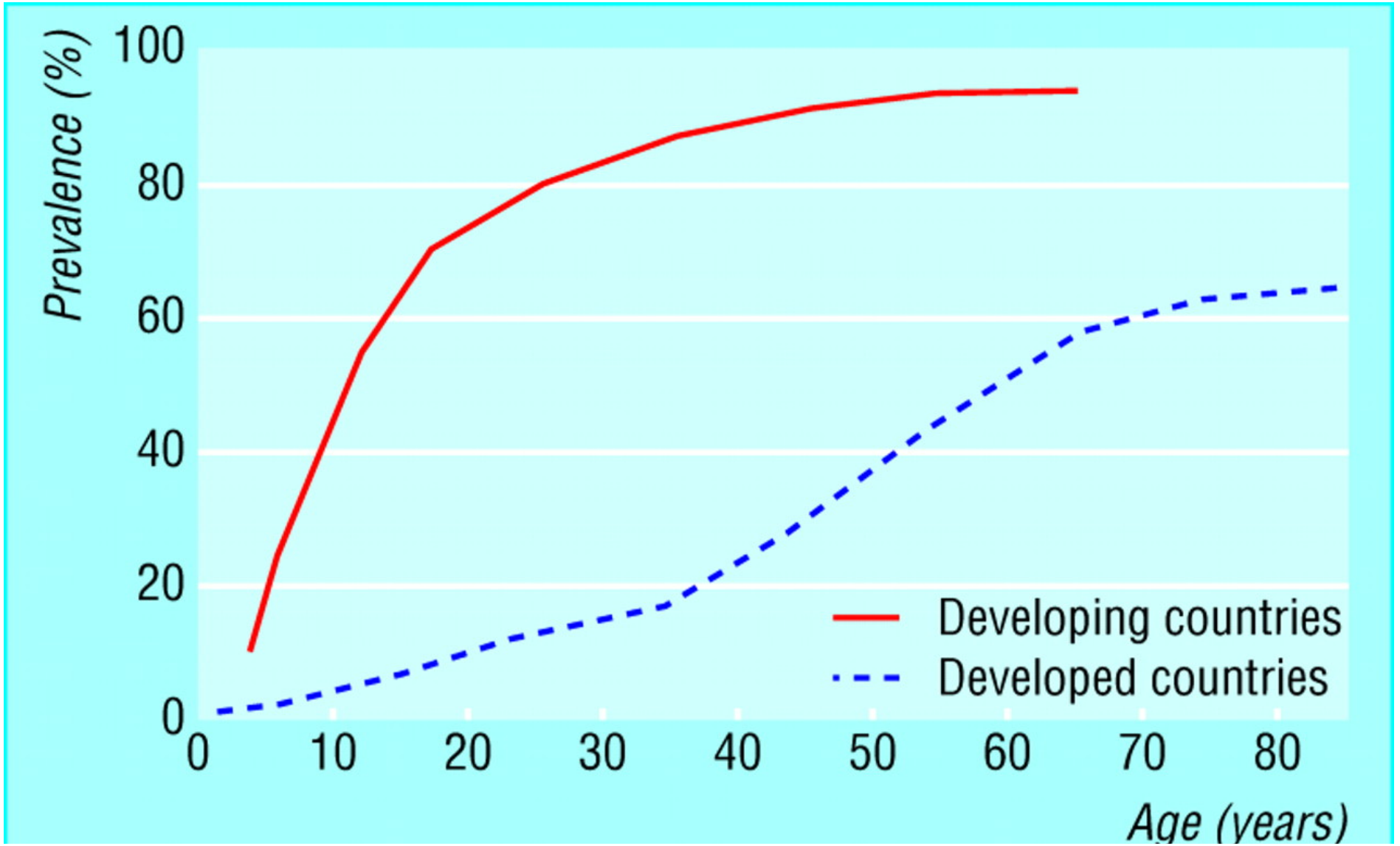
Causes of peptic ulcer disease (PUD)

Etiology:

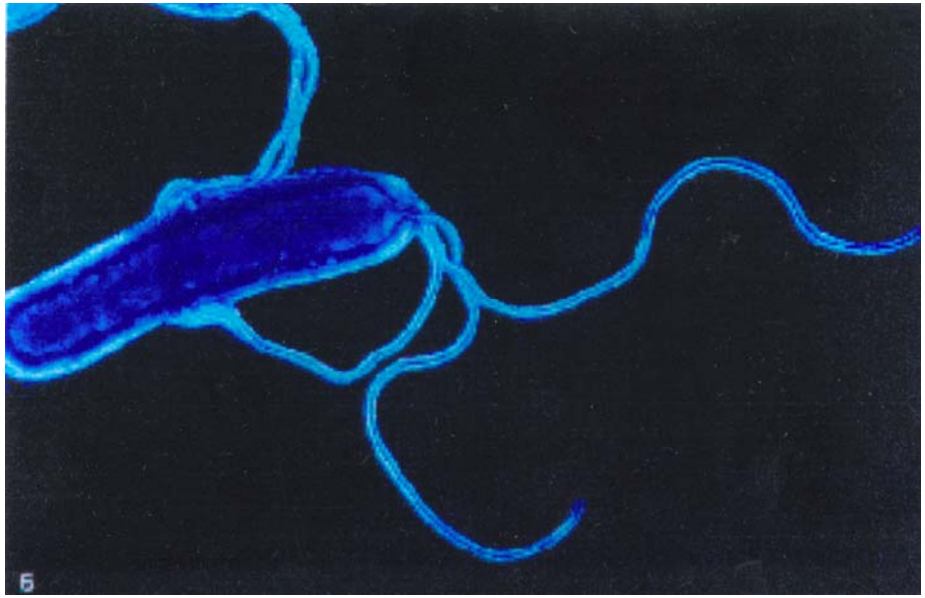
- Helicobacter Pylori (Gr- bacillus, urease production,)
- Drug therapy:
  - corticoids, nonsteroidal anti-inflammatory drugs (NSAIDS)
- Endocrine
  - Zollinger-Ellison sy. (gastrin), hyperparathyreosis
- Stress
- Hepatic failure
  - disordered metabolism and circulation
- Smoking?

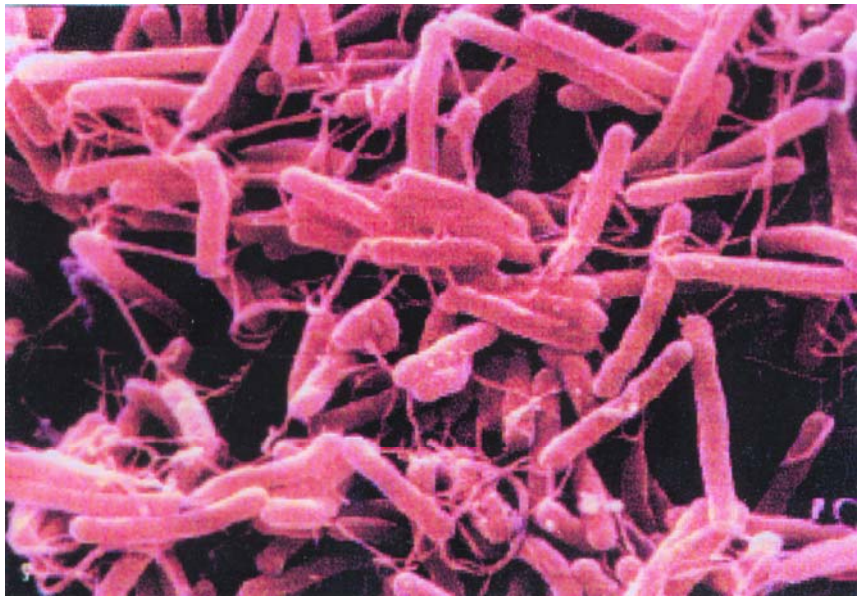
Helicobacter Pylori

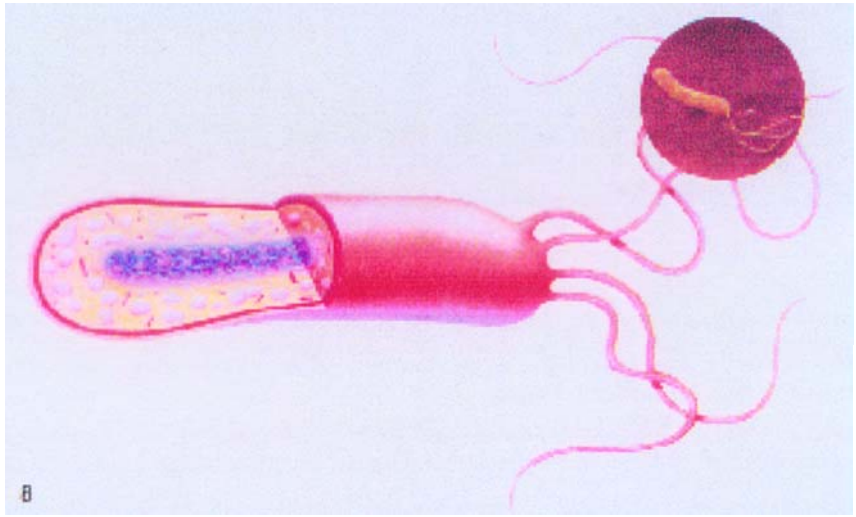
The most common human infection (increase with age)



Helicobacter Pylori



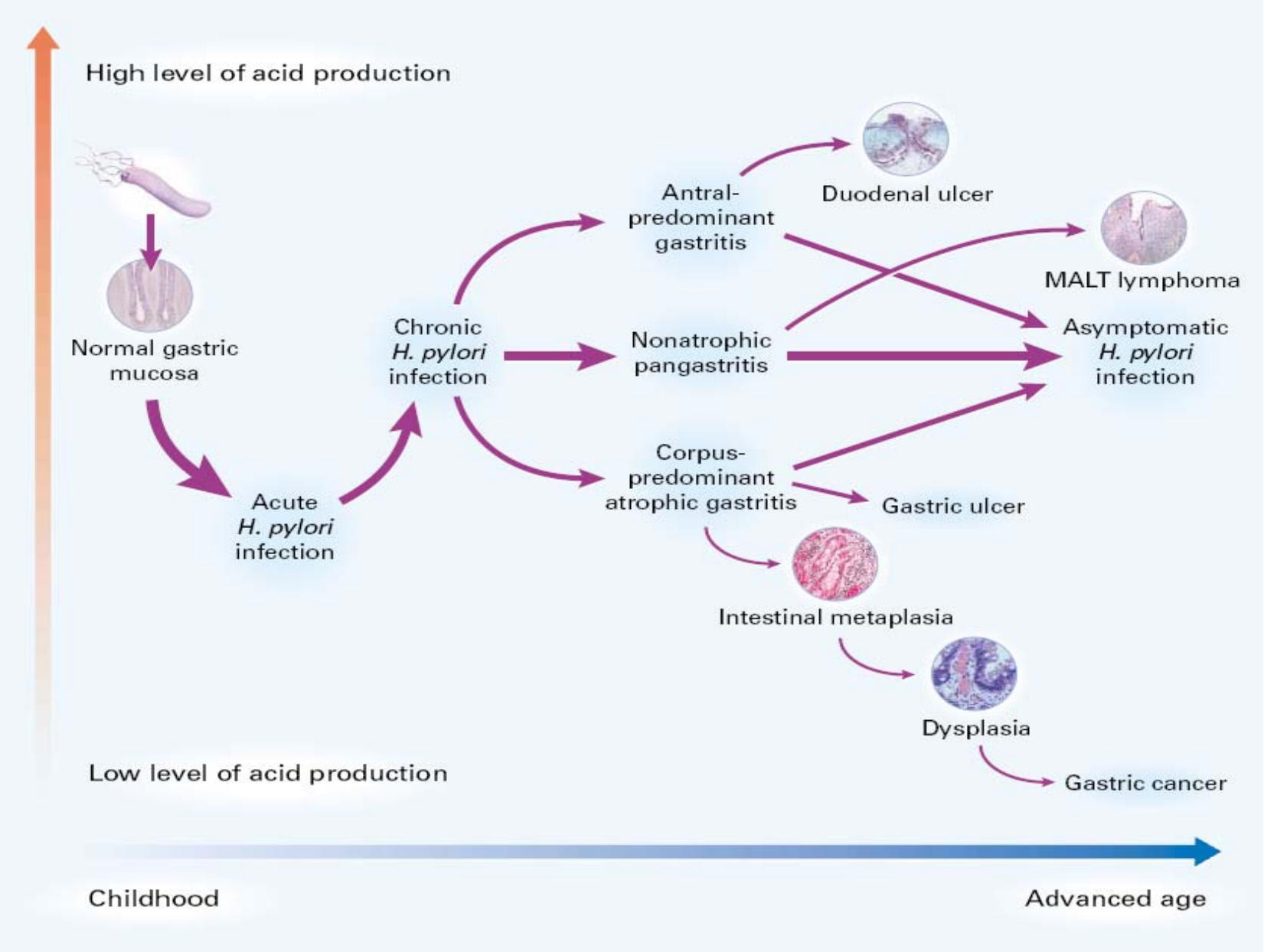




Gram-negative, microaerophilic bacterium

H pylori and acid production?

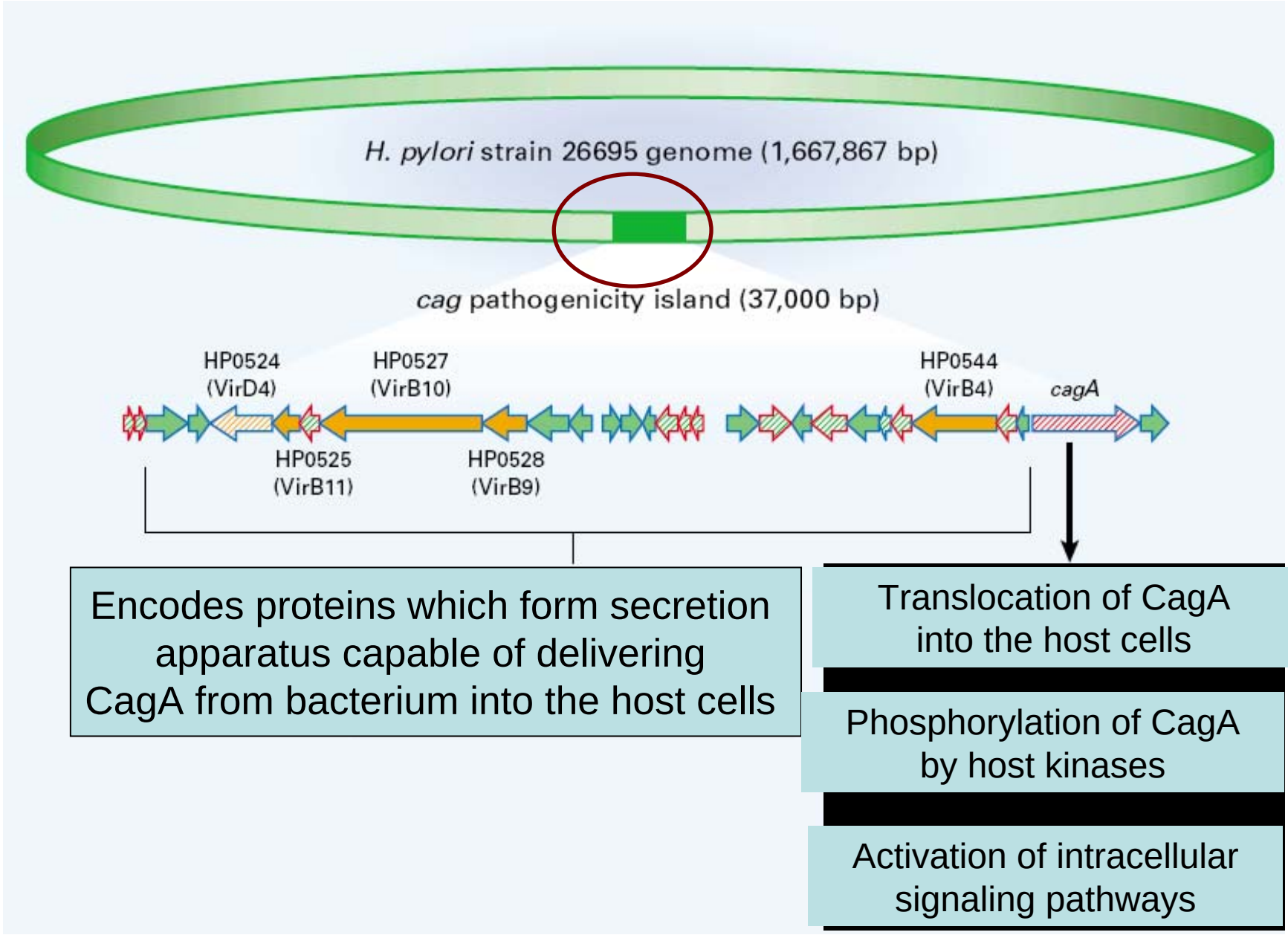
Natural History of *Helicobacter pylori* Infection



Helicobacter Pylori

- ”Discovered” 1982 Warren and Marshall
- Not all infected individuals have disease manifestation (15-20% HP positive have PUD)
- **Bacterial strains that cause ulcers:**
  - have the cagA (cytotoxin associated gene A)

The *cag* Pathogenicity Island



Helicobacter Pylori

- ”Discovered” 1982 Warren and Marshall
- Not all infected individuals have disease manifestation (15-20% HP positive have PUD)
- **Bacterial strains that cause ulcers:**
  - have the cagA (cytotoxin associated gene A)
- **How H. pylori survives in low pH of stomach?**
  - Urease (allow to survive extremely low pH ~ 1.0)
  - Cleaves urea to ammonium (which protects bacteria from HCl) and CO_2

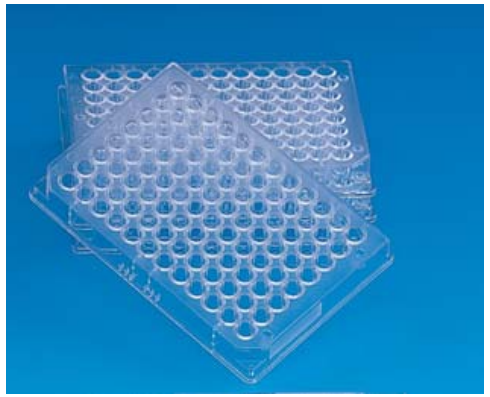
Diagnosis of *H. pylori* infection

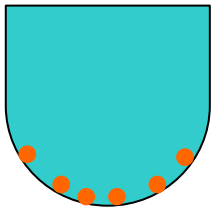
- Noninvasive:
  - serologic testing (serum IgG to H. pylori antigens)
  - breath test with isotype-labeled urea

- Invasive: Endoscopy + biopsy +
  + histological analysis of bioptic material
  + confirmation of urease activity (Clotest)
  + cultivation of H. pylori from the sample
  + PCR detection of H. pylori DNA in the sample

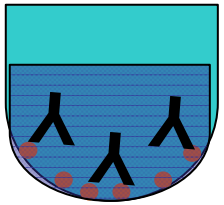
IgG to *H. pylori antigens* (ELISA)

- 96-well ELISA plate

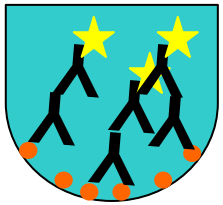




H. pyplori antigen

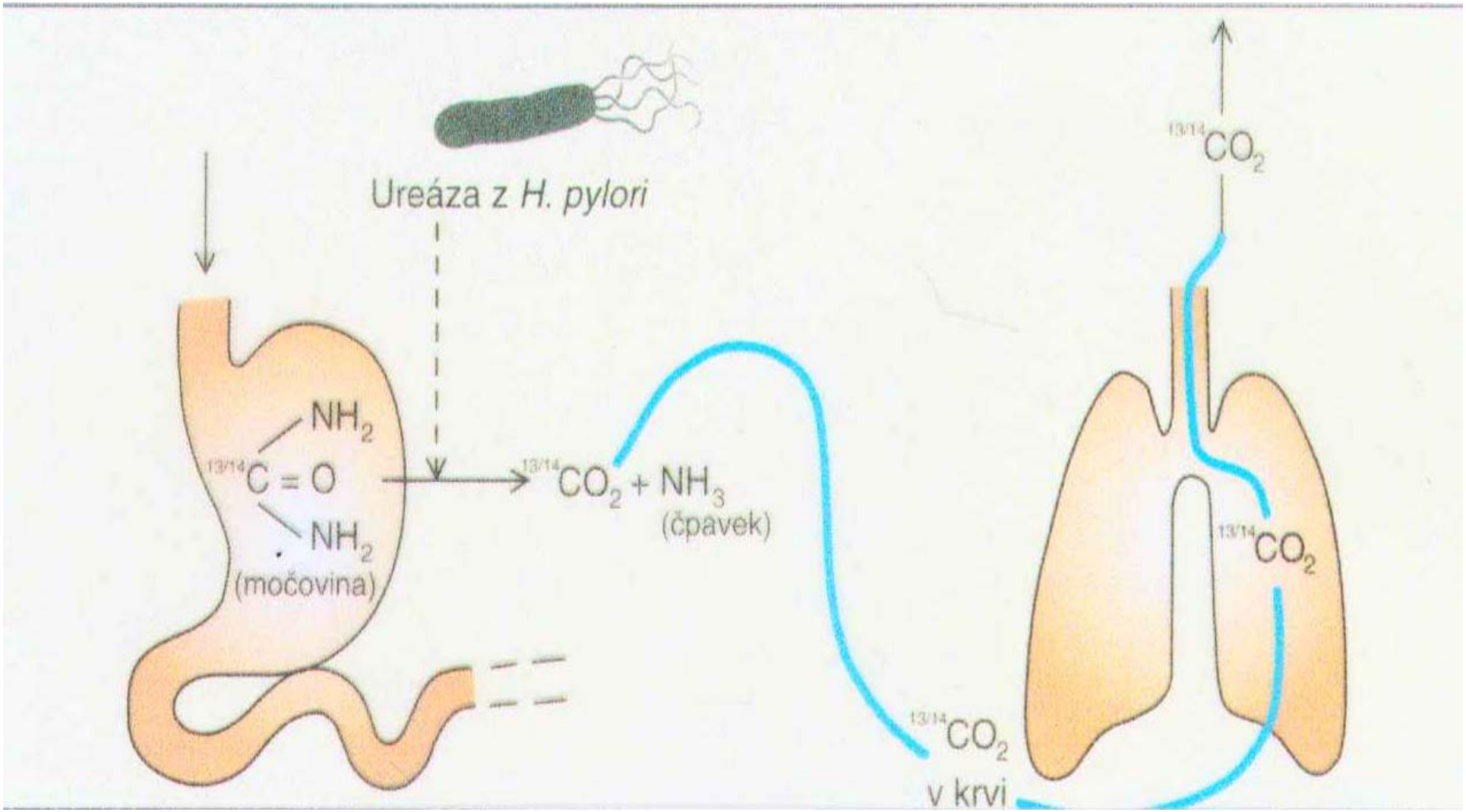


Pacient’s serum/plasma

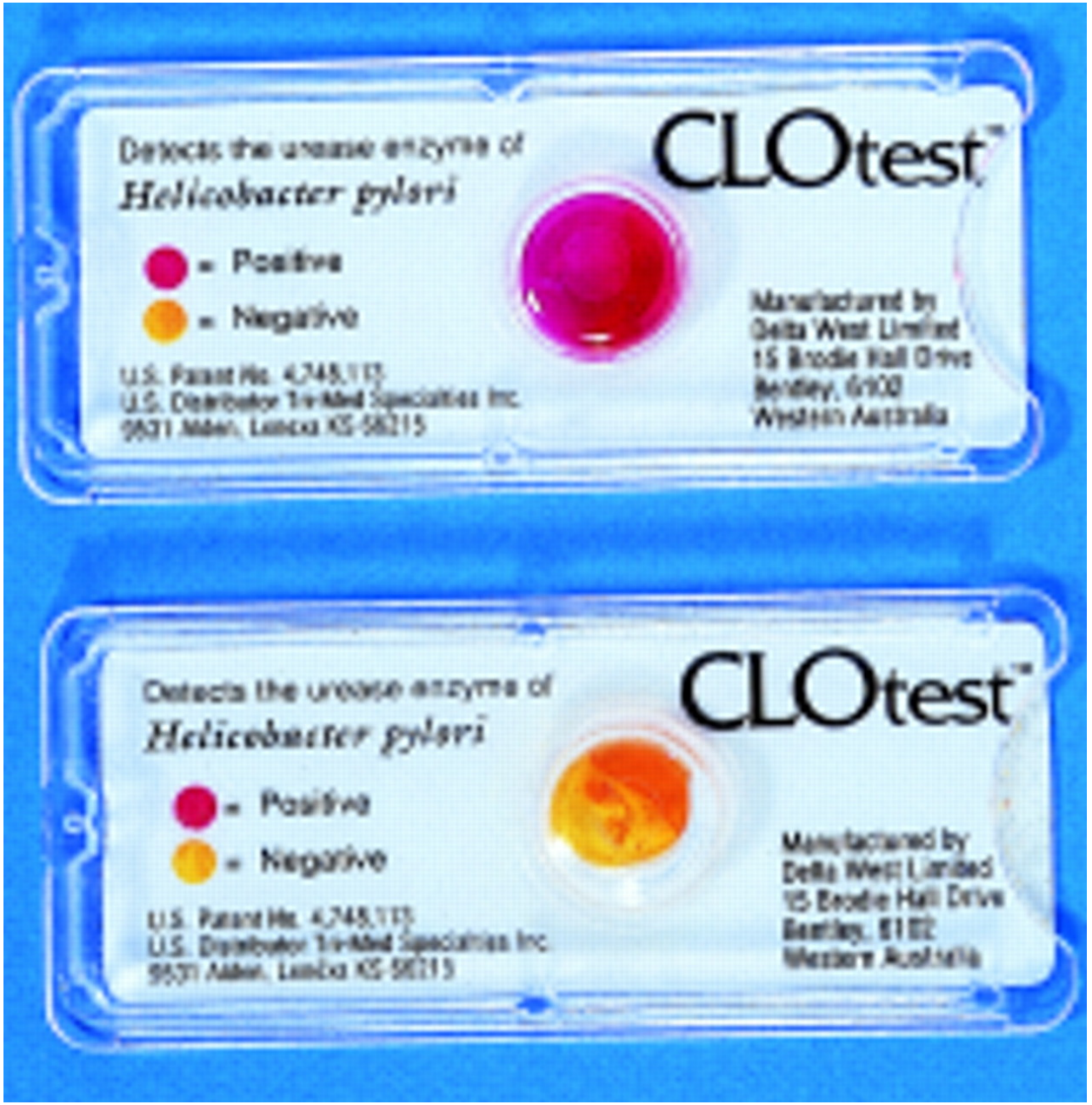


Anti-IgG (Ig) –HRP (AP)

Breath test with isotope-labeled urea (^{13}C or ^{14}C)



Positive and negative results of CLO test for *H pylori*



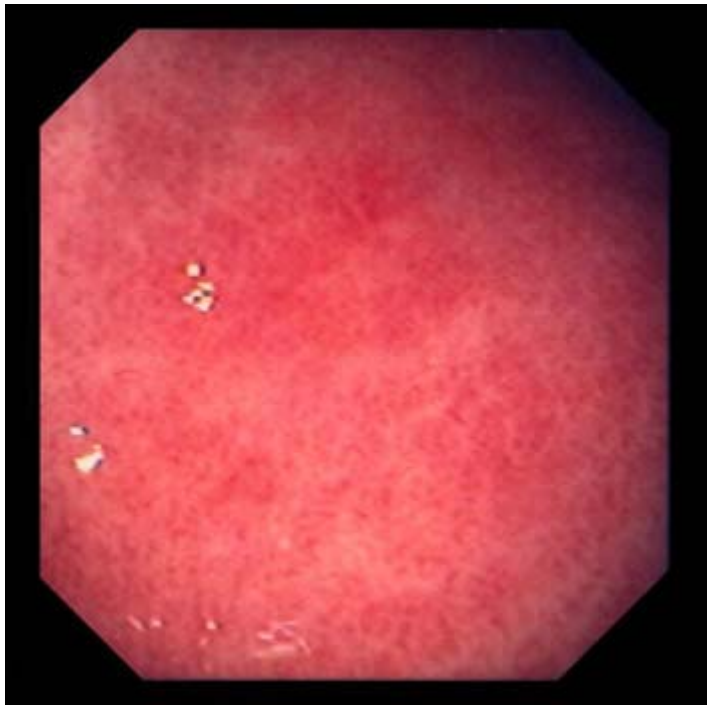
Endoscopic Examination
(gastroscopy, fibroscopy)
(Esophagogastroduodenoscopy = EGD)

- Risk of serious complications 1:800
- Risk of patients death 1:5000

- Direct observation
- Biopsy (followed by histology)
- Therapy
  - Lesions
  - Acute hemorrhage
  - Foreign element ingestion
  - Tumors

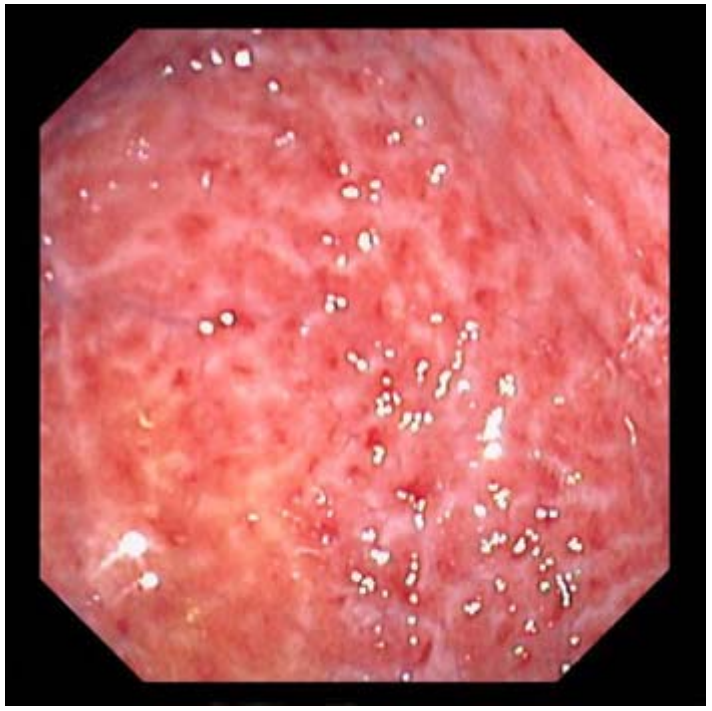
Gastritis

Acute gastritis



Patient tested positive for H. pylori

Chronic gastritis

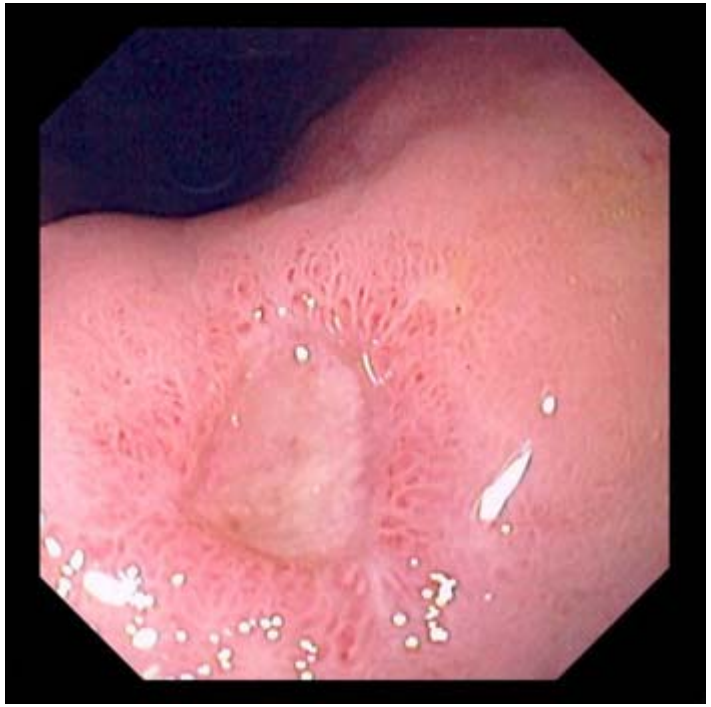


Chronic erosive gastritis may be idiopathic or caused by drugs, Crohn's disease or viral infections.
Helicobacter pylori does not appear to have a major role in the pathogenesis of this condition.

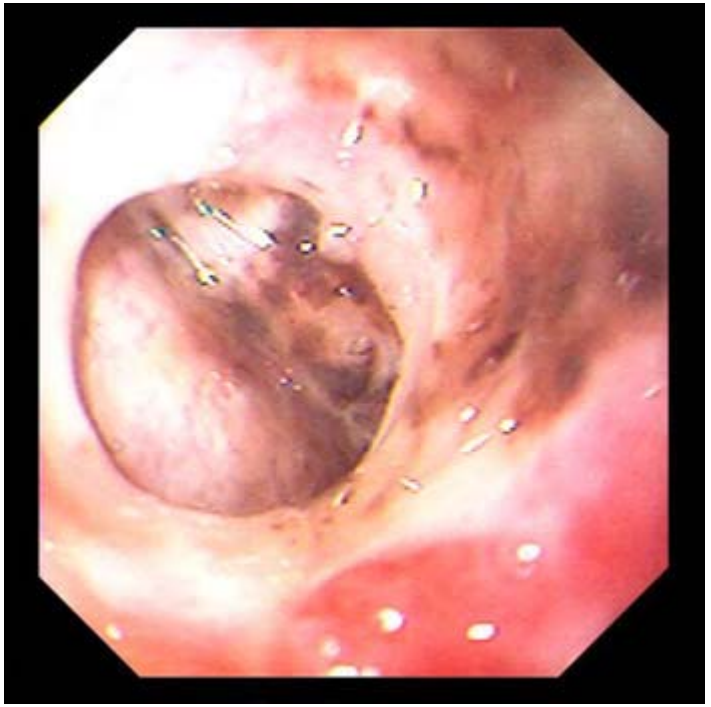
Peptic Ulcer Disease

An excoriated segment of the GI mucosa, typically in the stomach (gastric ulcer) or first few centimeters of the duodenum (duodenal ulcer), which penetrates through the muscularis mucosae

Gastric ulcer

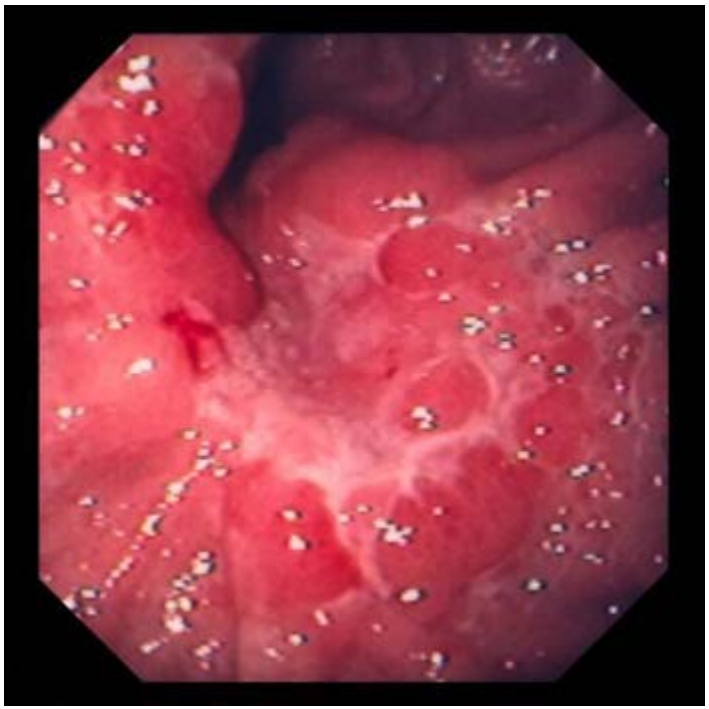


Gastric ulcer (confined perforation)



Gastric Tumors

Gastric adenocarcinoma (signet ring cell type)



Gastric adenocarcinoma (see Plate 34-3) accounts for 95% of malignant tumors of the stomach

Differential diagnosis commonly involves peptic ulcer disease

Endoscopy:
- direct inspection
- biopsy of suspicious areas

Cytology on gastric washings
- together with biopsy improves results.

X-rays
- unreliable in finding small, early lesions (<1 cm in diameter)

SMALL INTESTINE

Resorption Tests

- Direct methods
  - analysis of stool compounds (fat > 6g / day steatorhea ~ malabsorption)
- Indirect methods
  - measurement of the concentrations of p.o. administered compounds in:
    - urine
    - serum

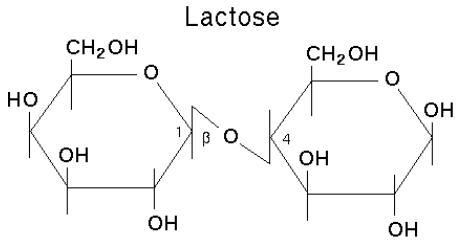
Xylose test

- measurement of xylose in urine or blood after p.o. administration (25 g)
- resorption defects in proximal intestine
- steatorhea, malabsorption sy., Cohn’s disease
- Blood 300 mg/L (2mmol/L) 2 h after p.o. xylose
- Urine >4g in 5h

Schilling test

- Test for pernicious anemia
  - B12 deficiency caused by defect in B12 resorption - intrinsic factor deficiency?
- p.o. administration of radio-labeled vitamin B12 (Co57 or Co58)
- An intramuscular injection of unlabeled vitamin B12
  - to temporarily saturate B12 receptors to prevent radioactive vitamin B12 binding in body tissues
- Measurement of B12 radio-activity in urine or blood

Lactose Intolerance



- The diagnosis may be suspected when chronic or intermittent diarrhea is acidic (pH < 6)
- The lactose tolerance test:
  - Lactose 50 g p.o.
    - Diarrhea with abdominal bloating and discomfort within 20 to 30 min
    - Blood glucose flat curve with no significant peak (peak 1-2 hours)
    - The hydrogen breath test
      - Interval measurement of breath hydrogen by mass spectrometry
  - Small-bowel biopsy
    - lactase activity in a jejunal biopsy specimen confirms the diagnosis

Identification of significant GI tract bleeding

- Technetium-99m labeled erythrocytes:
  - patients with susceptive lower GI bleeding after an exclusion of upper GI bleeding
  - sensitivity ~ 0.1 mL/min

Coeliac disease
Gluten-sensitive enteropathy

- Strong genetic component to coeliac disease ~ 90% of patients carry genes encoding HLA DQ2 and ~ 10% HLA DQ8 haplotype
- Patients with a first degree relative with coeliac disease have a 5-11% chance of being affected
- More common in females than males (1.5-2:1).
- Until the 1980s, coeliac disease was considered a rare condition that usually presented in childhood with symptoms of malabsorption (weight loss, chronic diarrhoea, or failure to thrive)
- Now known to be common, presenting in adulthood usually in the fourth or fifth decade of life with “non-classical” symptoms (irritable bowel syndrome-type symptoms, abdominal pain, altered bowel habit, and anaemia)

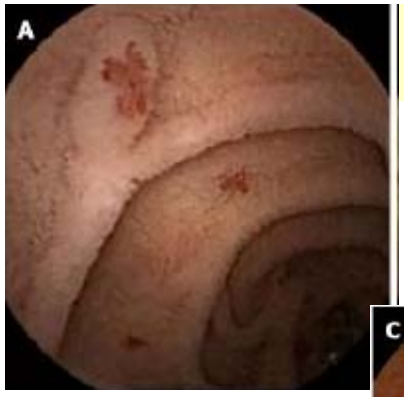
Coeliac disease
Gluten-sensitive enteropathy

- Laboratory testing
  - Antibody testing
    - Immunoglobulin A anti-tissue transglutaminase antibody (IgA TTG)
    - endomysial IgA
    - IgG-deamidated gliadin peptides (esp. children younger than 2 years)
    - genetic testing (to assess the likelihood that celiac sprue is present)
- Endoscopy
  - Capsule endoscopy (CE)
- Biopsy
  - Confirmation of diagnosis
- Clinical testing
  - gluten-free diet

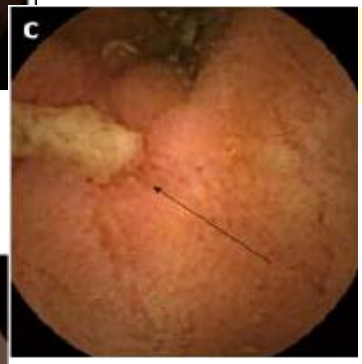
Capsule endoscopy

- A swallowable pill camera
- Introduced in 2000
- Non-invasive means of imaging the, previously difficult to access, small bowel
- Limitations
  - Contraindicated in patients with swallowing disorders (risks of aspiration)
  - Contraindicated in patients with known gastro-intestinal obstruction (capsule retention)
  - Theoretical risk of interference with permanent pacemakers and implantable cardiac defibrillators
  - Time consuming procedure
  - Currently has no biopsy or therapeutic capability

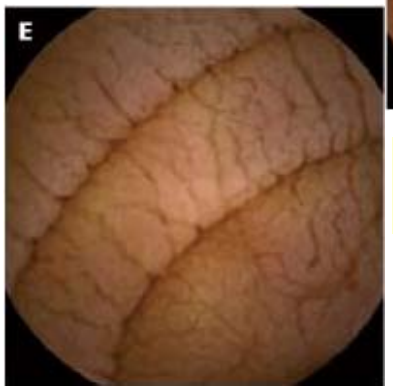
Capsule endoscopy



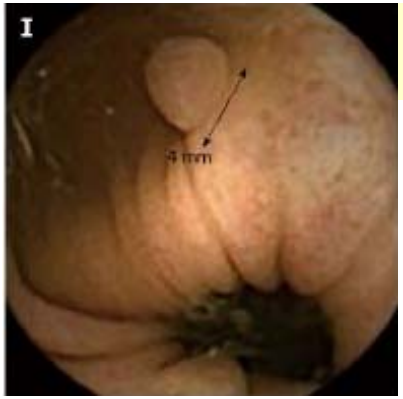
Multiple angioectasia



Ulceration due to Crohn’s disease



Mucosal changes associated with coeliac disease



Colonic polyp

World J Gastroenterol.2014 June 28; 20(24): 7752-7759.

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