

# Digestive system

University of Jordan  
Faculty of Medicine  
Batch of 2013-2019



☒ Slide ☐ Sheet ☐ Handout ☐ Other

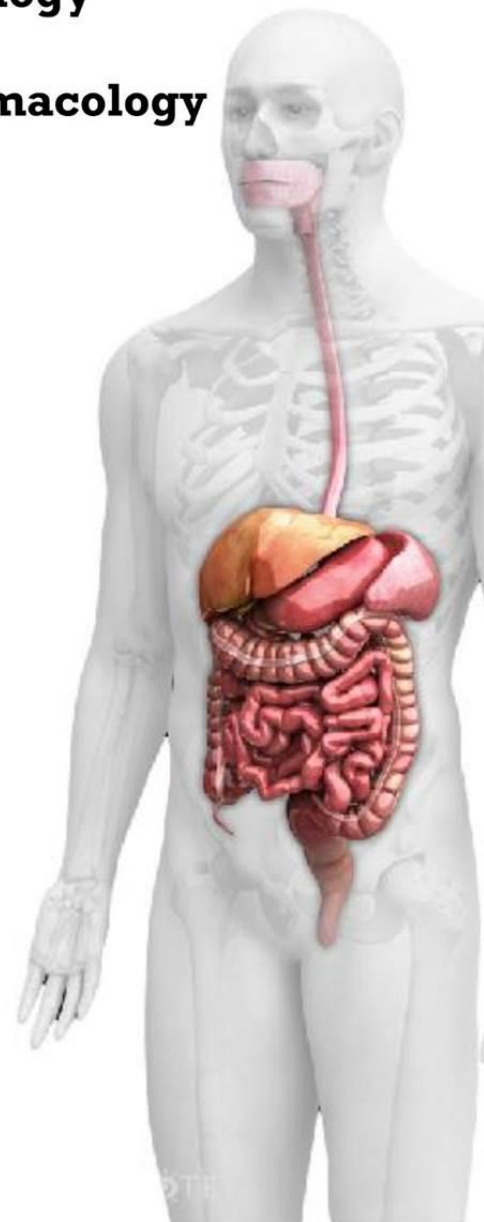
- |   |                                       |
|---|---------------------------------------|
| <input type="checkbox"/> Anatomy              | <input type="checkbox"/> Embryology   |
| <input type="checkbox"/> Physiology           | <input type="checkbox"/> Histology    |
| <input checked="" type="checkbox"/> Pathology | <input type="checkbox"/> Pharmacology |
| <input type="checkbox"/> Microbiology         | <input type="checkbox"/> PBL          |

Slide #: 2

Doctor's name: Dr Mazen

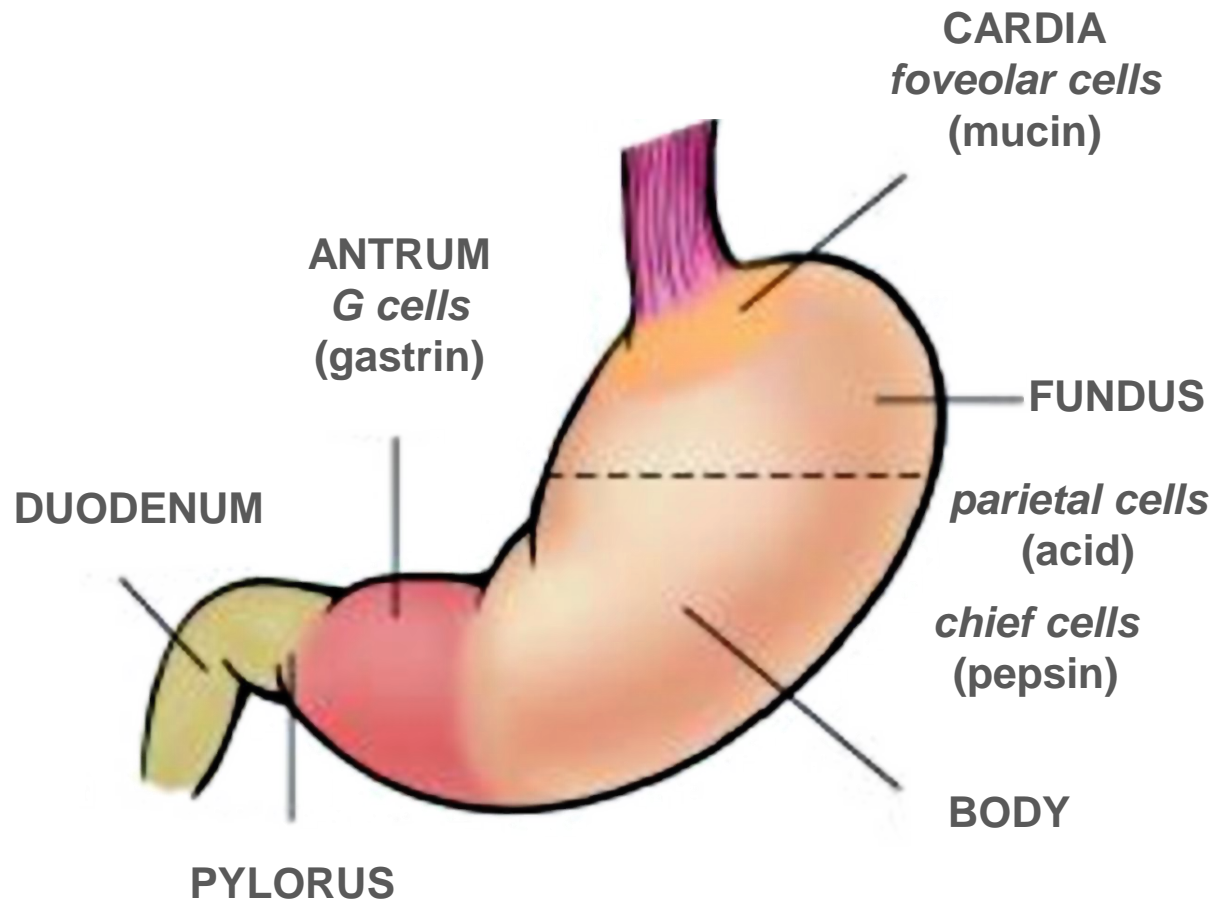
Date: 31/3/2015

Price:





Stomach



## The Stomach

Gastrin stimulates luminal acid secretion by parietal cells within the gastric fundus and body

## NORMAL

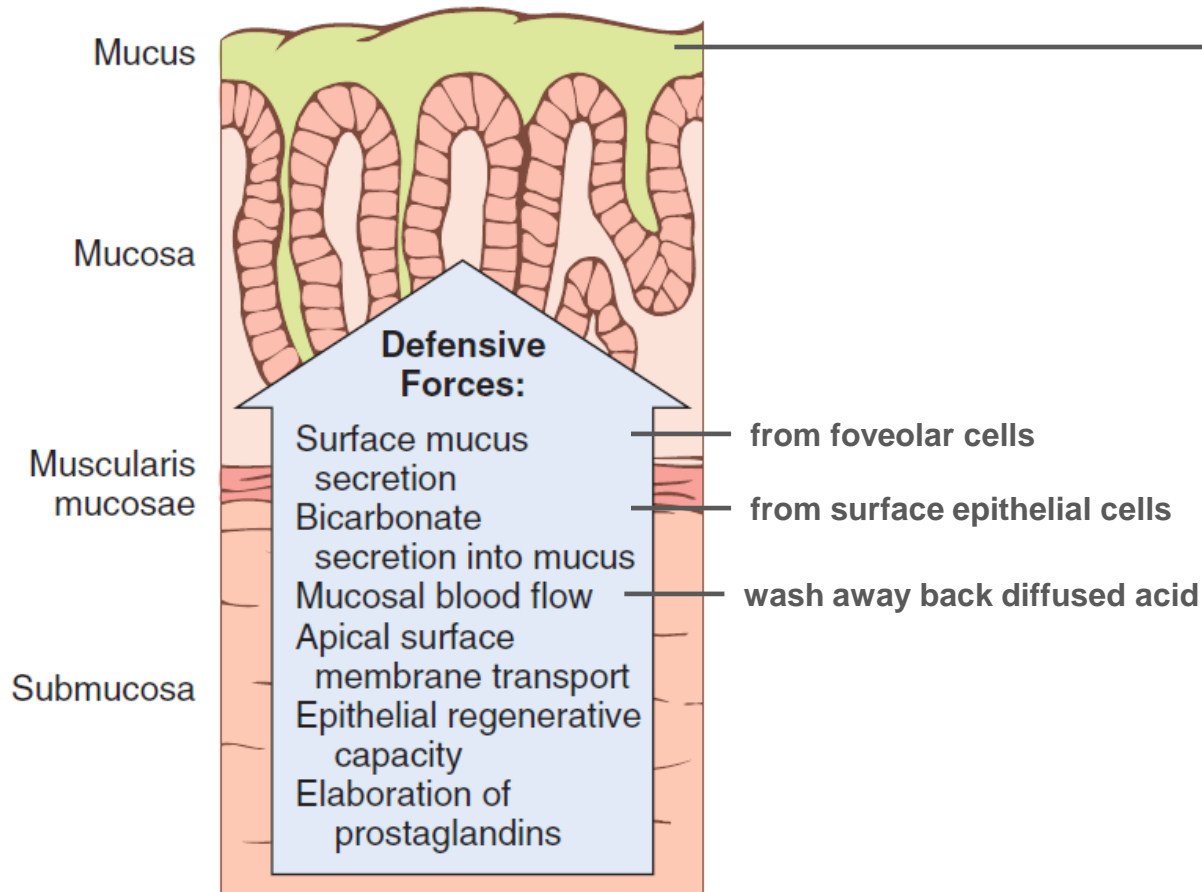
### Damaging Forces:

Gastric acidity  
Peptic enzymes

## Protective forces of the stomach

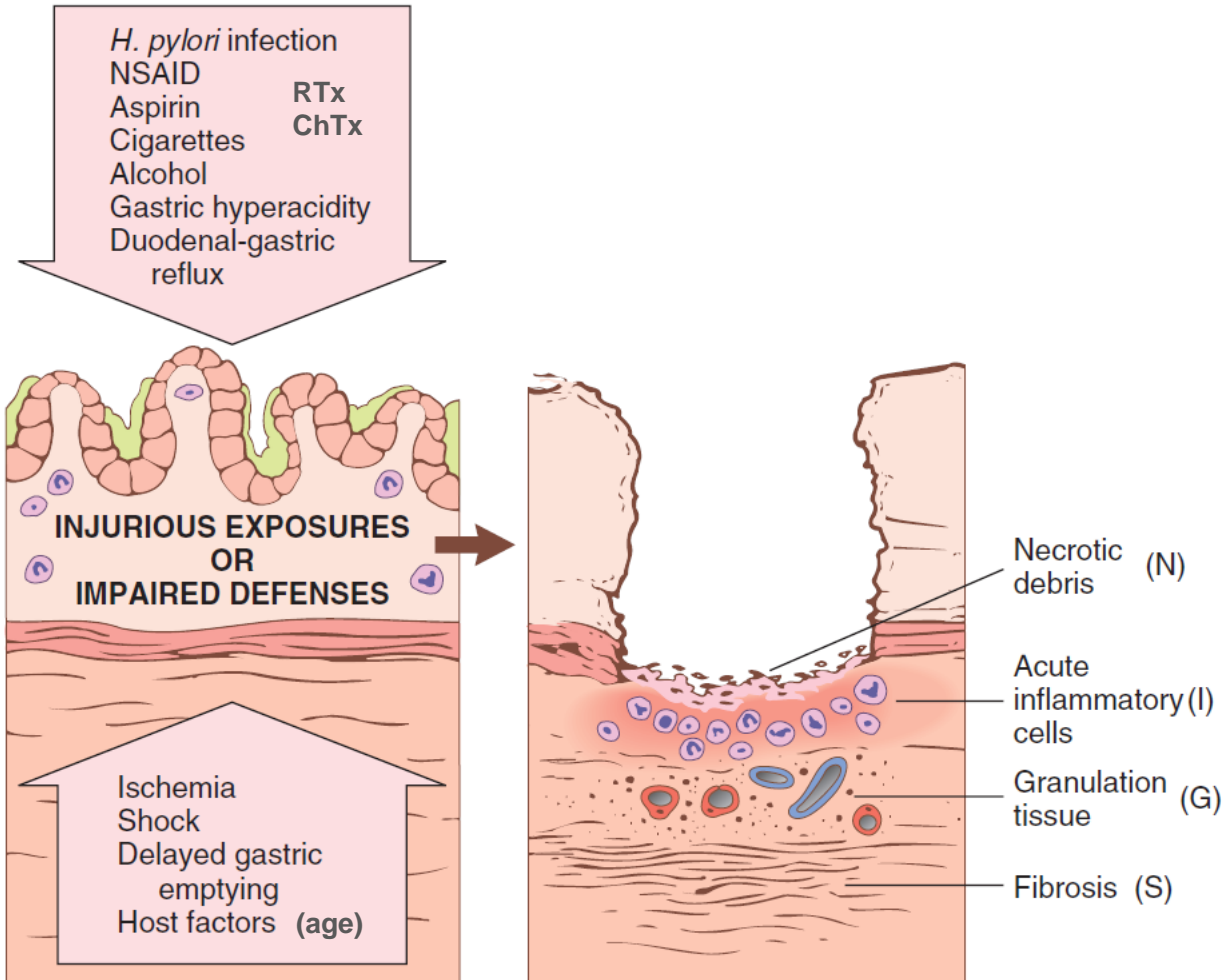
pH ~1

“unstirred” layer to prevent cell damage



## INJURY

## ULCER



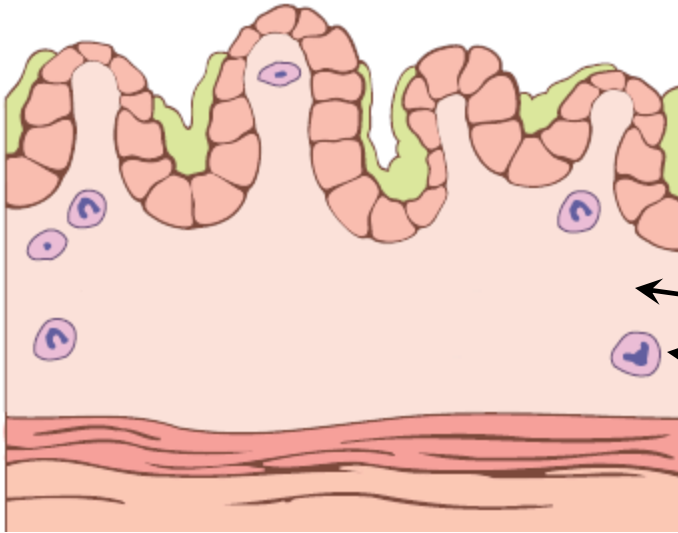
## Acute Gastritis

Disruption of protective barrier → transient mucosal inflammatory process:

- Asymptomatic or
- Epigastric pain + N/V
- Erosion/ulceration
- Bleeding

Morphology:

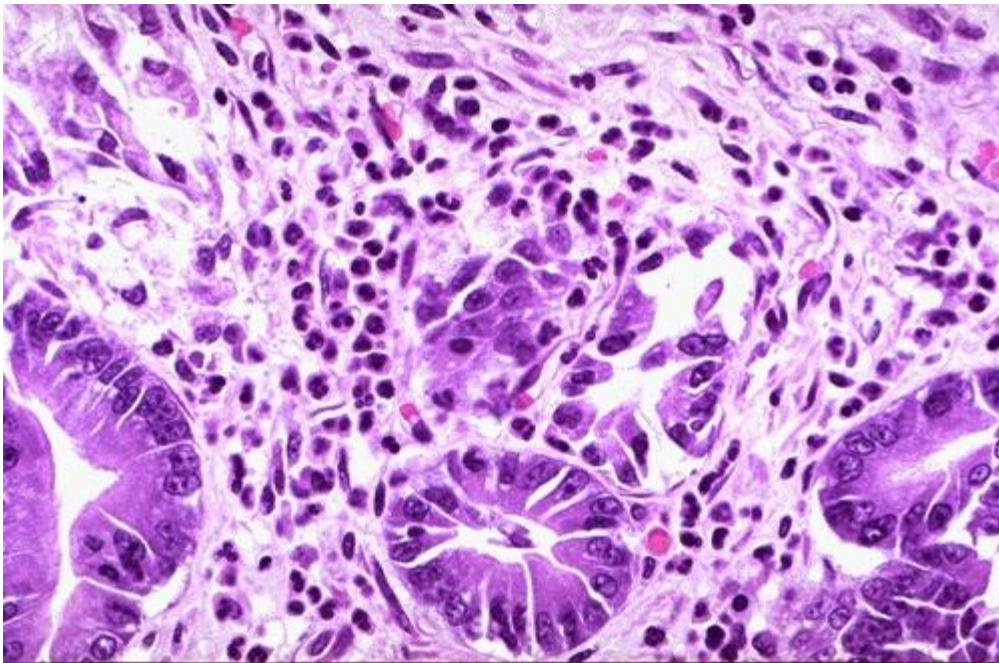
- Edema/congestion
- Erosion
- Hemorrhage



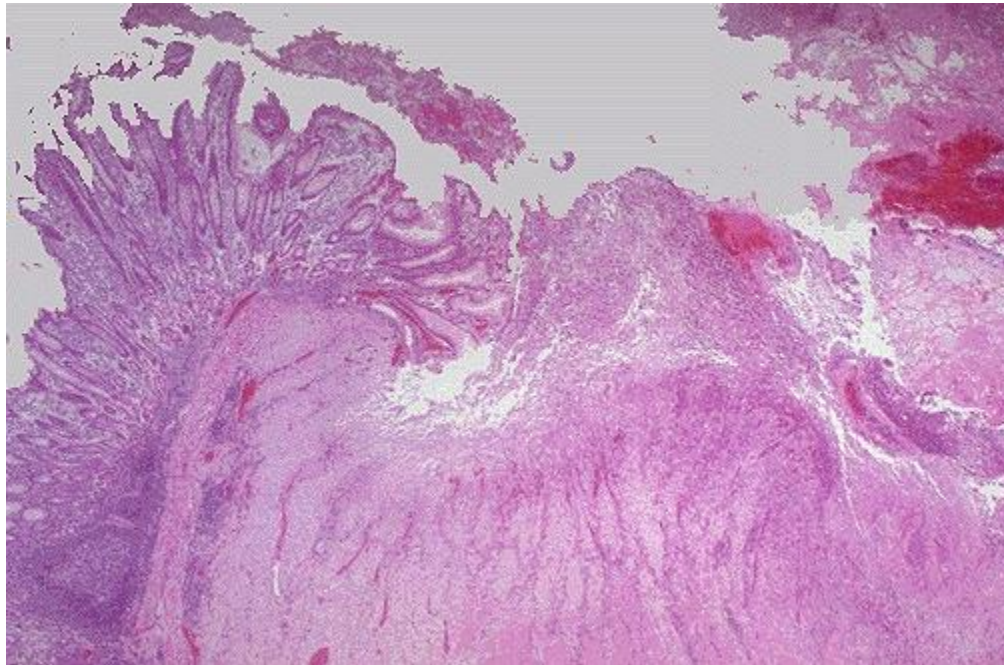
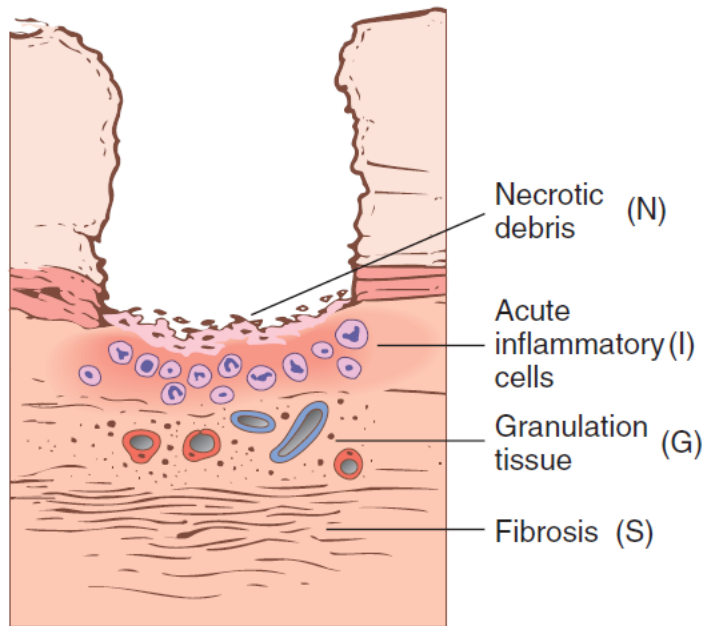
## Acute Gastritis

Mild acute gastritis

- Intact epithelium
- Edema/congestion
- Scattered neutrophils







## Acute Peptic Ulceration

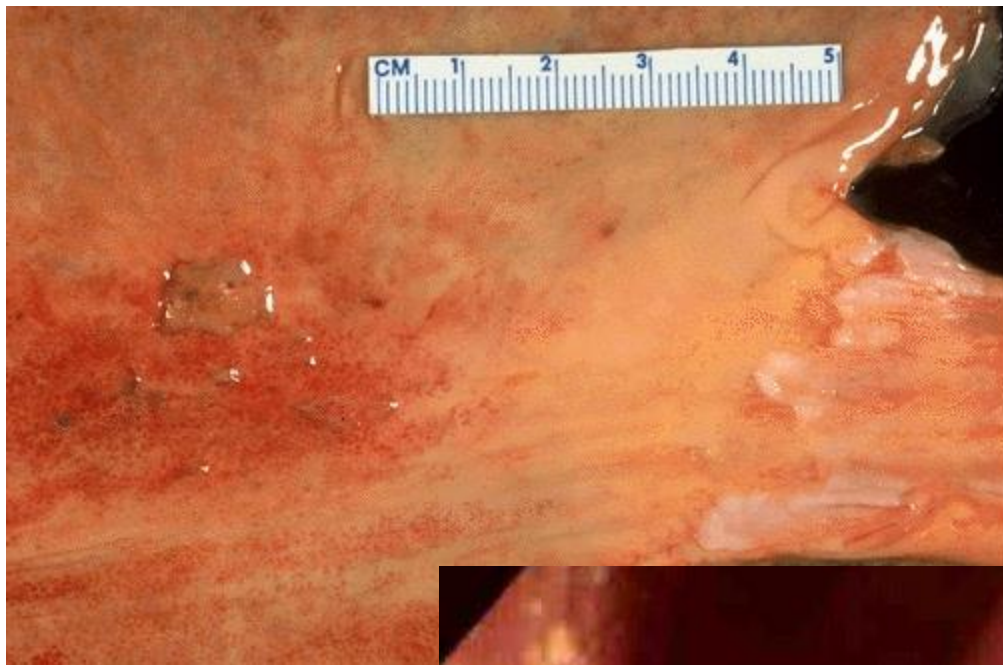
NSAIDs (direct +  $\downarrow$ COX)  
Severe physiologic stress

Stress ulcers: critically ill patients (shock, sepsis, severe trauma ~ acidosis)

Curling ulcers: with severe burns or trauma

Cushing ulcers: vagal stimulation with intracranial injury

Shallow erosions to mucosal penetration



## Acute Peptic Ulceration

Range from erosions to mucosal penetration

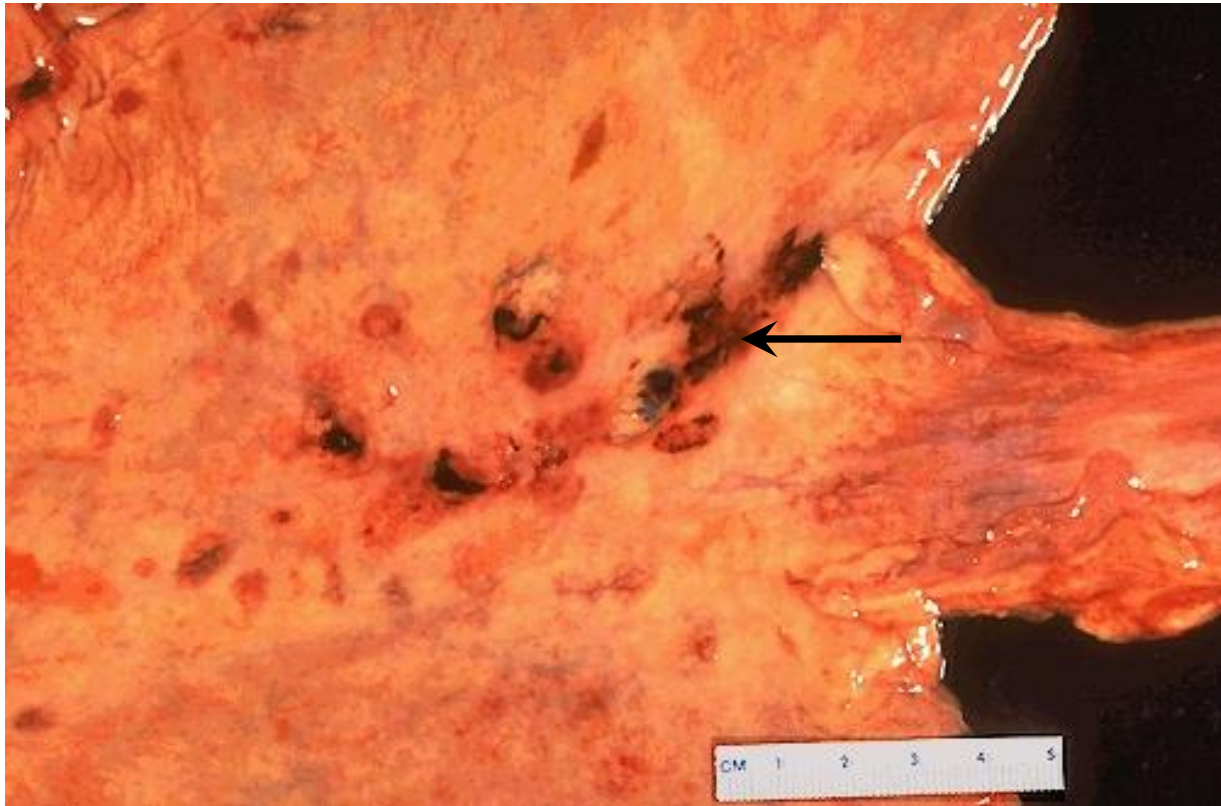
Rounded <1cm

Sharply demarcated

Normal adjacent mucosa

Singly or more commonly multiple ulcers





## Acute Peptic Ulceration

The ulcer base frequently is stained brown to black by acid digested RBCs

### Symptoms

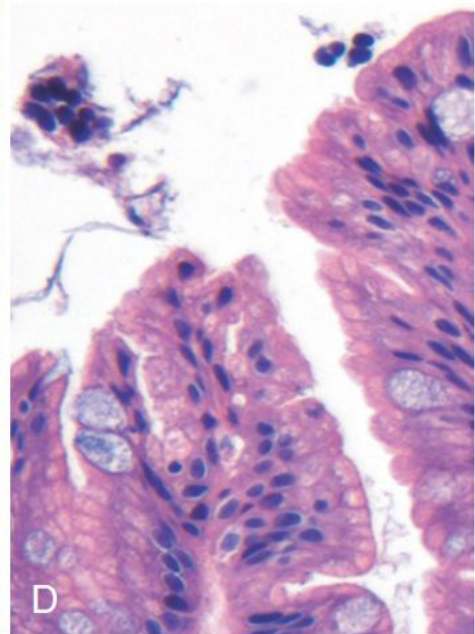
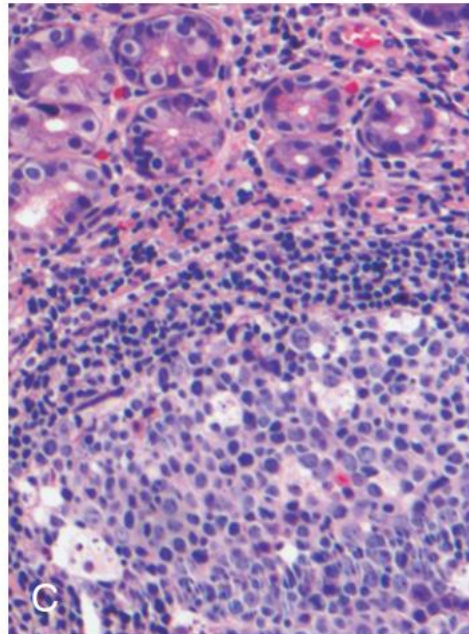
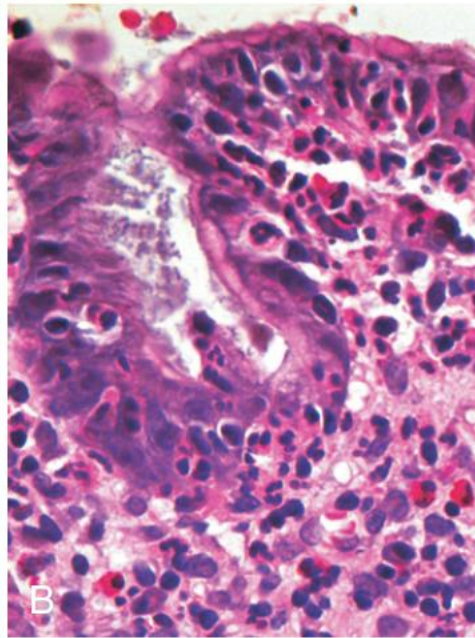
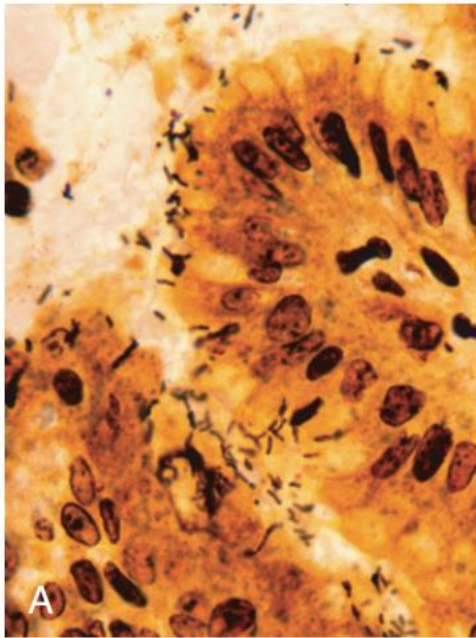
- N/V
- Hematemesis (coffee ground)

### Complications

- Excessive bleeding
- Perforation

### Tx

- PPI
- H2 receptor antagonist



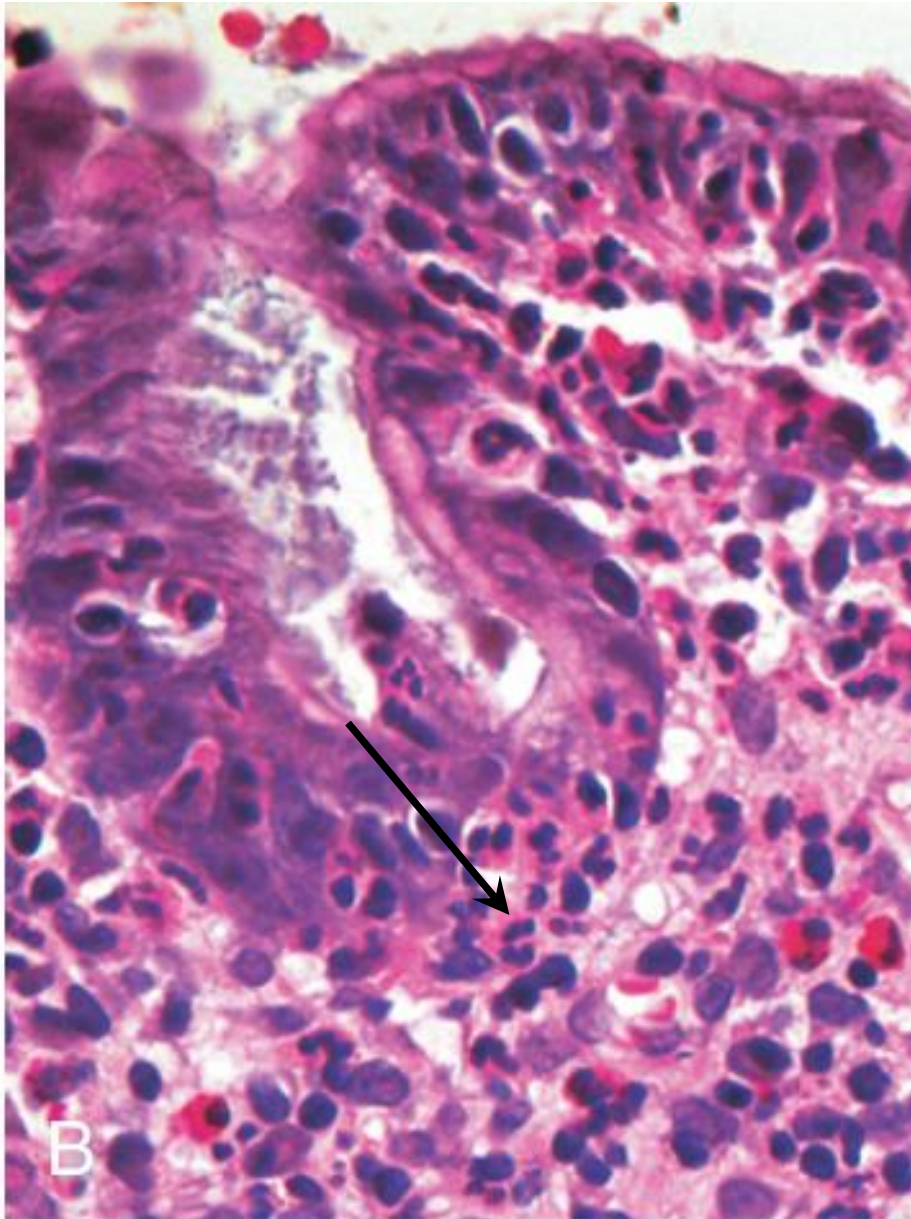
## Chronic Gastritis

Signs & symptoms less severe but more persistent

Hematemesis rare

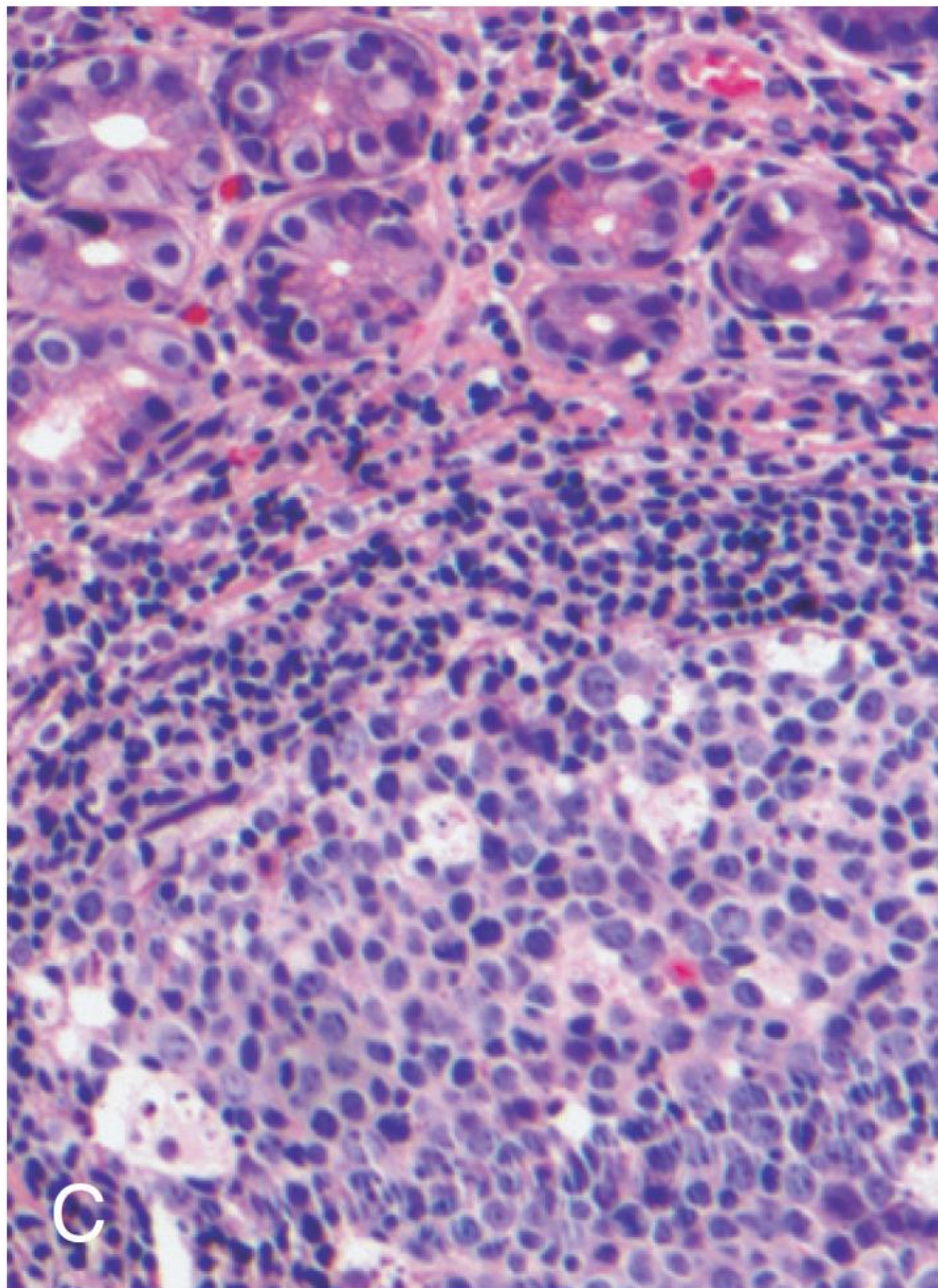
- *H. pylori*
- Autoimmune (atrophic)
- Radiation
- Chronic bile reflux





## Chronic Gastritis

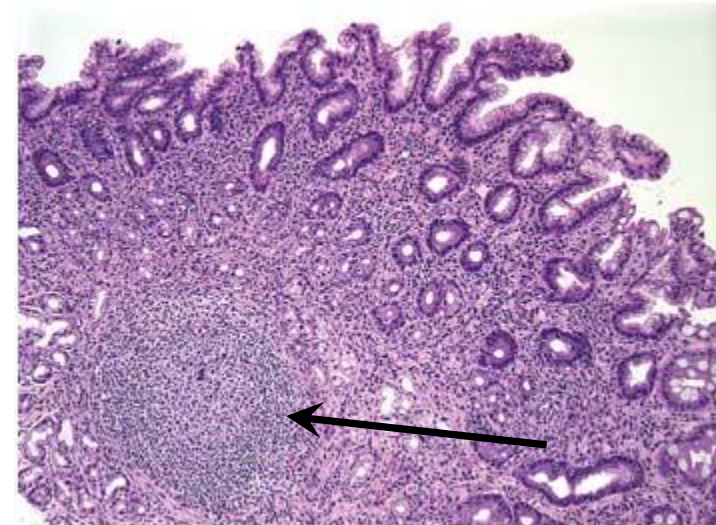
Intraepithelial and lamina propria neutrophils are prominent



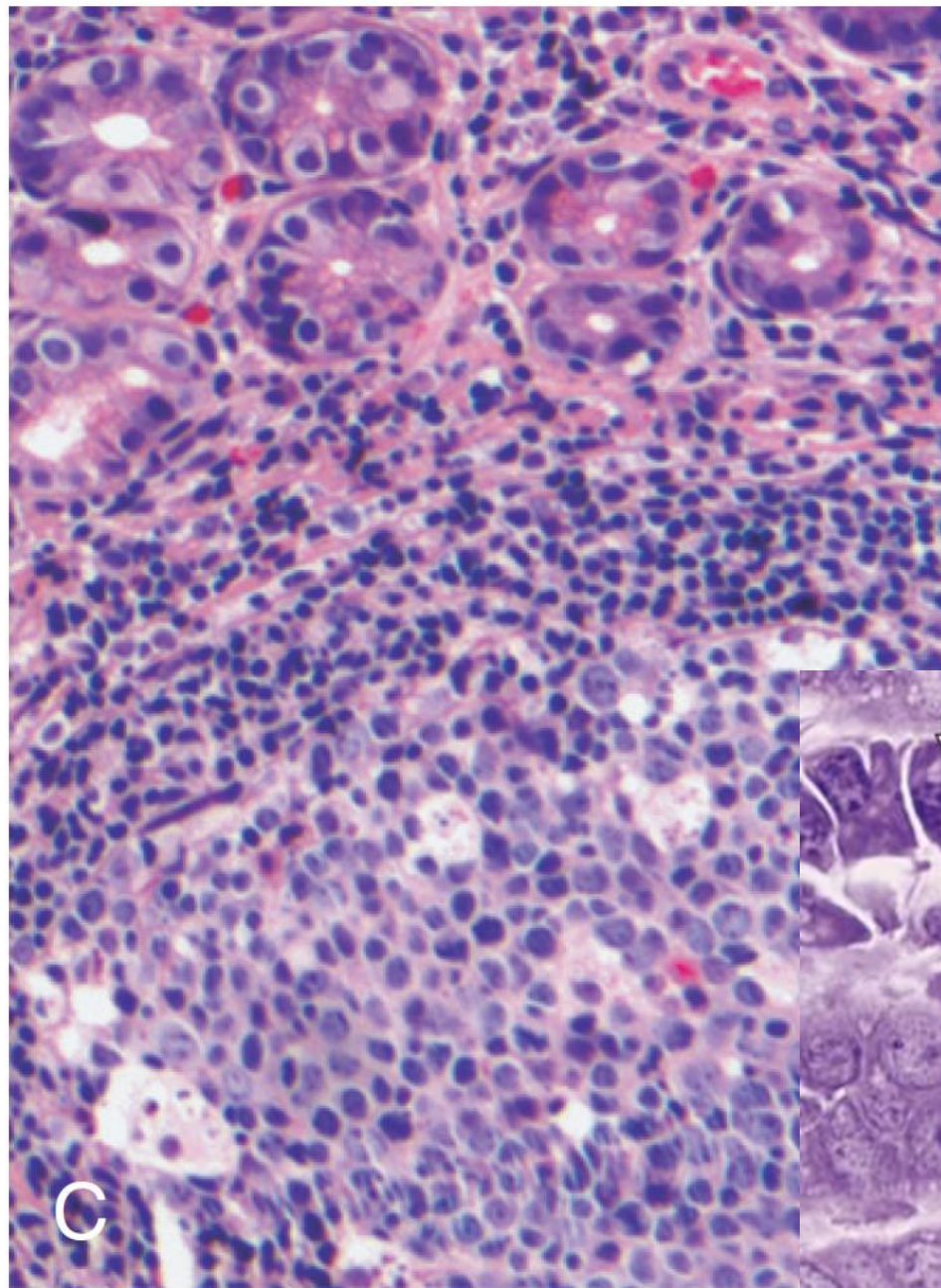
## Chronic Gastritis

Lymphoid aggregates  
with germinal centers

Subepithelial plasma  
cells



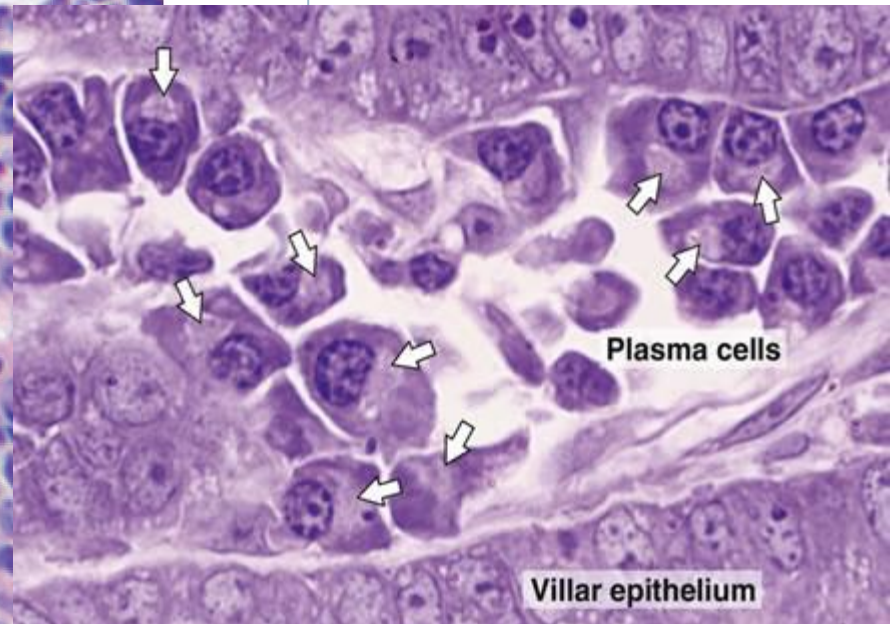




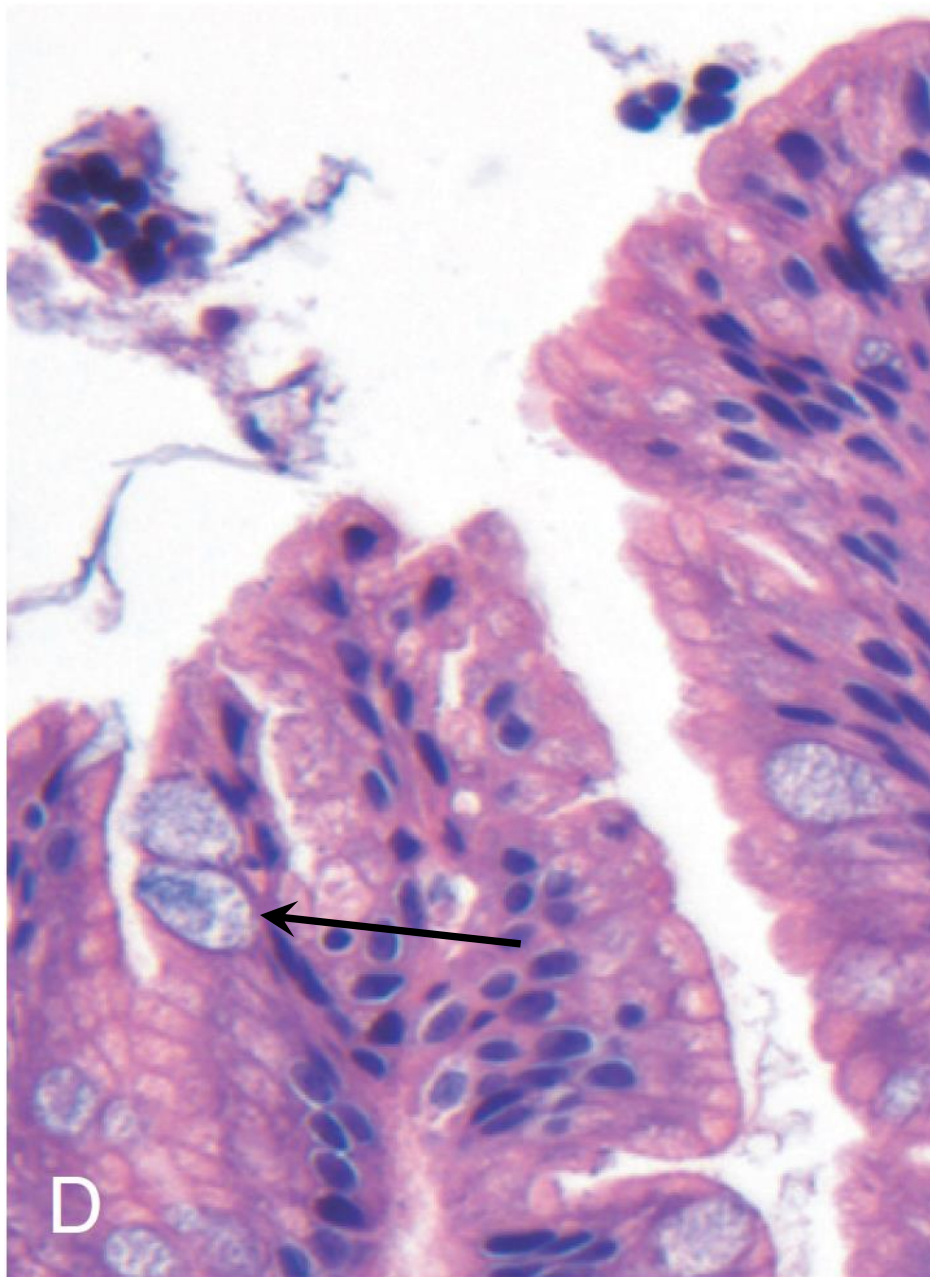
## Chronic Gastritis

Lymphoid aggregates  
with germinal centers

Subepithelial plasma  
cells





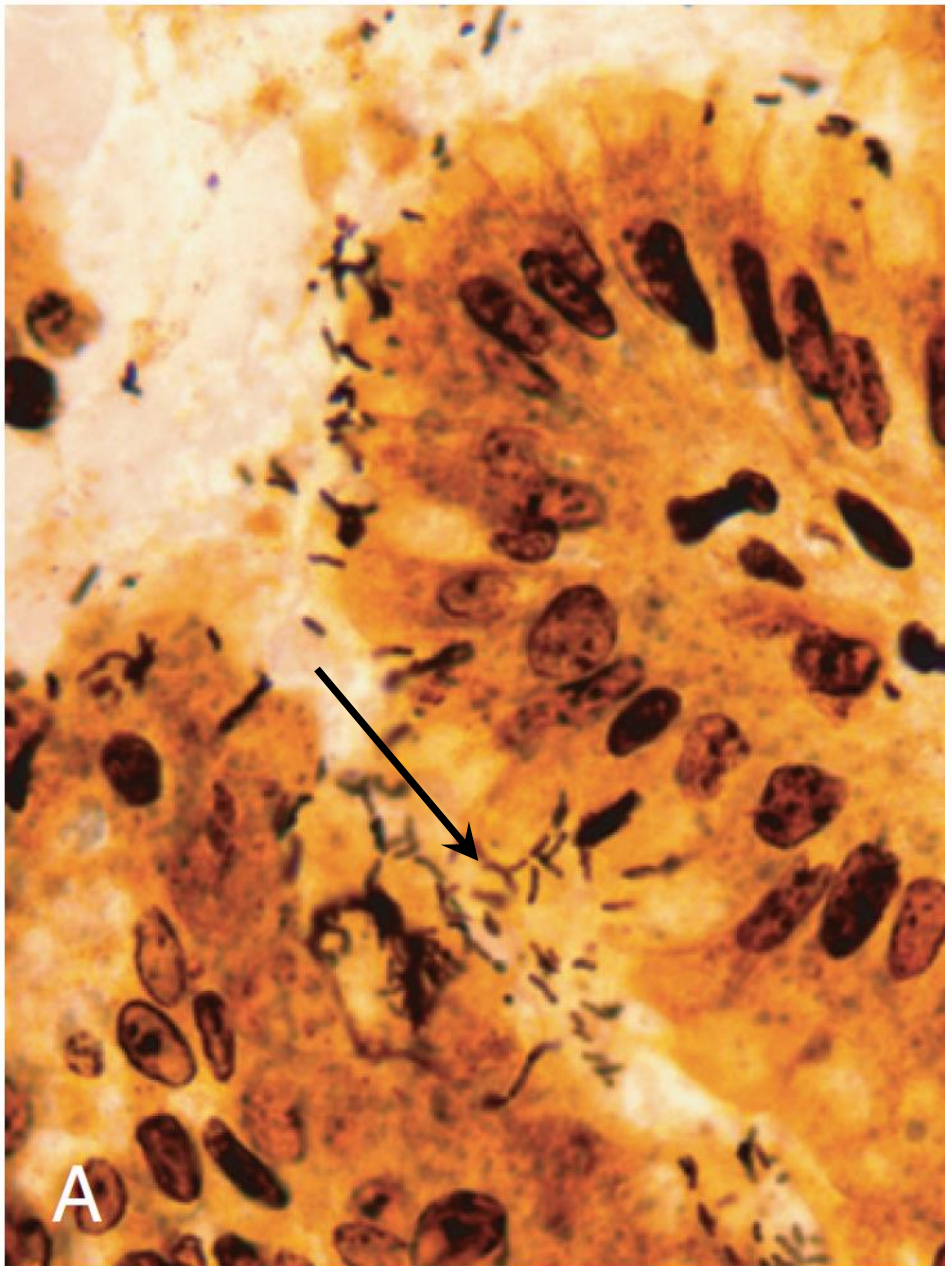


## Chronic Gastritis

Intestinal metaplasia

Goblet cells

Increased risk for  
adenocarcinoma

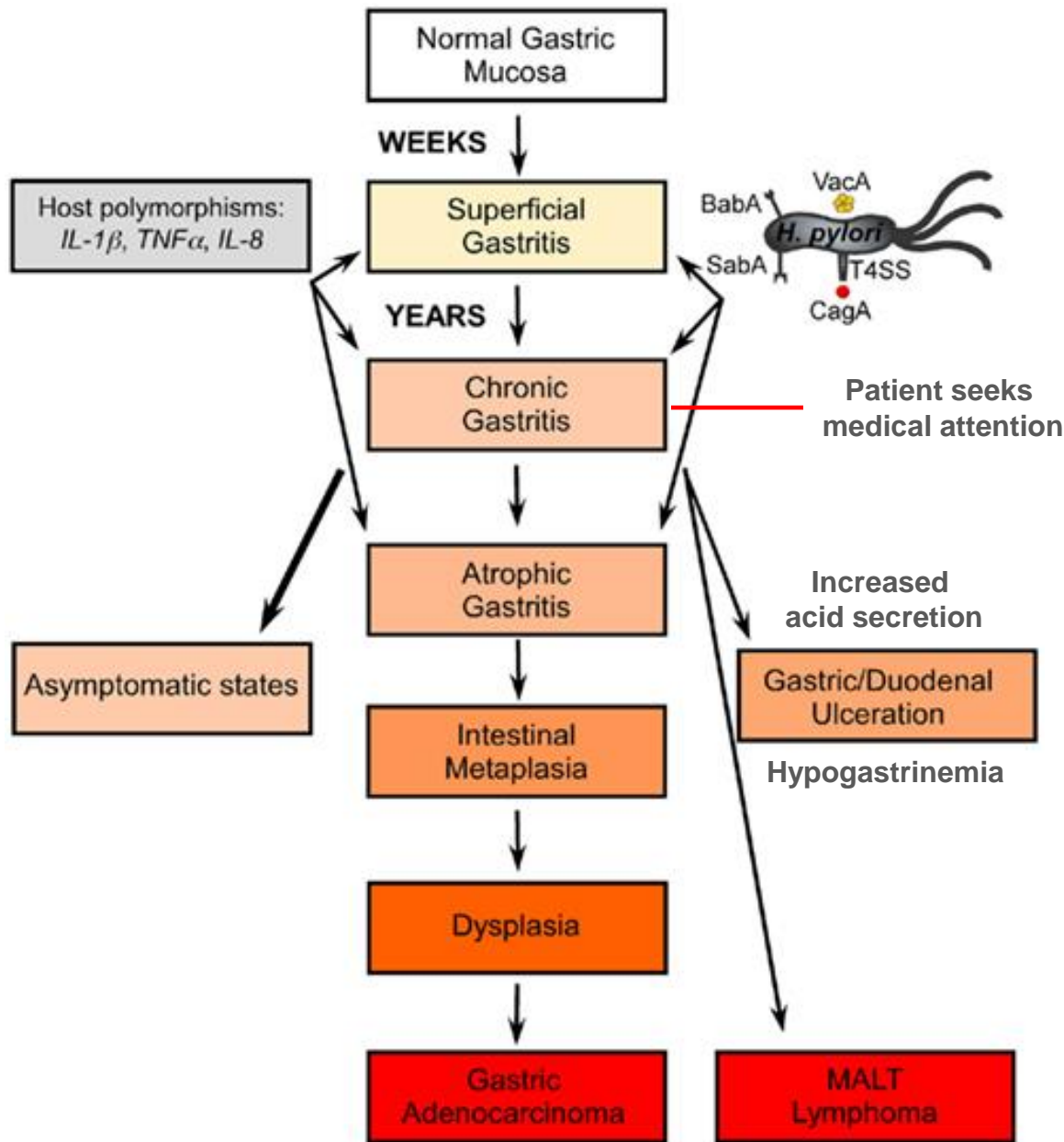


## ***H. pylori* Gastritis**

Warthin-Starry silver stain

Spiral-shaped or curved  
bacilli abundant within  
surface mucus

Tropism for foveolar  
epithelium



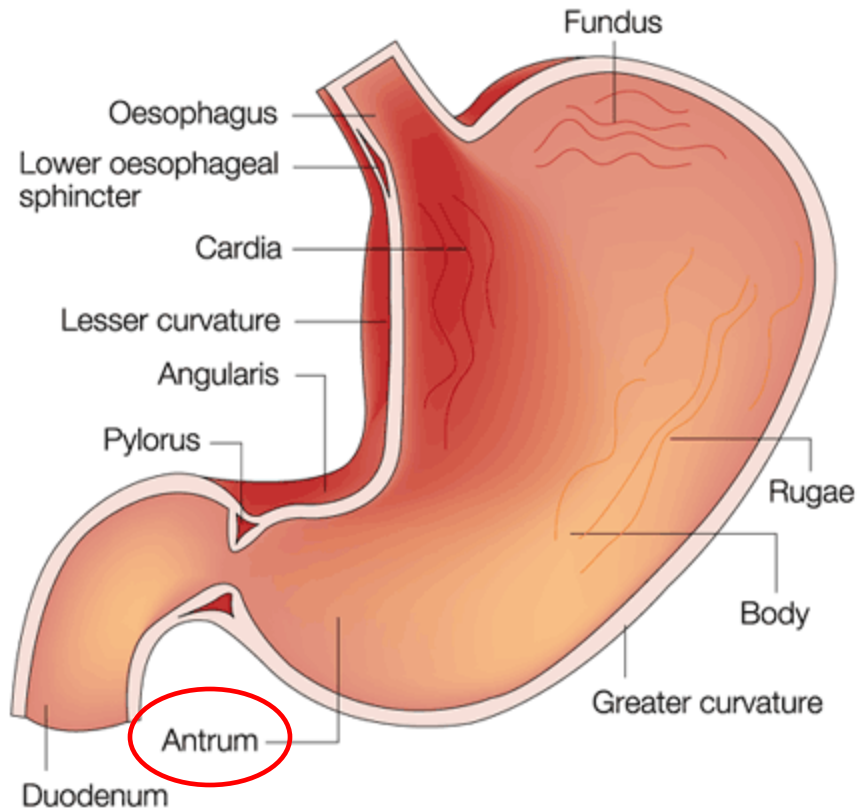
## *H. pylori* Gastritis

Acute: insufficient symptoms to require medical attention

Associated with poor hygiene (poverty, overcrowding...etc.)

Pathogenicity:

- Flagella
- Urease (ammonia pH)
- Adhesins
- Toxins (CagA)



Nature Reviews | Cancer

## *H. pylori* Gastritis

Not present in acid producing mucosa of gastric body, duodenum, or areas of intestinal metaplasia

Dx:

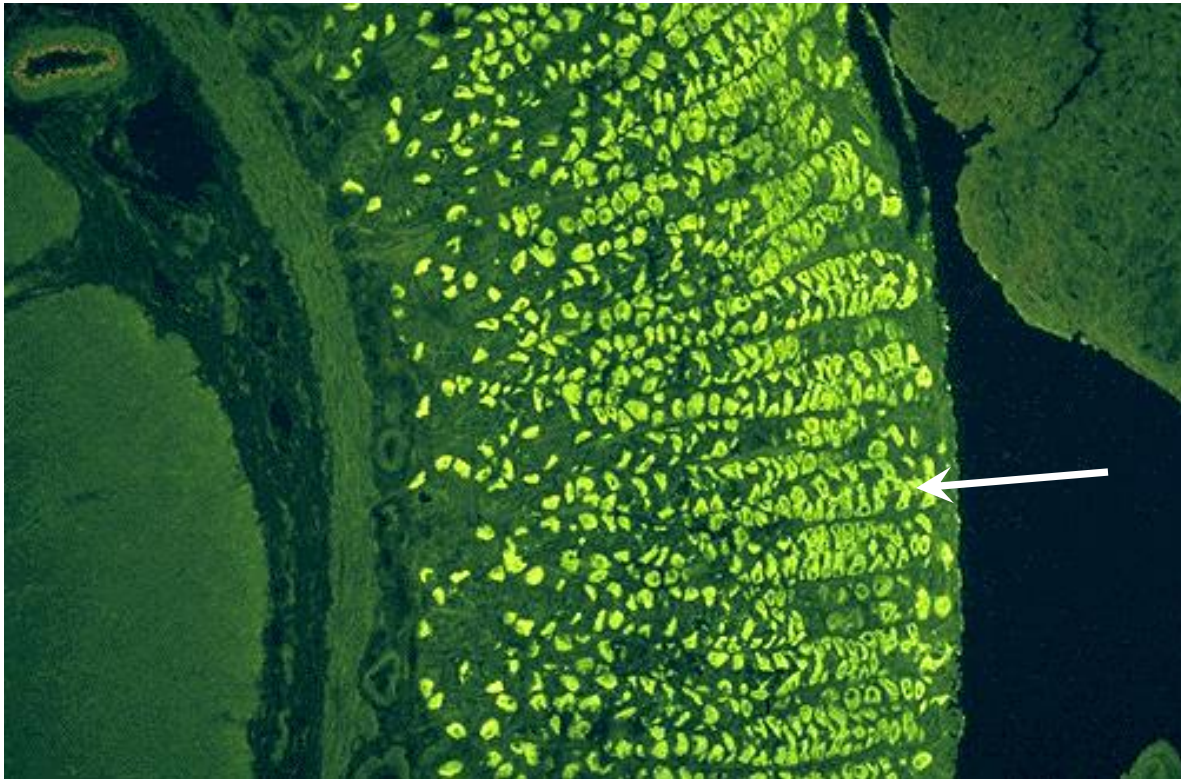
- Biopsy antrum
- Serological (Ab)
- Fecal detection
- Urea breath test

Tx:

Antibiotics+PPI

Relapse/reinfection





## Autoimmune Gastritis

Antrum spared but with G cell hyperplasia → Hypergastrinemia

Antibodies to parietal cells and intrinsic factor → Achlorhydria & pernicious anemia

Reduced serum pepsinogen I levels (chief cell loss)

Atrophy seen as loss of rugal folds



# Autoimmune vs H.pylori Gastritis

Feature	<i>H. pylori</i> –Associated	Autoimmune
Location	Antrum	Body
Inflammatory infiltrate	Neutrophils, plasma cells	Lymphocytes, macrophages
Acid production	Increased to slightly decreased	Decreased ( <b>achlorhydria*</b> )
Gastrin	Normal to decreased	Increased ( <b>hypergastrinemia</b> )
Other lesions	Hyperplastic/inflammatory polyps	Neuroendocrine hyperplasia ( <b>antrum</b> )
Serology	Antibodies to <i>H. pylori</i>	Antibodies to parietal cells (H <sup>+</sup> ,K <sup>+</sup> -ATPase, intrinsic factor)
Sequelae	Peptic ulcer, adenocarcinoma, lymphoma	Atrophy*, pernicious anemia ( <b>B<sub>12</sub>-IF</b> ), adenocarcinoma, carcinoid tumor
Associations	Low socioeconomic status, poverty, residence in rural areas	Autoimmune disease; thyroiditis, diabetes mellitus, Graves disease

\*diffuse damage of the oxyntic (acid-producing) mucosa within the body and fundus

