Medical Academy named after
S.I. Georgievsky of Vernadsky CFU

Department of Surgery №2
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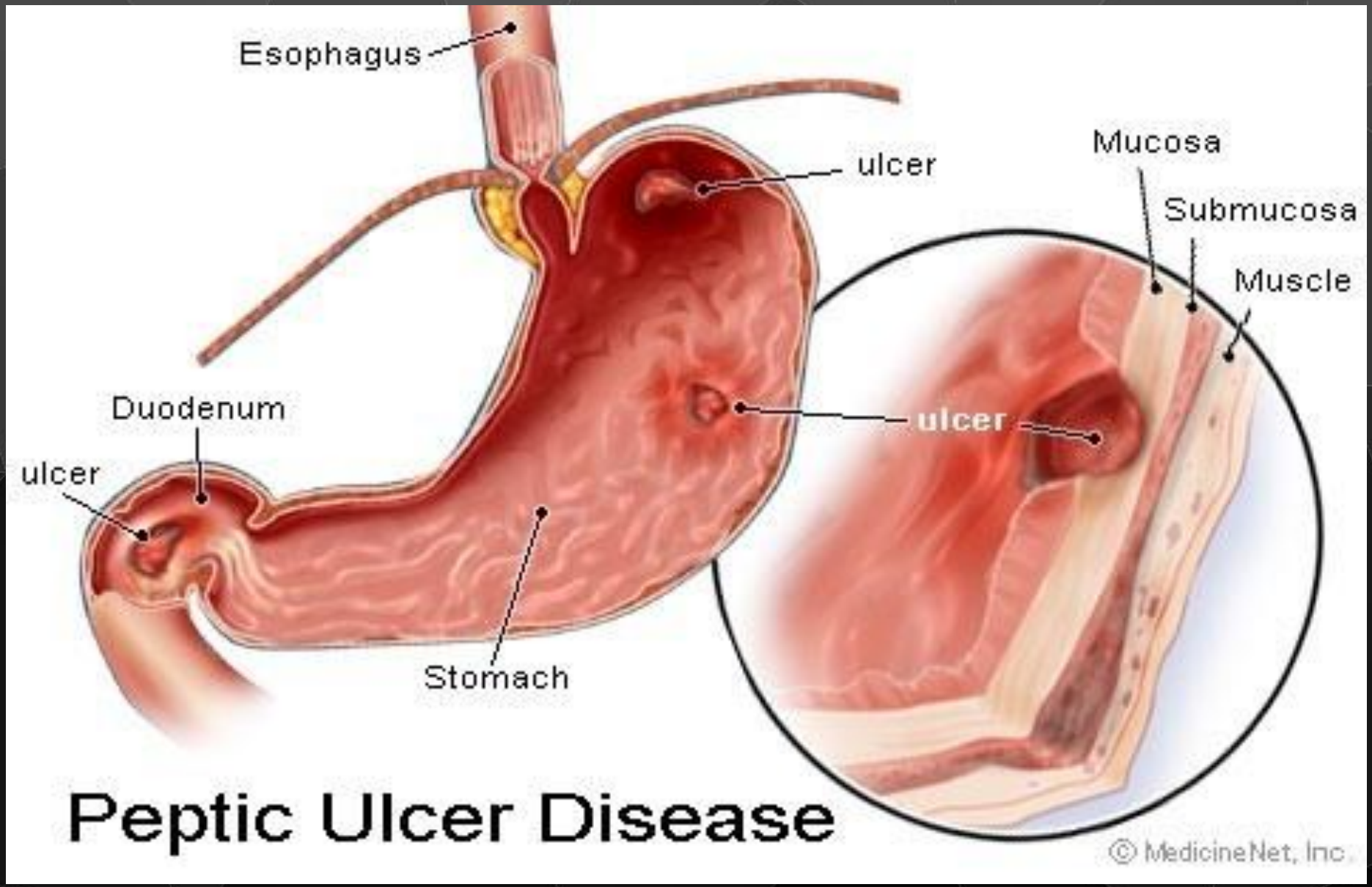
Peptic Ulcer Disease Complications

A **peptic ulcer** is an open sore in the upper digestive tract. There are two types of peptic ulcers, a gastric ulcer, which forms in the lining of the stomach, and a duodenal ulcer, which forms in the upper part of the small intestine.


Esophagus
ulcer
Mucosa
Submucosa
Muscle
Duodenum
ulcer
ulcer
Stomach
Peptic Ulcer Disease
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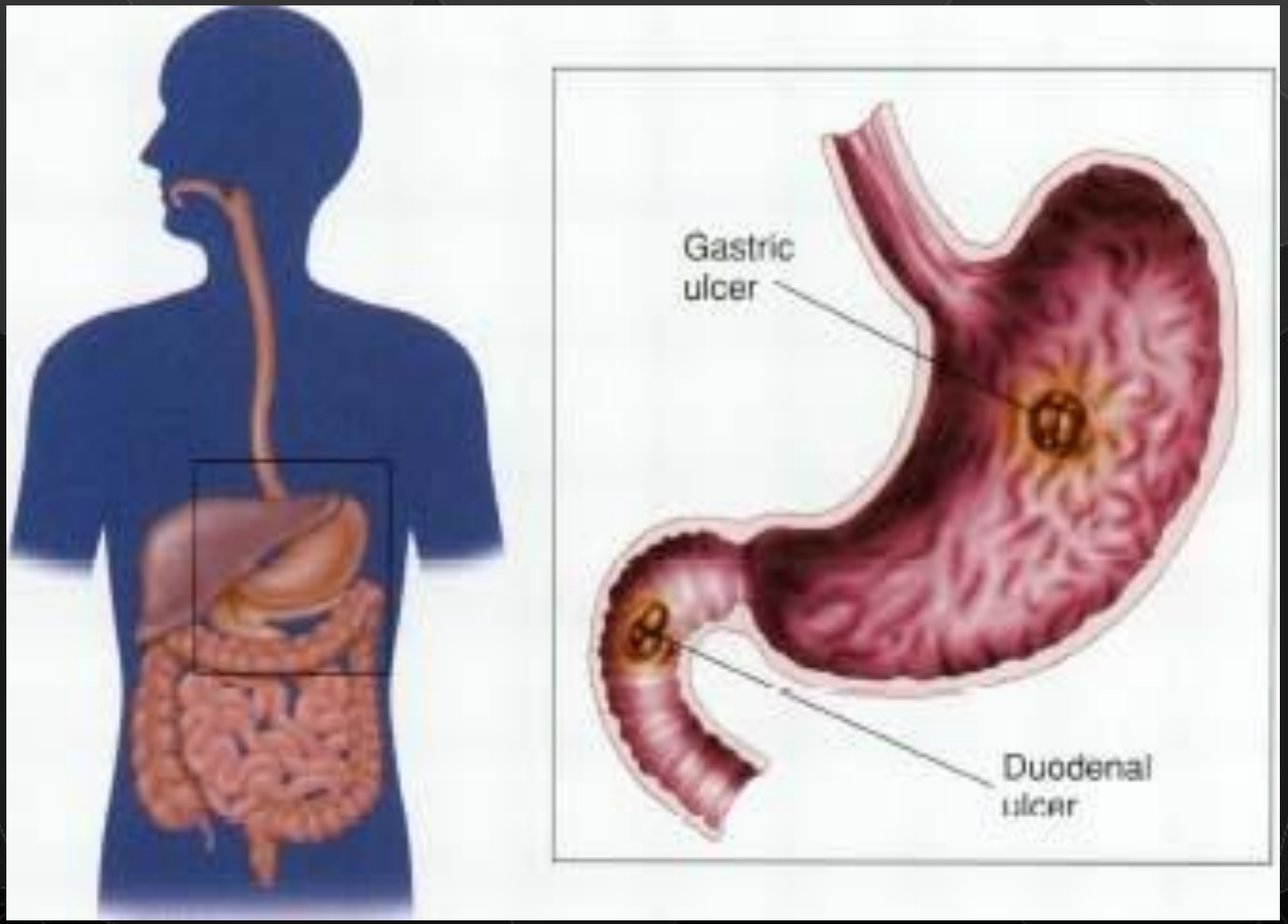
Classification

- Stomach (called **gastric ulcer**)
- Duodenum (called **duodenal ulcer**)
- Oesophagus (called **Oesophageal ulcer**)
- Types of peptic ulcers:
- Type I: Ulcer along the lesser curve of stomach
- Type II: Two ulcers present - one gastric, one duodenal
- Type III: Prepyloric ulcer
- Type IV: Proximal gastroesophageal ulcer
- Type V: Anywhere

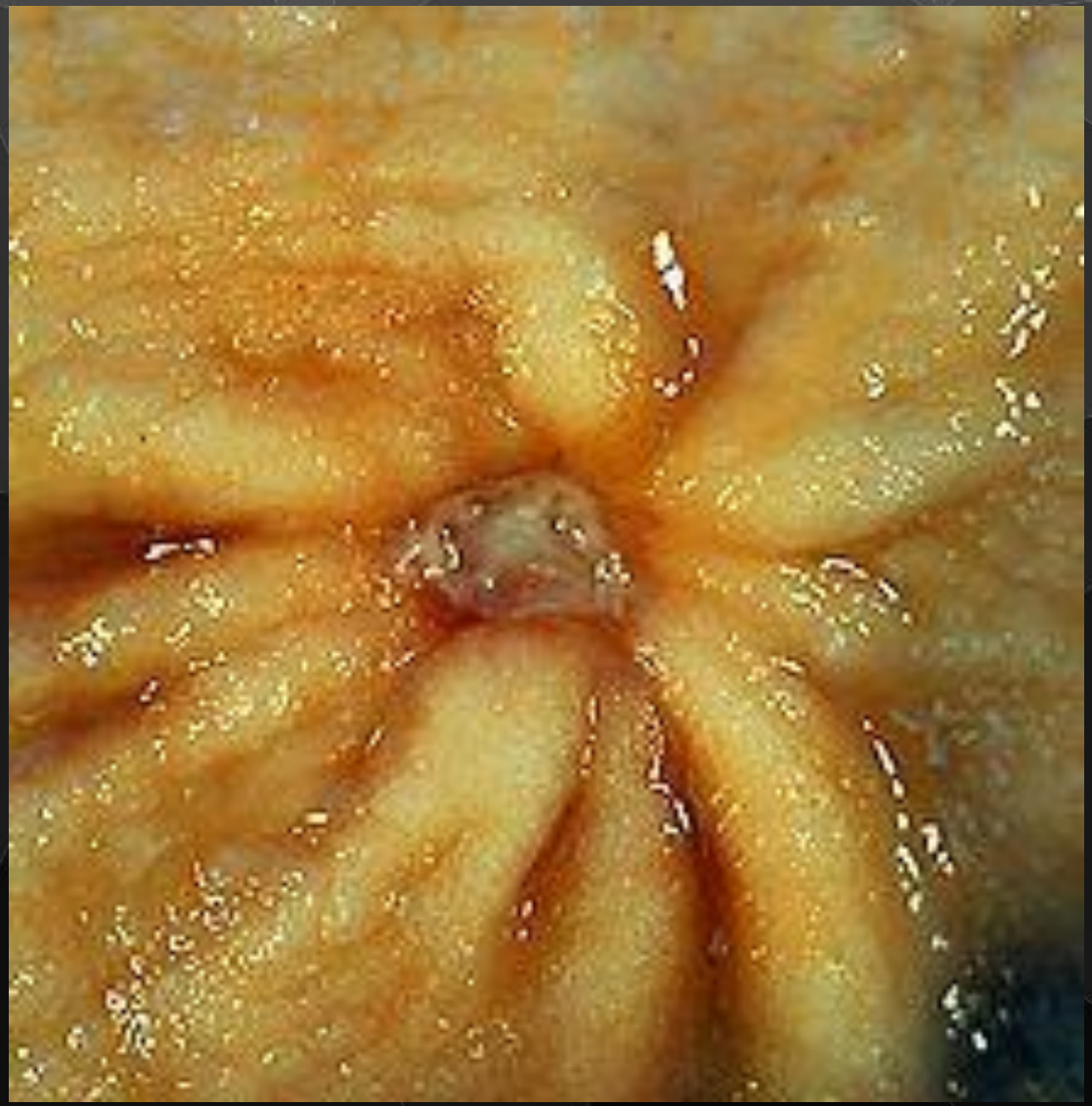
SYMPTOMS



SYMPTOMS

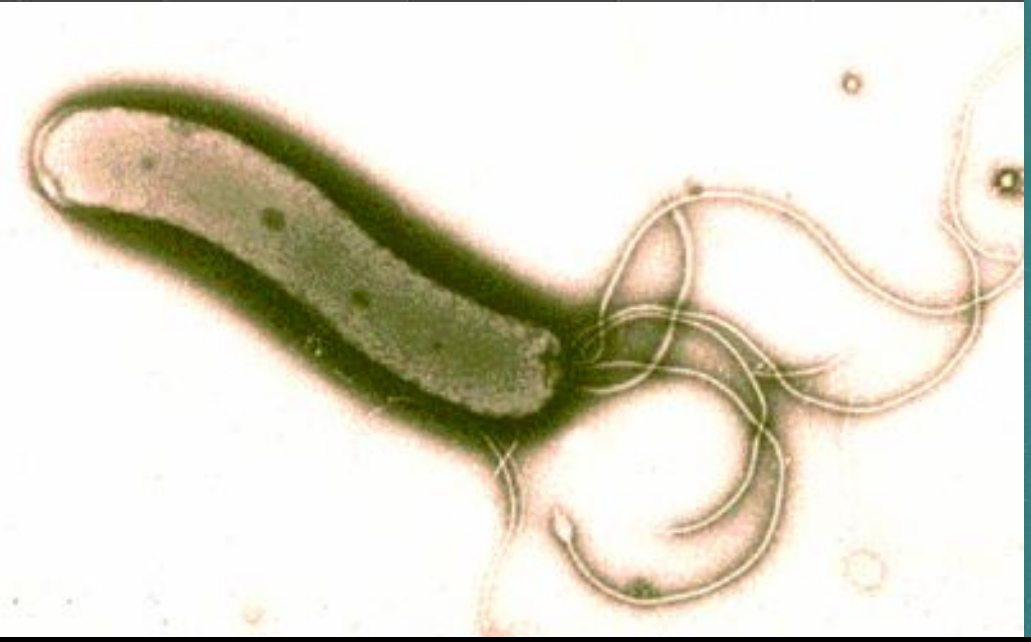
- Burning pain
- Bloating
- Nausea
- Water brash
- Unexplained weight loss
- Hematemesis (vomiting of blood)
- Appetite changes
- Melina
- Vomiting
- Blood in the stools
- Low blood cell count (anemia)
- Stomach pain wakes you up at night
- Frequent burping or hiccupping
- An early sense of fullness with eating

CAUSES



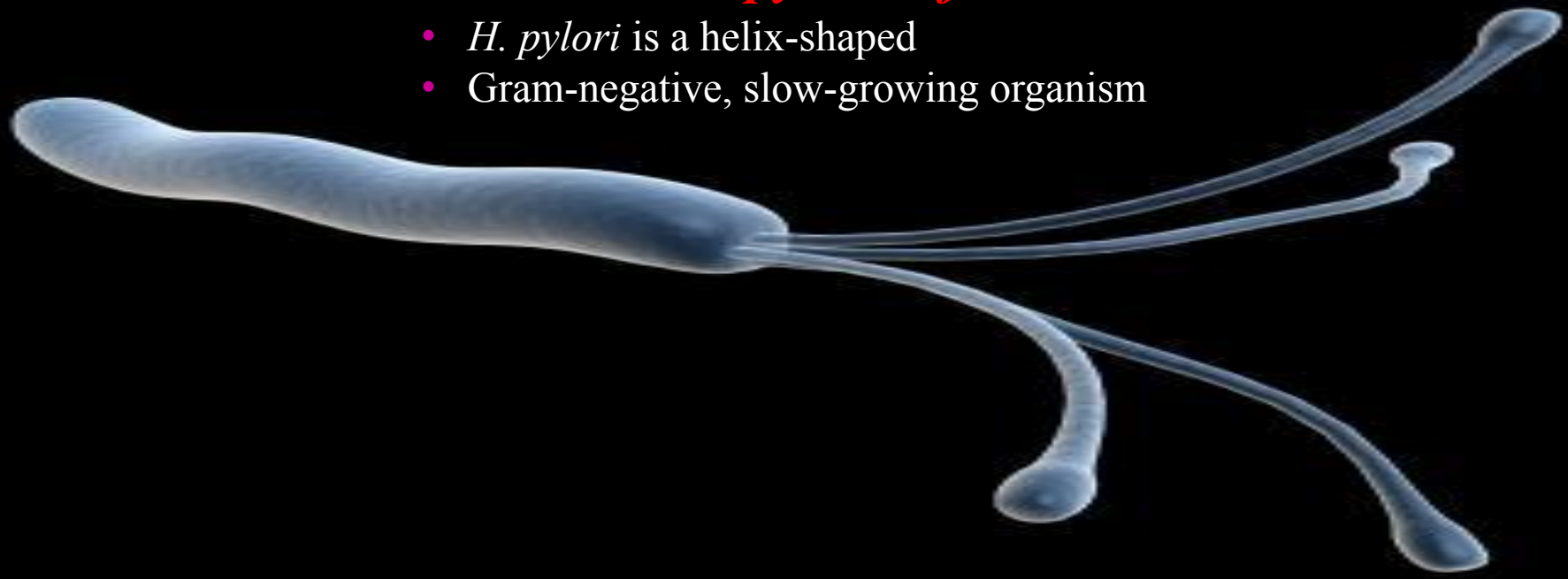
CAUSES

- Helicobacter pylori, a bacteria that is frequently found in the stomach
- Nonsteroidal anti-inflammatory drugs (NSAIDS) such as ibuprofen
- In addition, smoking and certain other genetic and environmental factors (such as medications) may influence the course of peptic ulcer disease.
- Psychological stress and dietary factors were once thought to be the cause of ulcers, although these factors are no longer thought have a major role.

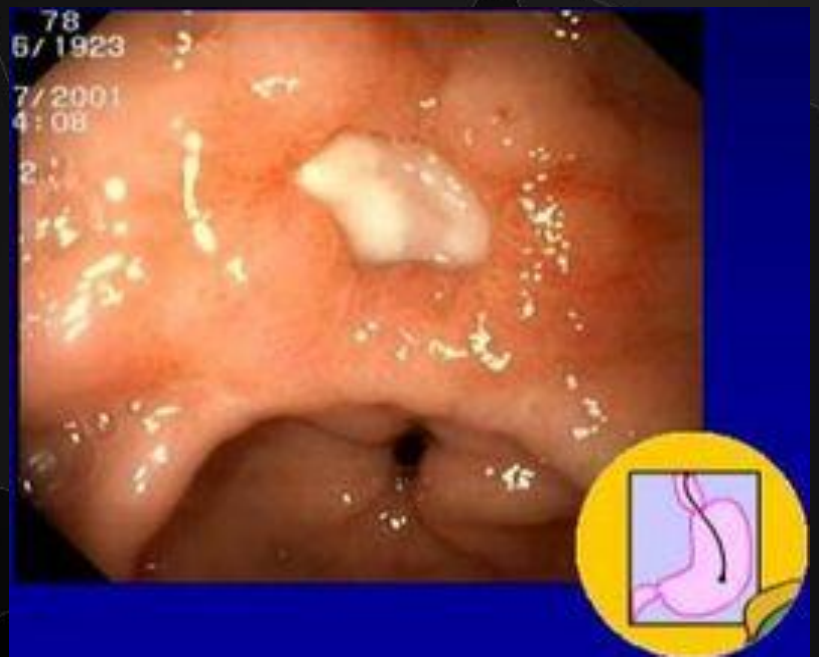


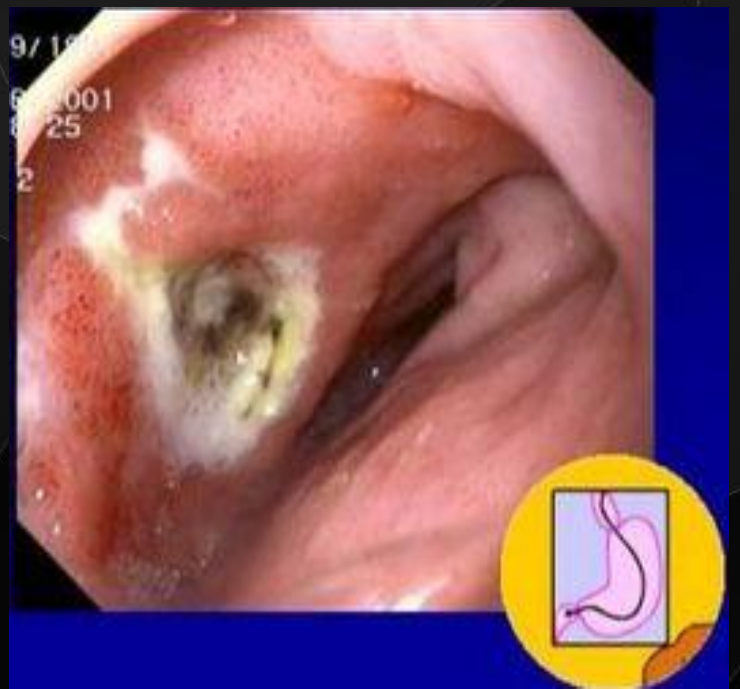
Helicobacter pylori infection

- *H. pylori* is a helix-shaped
- Gram-negative, slow-growing organism



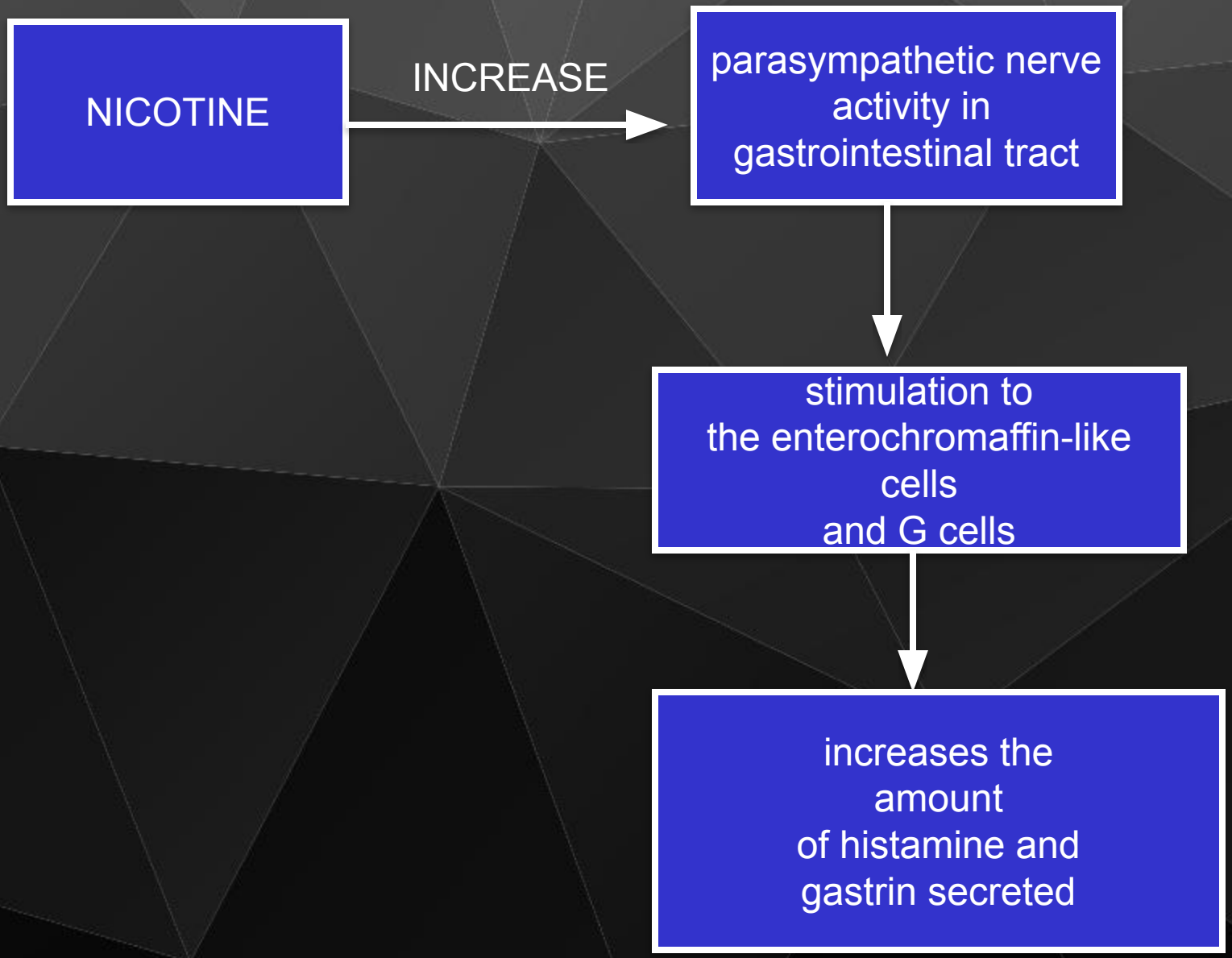
- The bacterium persists in the stomach for decades in most people. Most individuals infected by *H. pylori* will never experience clinical symptoms despite having chronic gastritis. Approximately 10-20% of those colonized by *H. pylori* will ultimately develop gastric and duodenal ulcers. *H. pylori* infection is also associated with a 1-2% lifetime risk of stomach cancer and a less than 1% risk of gastric MALT lymphoma.





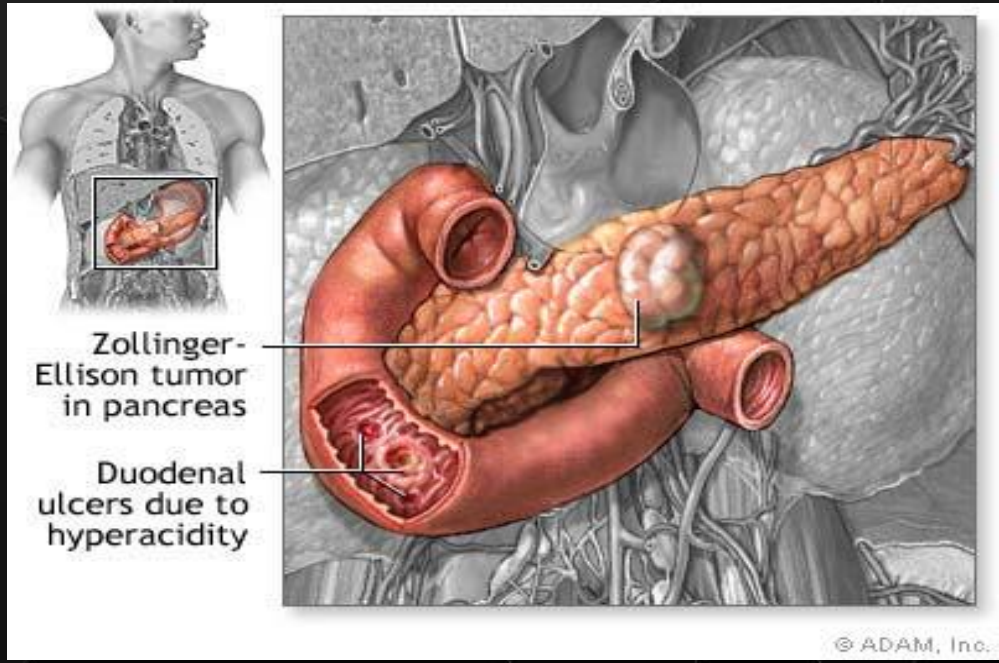
Effects of smoking on PUD

- Increased rate of gastric emptying
- Diminished pancreatic bicarbonate secretion
- Decreased duodenal pH
- Reduced mucosal blood flow
- Inhibition of mucosal prostaglandins


NICOTINE
INCREASE
parasympathetic nerve activity in gastrointestinal tract
stimulation to the enterochromaffin-like cells and G cells
increases the amount of histamine and gastrin secreted

Gastrinomas (Zollinger Ellison syndrome), rare gastrin-secreting tumors, also cause multiple and difficult to heal ulcers.

Excessive alcohol consumption Alcohol can irritate and erode the mucous lining of stomach and increases the amount of stomach acid that's produced. It's uncertain, however, whether this alone can progress into an ulcer or if it just aggravates the symptoms of an existing ulcer.




Caffeine

Beverages and foods that contain caffeine can stimulate acid secretion in the stomach. This can aggravate an existing ulcer, but the stimulation of stomach acid can't be attributed solely to caffeine.

The complications of Peptic Ulceration

The common complications are:

- Perforation
- Penetration
- Bleeding
- Stenosis

Perforation (a hole in the wall) often leads to catastrophic consequences. Erosion of the gastro-intestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain. Posterior wall perforation leads to pancreatitis; pain in this situation often radiates to the back

Perforation

Clinical Features

- History of peptic ulcer
- Sudden onset, severe, generalized abdominal pain
- Starts as chemical peritonitis, then bacterial peritonitis which will be accompanied by deterioration of the patient's condition

Perforation

Clinical symptoms

- Tachycardia, pyrexia
- Shock
- Board like rigidity of abdomen
- Abdominal splinting

Perforation

Clinical Features

- In elderly, the classical presentation of PPU may be absent
- Use of NSAID
- Board like abdominal rigidity may be not present
- Epigastric tenderness

Perforation

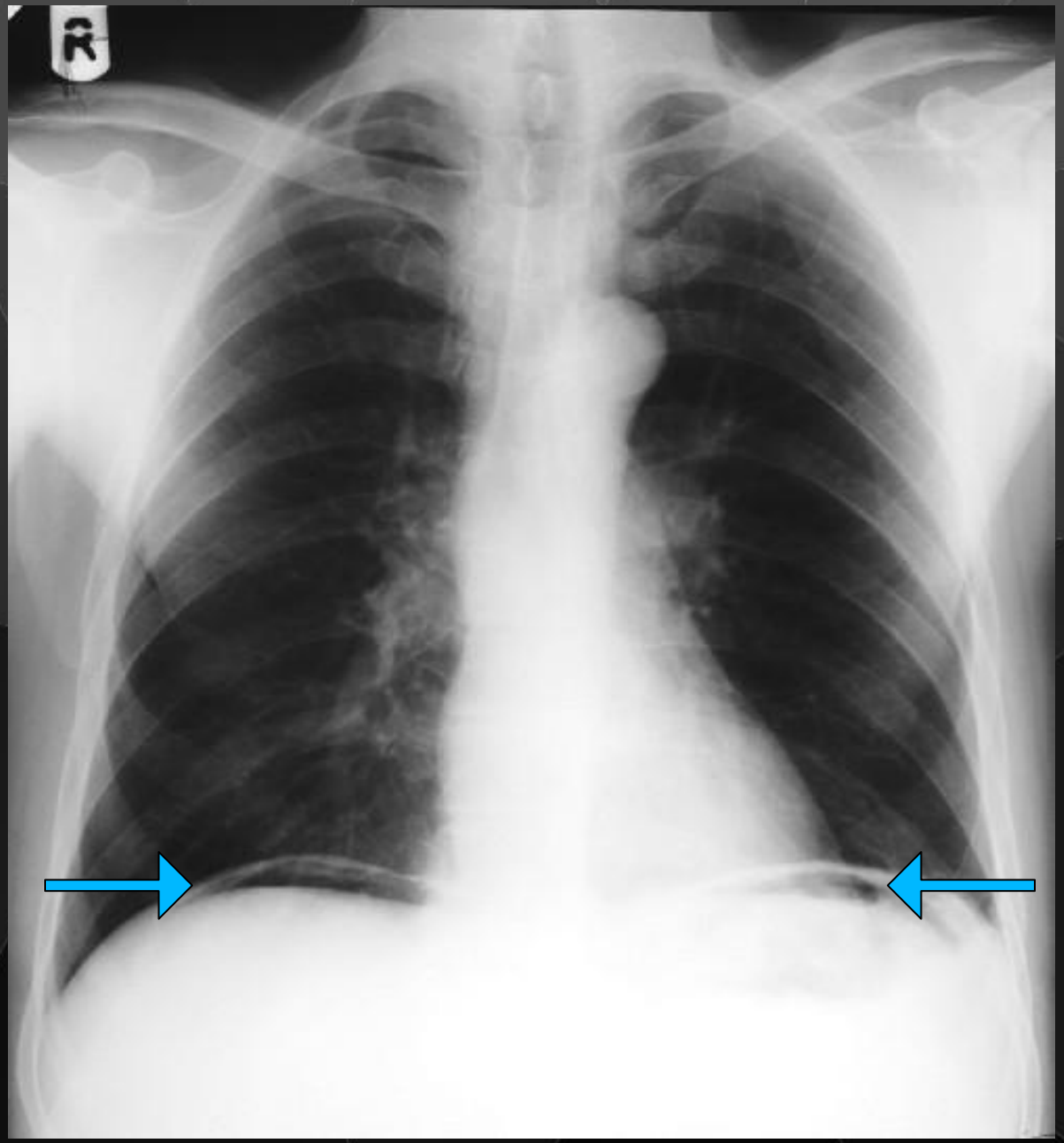
Clinical Features

- The most frequent place for perforation is the anterior wall of duodenum
- Anterior or incisural part of gastric ulcer may perforate
- Gastric ulcer may perforate in gland bag (difficult to diagnose)

Perforation

Investigations

- Observe chest X-ray will reveal free gas under the diaphragm in more than 50% of the cases
- Amylase level to R/O pancreatitis
- CT scan of the abdomen
- Endoscopy

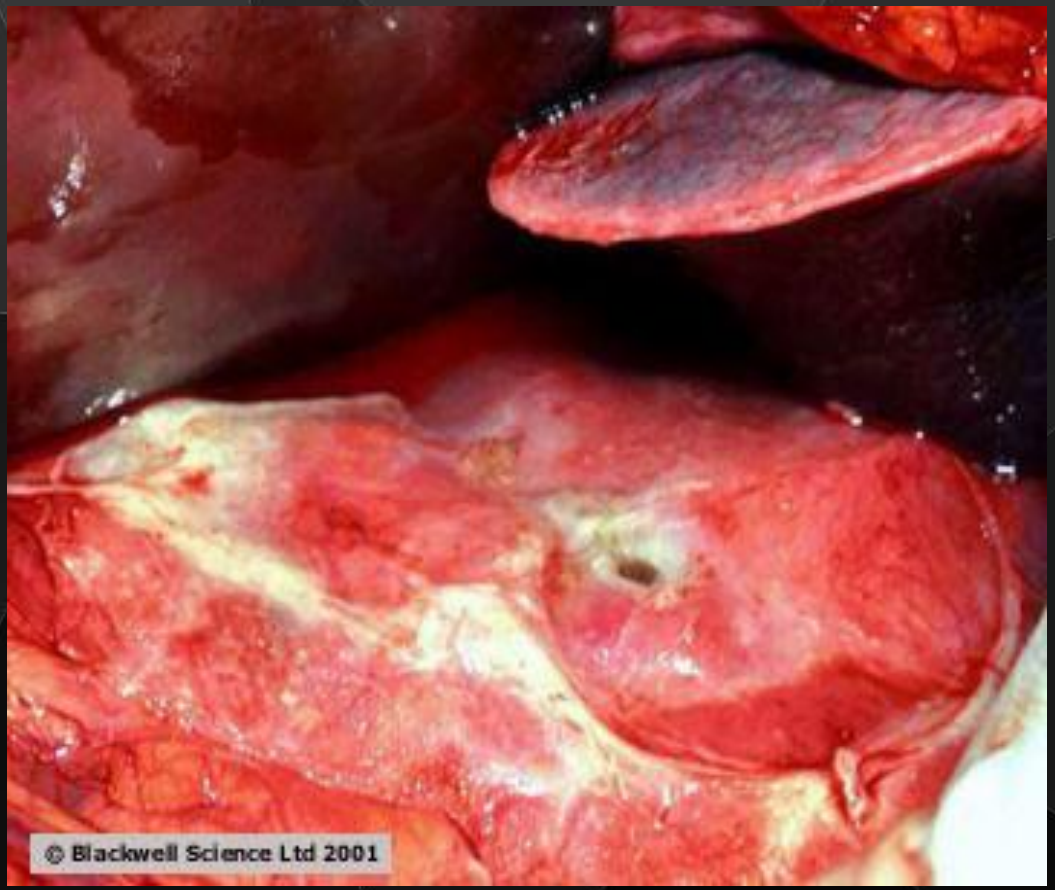


On X-ray is crescent-shaped illumination under the diaphragm

Perforation

Treatment

- Hospitalisation and analgesia
- The treatment is **principally surgical**
- Midline laparotomy
- Thorough peritoneal toilet
- Duodenal ulcer, close and patch with omentum
- Gastric ulcer, should if possible, excised and closed
- If suturing is not possible, Billroth resection.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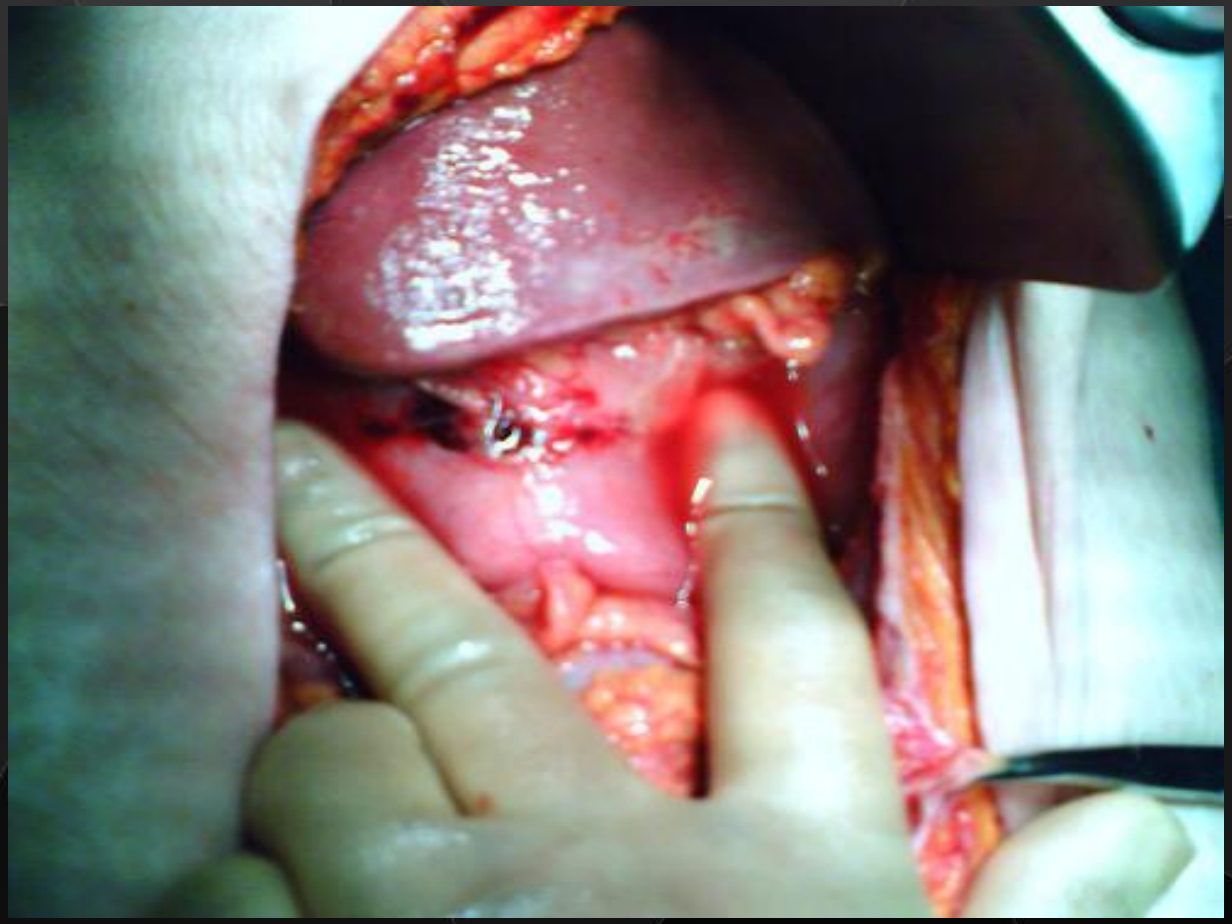
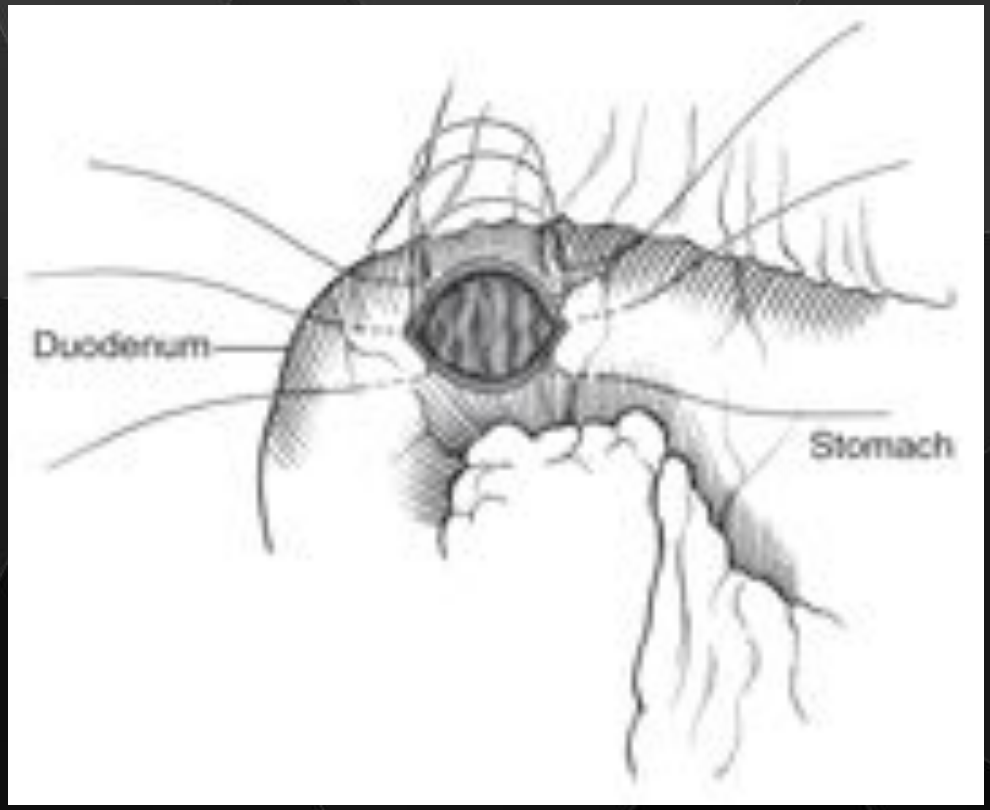
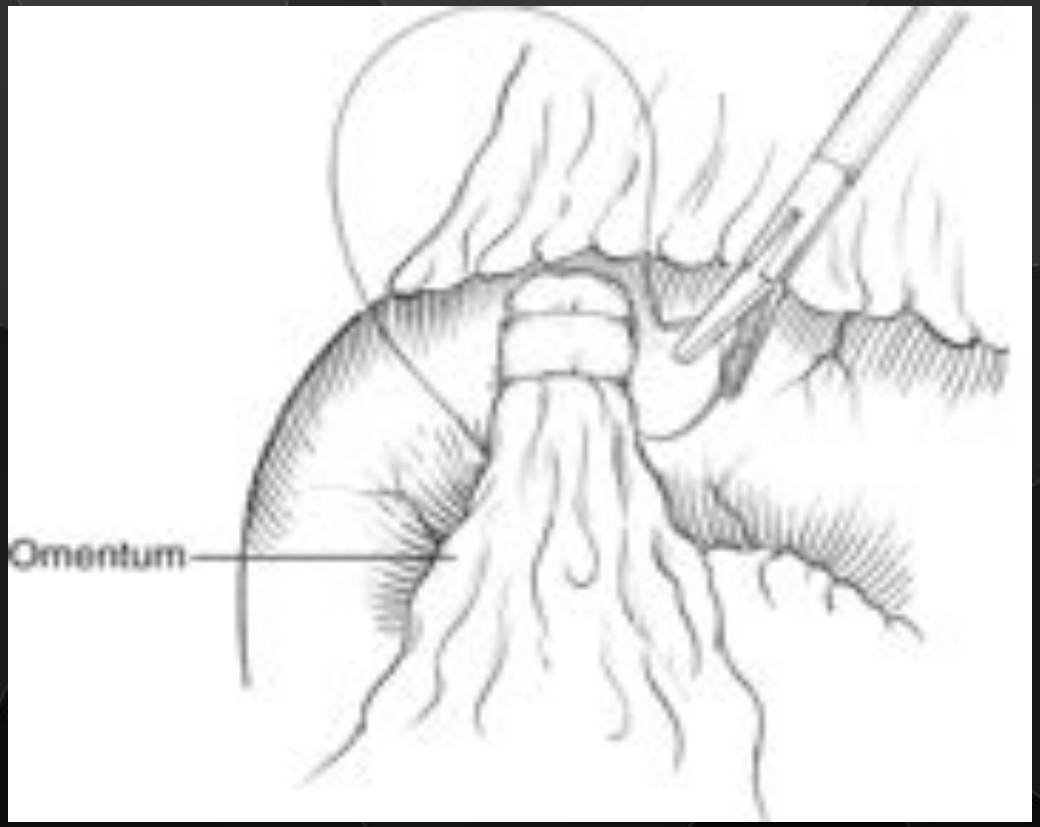


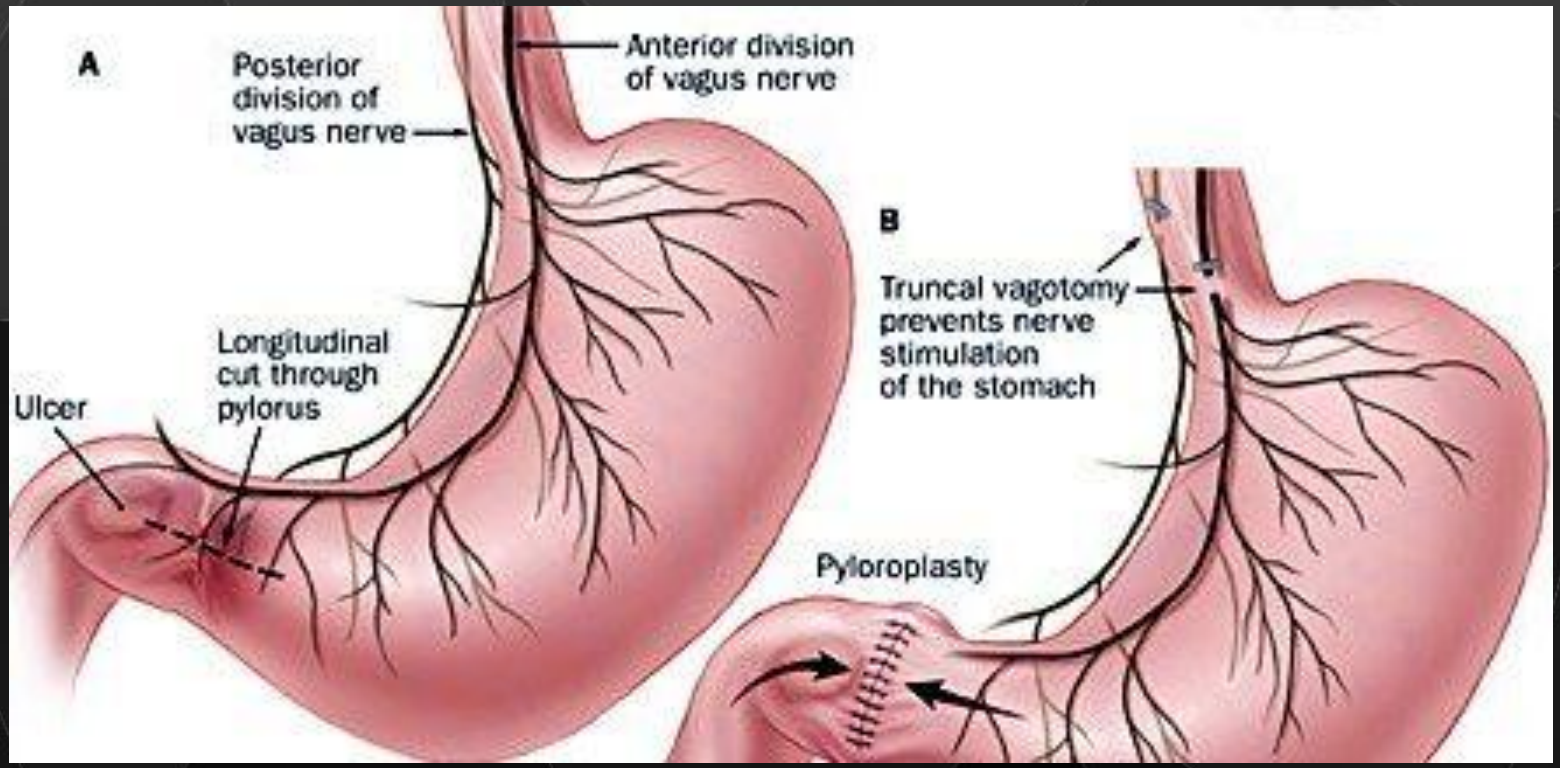
Suturing of Perforated Peptic Ulcer



Graham Omental Patching



Excision of ulcer with pyloroplasty by JADD



Perforation

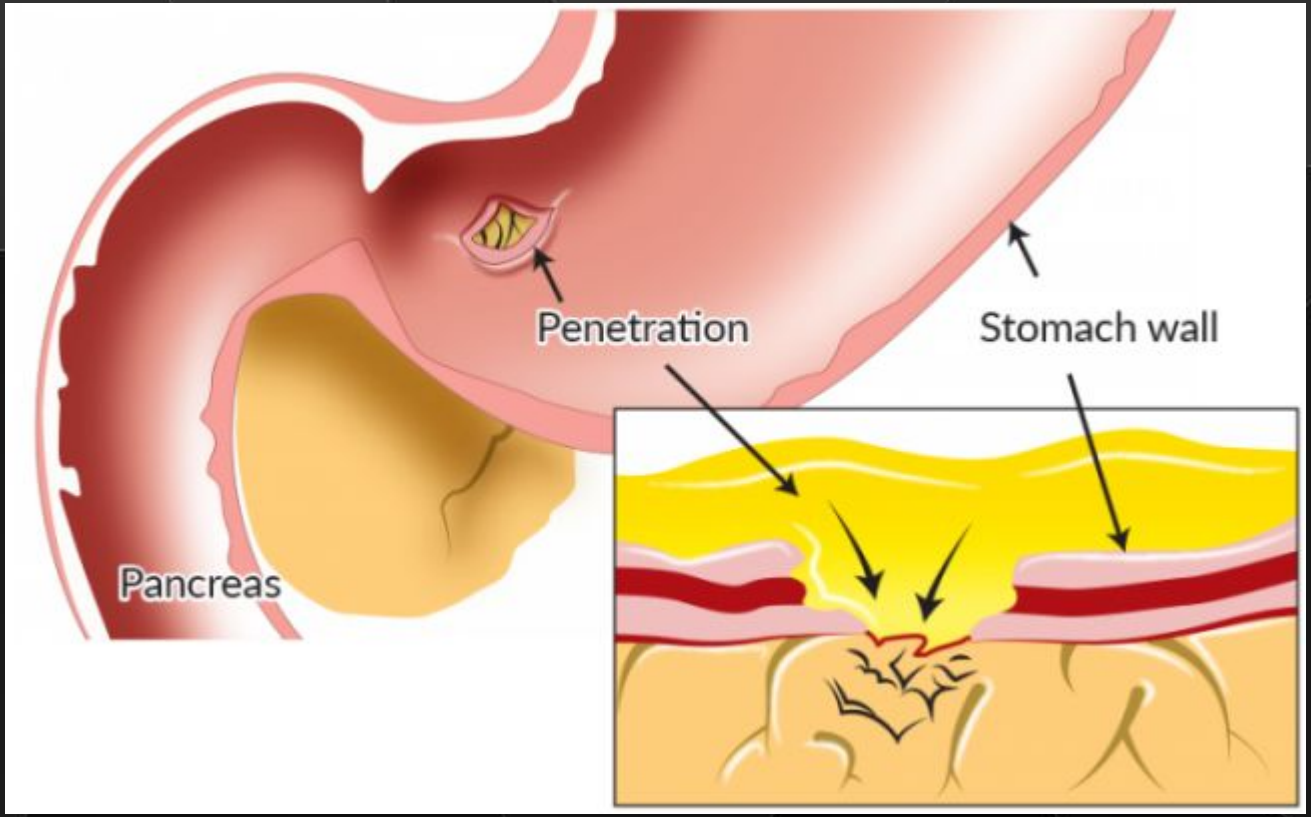
Treatment

- Systemic antibiotics
- Vagotomy, highly selective vagotomy
- Minimally invasive
- Conservative treatment
  - Small leak
  - Mild peritoneal contamination
  - I.V fluid, N/G tube
- Proton pump inhibitors lifelong especially if to continue on NSAID & H *pylori* eradication therapy

Bleeding

Gastrointestinal bleeding is the most common complication. Sudden large bleeding can be life-threatening. It occurs when the ulcer erodes one of the blood vessels. Bleeding can occur as slow blood loss that leads to anemia or as severe blood loss that may require hospitalization or a blood transfusion.

Penetration is when the ulcer continues into adjacent organs such as the liver and pancreas



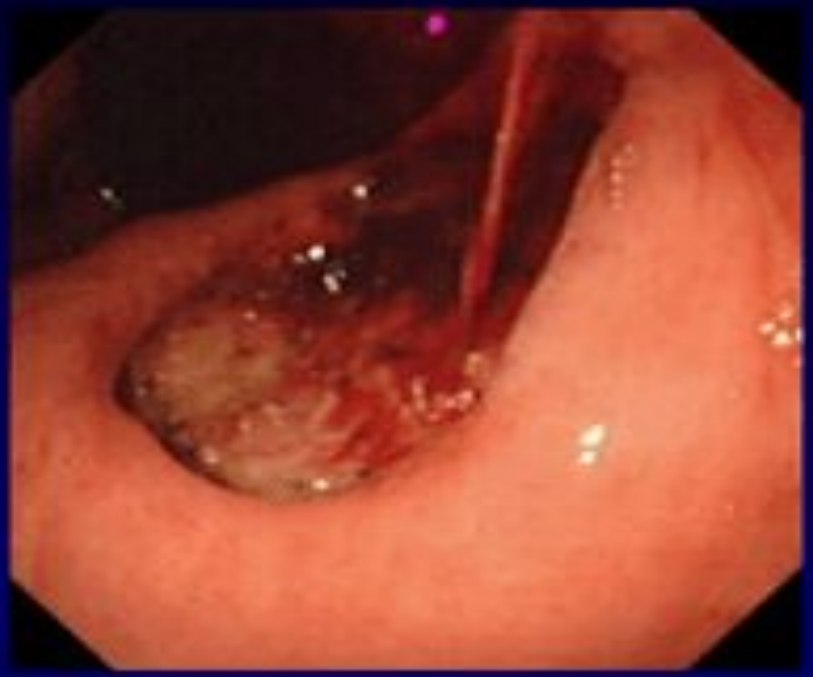
Penetration is a form of perforation in which the perforating ulcers erode the whole thickness of the stomach or duodenal wall, into adjacent abdominal organs such as liver, pancreas, bile duct or intestines. Pancreas is the most typical site of penetration. A combination of serious ulcer symptoms including abnormal pain distribution and decreased response to conventional treatment are signs of ulcer penetration.

Bleeding

- **Epidemiology**
- Mirror that of PPU
- NSAID

Peptic Ulcer Bleeding

- 5% of emergency admissions
- 80% stop spontaneously
- 10% of patients die
- Rebleeding increases mortality by 10x



Classification of bleeding according to J. Forrest (1974)

Classfication	Description
Acute hemorrhage	
Class Ia	Spurting hemorrhage
Class Ib	Oozing hemorrhage
Signs of recent hemorrhage	
Class IIa	Nonbleeding visible vessel
Class IIb	Adherent clot
Class IIc	Flat pigmented spot
Lesions without active bleeding	
Class III	Clean ulcer base

Bleeding

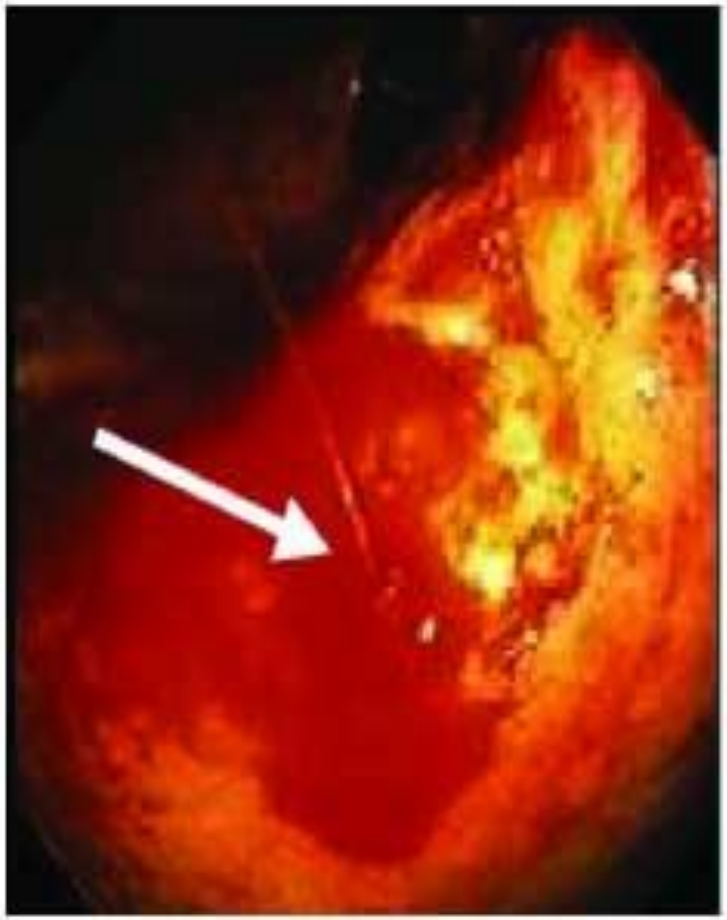
Treatment / Medical

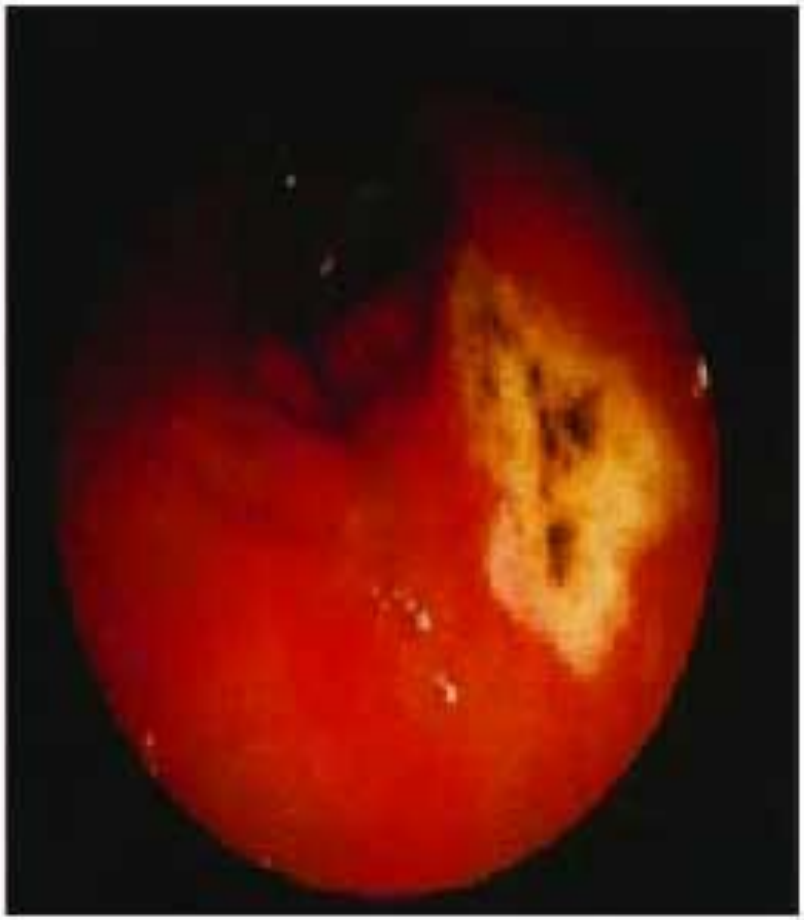
- Limited efficacy

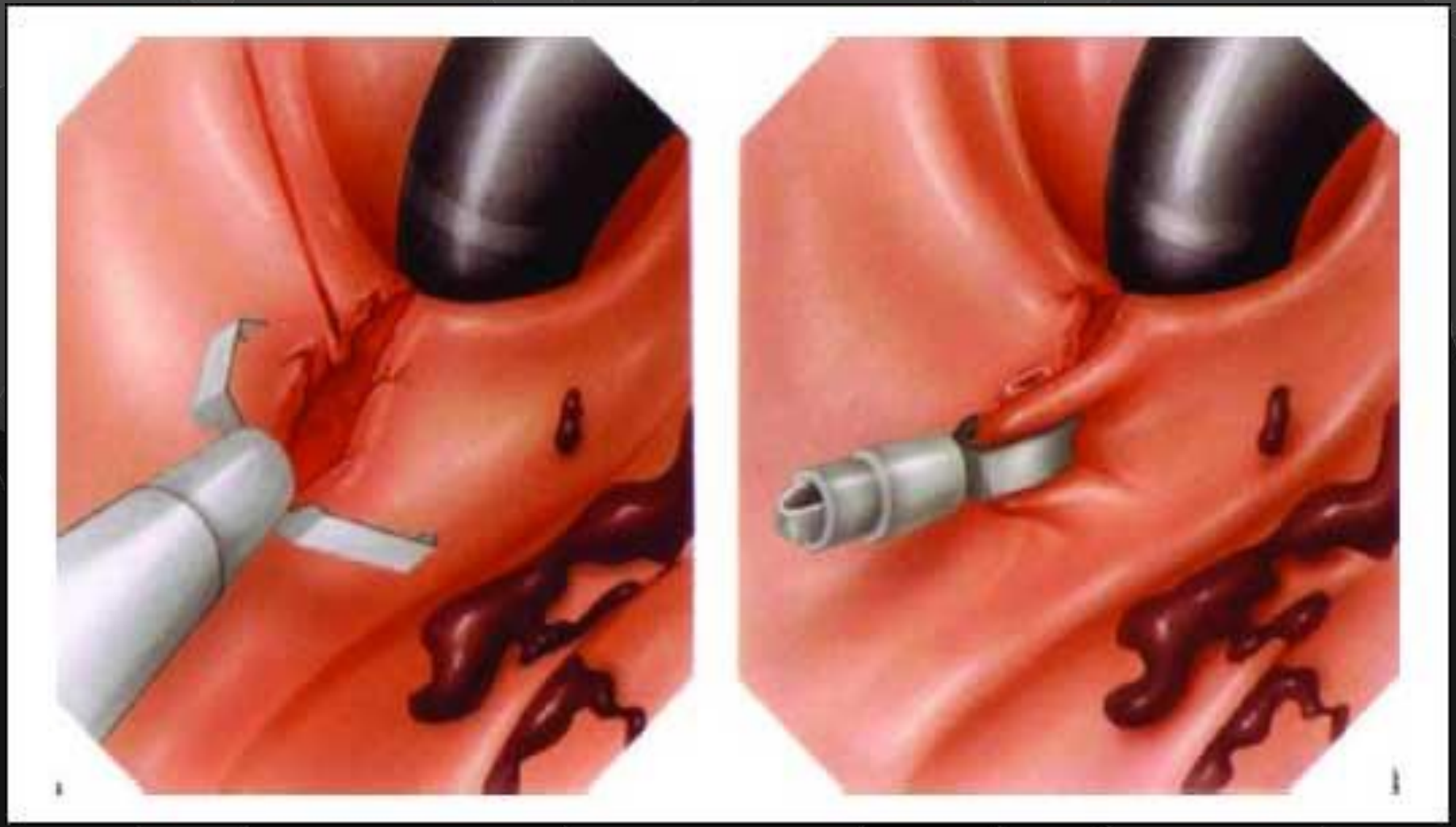
All patients are started on PPI (omeprasole)

- Endoscopic control
  - LASER & Argon diathermy
  - Injection
  - May have some value
  - Never effective in patients who are bleeding from large size vessels

Bleeding Peptic Ulcer







Bleeding

<u>Treatment / Surgical</u>

Indications

- Patient continue to bleed
- Visible vessel in ulcer base
- Spurting vessel
- Ulcer with a clot
- Elderly
- Patient who has required more than 6 units of blood

Bleeding

Treatment / Surgical

- Aim to stop bleeding
- Upper midline incision
- Site usually localized by prior Endoscopy
- Duodenal mobilization
- Pyloro-duodenotomy
- Suture that under-run the bleeding vessel
- Gastric ulcer, excise ulcer if possible, if not, under-run bleeding vessel and take biopsies

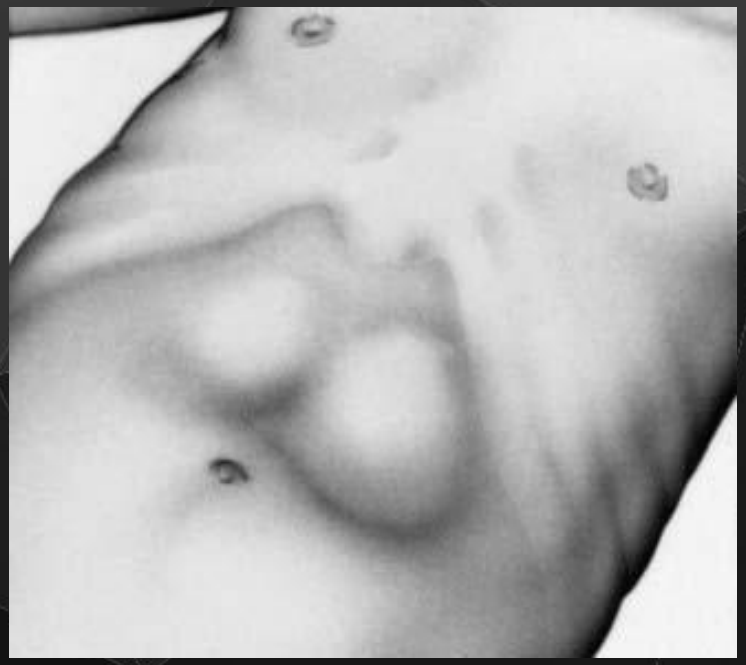
Bleeding

- **Treatment / Surgical**
- Definitive acid lowering surgery is not required
- PPI (omeprasole)
- Anti H *pylori*

Stenosis

Stenosis is usually found in the 1st part of duodenum

This condition occurs less and less nowadays



Scar tissue Scarring and swelling due to ulcers causes narrowing in the duodenum and **gastric outlet obstruction**. Patient often presents with severe vomiting. Peptic ulcers can also produce scar tissue that can obstruct passage of food through the digestive tract, causing you to become full easily, to vomit and to lose weight.

ULCEROUS STENOSIS CLASSIFICATION

A

I — compensated;

II — subcompensated;

III — decompensated.

B

I — stenosis of goalkeeper;

II — stenosis of bulb of duodenum;

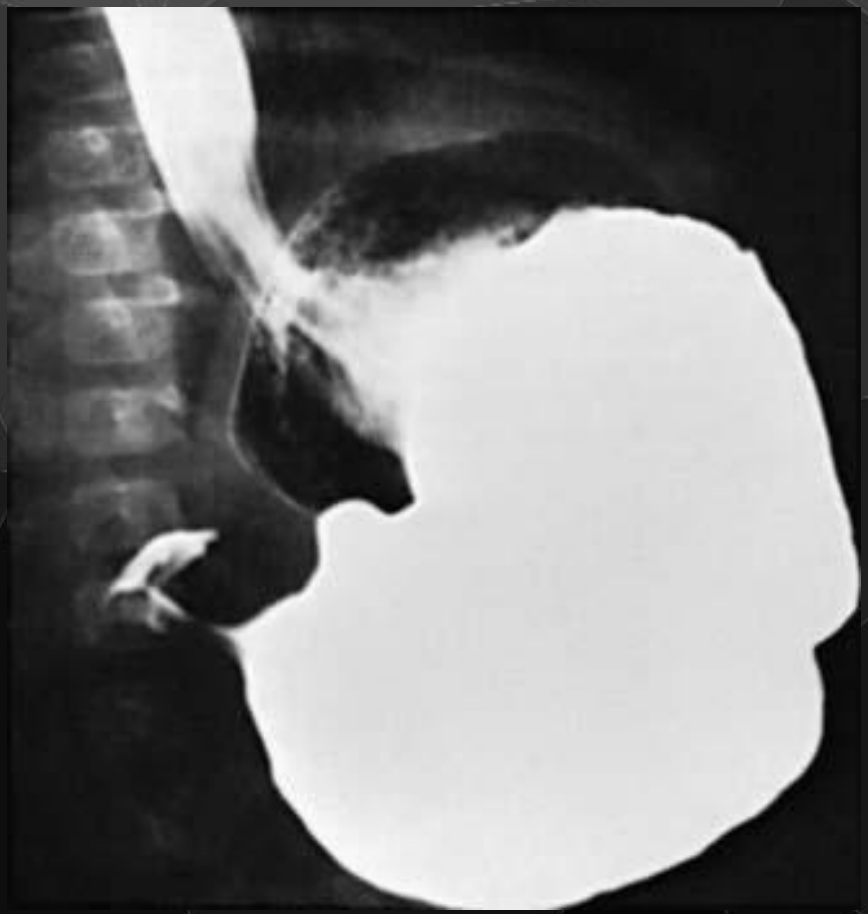
III — postbulbar duodenal stenosis.

Stenosis

Clinical Features

- Long history of peptic ulcer disease
- Vomiting, unpleasant in nature, totally lacking in bile, containing foodstuff taken several days previously
- Weight loss
- Patient looks unwell and dehydrated
- On examination you can see distended stomach, succussion splash may be audible on shaking the patient's abdomen

X-ray of Pyloric Stenosis



Stenosis

Metabolic effects

- Vomiting of HCl results in hypochloremic acidosis
- Initially Na^+ & K^+ levels are normal
- With dehydration, more profound metabolic abnormalities arise
- Renal dysfunction
- Initially urine has low chloride and high HCO_3 content , HCO_3 is excreted with Na^+, so patient become hyponatremic and more dehydrated

Stenosis

Metabolic effects

- Then because of dehydration, a phase of Na^+ retention follows and K^+ and Hydrogen are excreted in preference
- Paradoxical aciduria
- Hypokalemia
- Alkalosis leads to lowering of circulating ionized calcium and tetany may occur

Stenosis

Management

1) Correct metabolic abnormality

- Rehydration with isotonic saline with K^+ supplementation
- Replacing NaCl and water allows kidney to correct the acid-base abnormality
- Correct anemia which may appear after rehydration

Stenosis

Management

2) Empty the stomach with wide-bore N/G tube, may need lavage

3) Endoscopy and contrast radiology to confirm and R/O malignancy

4) Parenteral anti-secretory agent

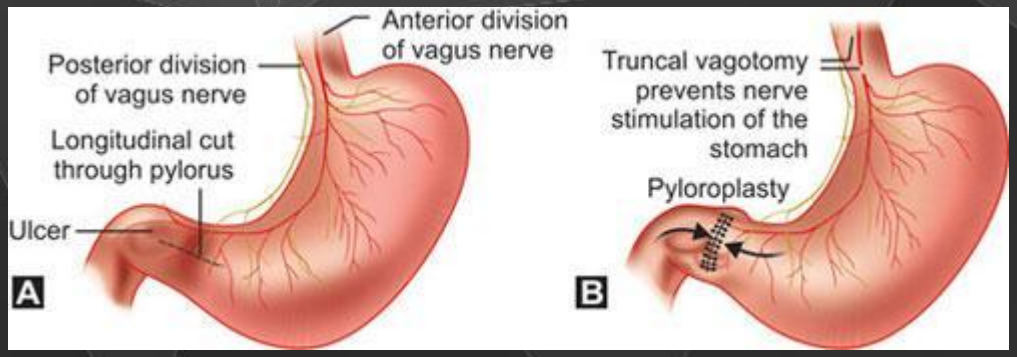
Stenosis

Management

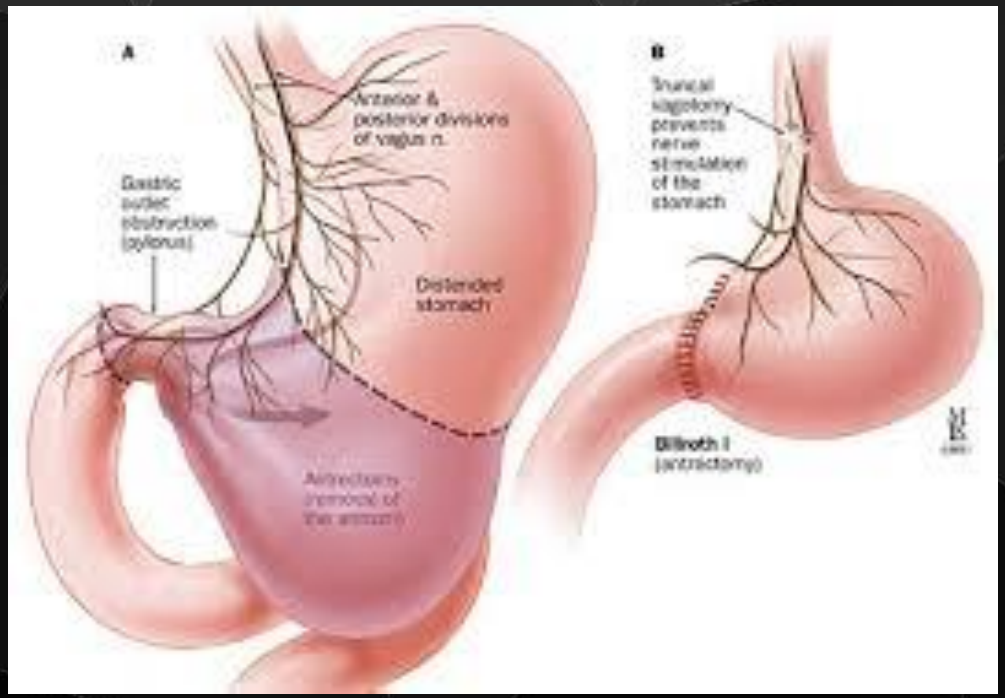
- Early cases may settle with conservative measurement, presumably as the edema around the ulcer diminishes as the ulcer is healed
- Gastroenterostomy
- Endoscopic balloon dilatation
  - Effective in early cases
  - Risk of perforation
  - Dilatation may have to be performed several times

Treatment / Surgical

Pyloroplasty with vagotomy (for I & II type)



Choice of resection treatment (for III type)



The End