

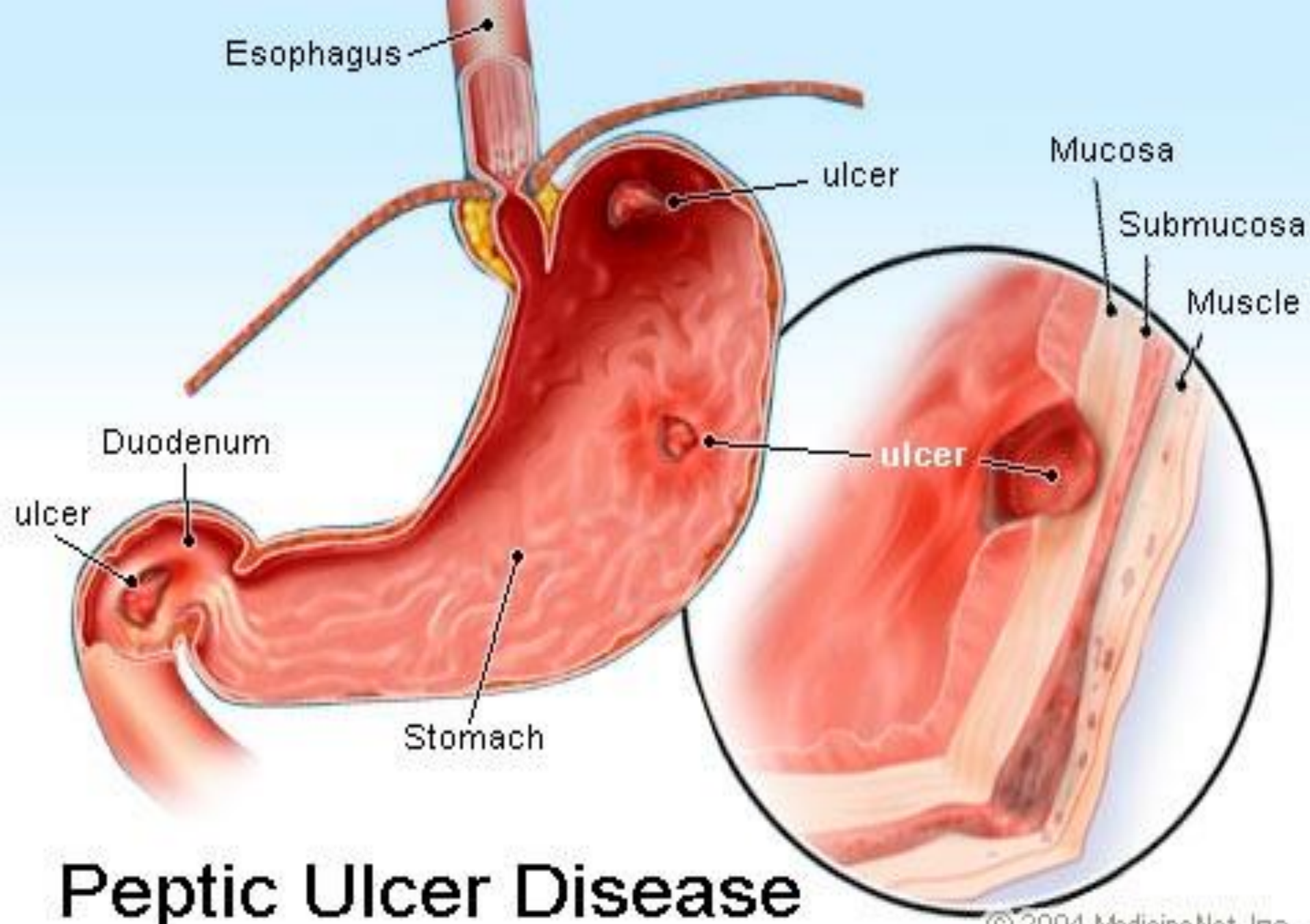
PEPTIC ULCER DISEASE



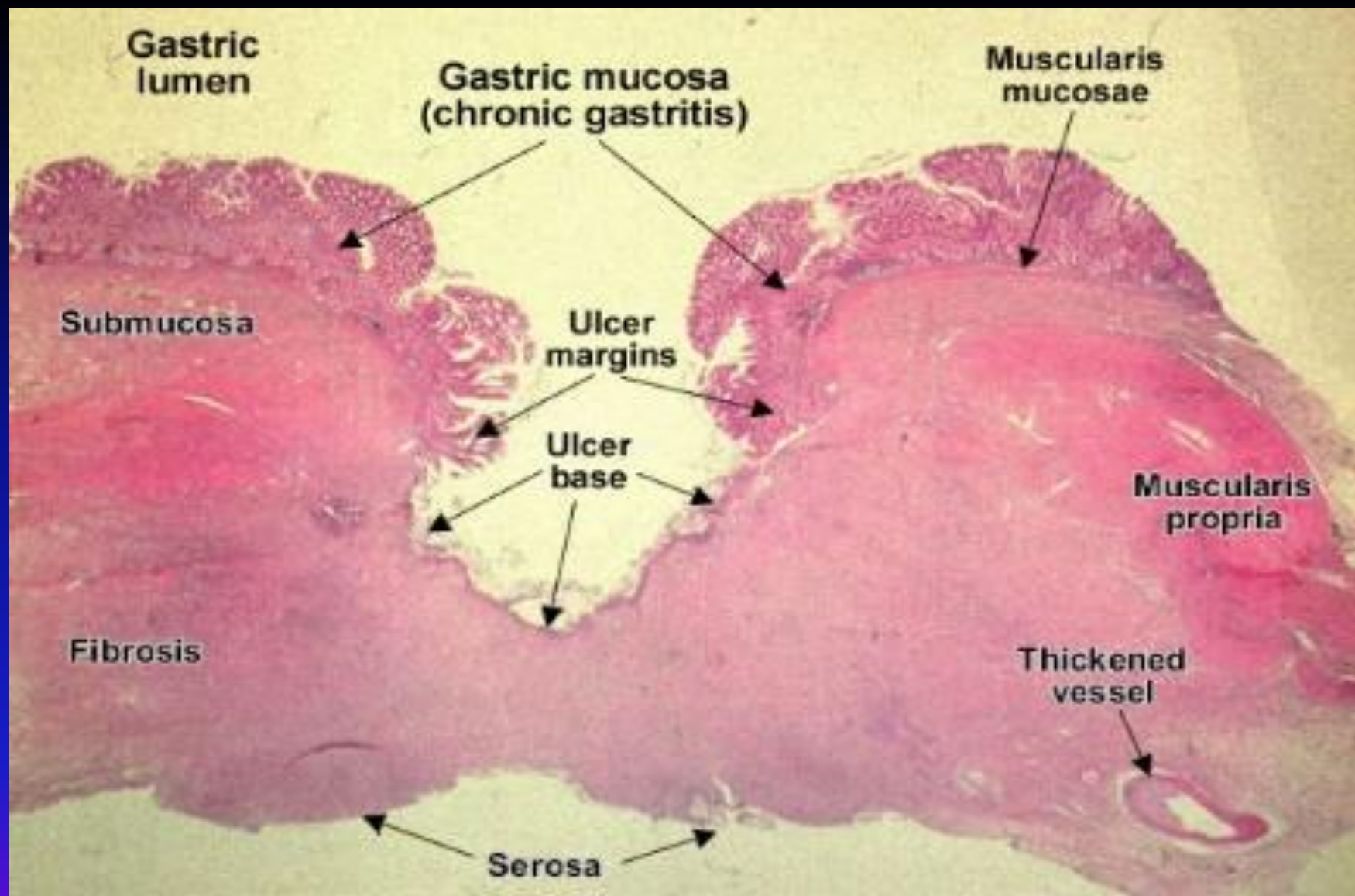
- PhD. assoc. prof. Rodica Bugai

Definition

- A **peptic ulcer** is a defect in the lining of the stomach or the first part of the small intestine, an area called the duodenum.
- A peptic ulcer in the stomach is called a **gastric ulcer**.
- An ulcer in the duodenum is called a **duodenal ulcer**.



Peptic Ulcer Disease



Frequency

- One-year point prevalence is 1.8%.
- Lifetime prevalence is approximately 10%.
- PUD affects approximately 4.5 million people annually.
- Lifetime prevalence is approximately 11-14% for men.
- Lifetime prevalence is approximately 8-11% for women.

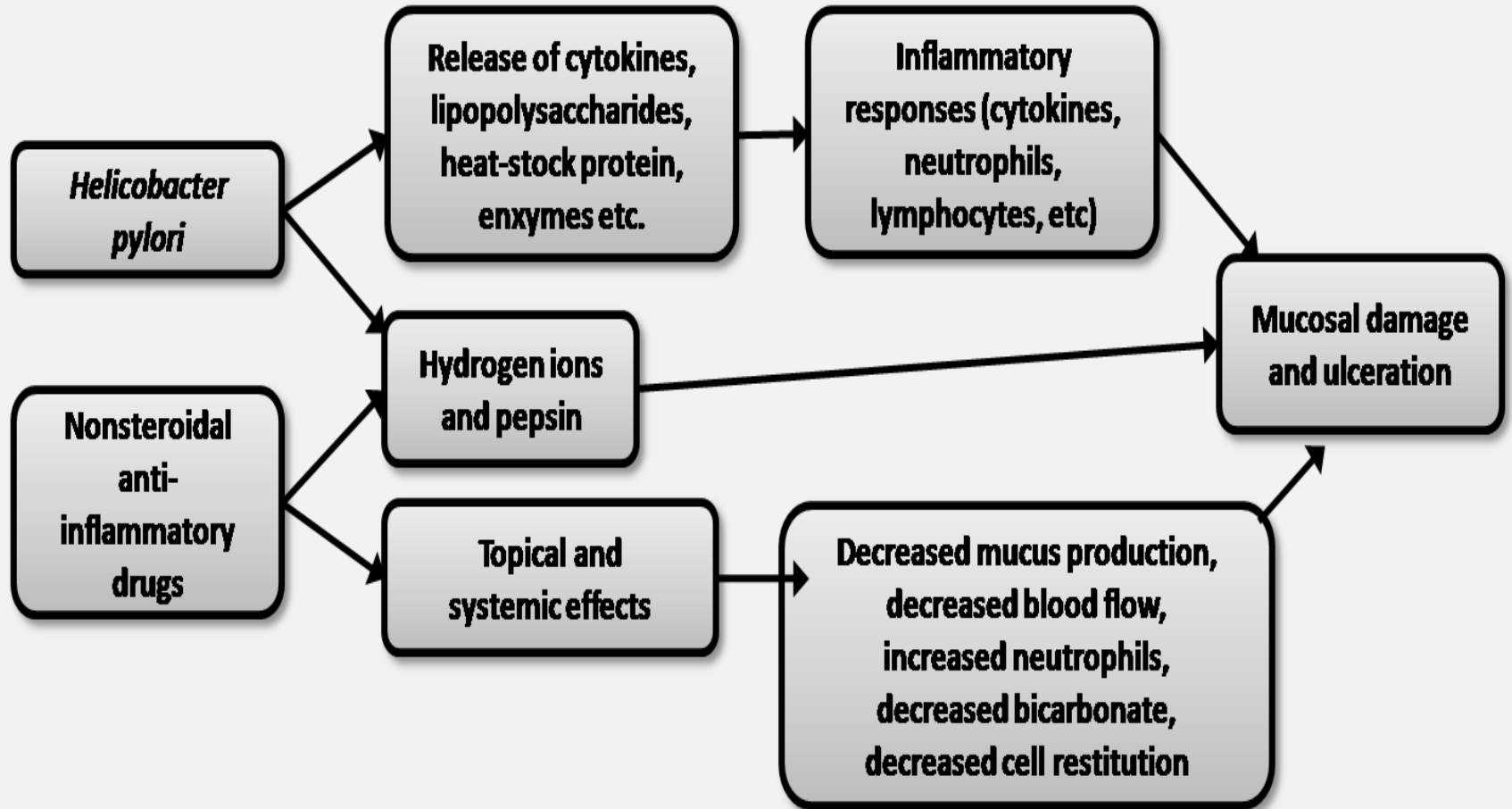
Mortality/Morbidity

- Physician office visits and hospitalizations for PUD have decreased in the last few decades.
- The mortality rate has decreased modestly in the last few decades and is approximately 1 death per 100,000 cases.
- The hospitalization rate is approximately 30 patients per 100,000 cases

Causes

The common mechanism of injury is an imbalance between the **aggressive** and the **defensive factors** that maintain the integrity of the gastric lining (mucosa).

pathogenesis



Peptic Ulcer Disease

NORMAL

Aggressive Forces:

Gastric acidity
Peptic activity



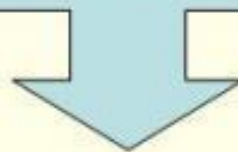
Defensive Forces:

Surface mucus secretion
Bicarbonate secretion into mucus
Mucosal blood flow
Apical surface membrane transport
Epithelial regenerative capacity
Elaboration of prostaglandins

INCREASED AGGRESSION

Aggravating Causes:

H. pylori infection
NSAID, aspirin
Cigarettes, alcohol
Impaired regulation of
acid-pepsin secretion



IMPAIRED DEFENSE



Impaired Defense:

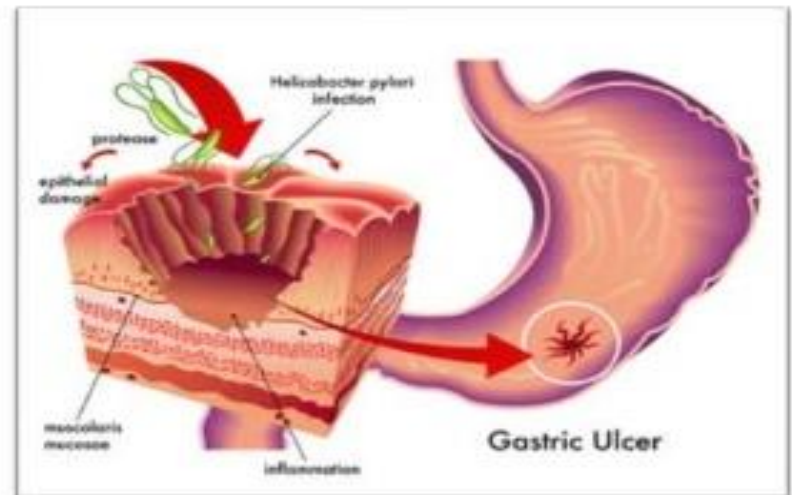
Ischemia, shock
Delayed gastric emptying
Duodenal-gastric reflux:

Causes

- The most common cause of such damage is infection of the stomach by bacteria called *Helicobacter pylori* (*H.pylori*) -Assoc. with as many as 90% of duodenal ulcers and 75% of gastric ulcers
- Yet, many people who have these bacteria in their stomach do not develop an ulcer.

Helicobacter pylori & Ulcer

- *H. pylori* -- causes chronic and indolent inflammation by damaging the mucosal defense system by reducing the thickness of the mucus gel layer, diminishing mucosal blood flow, and interacting with the gastric epithelium throughout all stages of the infection.
- *H. pylori* infection can also increase gastric acid secretion by producing various antigens, virulence factors, and soluble mediators

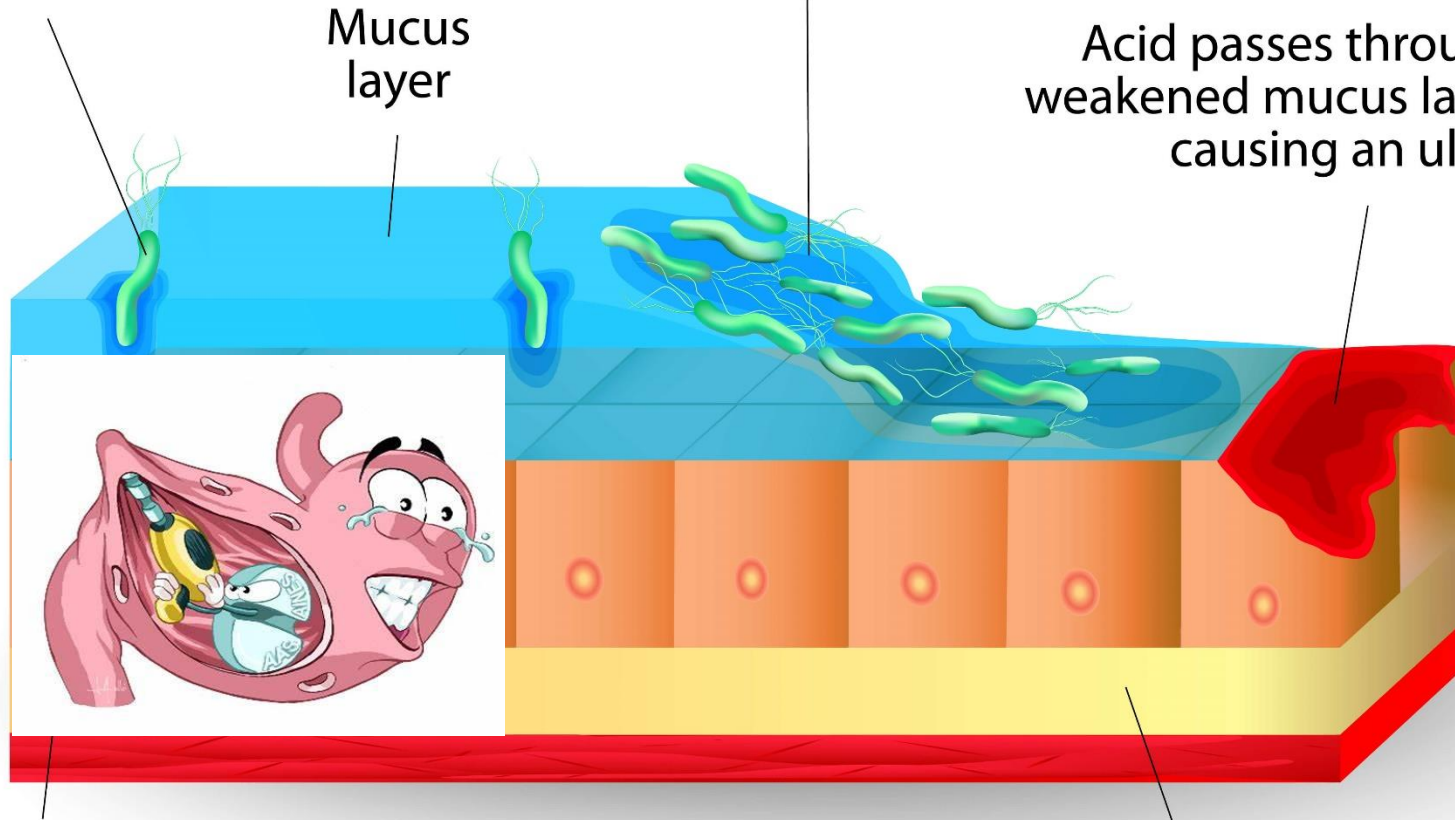


PEPTIC ULCER

Helicobacter pylori
damage protective
mucus layer

The bacteria colonize
the stomach mucosa

Acid passes through
weakened mucus layer
causing an ulcer



Epithelial
cells

Connective
tissue

HELICOBACTER PYLORI INFECTION

COMMON PRECURSOR
OF GASTRITIS AND
PEPTIC ULCERS

RISK FACTOR FOR
GASTRIC CARCINOMA

CURVED
GRAM-NEGATIVE
ROD

ORGANISMS SYNTHESIZE
UREASE, WHICH PRODUCES
AMMONIA THAT DAMAGES
THE GASTRIC MUCOSA

TREATMENT:
AMOXICILLIN,
METRONIDAZOLE,
AND BISMUTH

AMMONIA ALSO
NEUTRALIZES ACID PH,
WHICH ALLOWS THE ORGANISM
TO LIVE IN THE STOMACH

Causes

The following also raise the risk for peptic ulcers:

- Drinking too much alcohol
- Regular use of aspirin, ibuprofen, naproxen, or other nonsteroidal anti-inflammatory drugs (NSAIDs). Taking aspirin or NSAIDs once in a while is safe for most people.
- Smoking cigarettes or chewing tobacco
- Having radiation treatments
- Severe physiologic stress – burns/surgery
- Diseases associated with an increased risk of PUD include cirrhosis, chronic pulmonary disease, renal failure, and renal transplantation.

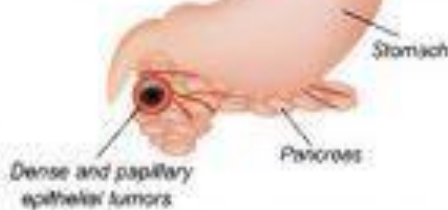
Causes

- A rare condition called [Zollinger-Ellison syndrome](#) causes stomach and duodenal ulcers. Persons with this disease have a tumor in the pancreas. This tumor releases high levels of a hormone that increases stomach acid.

HELICOBACTER
PYLORI BACTERIAL
INFECTION



NONSTEROIDAL
ANTI-INFLAMMATORY
DRUGS



ZOLLINGER-ELLISON
SYNDROME

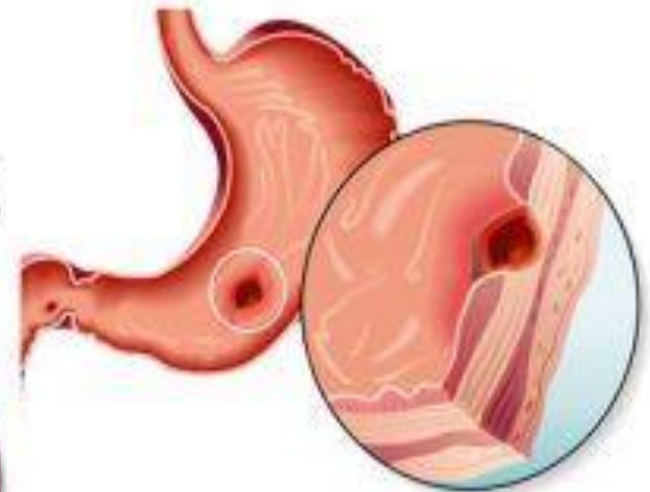
CAUSES
THAT LEAD TO
**PEPTIC
ULCERS**



ALCOHOLIC
BEVERAGES



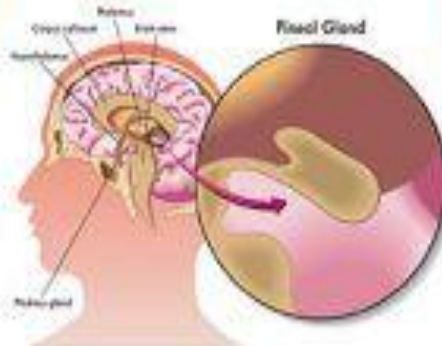
HYPERCALCEMIA



FAMILY HISTORY



EXCESS
STRESS



LOW LEVELS
OF MELATONIN



Symptoms

An ulcer may or may not have symptoms.

When symptoms occur, they include:

- A gnawing or burning pain in the middle or upper stomach between meals or at night
- Bloating
- Heartburn
- Nausea or vomiting

In severe cases, symptoms can include:

- Dark or black stool (due to bleeding)- melena
- Vomiting blood (can have a "coffee-grounds" appearance)
- Weight loss

Symptoms

- **Duodenal ulcer** — "Classic" symptoms of a duodenal ulcer include **burning, gnawing, aching, or hunger-like pain**, primarily in the upper middle region of the abdomen below the breastbone (the epigastric region). **Pain may occur or worsen when the stomach is empty, usually two to five hours after a meal. Symptoms may occur at night between 11 PM and 2 AM**, when acid secretion tends to be greatest.
- **Gastric ulcer** — Symptoms of a gastric ulcer typically include **pain soon after eating**. Symptoms are sometimes not relieved by eating or taking antacids.

Stomach Ulcer Symptoms



Burning



Fatigue



Heartburn



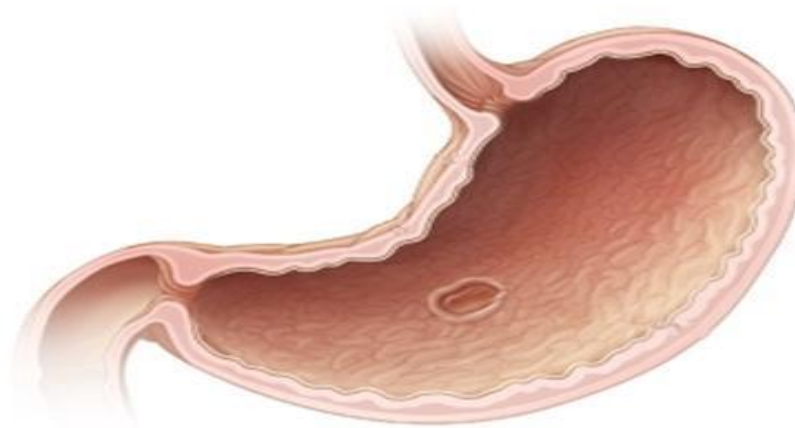
Dull pain



Loss of appetite



Weight loss



Vomiting



Burping



Bloating



Nausea

PEPTIC ULCER DIAGNOSIS

Not everyone with ulcer symptoms has an ulcer.

Similar symptoms can be caused by a wide variety of conditions such as functional dyspepsia (ie, the presence of ulcer-symptoms without a specific cause), abnormal emptying of the stomach, acid reflux, gallbladder problems, and, much less commonly, stomach cancer.

Thus, the process needed to diagnose an ulcer depends upon the person's medical history and sometimes, use of specific tests.

PEPTIC ULCER DISEASE (PUD)



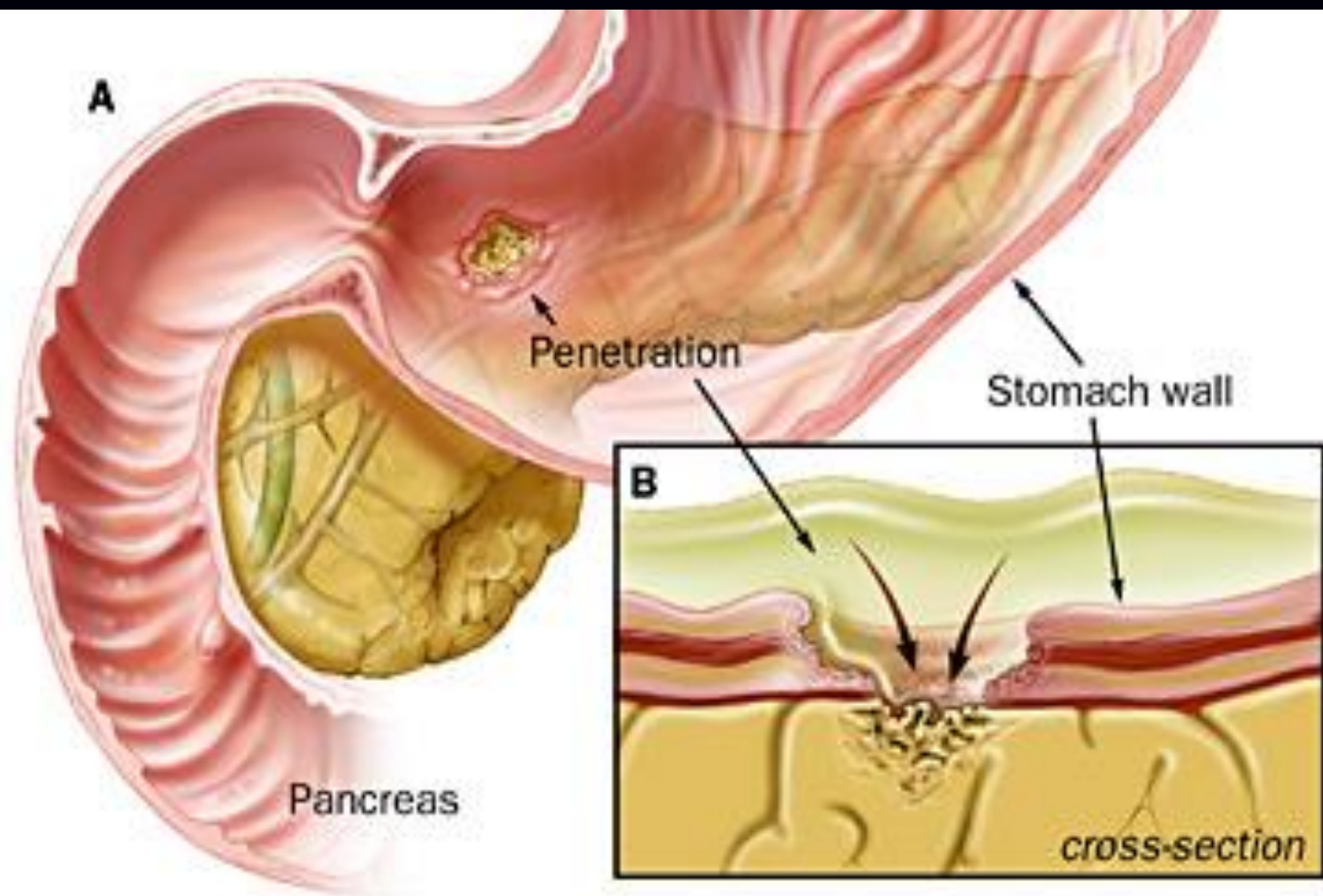
Physical

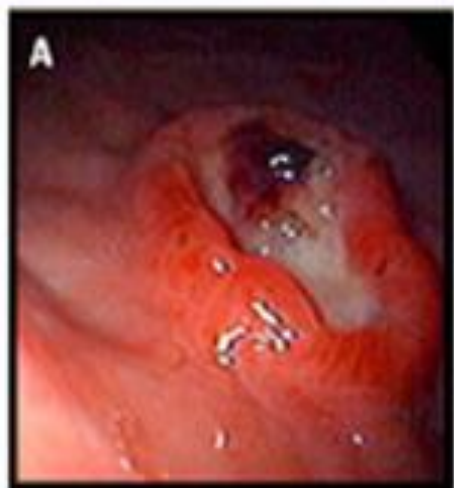
- Epigastric tenderness,
- Guaiac-positive stool resulting from occult blood loss or melena.

Complications

- Bleeding
- Perforation (a hole through the wall of the stomach)
- Gastric outlet obstruction from swelling or scarring that blocks the passageway leading from the stomach to the small intestine

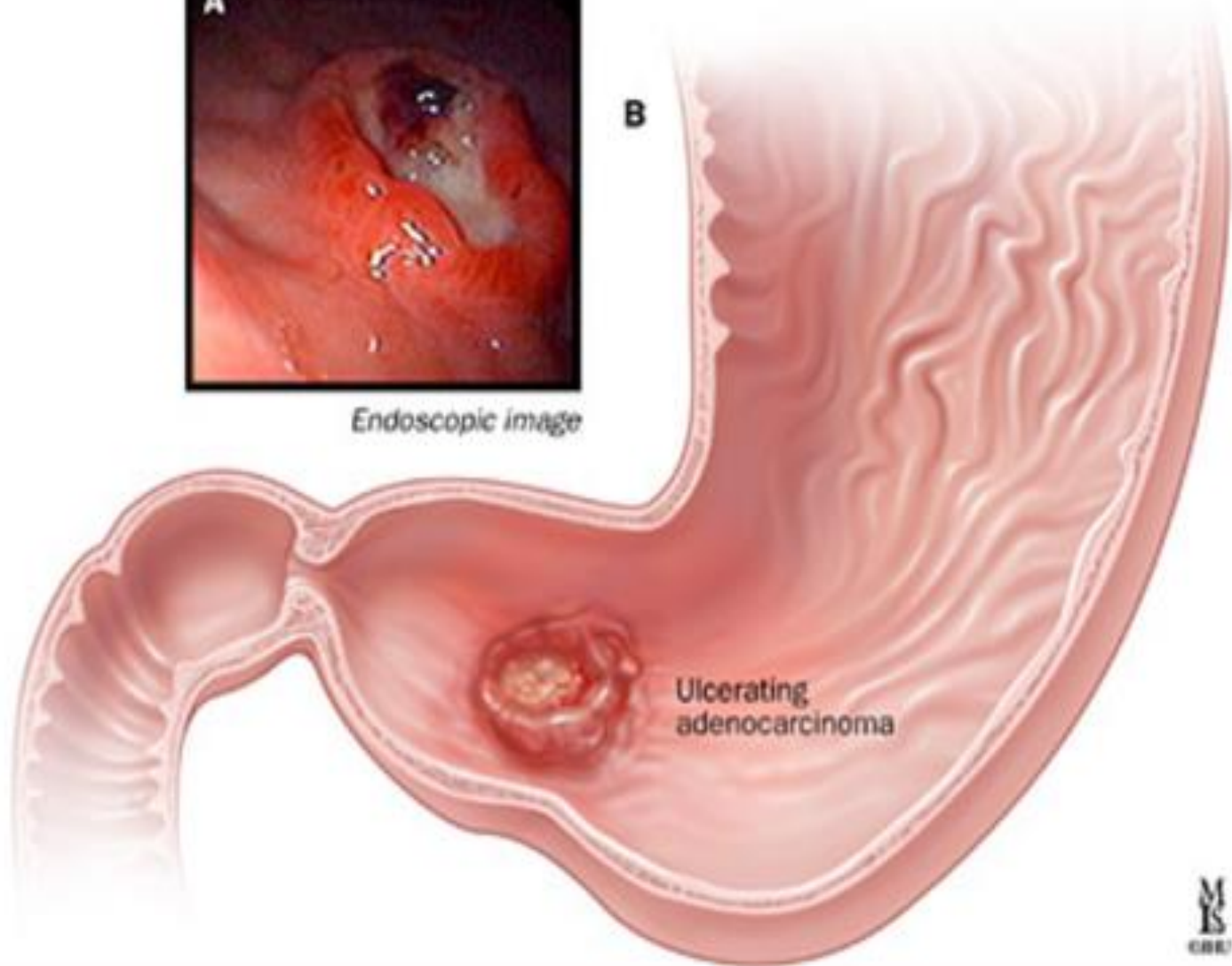






A

Endoscopic image



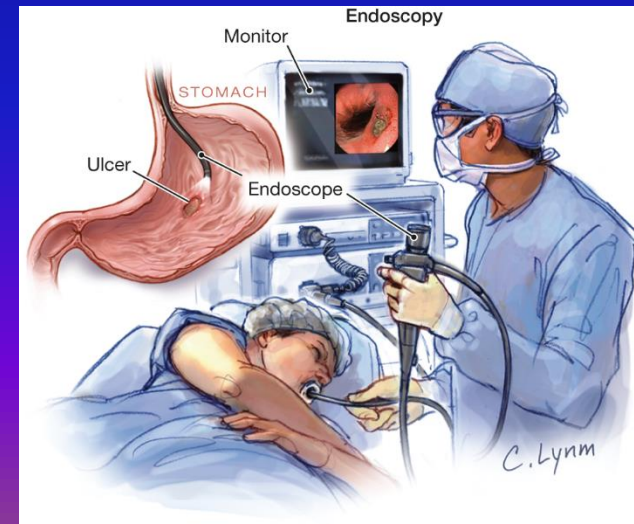
B

Ulcerating
adenocarcinoma

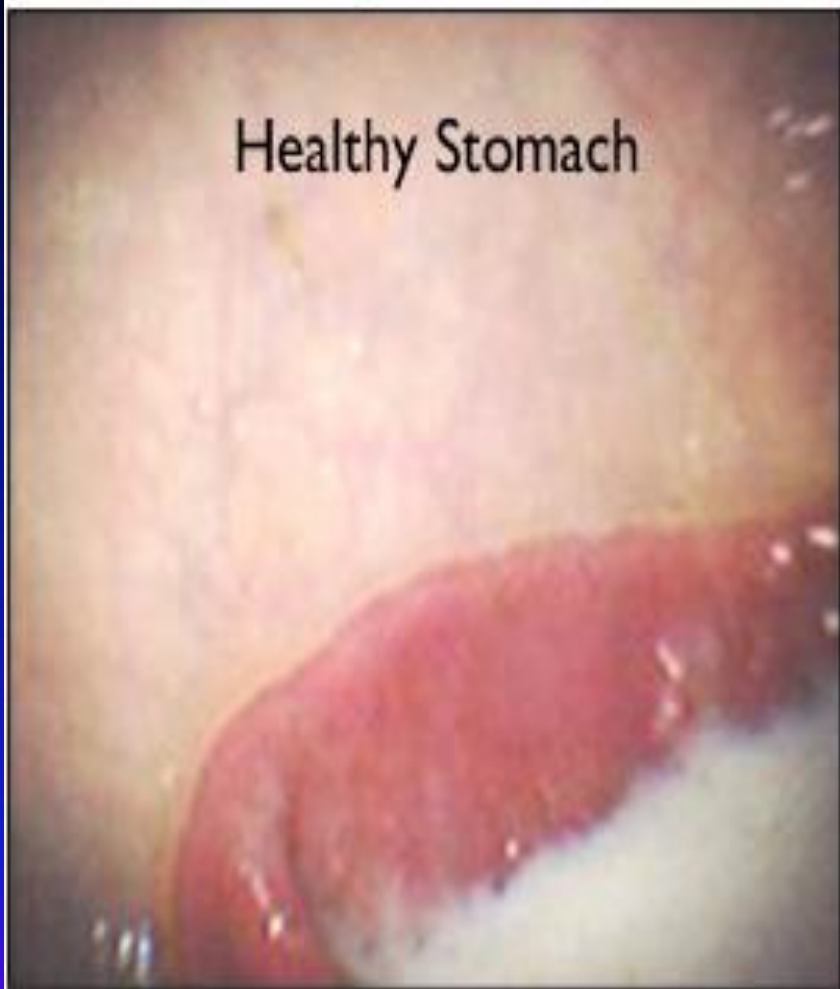


Diagnosis

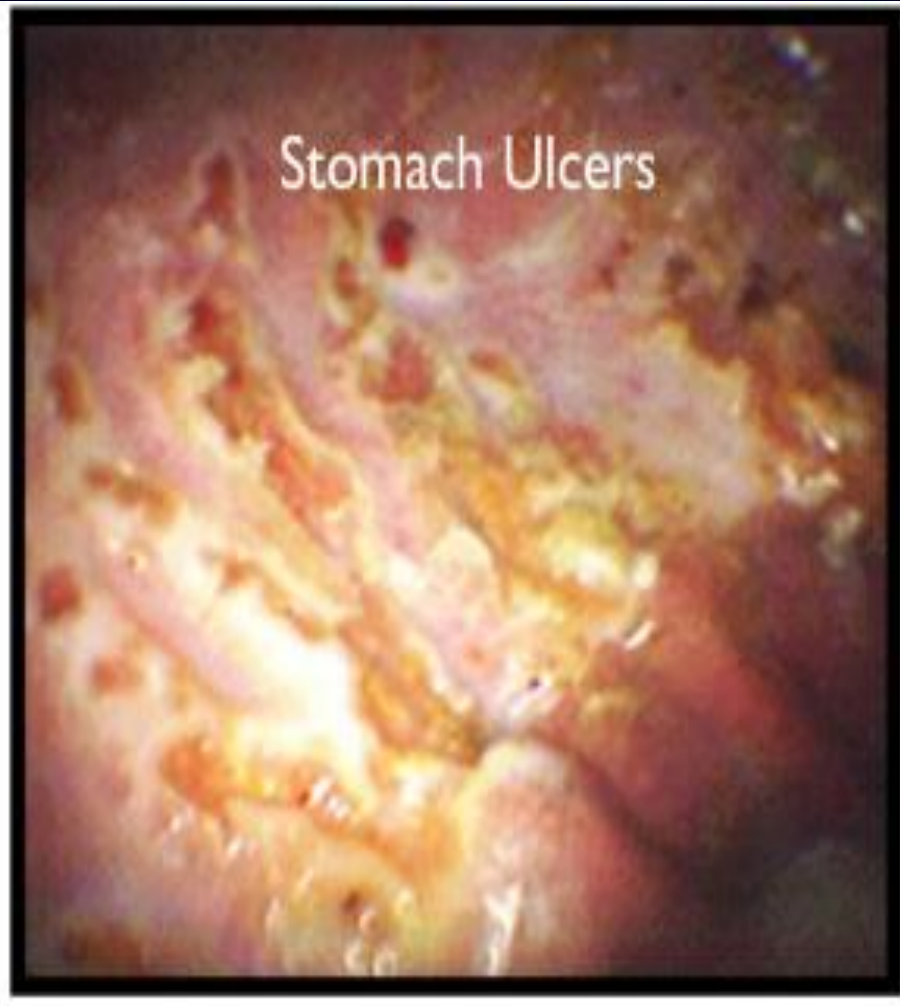
- The diagnosis of an ulcer is made by either a barium upper GI x-ray or an upper [endoscopy](#) (EGD-esophagogastroduodenoscopy)
- Upper endoscopy has the added advantage of having the capability of removing small tissue samples (biopsies) to test for *H. pylori* infection.
- Biopsies can also be examined under a microscope to exclude cancer
- *H pylori* tests



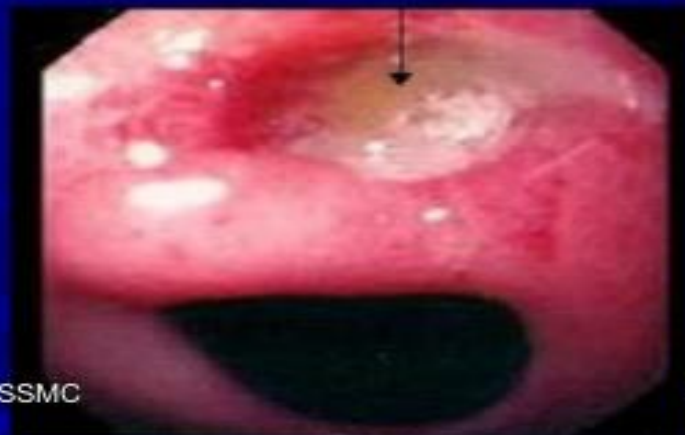
Healthy Stomach



Stomach Ulcers



Gastric Ulcer- Endoscopic Appearance

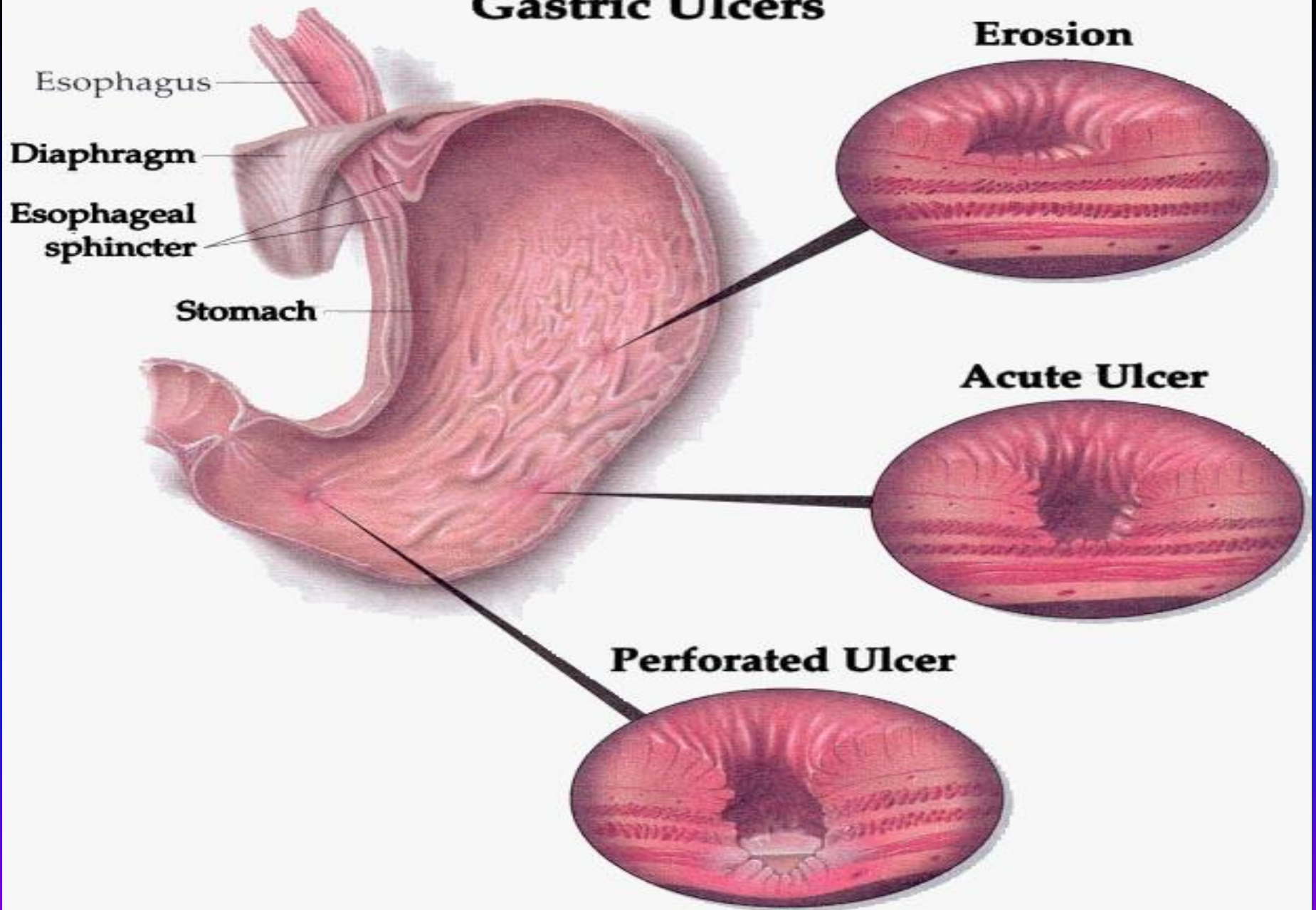


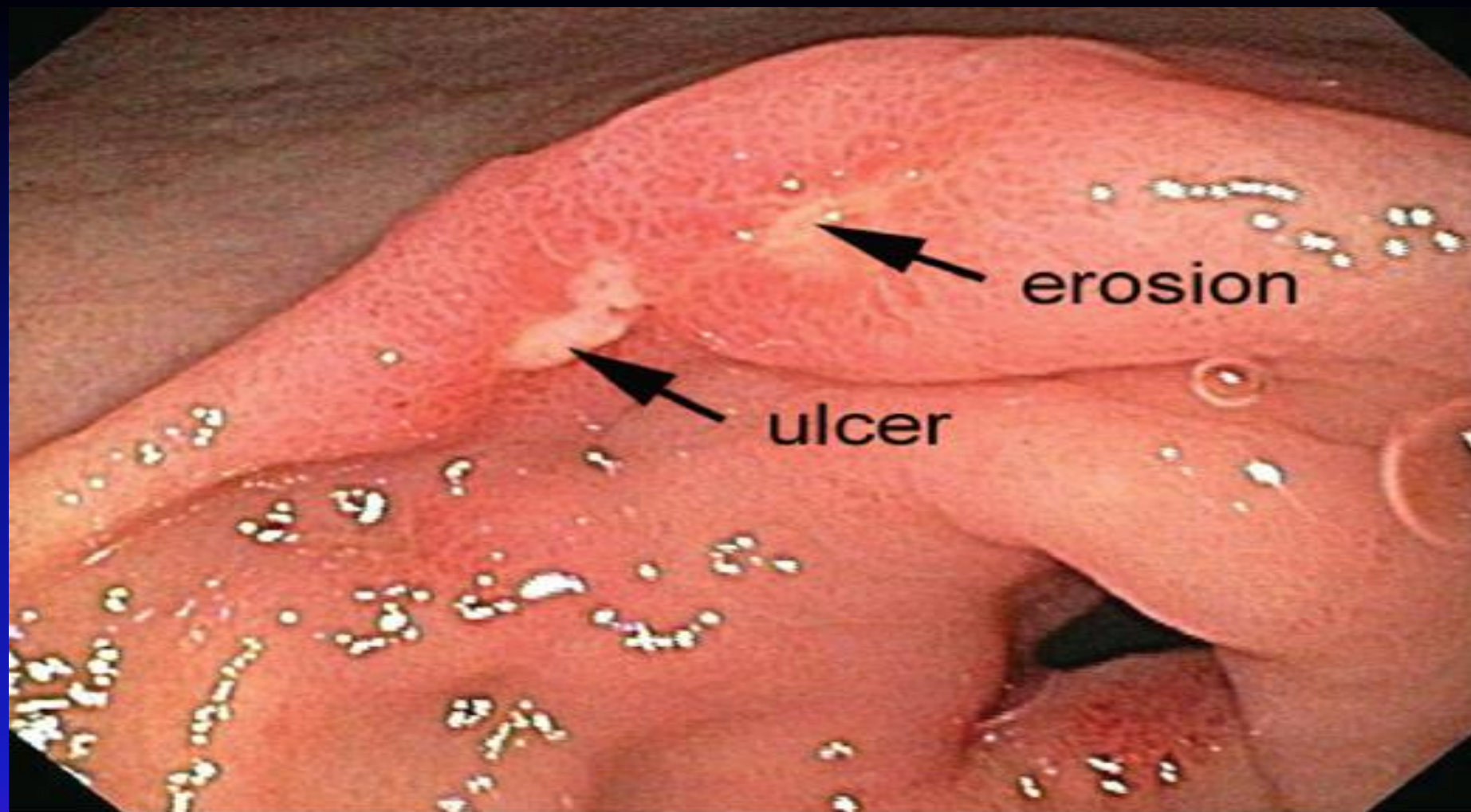
Sunday, November 18, 2012

DR. RUBEL, SSMC

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Gastric Ulcers





Source: Usatine RP, Smith MA, Mayeaux EJ, Chumley HS: *The Color Atlas of Family Medicine, Second Edition*; www.accessmedicine.com
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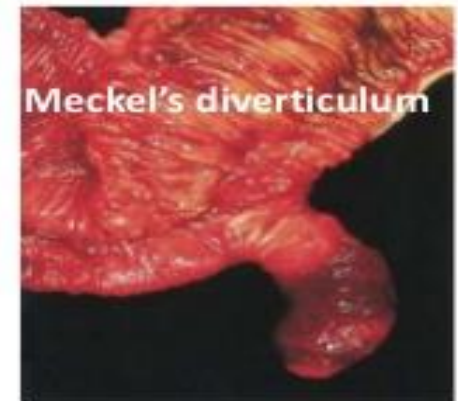
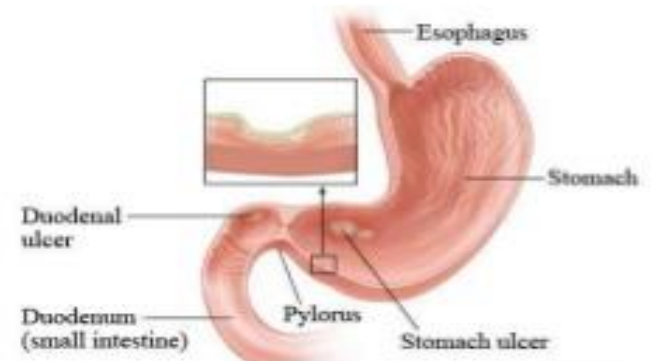
Gastric ulcer



Duodenal ulcer

Sites of peptic ulcer

- **Duodenum:** First portion, Anterior wall
- **Stomach:** usually antrum, lesser curvature (common), anterior and posterior wall, greater curvature (less common)
- In the margins of a gastroenterostomy (stomal ulcer)
- In the duodenum, stomach or jejunum of patients with [Zollinger- Ellison syndrome](#).
- Within or adjacent to a Meckel's diverticulum.

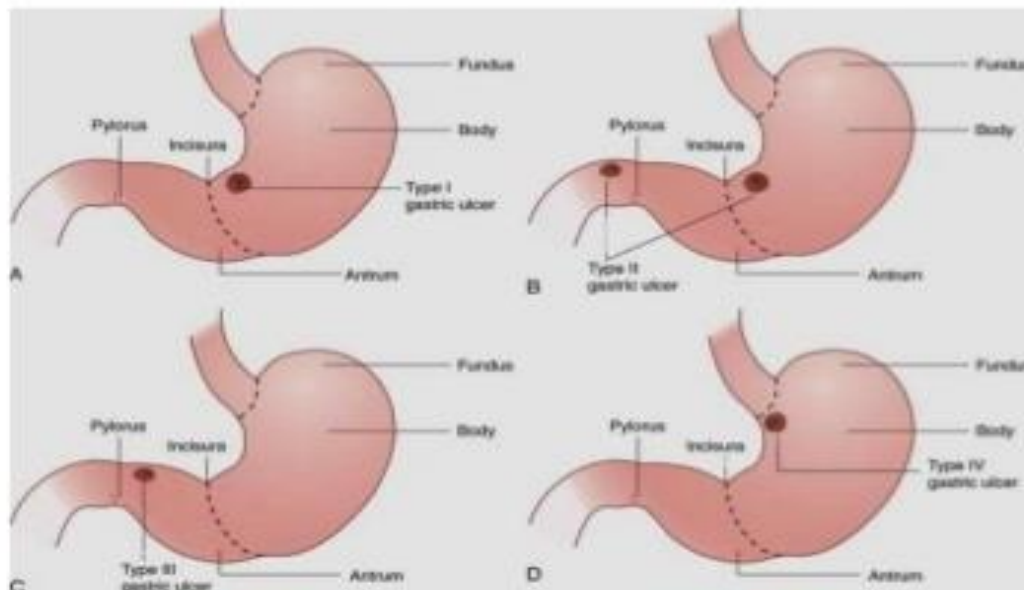


Types of Gastric Ulcer



55%

25%



15%

5%

Type I

in the antrum, near lesser curvature

Type II

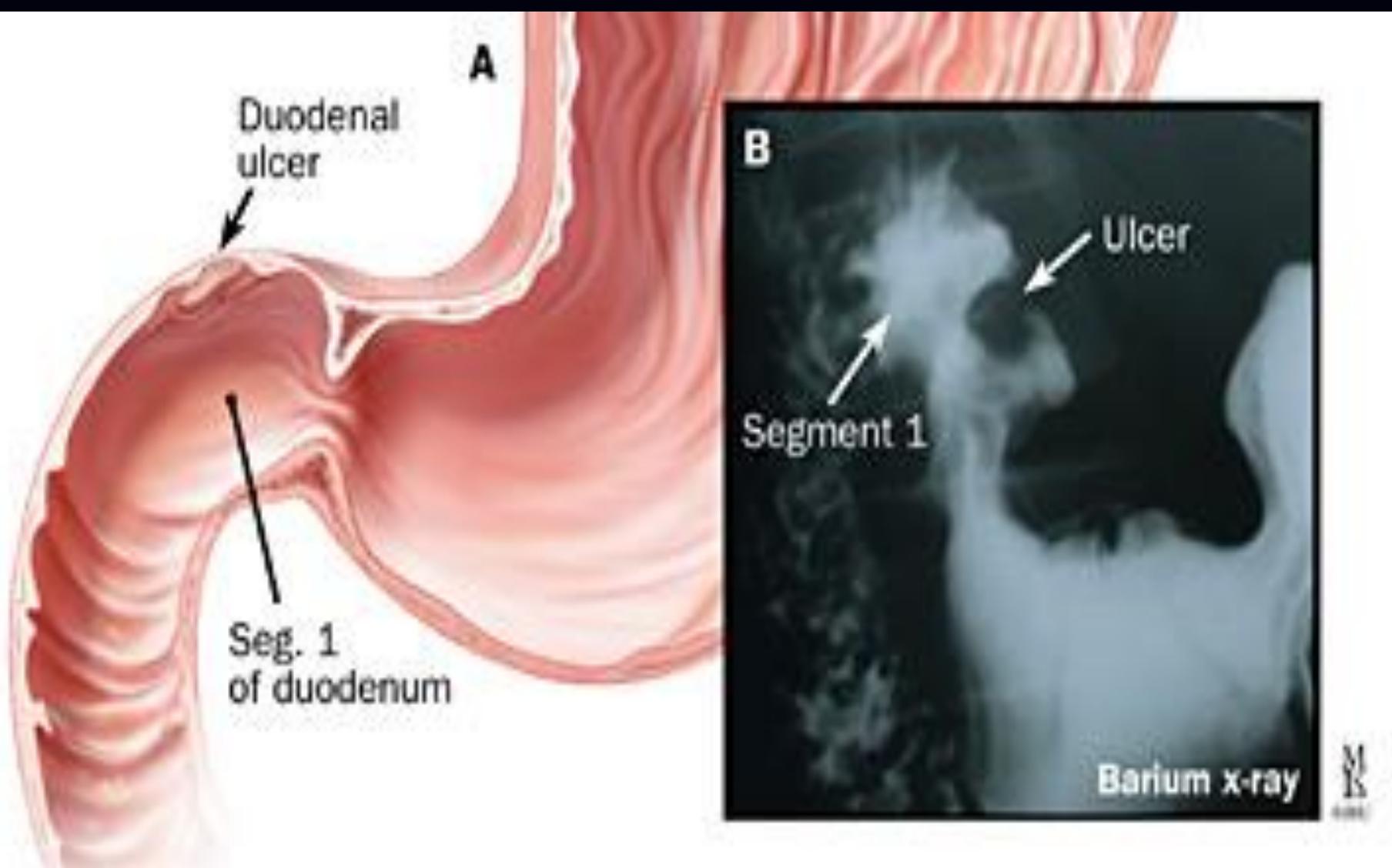
Combined gastric and duodenal ulcer

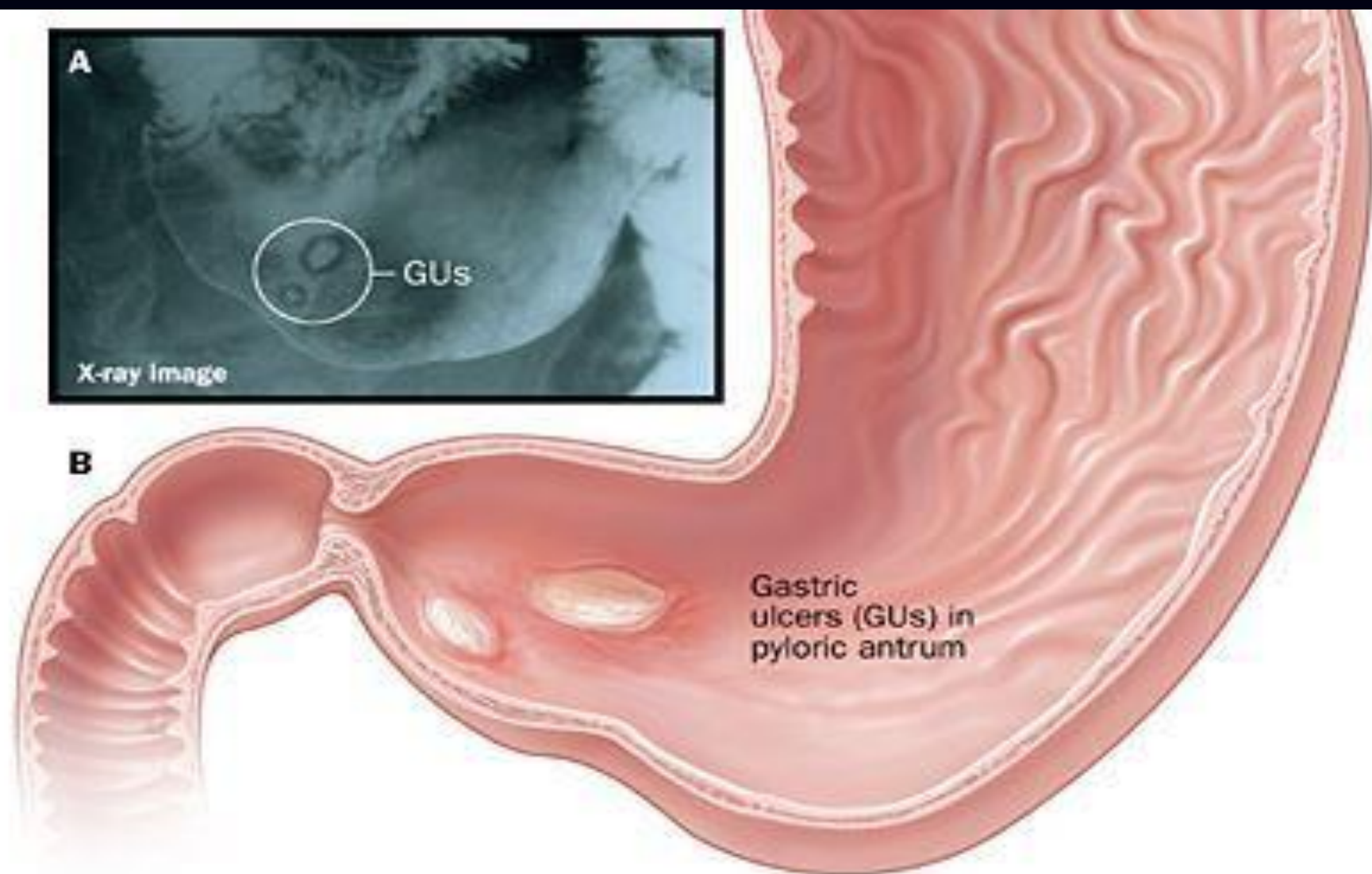
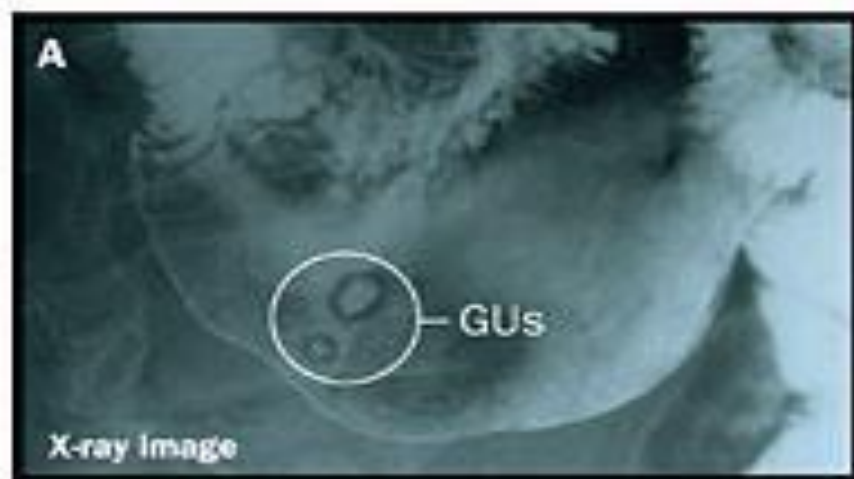
Type III

Prepyloric

Type IV

Ulcer in the proximal stomach and Cardia



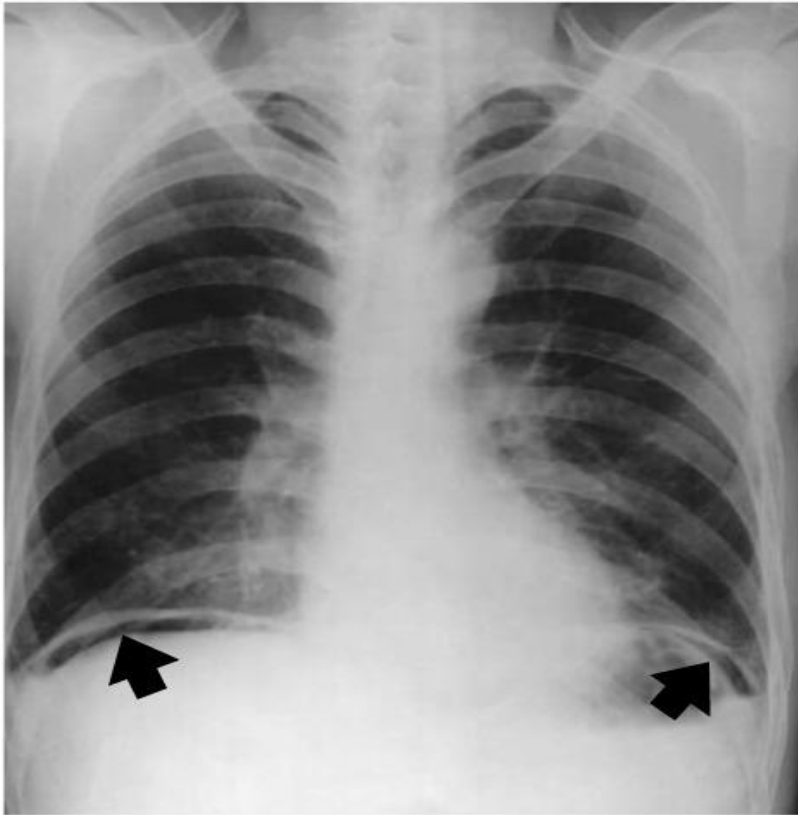


Benign Gastric Ulcer

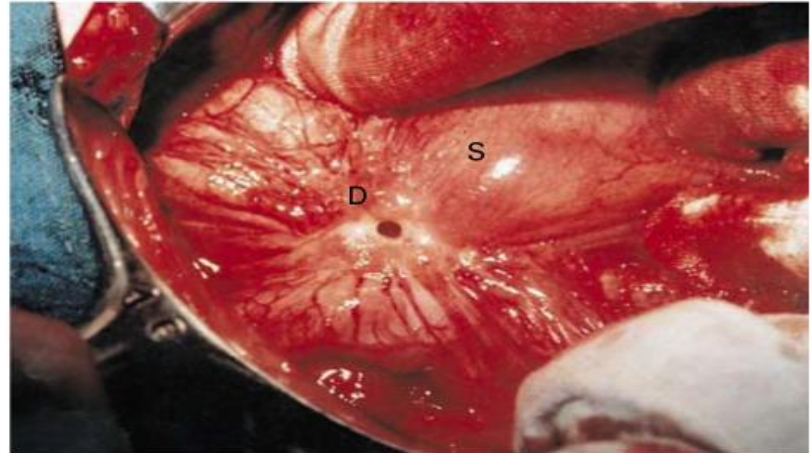
MUCOSAL FOLDS	Converging folds
SITE	95% in Lesser curvature
Margin	Regular
Floor	Granulation tissue in floor
Edges	NOT everted ,punched
Surrounding Area	Normal
Size and Extent	Small deep up to muscle layer



Perforated duodenal ulcer



A



B

Microscopic picture of peptic ulcer

Case 11: Chronic gastric ulcer

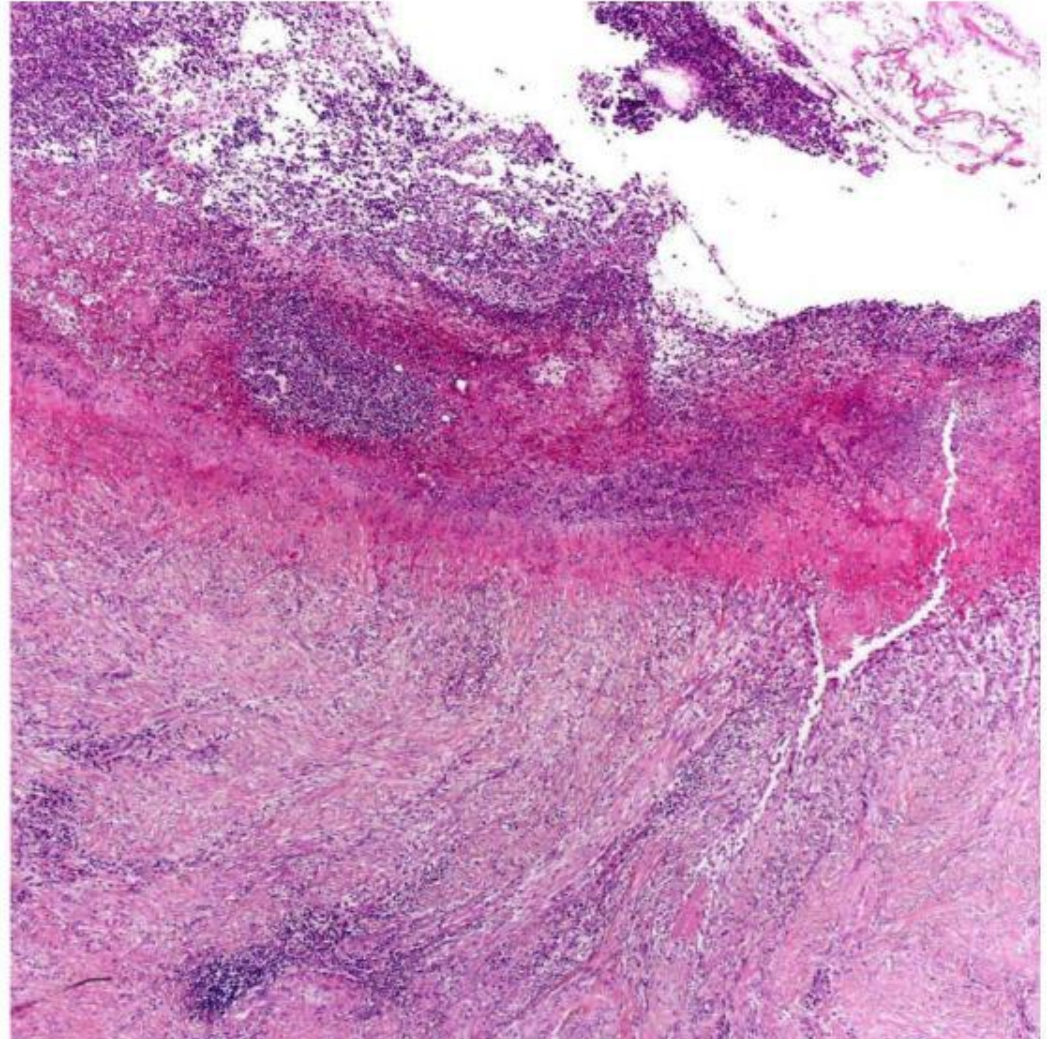
cellular debris



fibrinoid necrosis



granulation tissue



H pylori tests

Three nonendoscopy-based *H pylori* tests are available:

1. ***H pylori* stool antigen test (HpSA)**. It has sensitivity and specificity of greater than 90%. It can be used for both the diagnosis of *H pylori* and the confirmation of eradication after therapy.
2. **An urea breath test**. It uses ¹³C- or ¹⁴C-labeled urea taken orally. *H pylori* metabolizes the urea and liberates labeled carbon dioxide that is exhaled. This, in turn, can be quantified in breath samples. The sensitivity and specificity of the urea breath test is greater than 90%. This is considered the noninvasive diagnostic method of choice in situations where endoscopy is not indicated. It can also be used to confirm eradication after therapy
3. The third test depends on the presence of **antibodies to *H pylori* in the serum**. The major drawback to this test is that serologic assays may remain positive for as long as 3 years after eradication of the bacteria. Therefore, serologic assays are often unreliable to document eradication of *H pylori*. This test can be used for the diagnosis of *H pylori*, provided that the patient has not received any prior therapy for it.

H pylori tests

Three endoscopy-based *H pylori* tests are available:

1. **Rapid urease test (RUT).** It is performed by placing a gastric biopsy specimen, obtained on endoscopy, onto a gel- or membrane-containing urea and a pH-sensitive indicator. If *H pylori* is present, the bacterial urease hydrolyzes urea and changes the color of the media. The sensitivity and specificity of this test is greater than 90%.
2. **A bacterial culture *H pylori*.** It is highly specific but is not widely used because of the degree of expertise required. It is used when antibiotic susceptibilities are necessary.
3. **Histologic detection of *H pylori* in the biopsy specimen** is another endoscopy-based test. Appropriate staining is achieved using such stains as hematoxylin and eosin, Warthin-Starry, Giemsa, or Genta.

Studies

Special studies

- Obtaining a serum gastrin is useful in patients with recurrent, refractory, or complicated PUD and is useful in patients with a family history of PUD to screen for Zollinger-Ellison syndrome.
- A secretin stimulation test can be performed to distinguish Zollinger-Ellison syndrome from other conditions with a high serum gastrin, such as achlorhydria and antisecretory therapy with a proton pump inhibitor.

The treatment

Lifestyle

- To treat an ulcer, first eliminate substances that can be causing the ulcers. If you smoke or drink alcohol, **STOP !!!**.
- If the ulcer is believed to be caused by the use of NSAIDs, they need to be stopped.

The treatment



The diet:

A diet with avoidance of acid, hot or seasoned food might still be recommended.

The exclusion of coffee during the acute period may be recommended.

Aspirin intake, as well as NSAIDs and corticoids should be forbidden.

Treatment

- **Control stress.**
- **Don't smoke.**
- **Limit or avoid alcohol.**
- **Get enough sleep** (sleep can help your immune system, and therefore counter stress. Also, avoid eating shortly before bedtime.

Medical therapy

A) Antisecretory drugs

- H2 histaminic receptor blockers:

- Cimetidine 1000mg/day
- Ranitidine 300 mg/day
- Nizatidine(Axid) 300 mg/day
- Famotidine (Quamatel) 40mg/day
-

Famotidine will be preferred, given once or twice a day and without drug interferences of Cimetidine(cytochrome P450).

- HK ATP-ase pump blockers:

- Esomeprazole (Nexium) 40mg/day
- Omeprazole (Losec, Antra, Ultop) 40 mg/day
- Pantoprazole (Controloc) 40 mg/day
- Lanzoprazole (Lanzap) 30mg/day
- Rabeprazole (Pariet) 20mg/day
- The duration of antisecretory therapy will be for 6-8 weeks.

B) Gastric mucosal protectives

-sucralfate 4g/day (qid)might be associated.

C) Antacids

To neutralize the acid excess and reduce the painful symptoms, symptomatic medication such as Maalox, Almagel, Alfogel etc. are used.

The eradication of *Helicobacter Pylori* infection



- **The schemes** including proton pump blockers (zomeprazole, lansoprazole or pantoprazole), associated with two antibiotics indicated, triple or even quadruple schemes are used.

The triple therapy includes

OAM=Omeprazole(40mg/day)+Amoxicillin(2g/day)+Metronidazole(1500mg/day); or the ideal association is

OAC=Omeprazole+Amoxicilline+Clarithromicine(macrolide in dose of 1000mg/day).

The quadruple therapy is composed of Omeprazole+Subcitric bismuth (De-Nol)+Tetracycline+Metronidazole.

- The most widely used efficient therapies to eradicate *H pylori* are triple therapies, and they are recommended as first-line treatments; quadruple therapies are recommended as second-line treatment when triple therapies fail to eradicate *H pylori*.

The anti HP therapy duration is for 14 days.

Guidelines

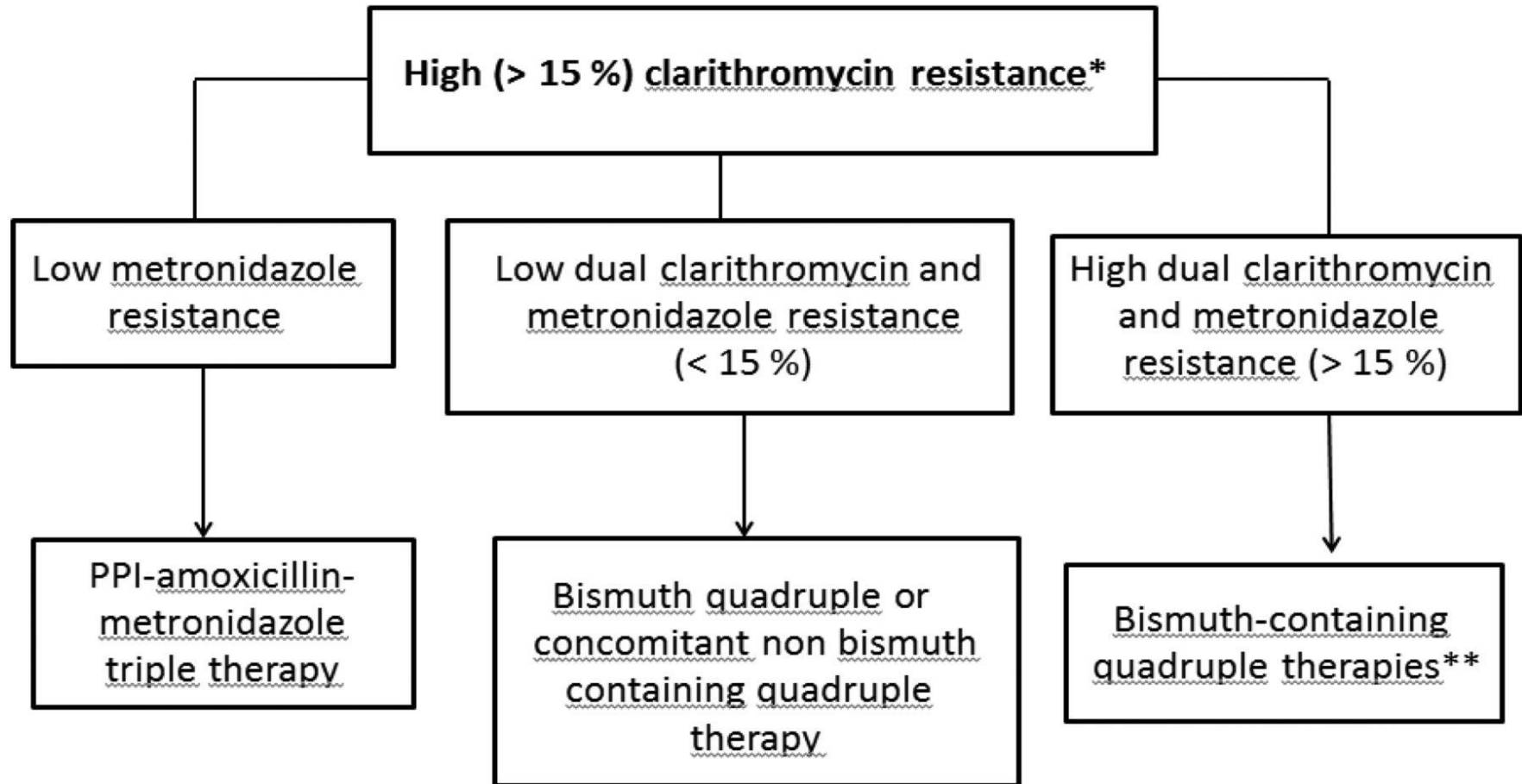
Management of *Helicobacter pylori* infection—the Maastricht V/Florence Consensus Report



- *H. pylori* resistance rates to antibiotics are increasing in most parts of the world.
- In areas of high (>15%) clarithromycin resistance, bismuth quadruple or non-bismuth quadruple, concomitant (PPI, amoxicillin, clarithromycin and a nitroimidazole) therapies are recommended.
In areas of high dual clarithromycin and metronidazole resistance, bismuth quadruple therapy (BQT) is the recommended first-line treatment.
- Currently, concomitant therapy (PPI, amoxicillin, clarithromycin, and a nitroimidazole administered concurrently) should be the preferred non-bismuth quadruple therapy, as it has shown to be the most effective to overcome antibiotic resistance.
- In areas of low clarithromycin resistance, triple therapy is recommended as first-line empirical treatment. Bismuth-containing quadruple therapy is an alternative.
- The treatment duration of PPI-clarithromycin based triple therapy should be extended to 14 days, unless shorter therapies are proven effective locally.

Guidelines

Management of *Helicobacter pylori* infection—the Maastricht V/Florence Consensus Report



* Regardless of their population expectations, individuals who have previously taken clarithromycin and/or metronidazole should be considered high risk patients for dual resistance.

** If bismuth is not available, levofloxacin, rifabutin and high dose dual (PPI + amoxicillin) therapies might be considered. If tetracycline is not available, bismuth-containing quadruple therapy combining furazolidone-metronidazole or amoxicillin-metronidazole can be considered.

Surgical treatment

With the success of medical therapy, surgery has a very limited role in the management of PUD.

Potential indications for surgery include:

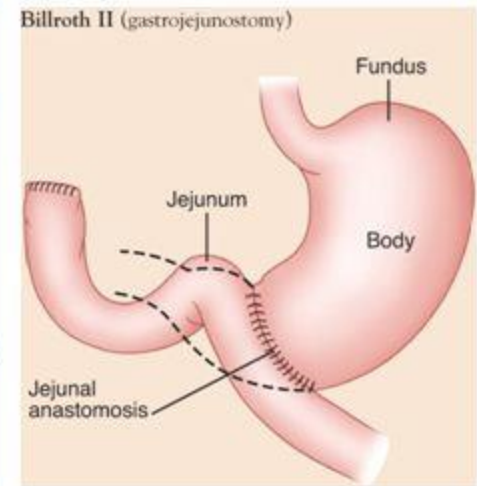
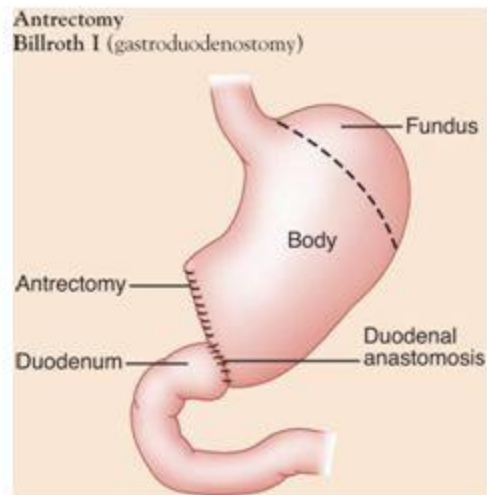
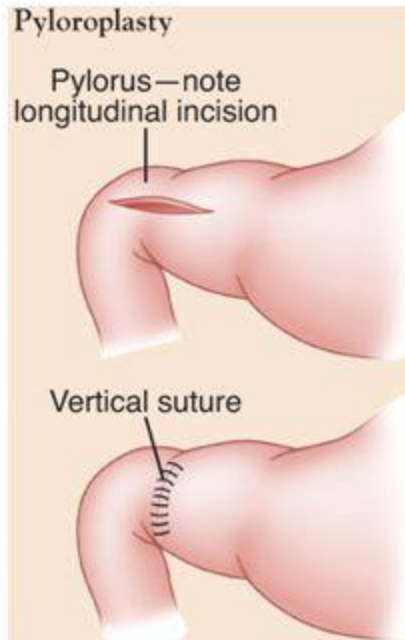
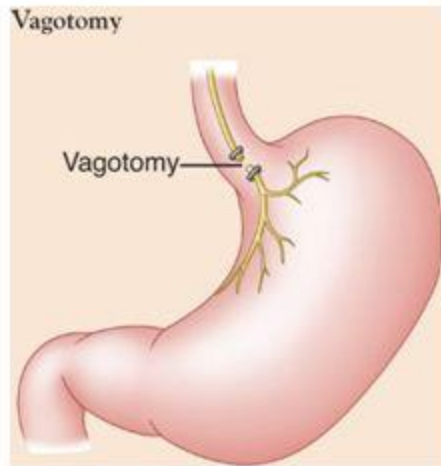
- refractory disease
- complications of PUD including the following:
 - **Refractory, symptomatic peptic ulcers are a potential complication of PUD**
 - **Perforation usually is managed emergently with surgical repair. However, this is not mandatory in all patients.**
 - **Obstruction can complicate PUD and may persist or recur despite endoscopic balloon dilation.**
 - **Bleeding, particularly in patients with massive hemorrhage.**

Surgical treatment

The appropriate surgical procedure depends on the location and nature of the ulcer.

- oversewing of the ulcer
- vagotomy and pyloroplasty
- vagotomy and antrectomy with gastroduodenal reconstruction (Billroth I)
- vagotomy and antrectomy with gastrojejunal reconstruction (Billroth II)
- highly selective vagotomy

Surgical Procedures for Peptic Ulcers



Treatment

- Endoscopy. Some bleeding ulcers can be treated through the endoscope.

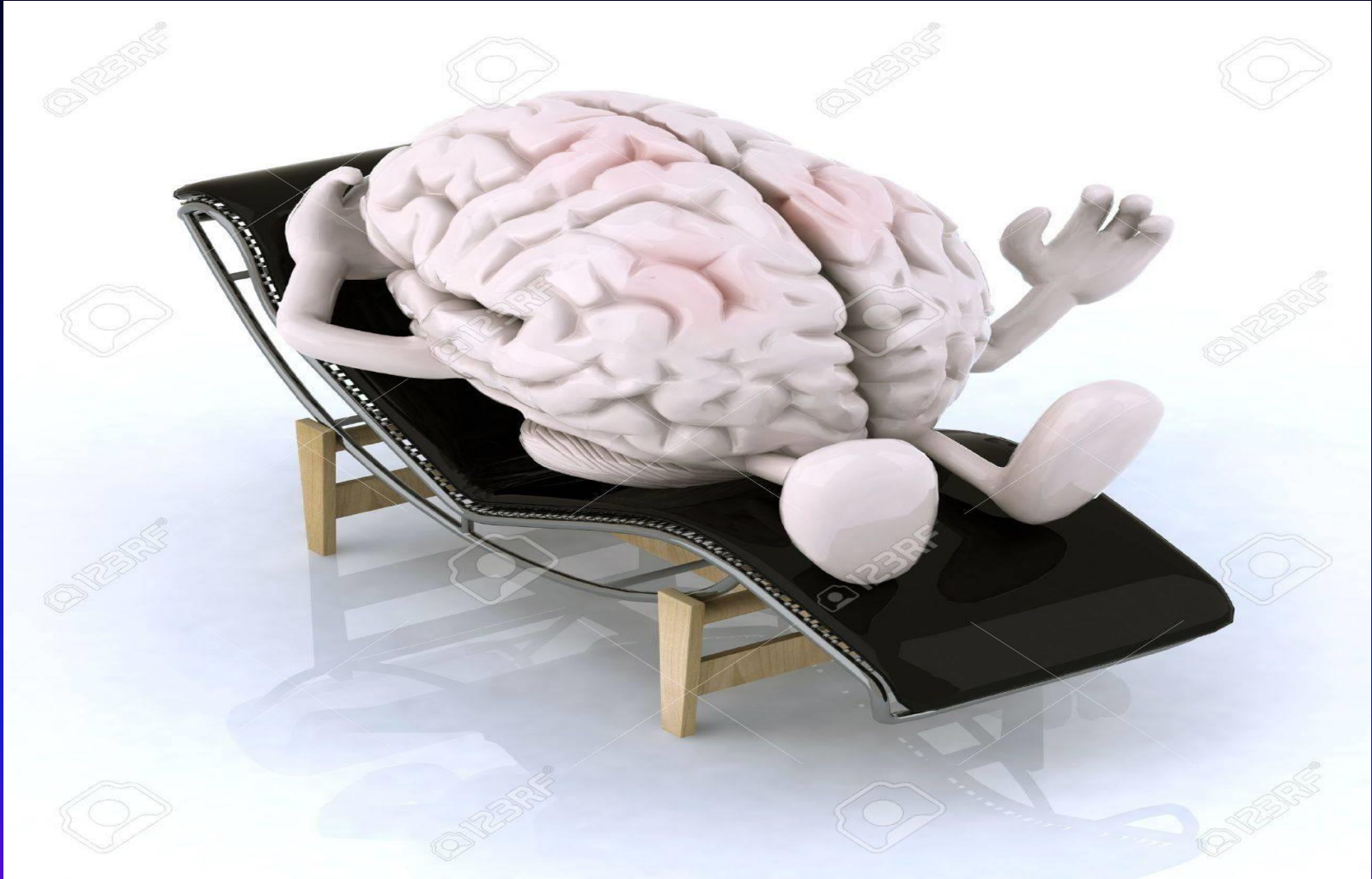
Further Outpatient Care

- Endoscopy is required to document healing of gastric ulcers and to rule out gastric cancer. This usually is performed 6-8 weeks after the initial diagnosis of PUD.

Prognosis:

- When the underlying cause is addressed, the prognosis is excellent.
- Most patients are treated successfully with the cure of *H pylori* infection, avoidance of NSAIDs, and the appropriate use of antisecretory therapy.
- Cure of *H pylori* infection changes the natural history of the disease, with a decrease in the ulcer recurrence rate from 60-90% to less than 10% per year (in some reports, recurrence is 1-2%).

Now it's time for a break



thank you