

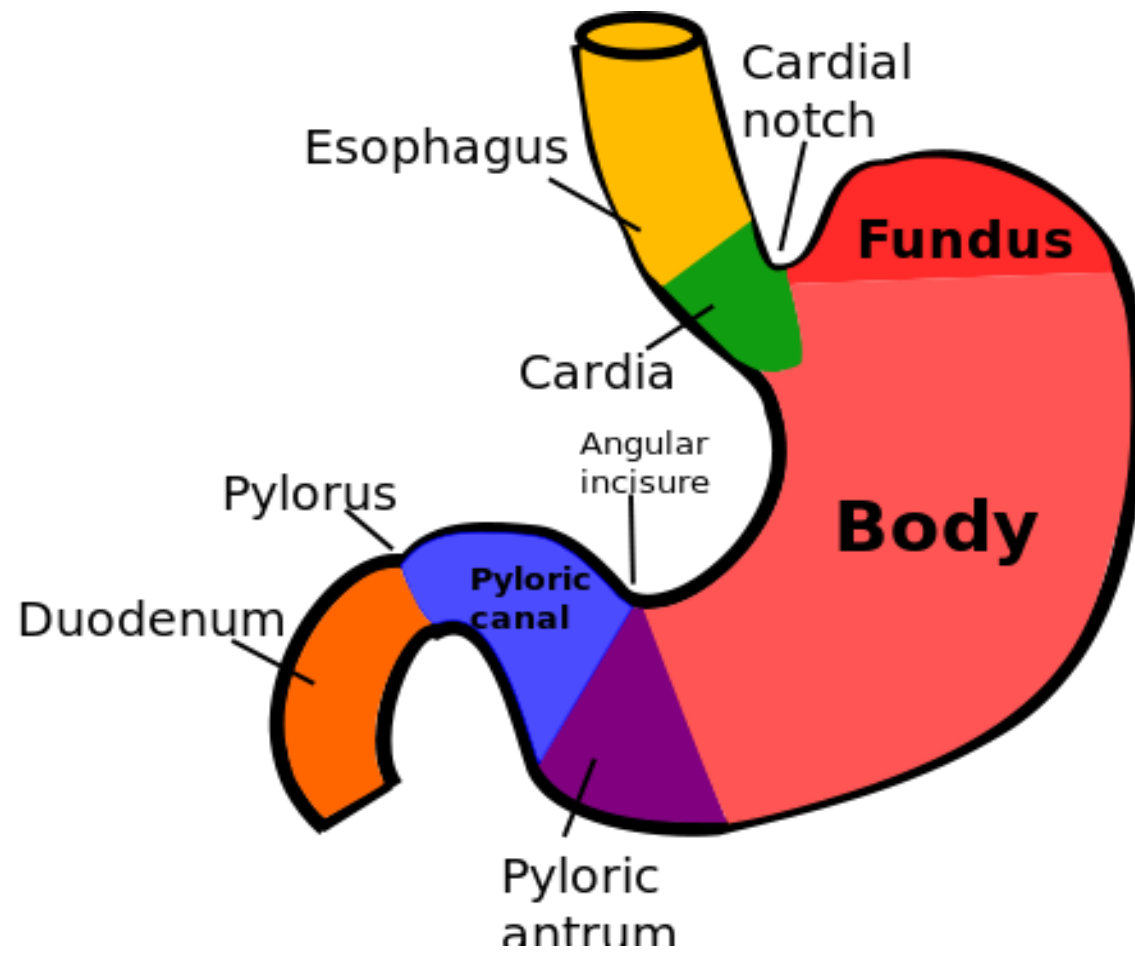
# Gastrointestinal pathology esophagus and stomach lecture 3

Dr Heyam Awad

FRCPath

# Diseases of the stomach





- Mucin is secreted mainly from the foveolar cells in the cardia
- Acids are secreted from the parietal cells in the fundus and body.
- Pepsinogen secreted from the chief cells in the body and fundus
- Gastrin is secreted from G cells in the antrum (endocrine cells)...  
gastrin stimulates acid secretion

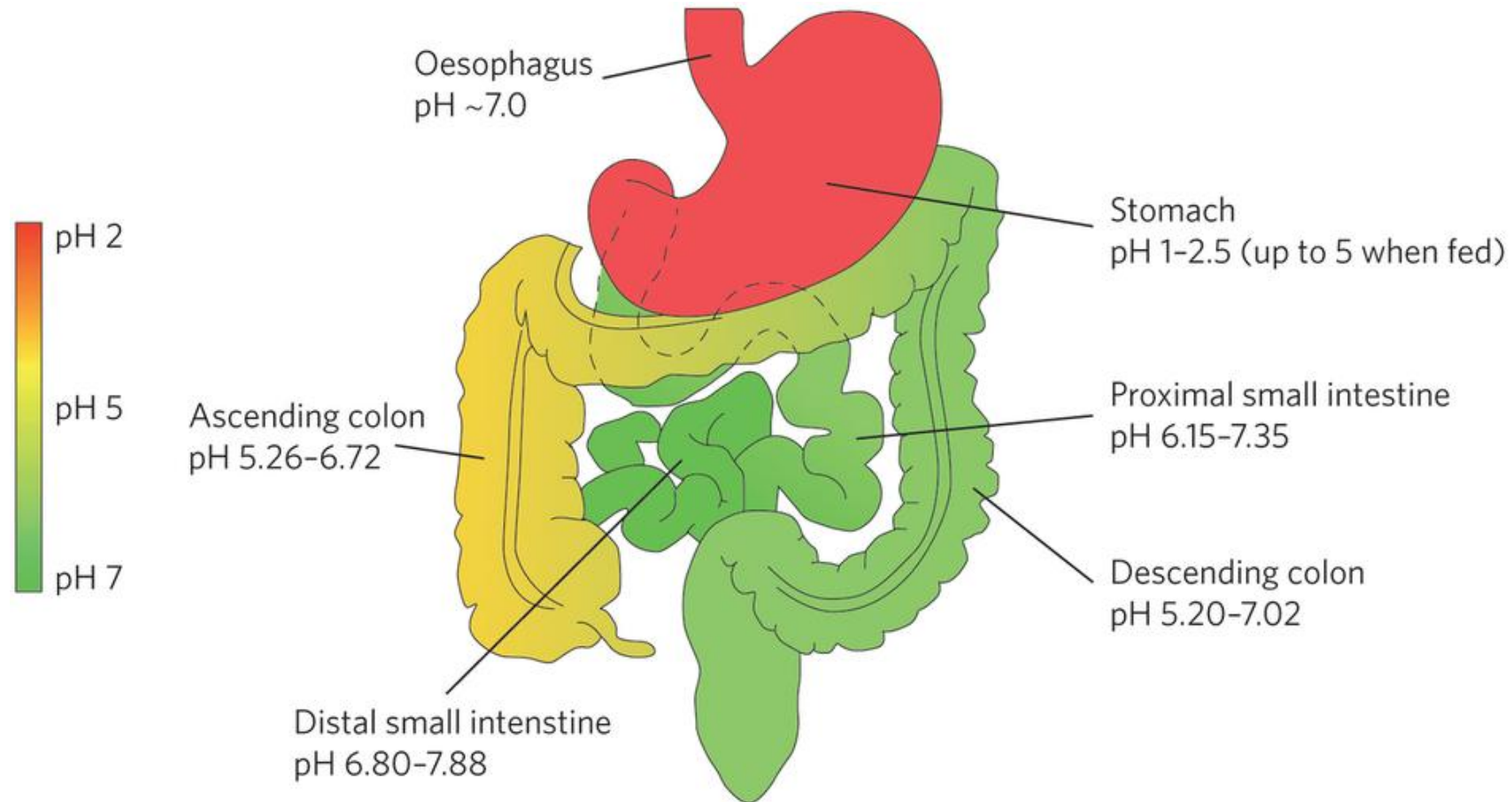
# Main diseases affecting the stomach

- Inflammations: acute gastritis, acute peptic ulcer, chronic gastritis, peptic ulcer disease (chronic ulcer)
- Neoplastic disease: gastric polyps and gastric malignancies.

# ACUTE GASTRITIS

- Is acute inflammation of the stomach which can be caused by any process that disrupts the mucosa.
- Disruption occurs due to direct mucosal injury or due to disturbance between acid secretion and protective mechanisms.

# Stomach acidity is important for digestion



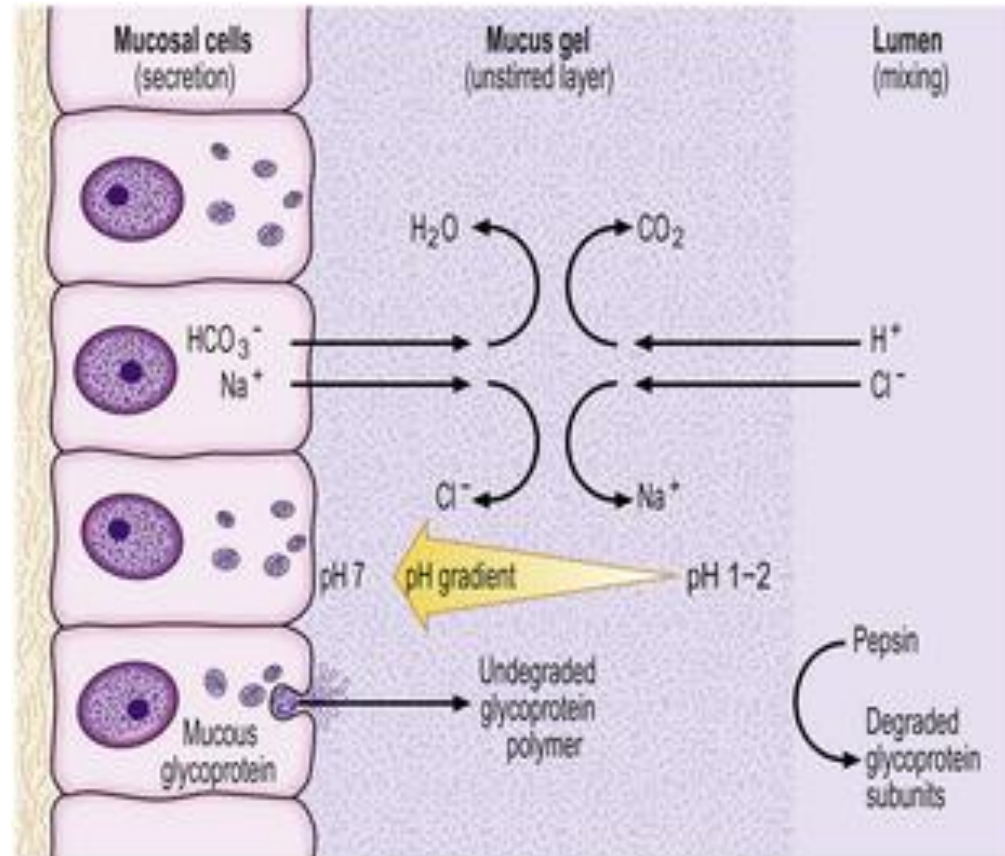
# pH of the stomach

|                      | pH | Example          |
|----------------------|----|------------------|
| <b>strong alkali</b> | 14 | drain cleaner    |
|                      | 13 | bleach           |
|                      | 12 | soapy water      |
|                      | 11 | ammonia          |
|                      | 10 | milk of magnesia |
|                      | 9  | baking powder    |
|                      | 8  | sea water        |
| <b>neutral</b>       | 7  | pure water       |
|                      | 6  | saliva           |
|                      | 5  | black coffee     |
|                      | 4  | acid rain        |
|                      | 3  | vinegar          |
|                      | 2  | lemon juice      |
|                      | 1  | stomach acid     |
| <b>strong acid</b>   | 0  | battery acid     |



- This high acidity is important for digestion.
- The stomach is protected from this high acidity by : mucus layer and bicarbonate release..... Both are secreted under the influence of protective prostaglandins.
- Inflammation in the stomach occurs when the balance between the damaging effect of acid and the protection mechanisms is disturbed.

# Protection of gastric mucosa against the acidity



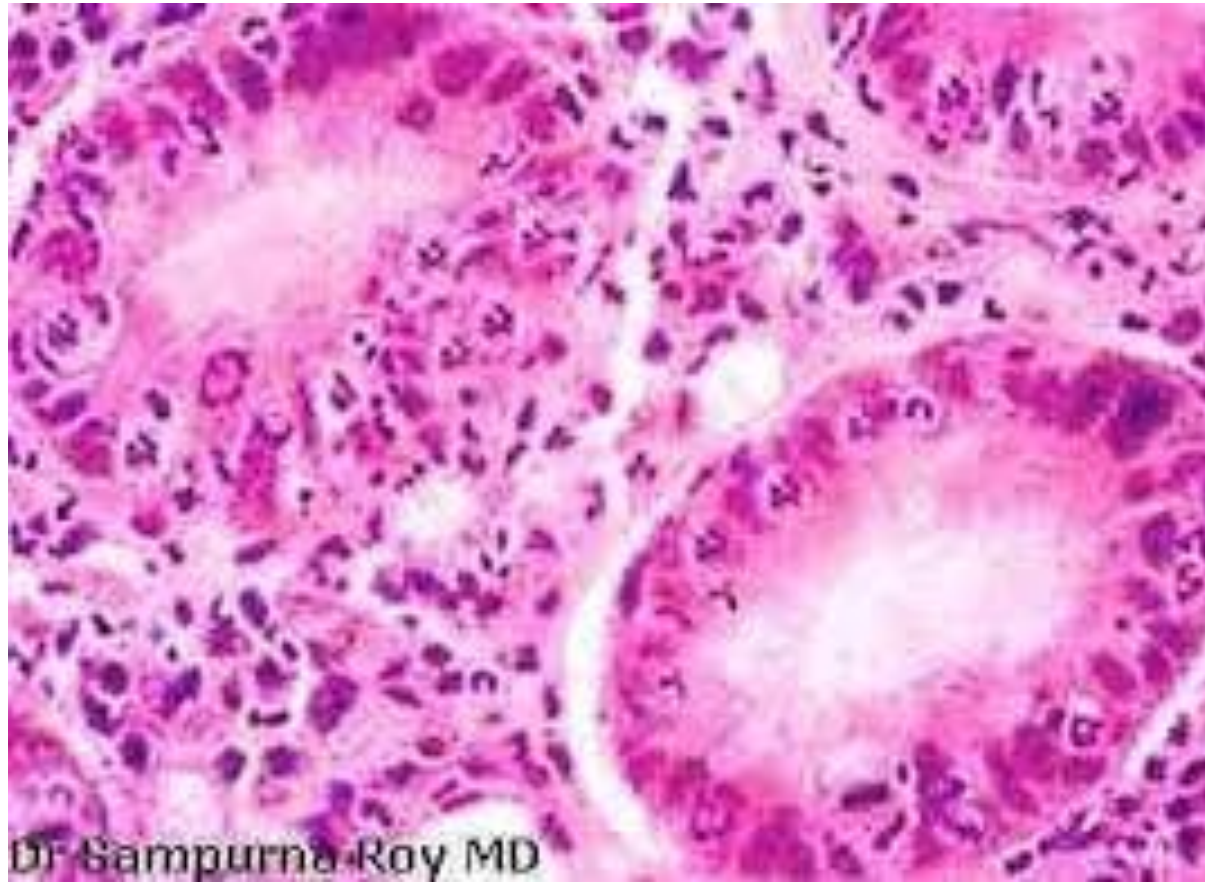
# Acute gastritis occurs when the protective mechanisms fail

- Reduced mucin secretion in the elderly
- NSAIDS interfere with protective prostaglandins or causes reduced bicarbonate synthesis
- Chemical ingestion can damage the mucosa
- Alcohol and Radio or chemotherapy can cause mucosal damage

# Signs and symptoms

- Nausea
- Vomiting
- Epigastric pain.

Morphology/ neutrophils are the main cells in a acute gastritis



# Acute peptic ulcer

- = focal loss of the mucosa.

## Causes:

1. NSAIDS
2. Stress ulcer ( physiologic stress in critically ill patients)
3. Curling ulcer, in burns and trauma
4. Cushing ulcer, occur in association with increased intracranial pressure.

# Stress ulcer

- Critically ill patients: shock or sepsis



Curling ulcer: usually seen in duodenum

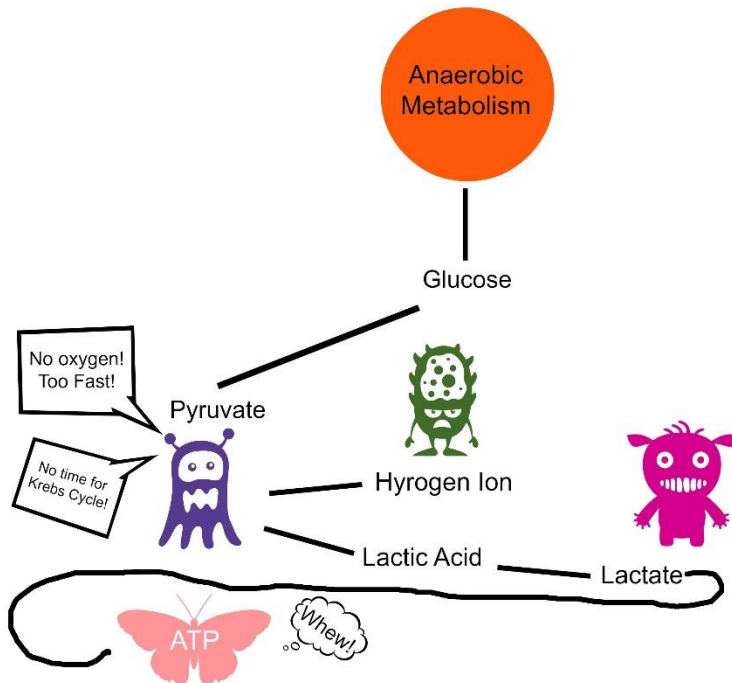




# Pathogenesis of stress and curling ulcers

- Critically ill patients or those with hypovolemia or shock have decreased blood volume
- To protect vital organs from hypoxia, some blood vessels constrict like those of the stomach
- This vasoconstriction causes hypoxia which damages gastric epithelial cells causing ulceration
- Also hypoxia causes decreased oxidative phosphorylation .. This results in anaerobic glycolysis with lactic acid production ... this results in acidosis and epithelial cell damage

# Curling and stress ulcers



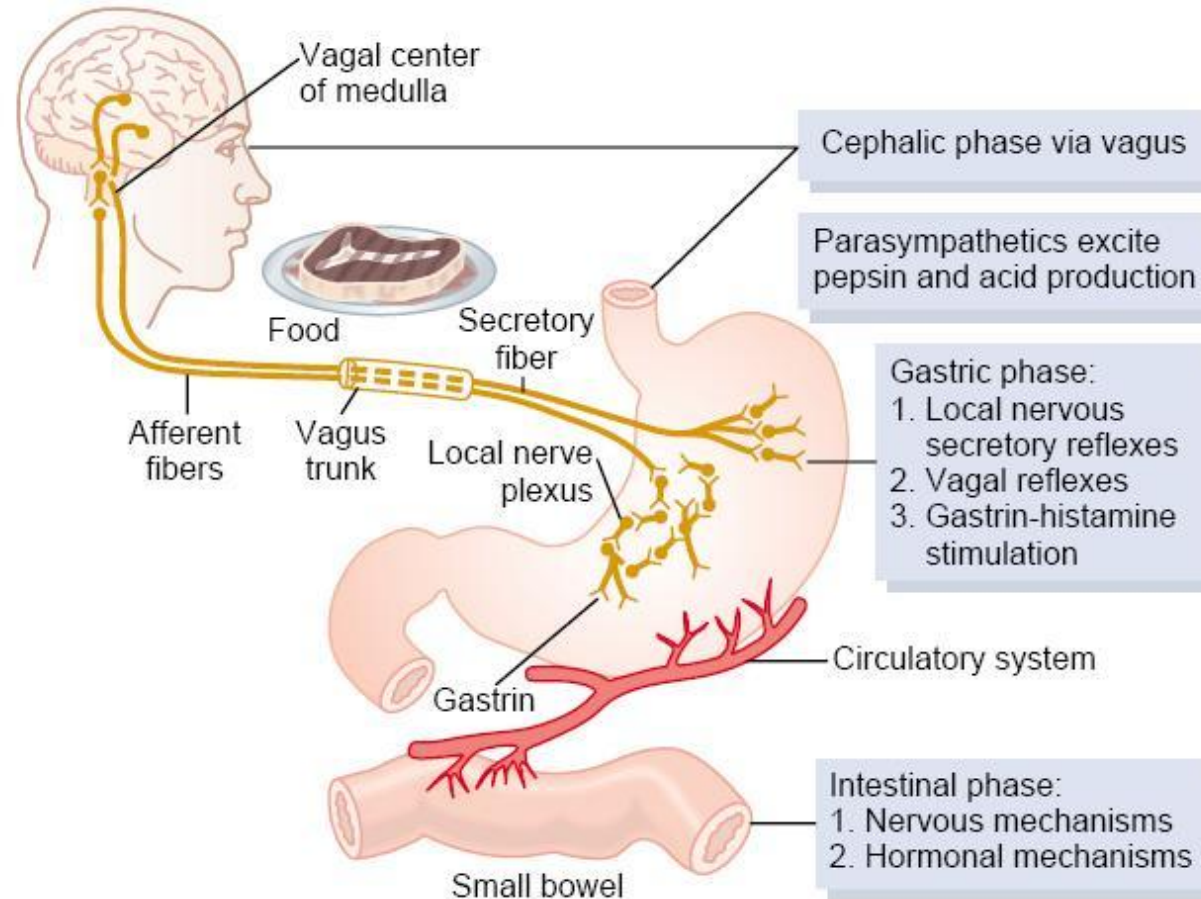
# Pathogenesis of Cushing ulcer

- Increased intracranial pressure due to any cause ( tumor, bleeding, stroke) can cause stimulation of the vagus nerve which will stimulate acid production in the stomach

# Cushing ulcer: affect stomach, duodenum or esophagus



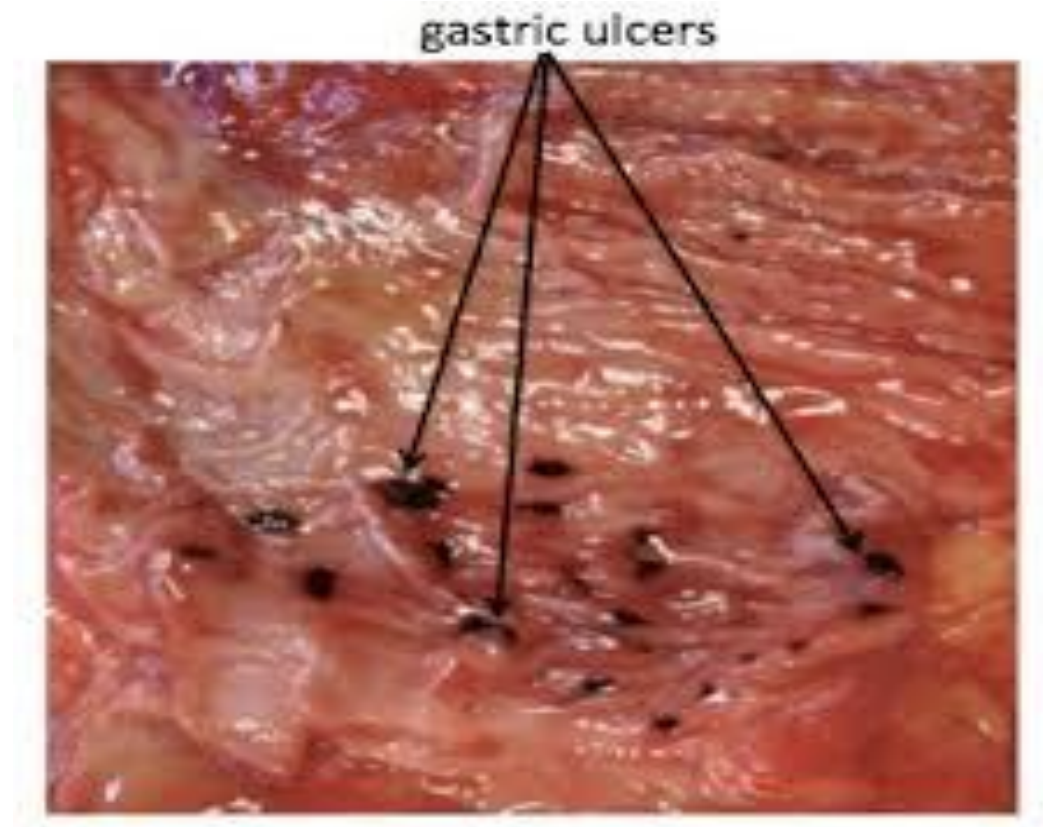
# Pathogenesis of Cushing ulcer



Morphology: ulcer with hemorrhagic base  
surrounding mucosa is normal



# morphology



# note

- ACUTE ULCERS HEAL AFTER REMOVING THE CAUSE



# Clinical picture

- Nausea
- Vomiting
- Coffee ground hematemesis
- **Complications:**
  - Bleeding
  - Perforation
- **Outcome** mainly determined by the severity of the underlying condition

# Chronic gastritis

- Symptoms less severe than acute gastritis.
- Nausea, vomiting, abdominal discomfort.
- Hematemesis is rare

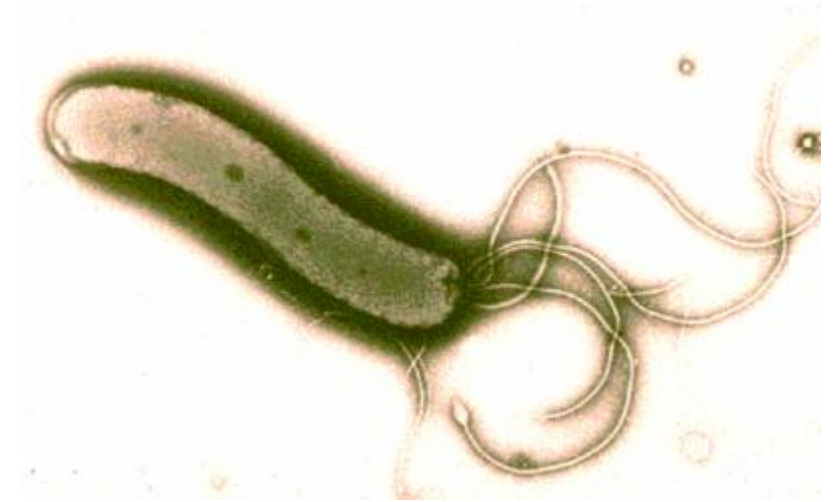
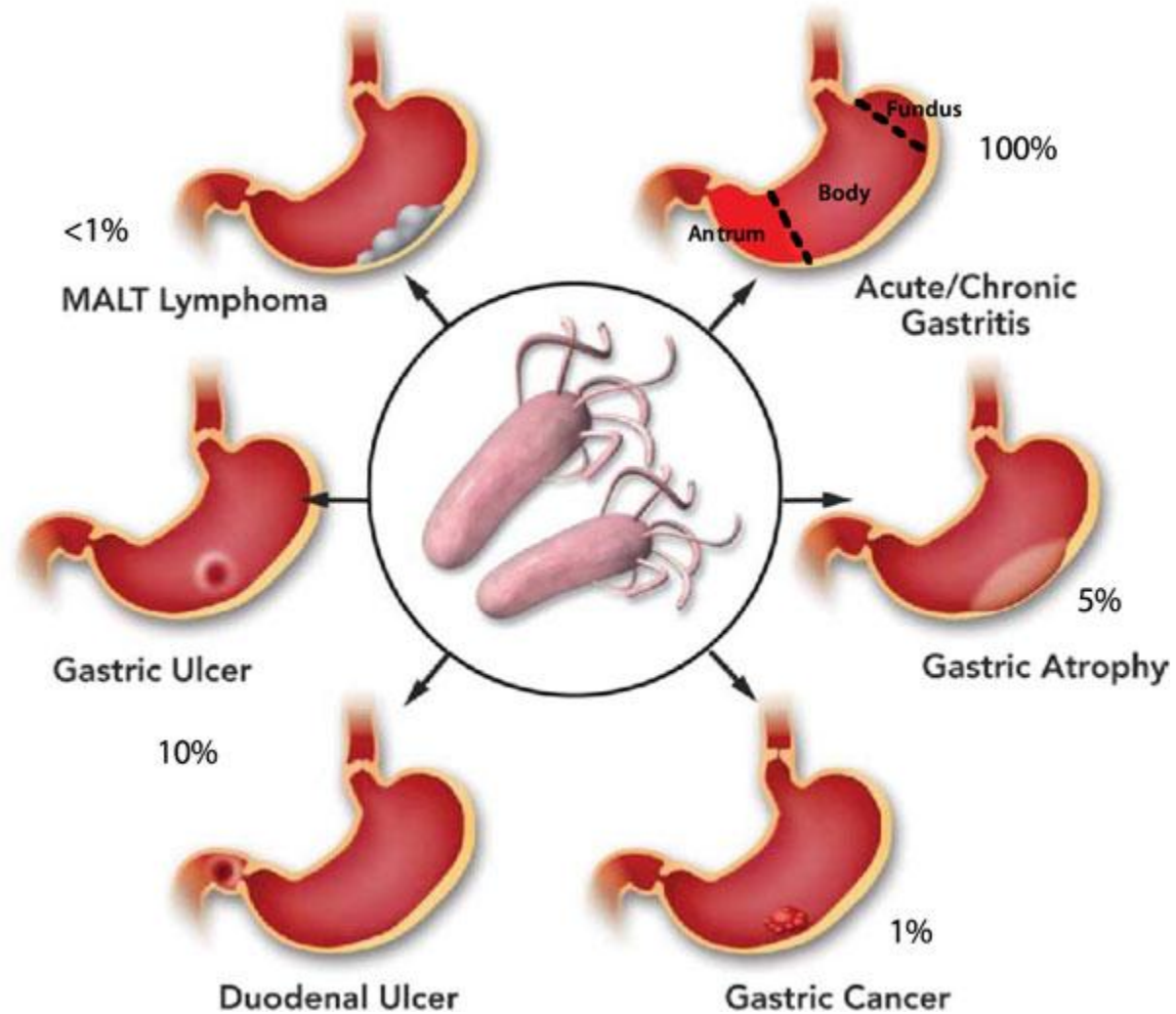
# Chronic gastritis

- H Pylori associated chronic gastritis.. Type B gastritis.. majority of cases..
- Autoimmune gastritis.. Type A, 10% of cases

# H pylori

- Gram negative bacilli
- Found in : duodenal ulcer, gastric ulcer, chronic gastritis
- Also associated with gastric carcinoma and lymphoma

# H pylori

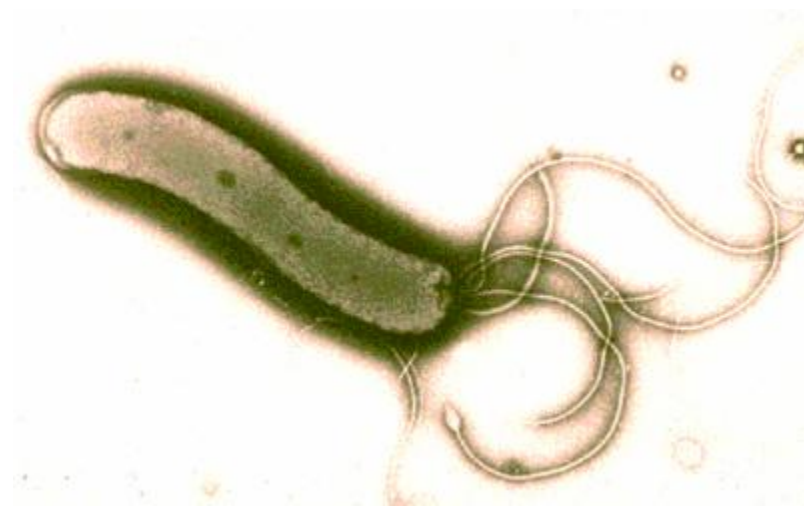


# H pylori infection

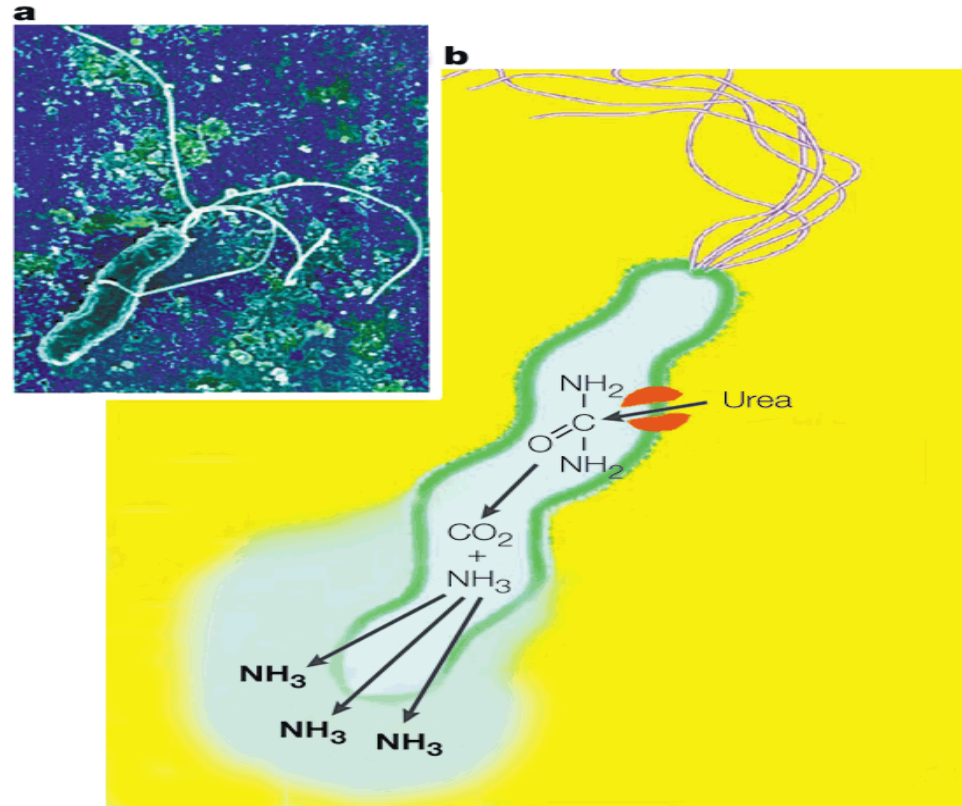
- Poverty
- Crowding
- Poor sanitation

# pathogenesis

- Flagella help the bacteria to move through the thick viscous mucin

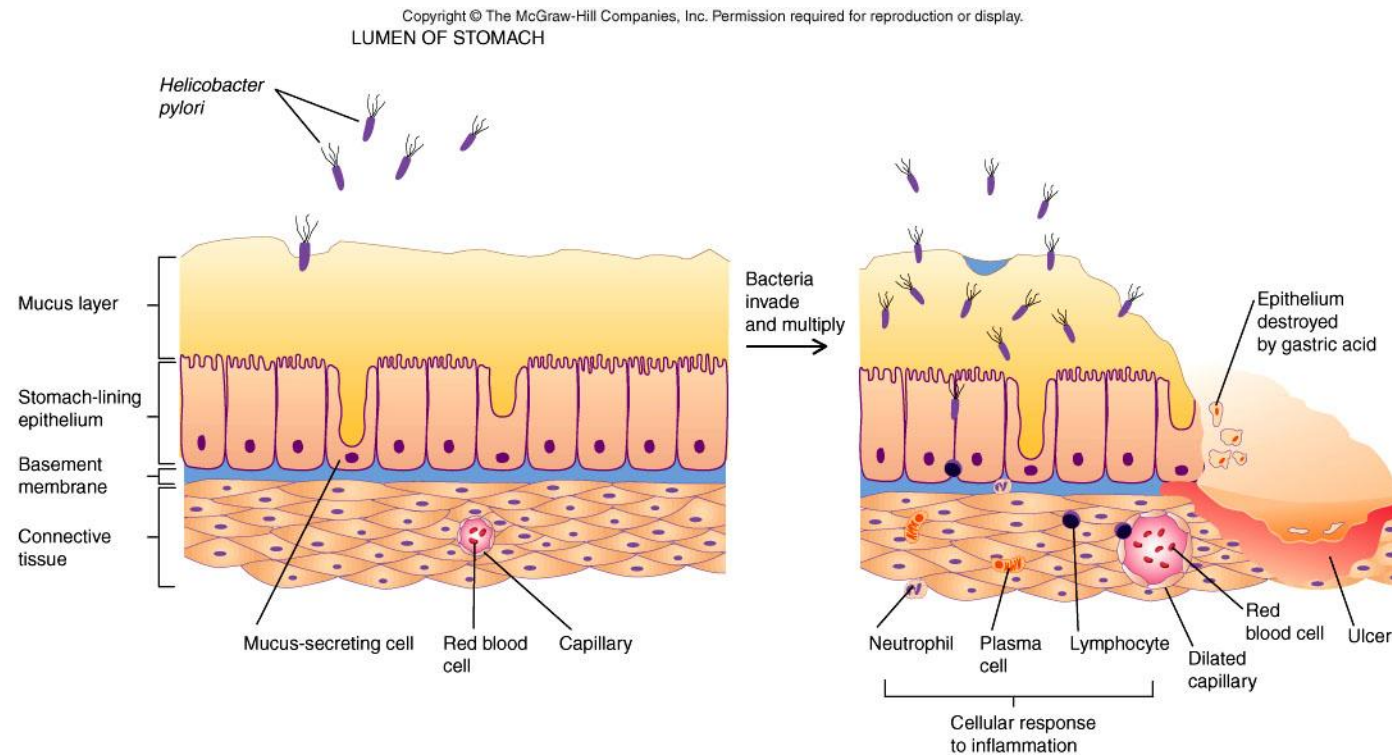


*H. pylori* urease allows the bacteria to produce  $\text{NH}_3$  from urea..  $\text{NH}_3$  is basic and helps buffer the acidity around the bacteria

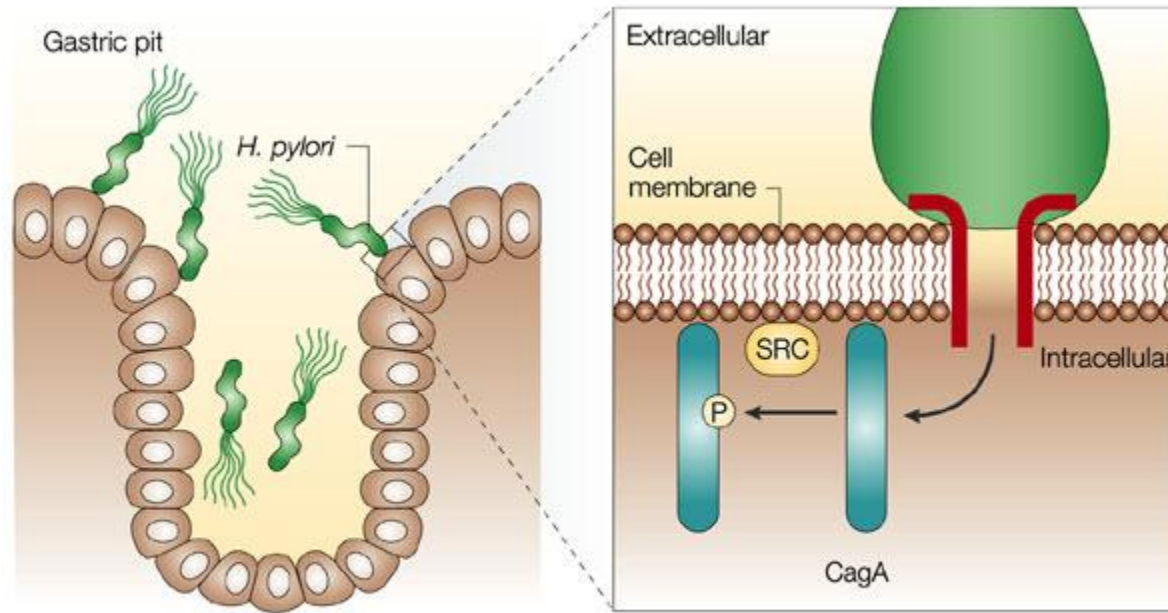




# Ability to attach to epithelium is also important in this bacterial pathogenesis



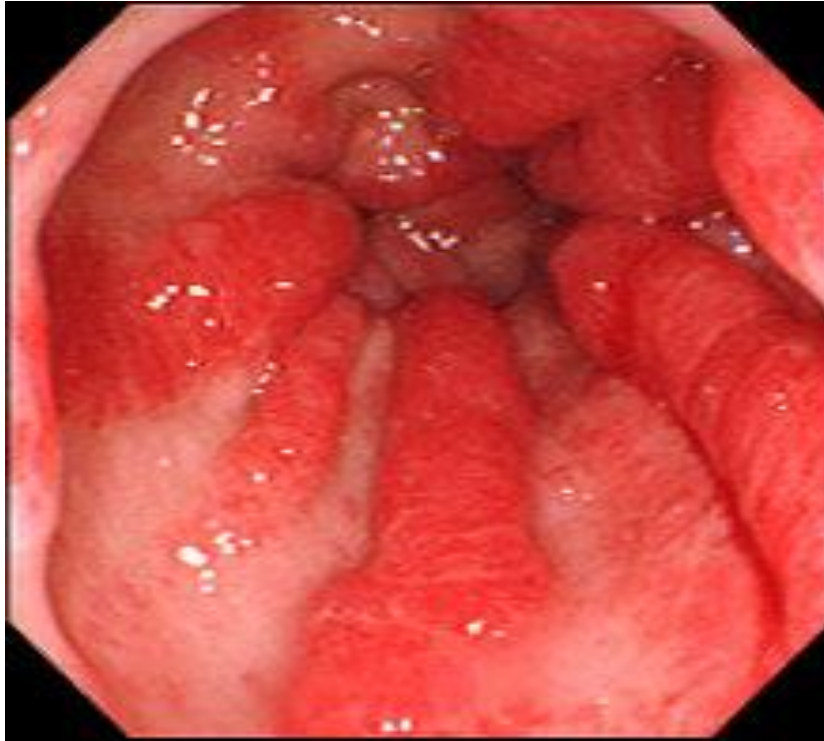
# CAG A toxin by *H. pylori* is thought to be important in gastritis and gastric carcinoma



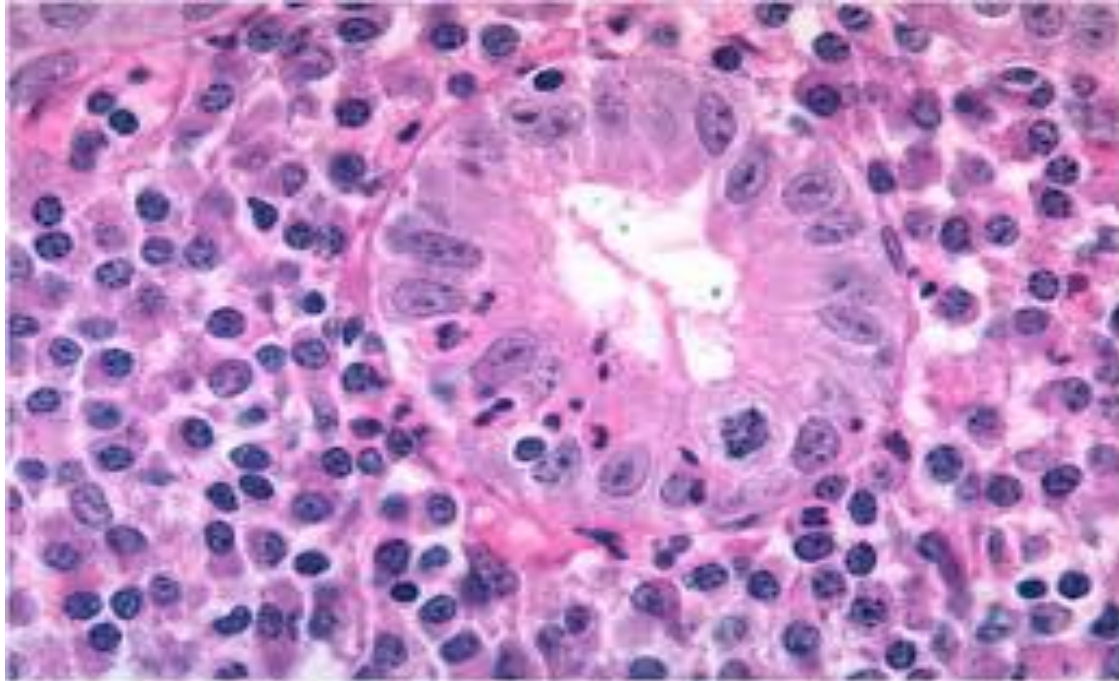
Morphology.. Chronic inflammation with redness and edema



# Chronic gastritis

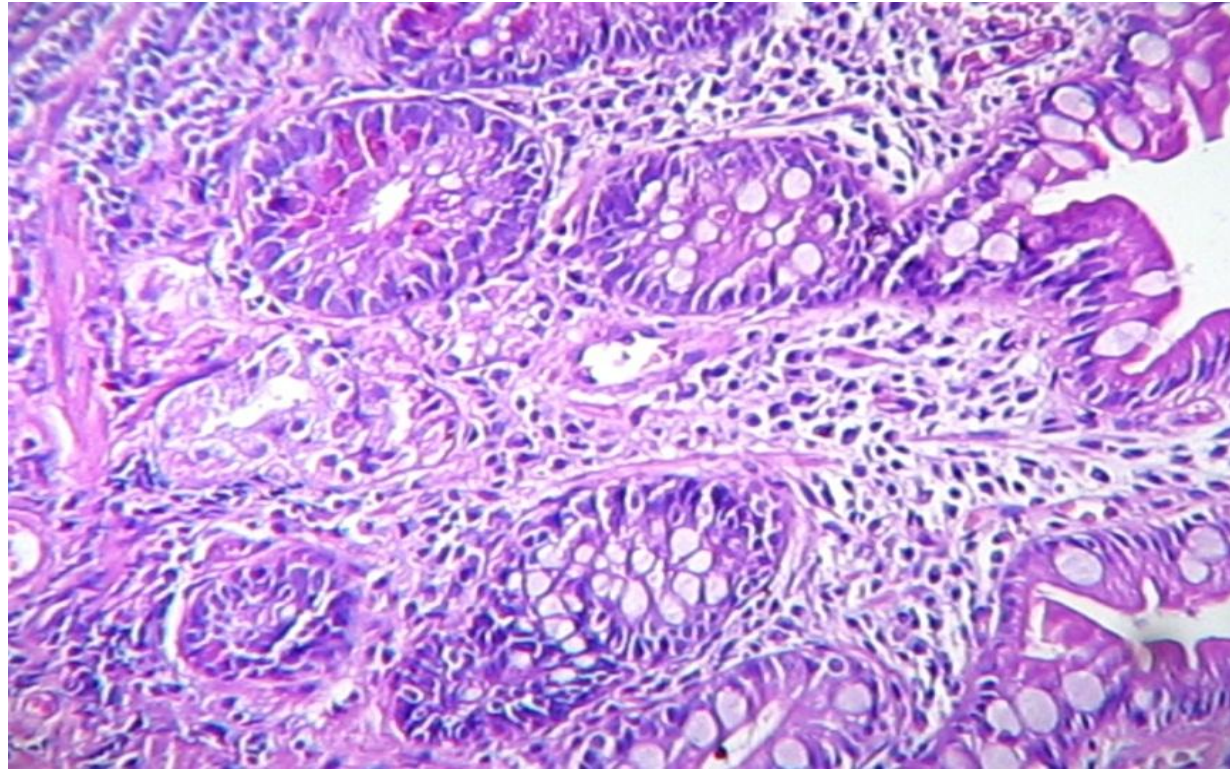


Chronic gastritis.. Increased chronic cell inflammatory infiltrate mainly lymphocytes and plasma cells

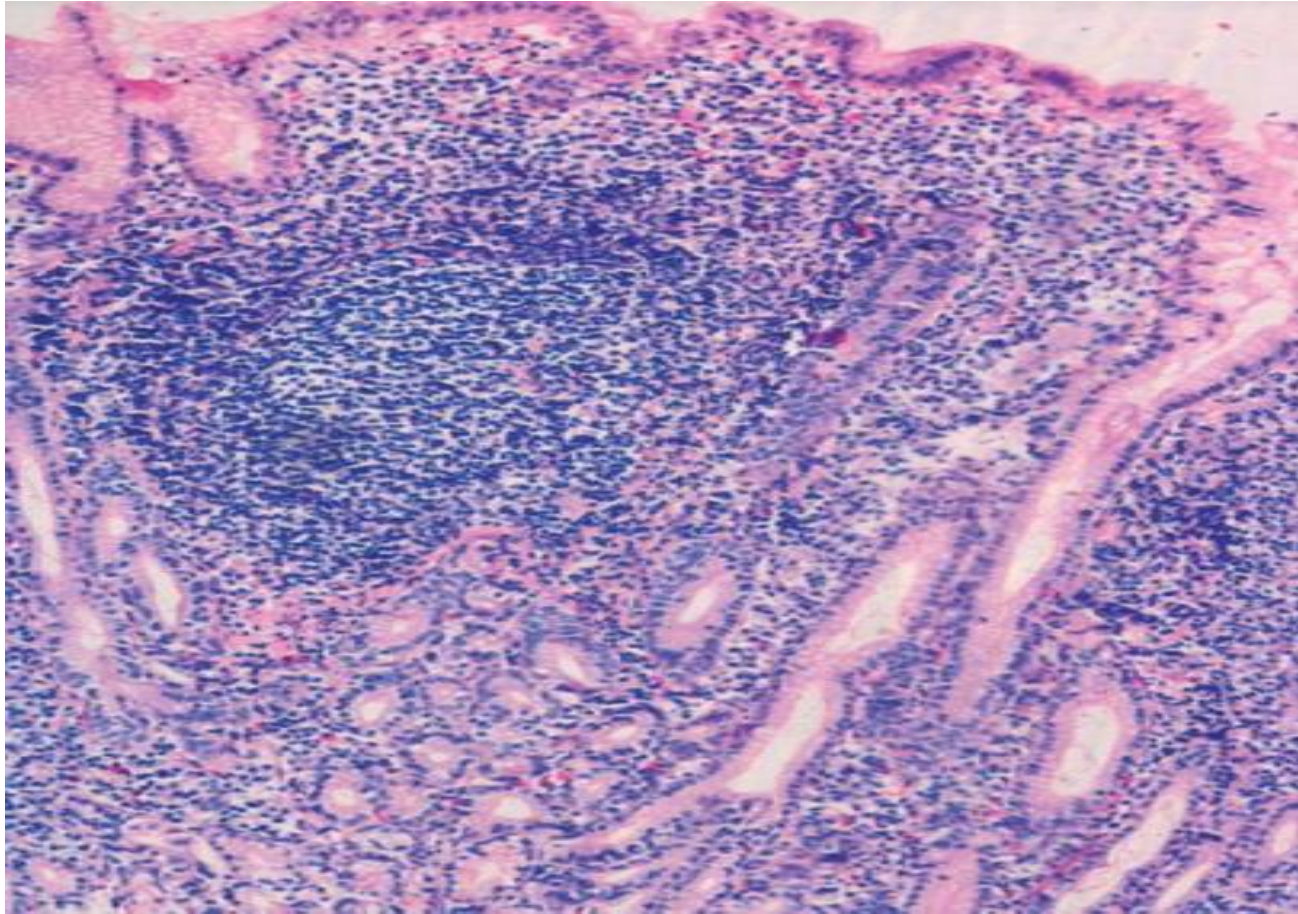




Intestinal metaplasia can complicate chronic gastritis.. And this predisposes to adenocarcinoma



# Lymphoid aggregates in chronic gastritis

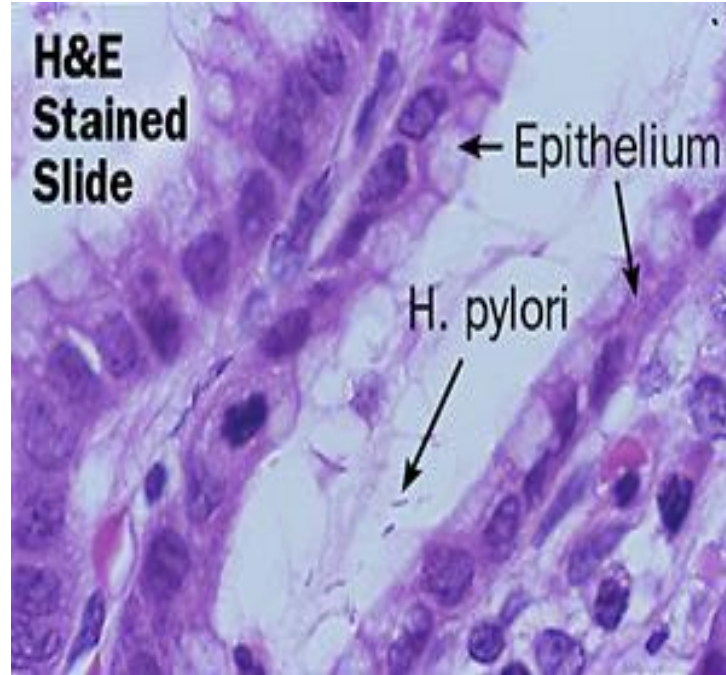
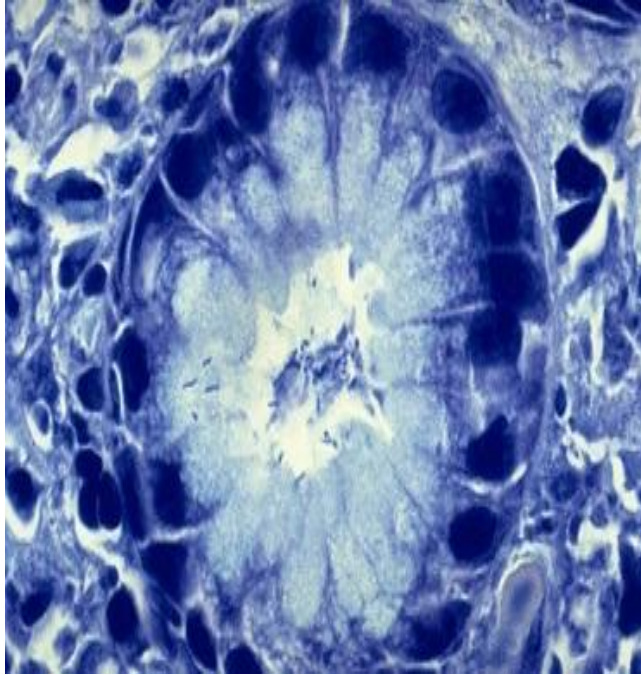


# How to diagnose H Pylori

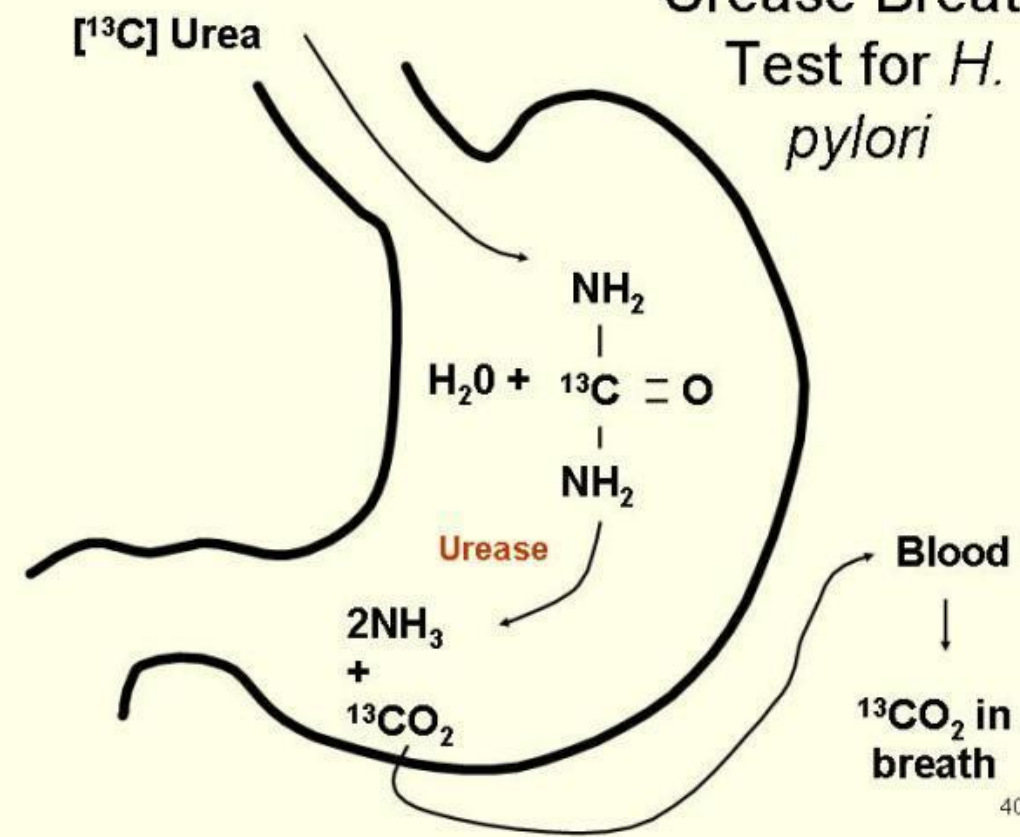
- Serology to detect anti H pylori antibodies
- Fecal bacterial detection
- Urea breath test
- Gastric biopsy
- PCR
- culture



# H pylori diagnosis



## Urease Breath Test for *H. pylori*



# serology



# Culture



Fig. *Helicobacter pylori* on Columbia blood agar

# Autoimmune gastritis

- Less than 10% of chronic gastritis
- Usually spares the antrum
- Induces hypergastrinemia

# Characteristics of autoimmune gastritis

- Antibodies to parietal cells and intrinsic factor
- Reduced pepsinogen 1 level
- Antral endocrine cell hyperplasia
- Vitamin B 12 def
- achlorohydria

# pathogenesis

## Autoimmune Gastritis

Anti parietal cell  $\pm$  anti intrinsic factor (IF) antibodies



Gland destruction



**ATROPHY**

Loss of Acid production  
(Achlorhydria)

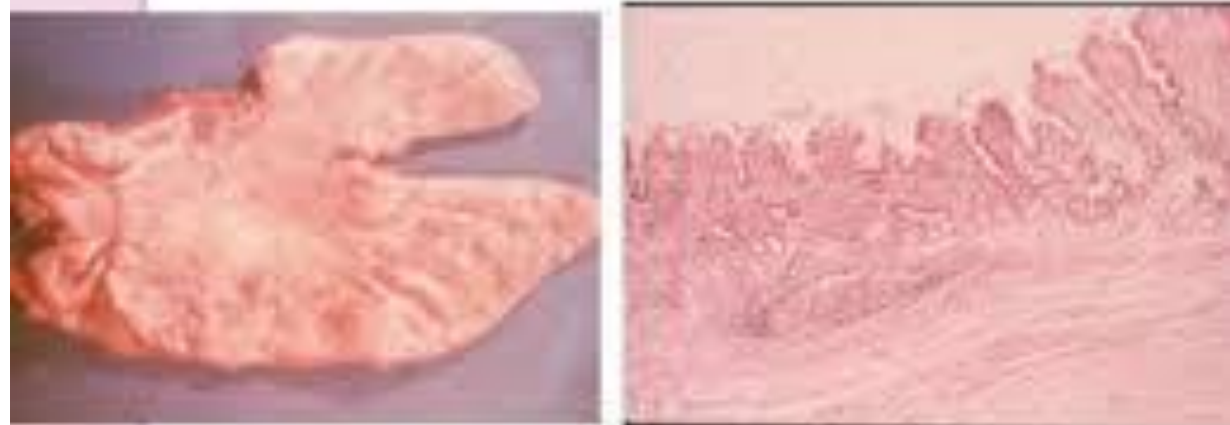
Loss of IF  
Vitamin B<sub>12</sub> deficiency  
(Pernicious anemia)

Mainly involves  
body and  
fundus



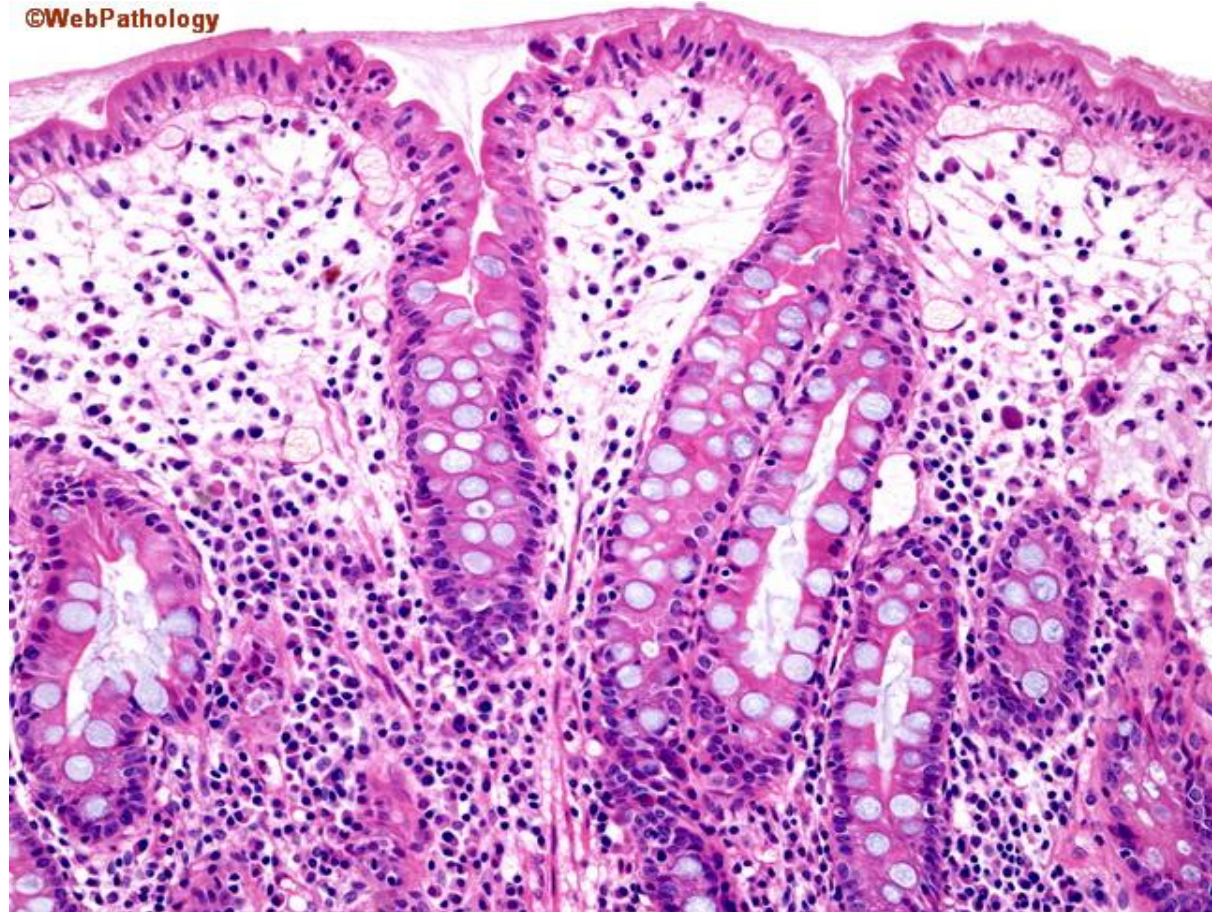
## Autoimmune Gastritis -Morphology

Diffuse mucosal damage of the **body and fundic mucosa**. Antrum less involved.

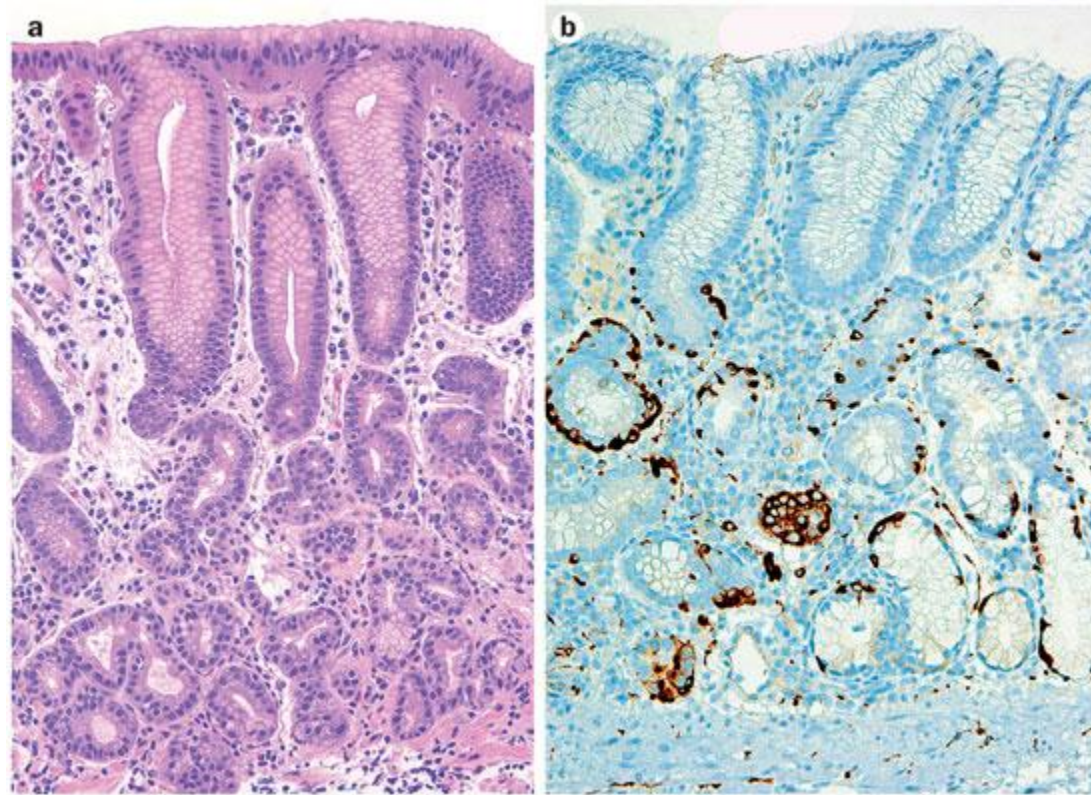




# Intestinal metaplasia can be seen in autoimmune gastritis



# G cell hyperplasia... a precursor to carcinoid tumor



|                 | H pylori gastritis                     | Autoimmune gastritis  |
|-----------------|--|---|
| site            | Mainly antrum                          | Body.. Spares antrum  |
| Acid production | Usually increased                      | decreased   |
| gastrin         | Normal or decreased                    | increased   |
| serology        | H pylori antibodies                    | Antibodies to parietal cells and intrinsic factor           |
| complications   | Peptic ulcer, adenocarcinoma, lymphoma | Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor |
| associations    | Poverty, low socioeconomic class       | Other autoimmune diseases: thyroiditis, diabetes,           |
|                 |  |   |