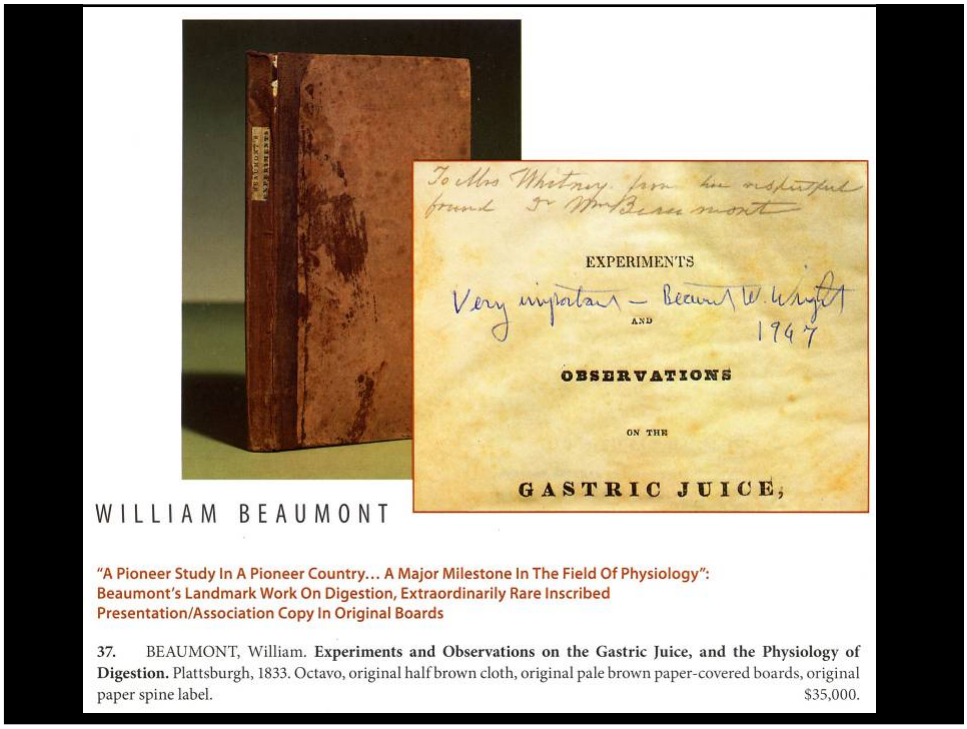


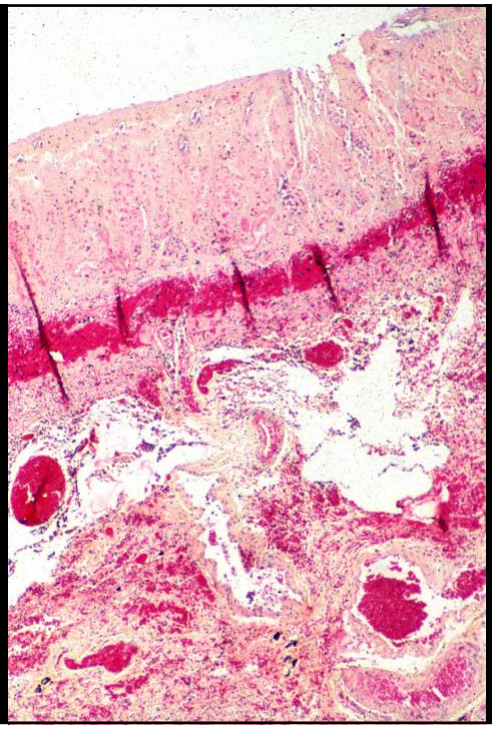

# Gastritis: Causes

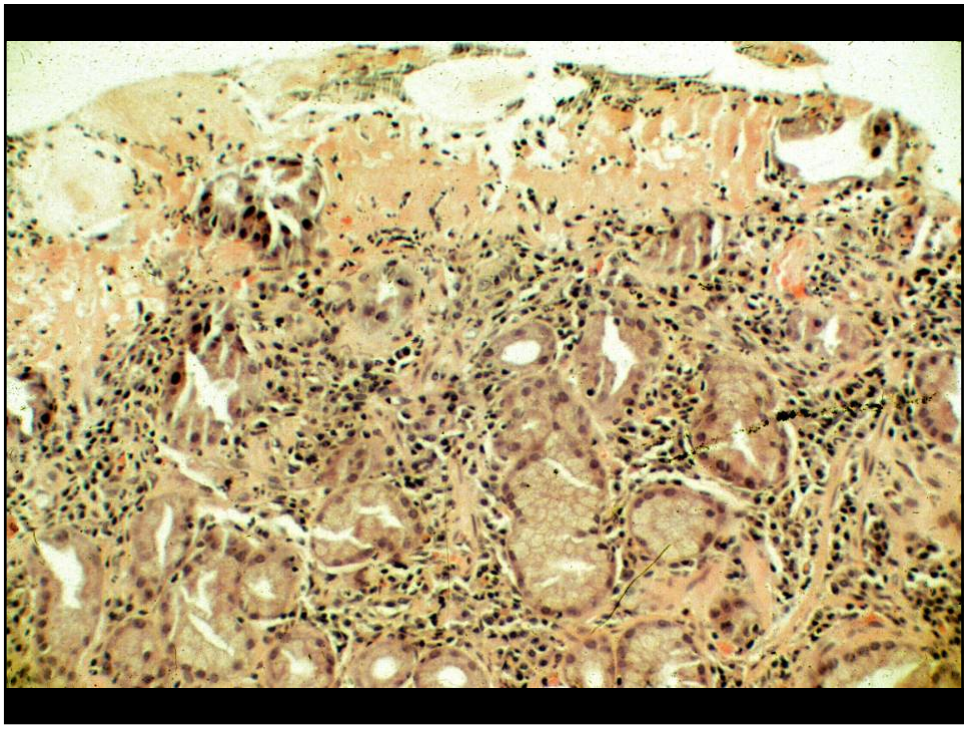
- ***Helicobacter pylori* infection**
- **NSAID use**
- Excessive alcohol consumption
- Heavy smoking
- Radiotherapy
- Cancer chemotherapy
- Systemic infections (*Salmonella*, CMV)
- Severe stress
- Ischemia and shock
- Suicide attempts with acids or alkali
- Mechanical trauma
- Distal gastrectomy

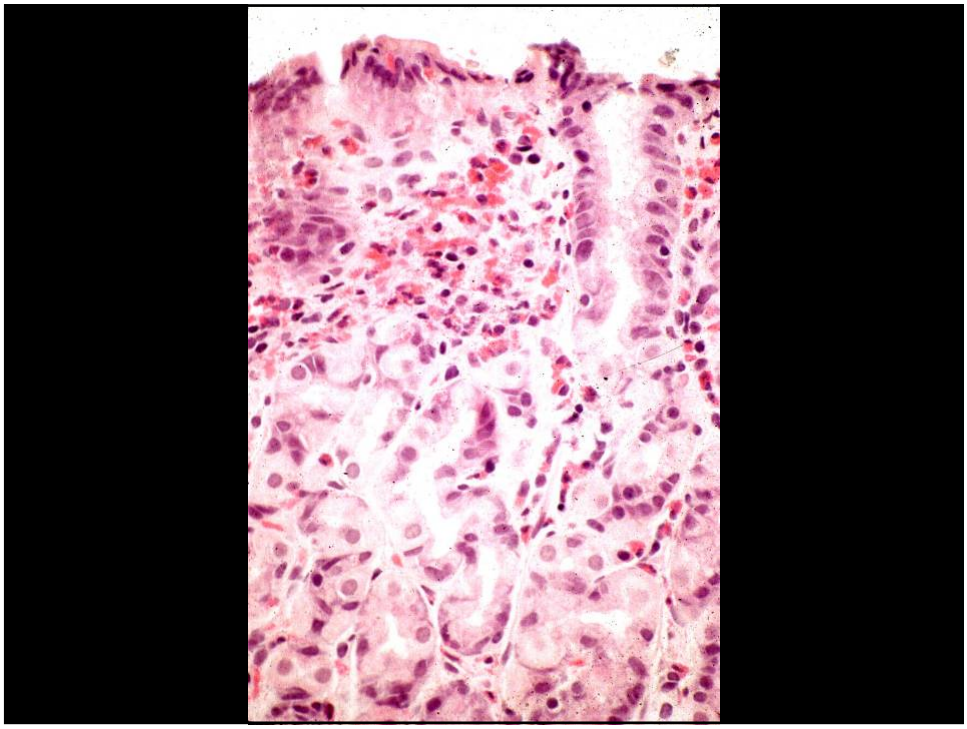
# GASTRITIS

Morphologic (descriptive) classification

1. Acute gastritis (neutrophils)
2. Hemorrhagic gastritis (fresh blood)
3. Erosive gastritis (destruction of parts of the mucosa)
4. Granulomatous gastritis
5. Eosinophilic gastritis
6. Chronic gastritis (most common)

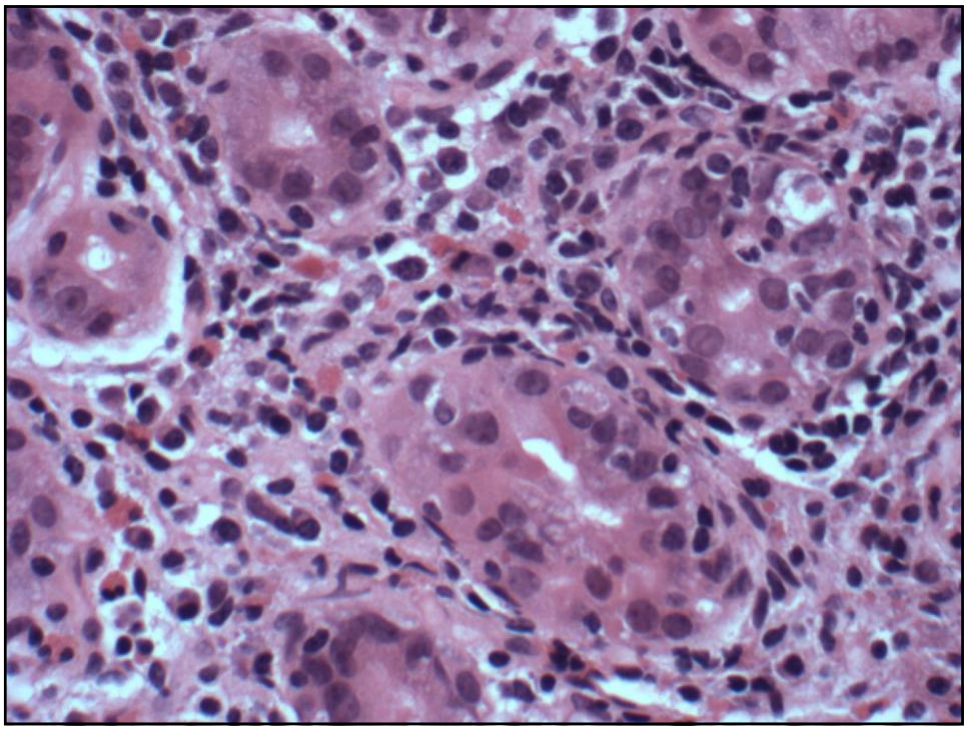


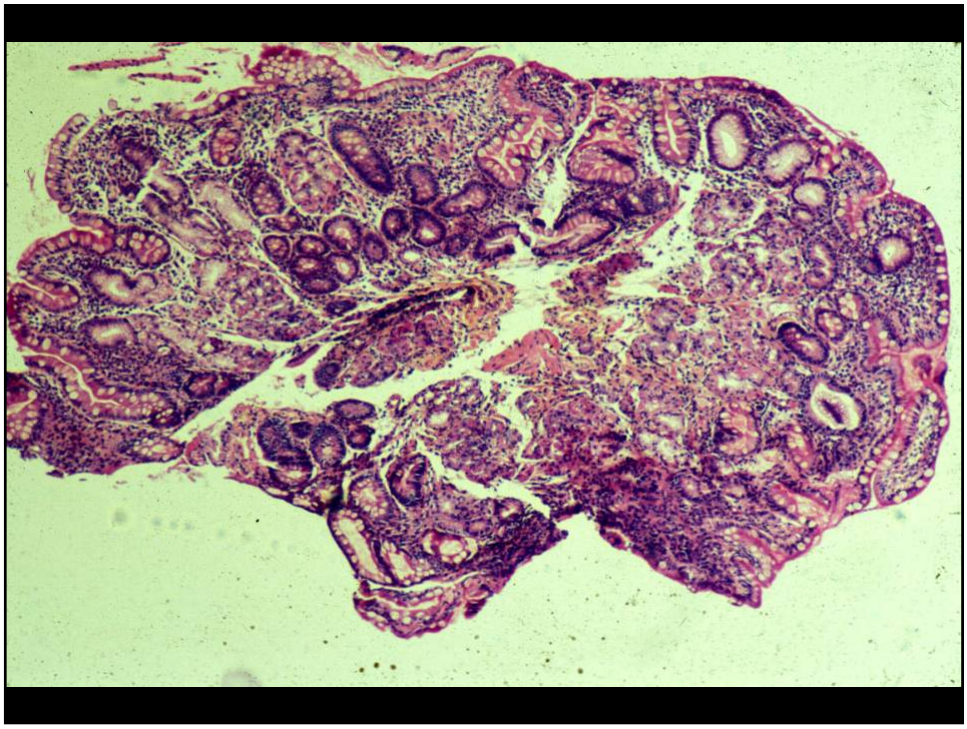


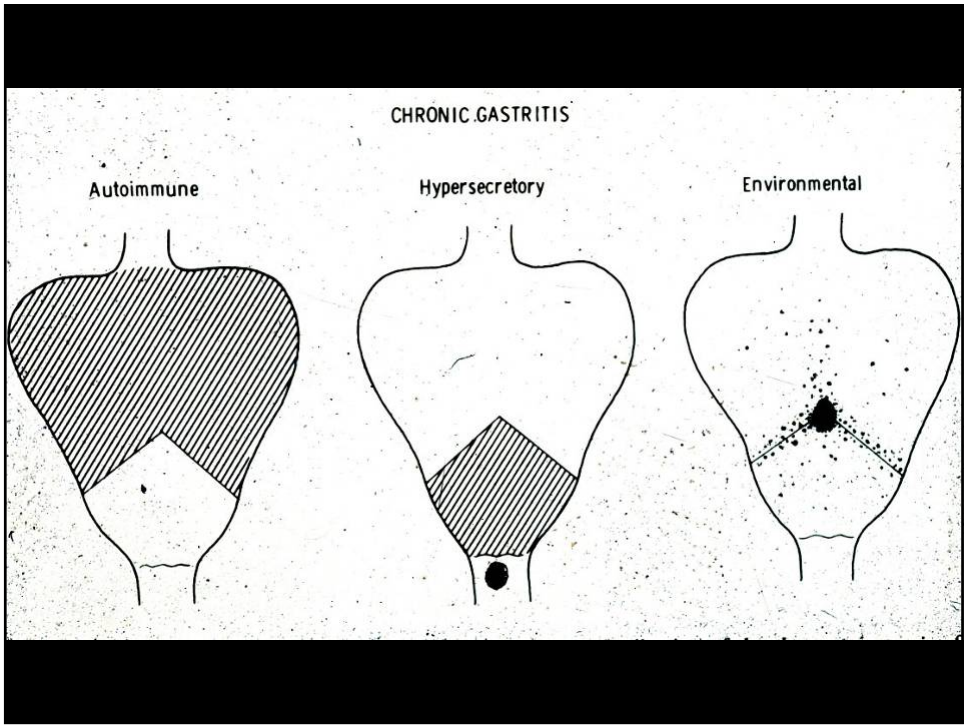


# Types of Chronic Gastritis

- **Autoimmune gastritis (type A gastritis)**: diffuse gastritis of corpus; antibodies to parietal cells and intrinsic factor; low acid, pernicious anemia; associated with other autoimmune disorders; uncommon.
- ***Helicobacter pylori* gastritis (type B gastritis)**: may affect all parts of the stomach, mostly antrum; 3 subtypes: antrum-predominant, corpus-predominant, pangastritis; very common.
- **Chemical gastritis (type C gastritis)**: due to repeated chemical or toxic injury (bile acids, duodenal contents, NSAIDs); common.

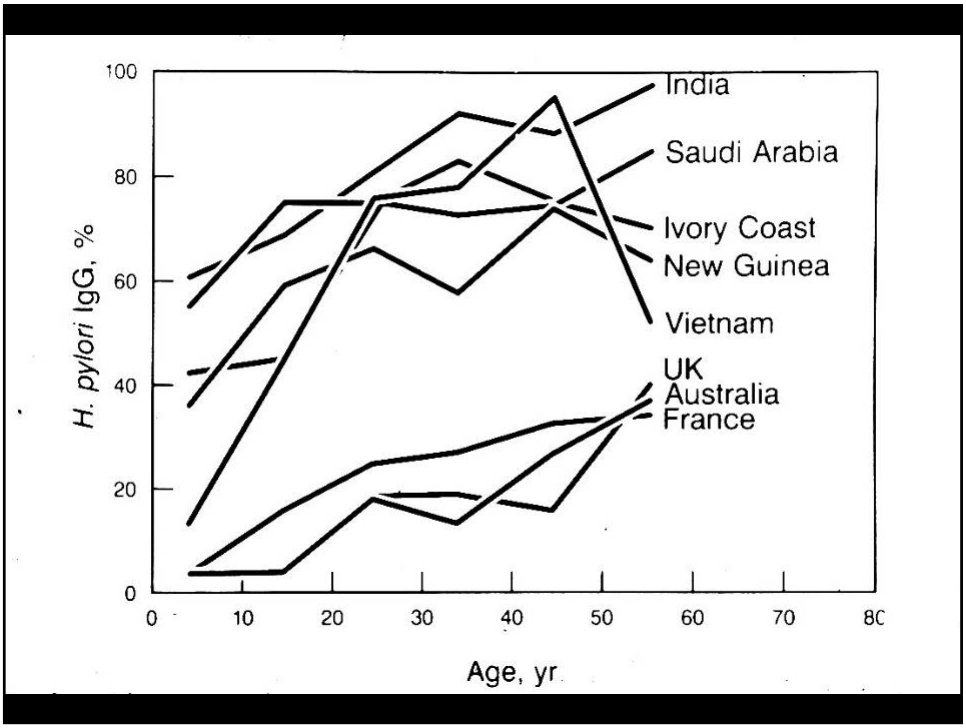





CHRONIC GASTRITIS
Autoimmune
Hypersecretory
Environmental

## Prevalence of Biopsy-proven *H. pylori* Gastritis

| | |
|---|---|
| Asymptomatic adults: | 30% |
| Non-ulcer dyspepsia: | 67% |
| Gastric ulcer: | 65% |
| Duodenal ulcer: | 86% |



## Helicobacter Pylori Infection

**Cofactor:** Time of life when infection was acquired

**Childhood:** Multifocal atrophic gastritis
Gastric ulcer
Gastric cancer

**Adulthood:** Chronic active gastritis
Duodenal ulcer

## H. Pylori Gastritis

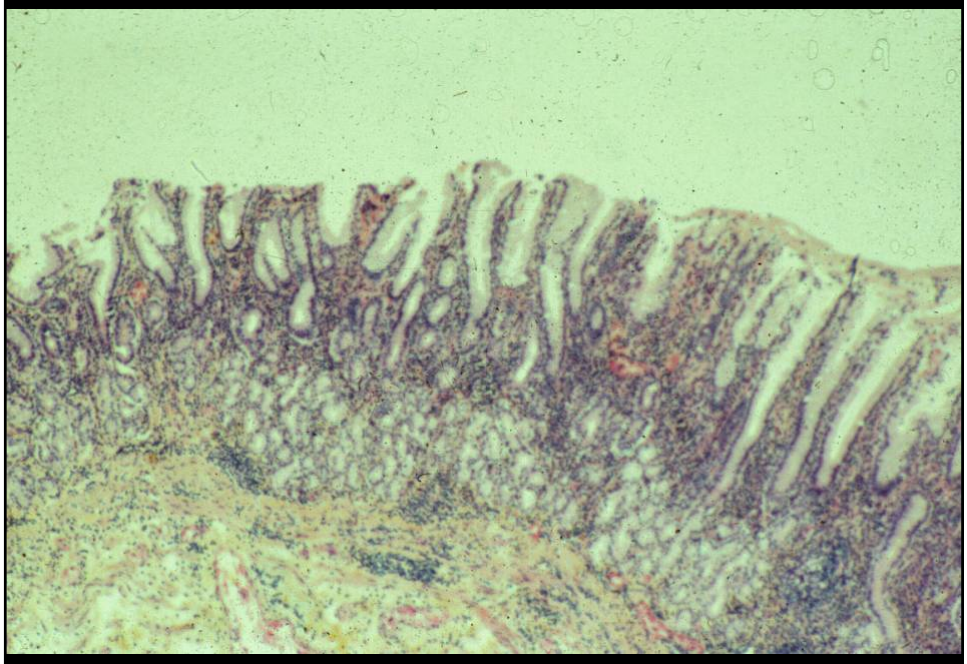
**Topographic Types**

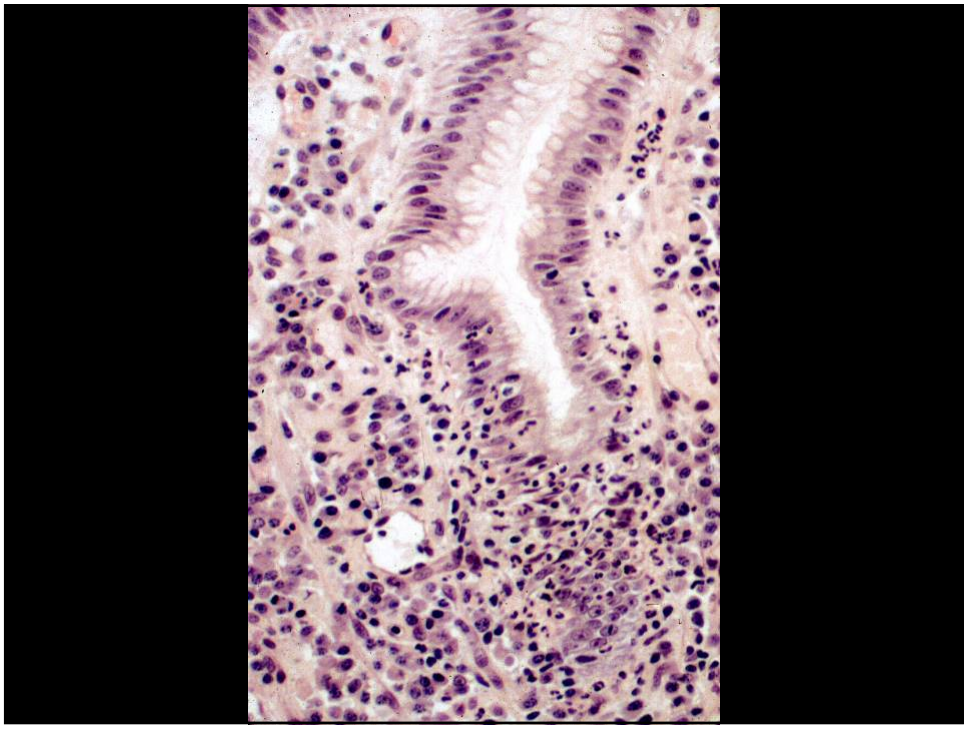
Chronic gastritis of antrum and corpus
Chronic gastritis, antrum-predominant
Chronic gastritis, corpus-predominant

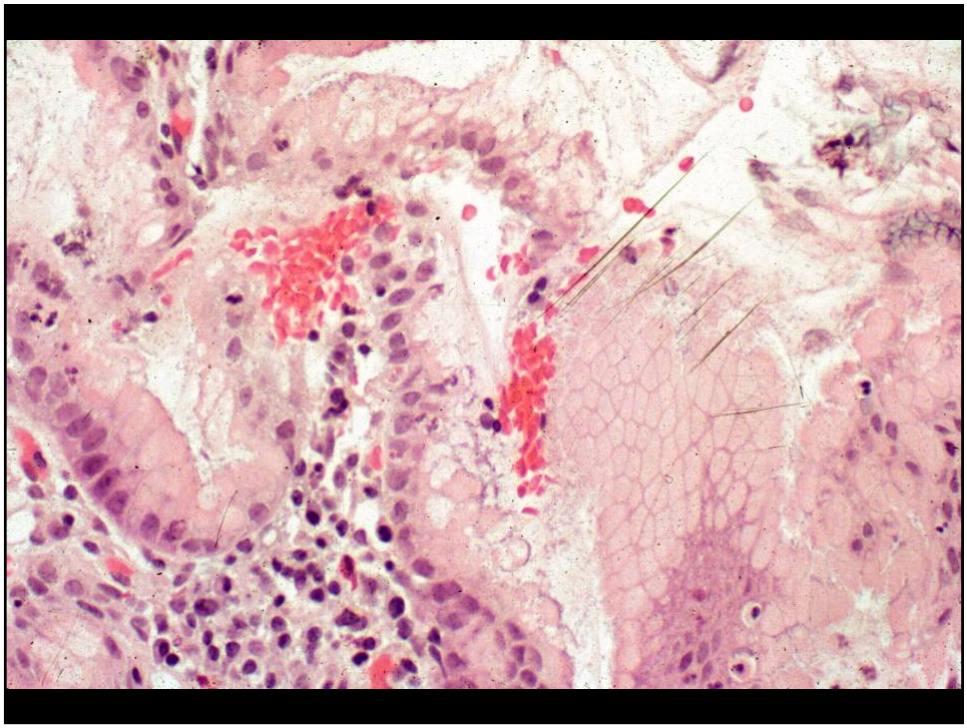
# H. PYLORI
# UNUSUAL FEATURES

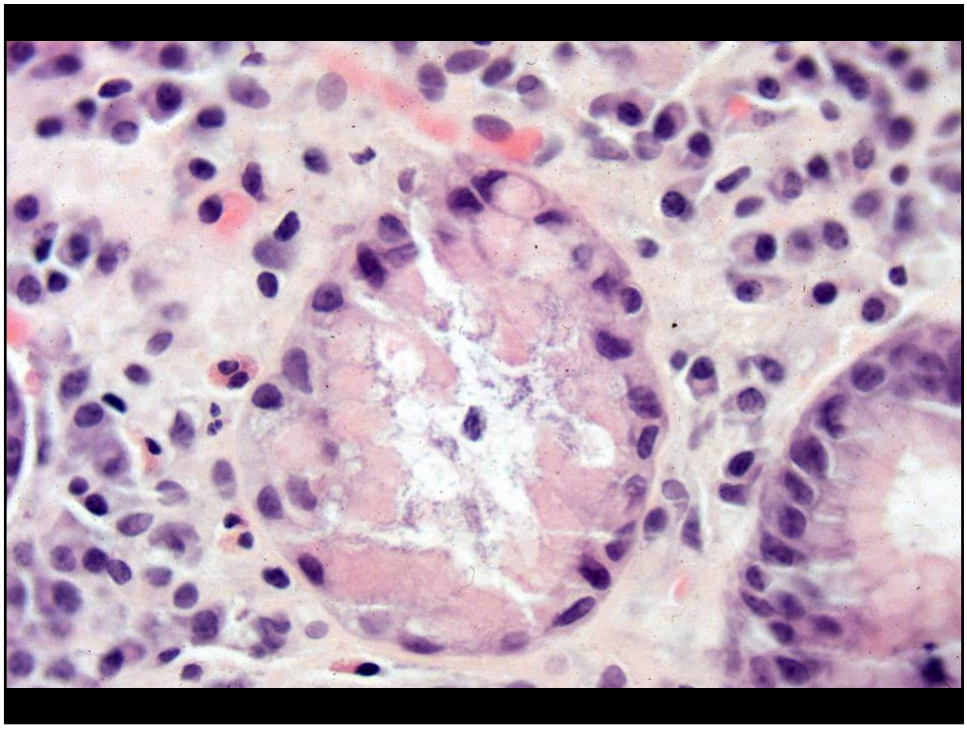
| | |
|---|---|
| First cultured in | 1982 (April 14) |
| "Unidentified curved bacilli" | 1983 |
| Campylobacter pyloridis | 1984 |
| Campylobacter pylori | 1987 |
| HELICOBACTER pylori | 1989 |

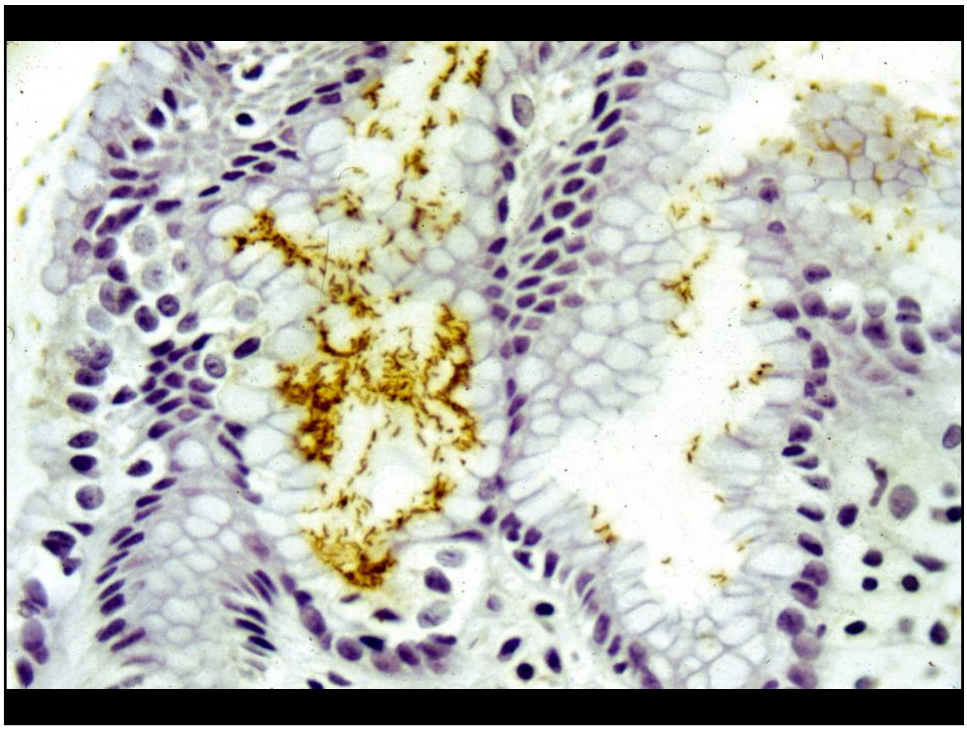
Natural habitat: human stomach

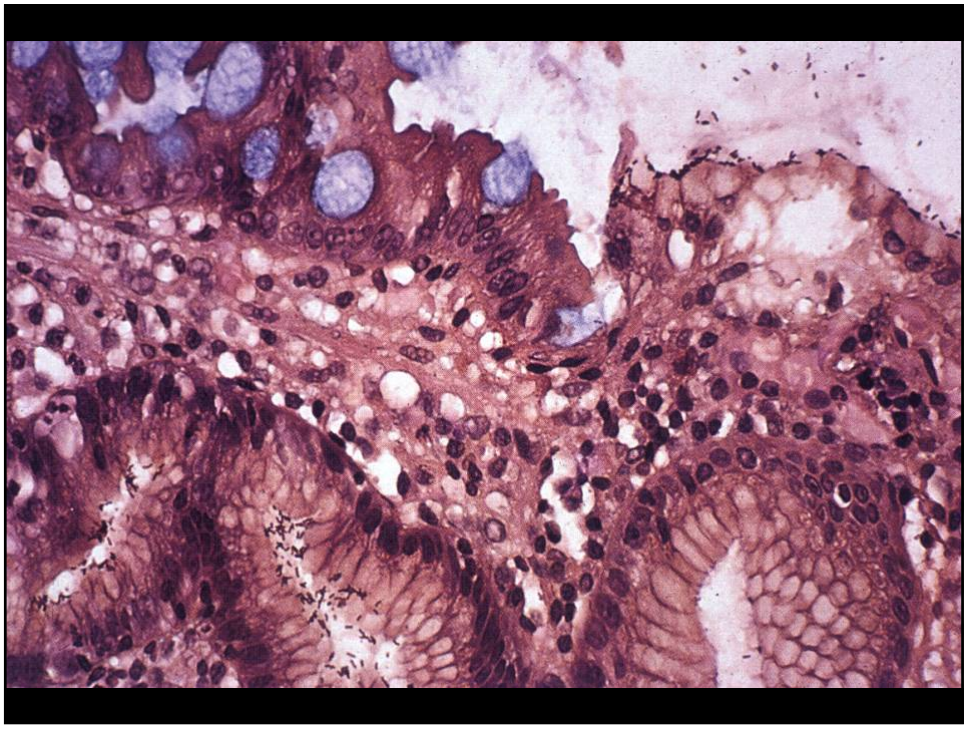












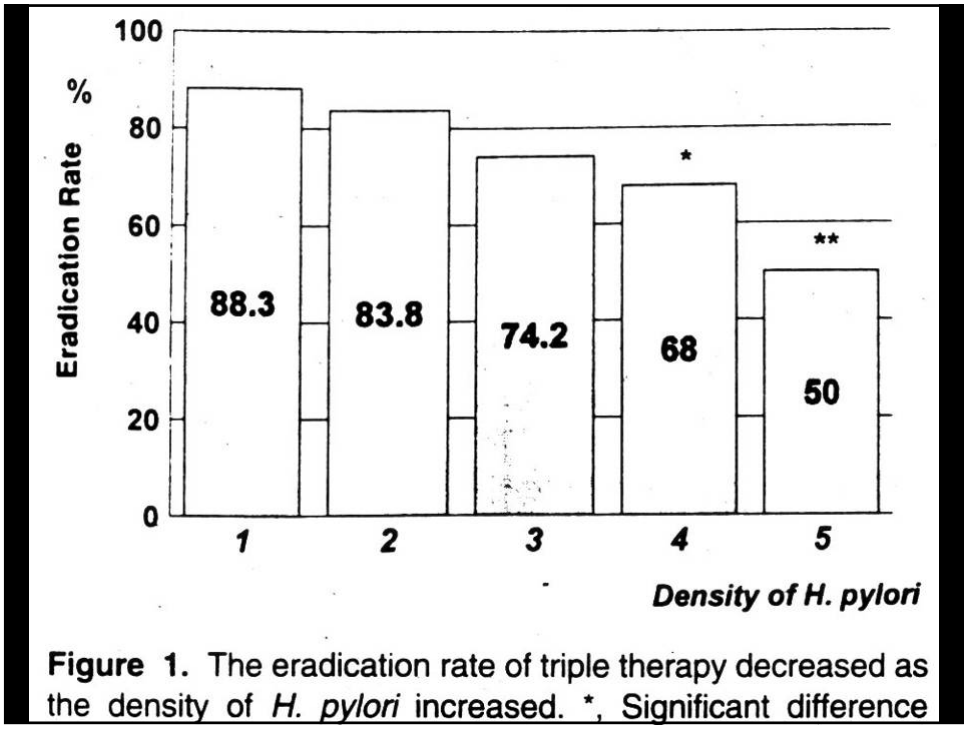
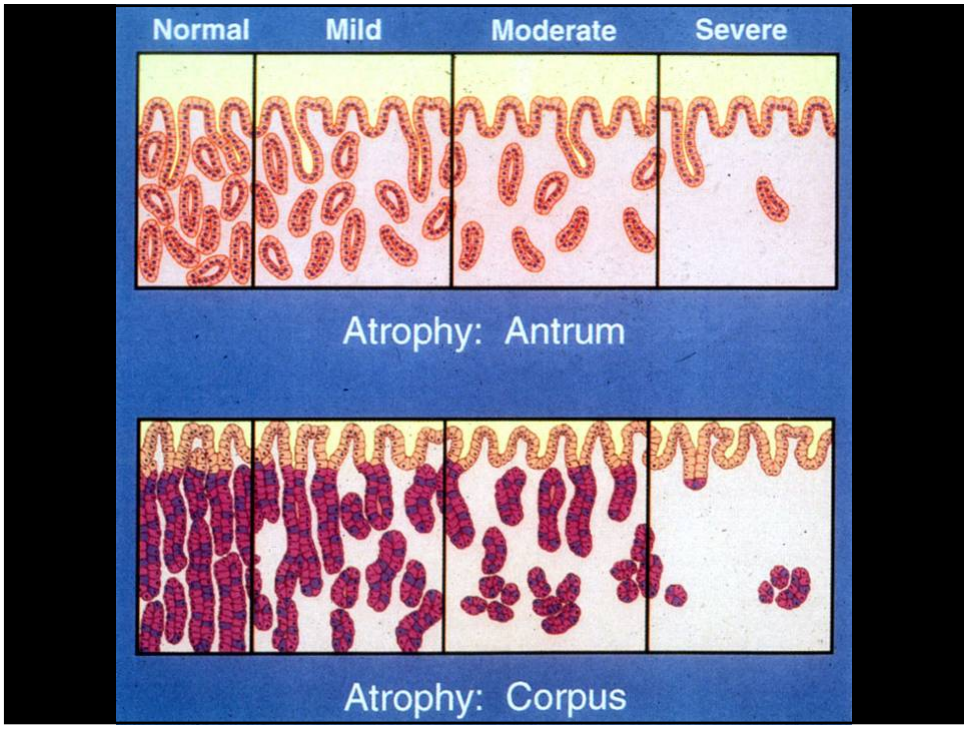
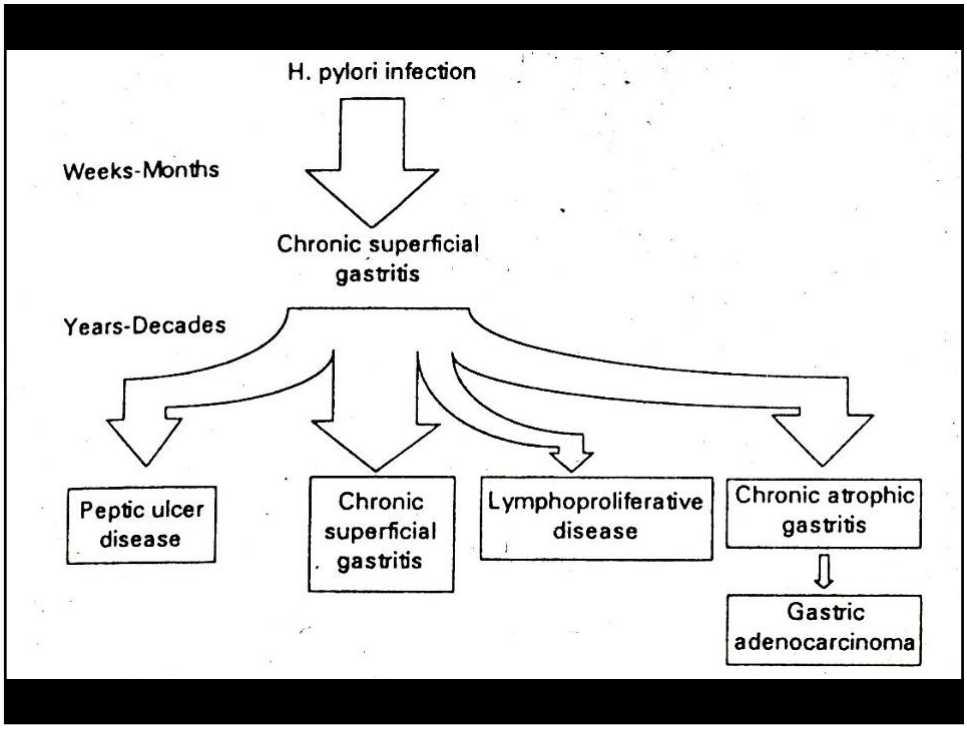



**Figure 1.** The eradication rate of triple therapy decreased as the density of *H. pylori* increased. *, Significant difference



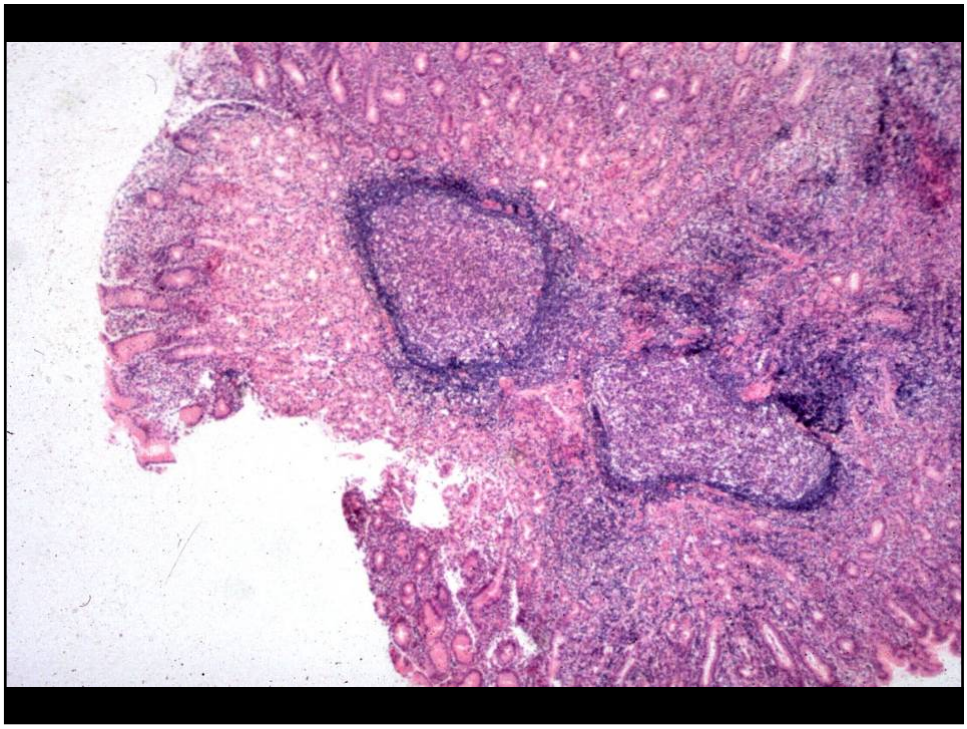

**Some Characteristics and Outcomes of the Chronic Gastritides**

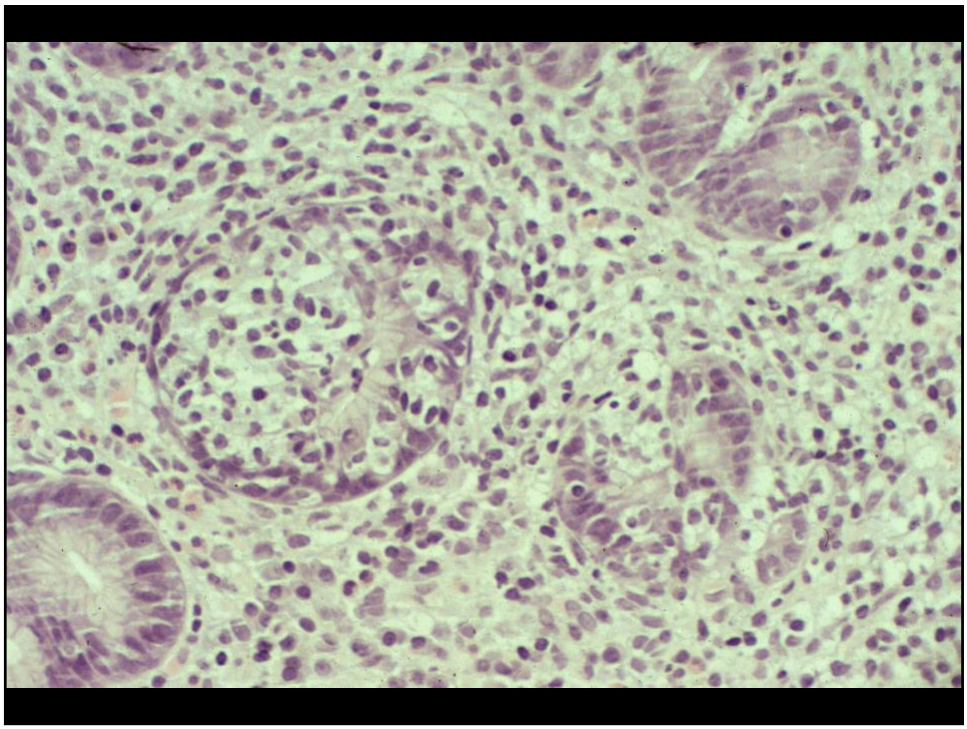
| | Topography of the Atrophy | | | |
|---|---|---|---|---|
| **Parameter** | **Severe antral atrophy** | **None** | **Severe panatrophy** | **Severe corpus atrophy** |
| HP related | Yes[a] | Yes | Yes[a] | No<br>Autoimmune pathogenesis |
| Gastrin output | Impaired | Mild increase | Low | High |
| Acid output | Normal | Normal | Low | Achlorhydria |
| Peptic ulcer | Increased | Increased | Slight | No |
| Relative risk | 30–40 | 10–40 | 1–2 | 0 |
| Gastric carcinoma | Markedly increased | Slightly increased | Greatly increased | Increased |
| Relative risk | 18 | 2 | Up to 90 | 3–5 |
| Other features | | | | Polyps (hyperplastic or inflammatory origin) |




## Complications of *H.pylori* gastritis: Frequency

- **Lymphoma: 0.1%**
- **Duodenal ulcer: 13%**
- **Carcinoma: 1%**





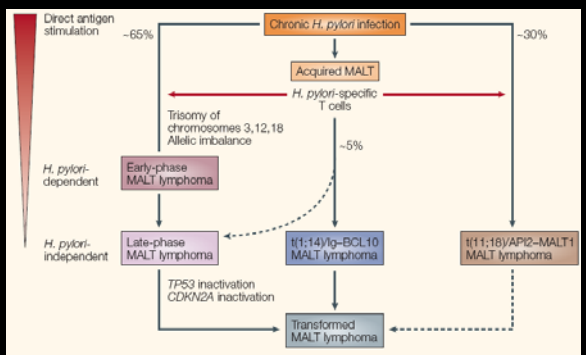
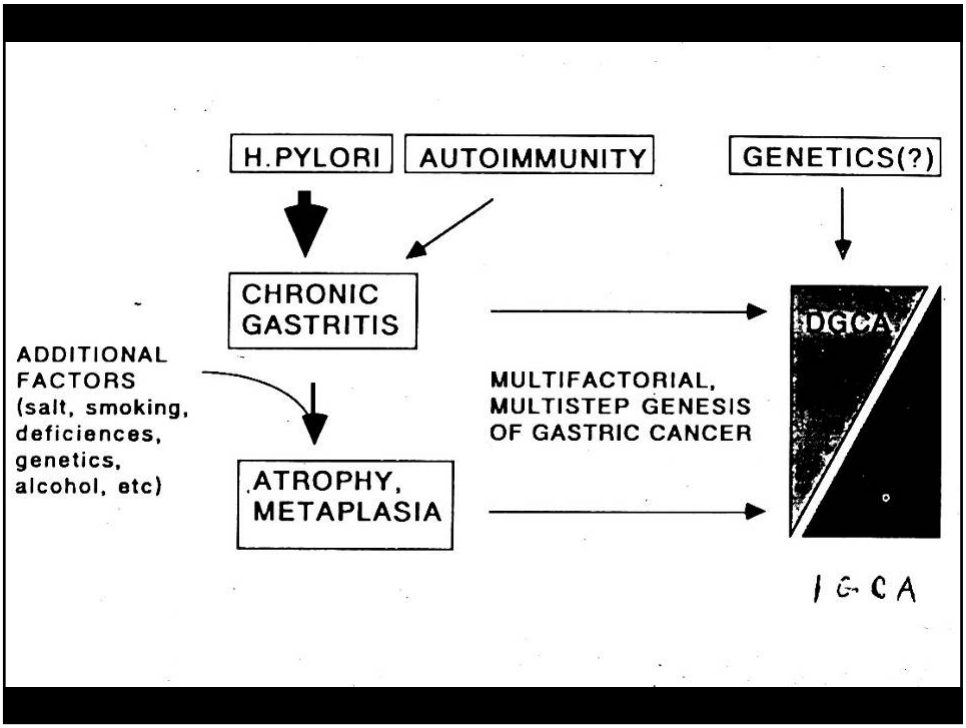



Figure 1 | **Multistage development of gastric MALT lymphoma.** *H. pylori* infection stimulates the production of lymphoid infiltrates, which leads to the formation of acquired mucosa-associated lymphoid tissue (MALT) in the gastric mucosa. As a result of both direct and indirect immunological stimulation (by auto-antigens *and Helicobacter-pylori*-specific T cells, respectively), infiltrating B-cells actively proliferate and occasionally undergo malignant transformation because of the acquisition of genetic abnormalities. Lymphomas with t(11;18)(q21;q21)/API2–MALT1 gain autonomous growth ability and do not respond to *H. pylori* eradication, but rarely develop into high-grade tumours (transformed MALT lymphoma). Lymphomas with t(1;14)(p22;q32)/Ig–BCL10 are probably *H. pylori*-independent and might undergo high-grade transformation. MALT lymphomas without these chromosomal translocations, sometimes carrying trisomies of chromosomes 3, 12 and 18, are *H. pylori*-dependent at early stages and can be effectively treated by *H. pylori* eradication. However, they can progress and become *H. pylori*-independent, and transform into high-grade tumours following inactivation of the tumour-suppressor genes *TP53* and *CDKN2A*.



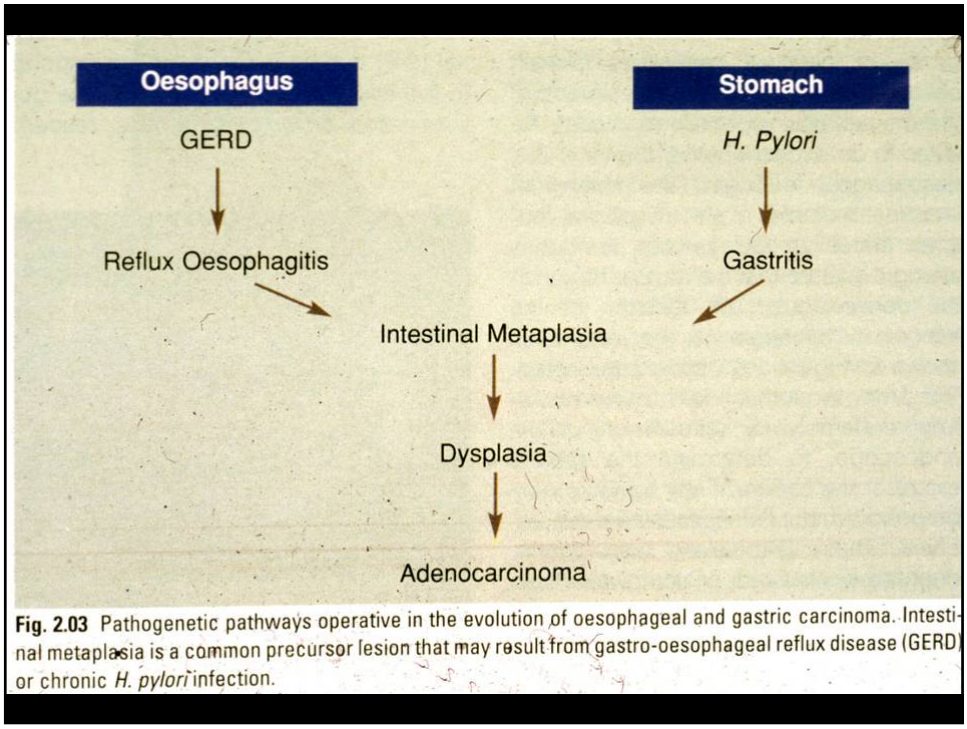
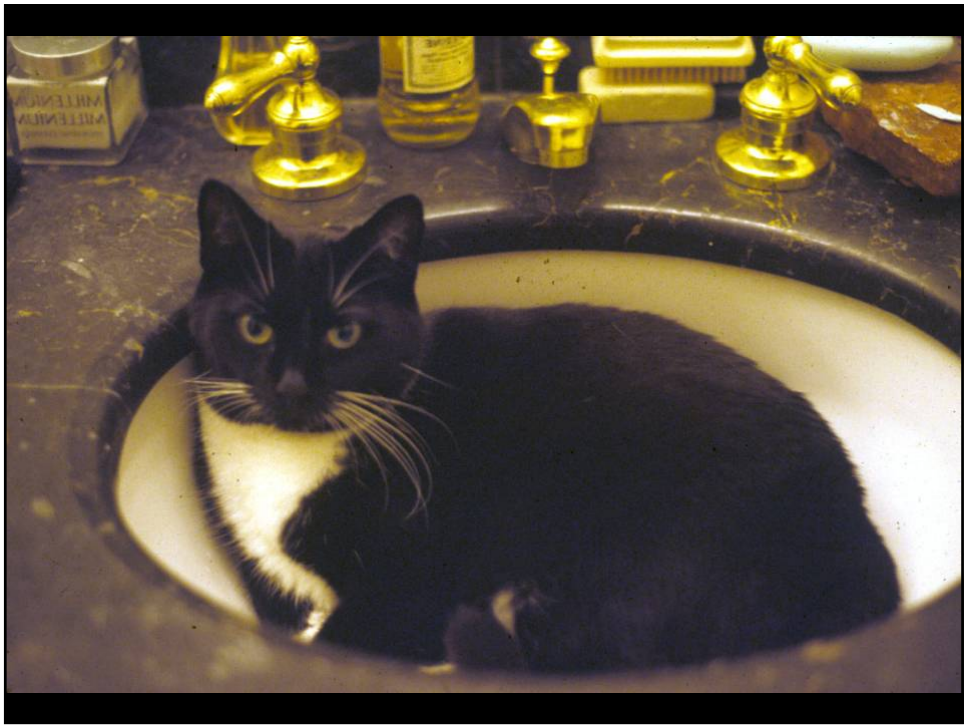
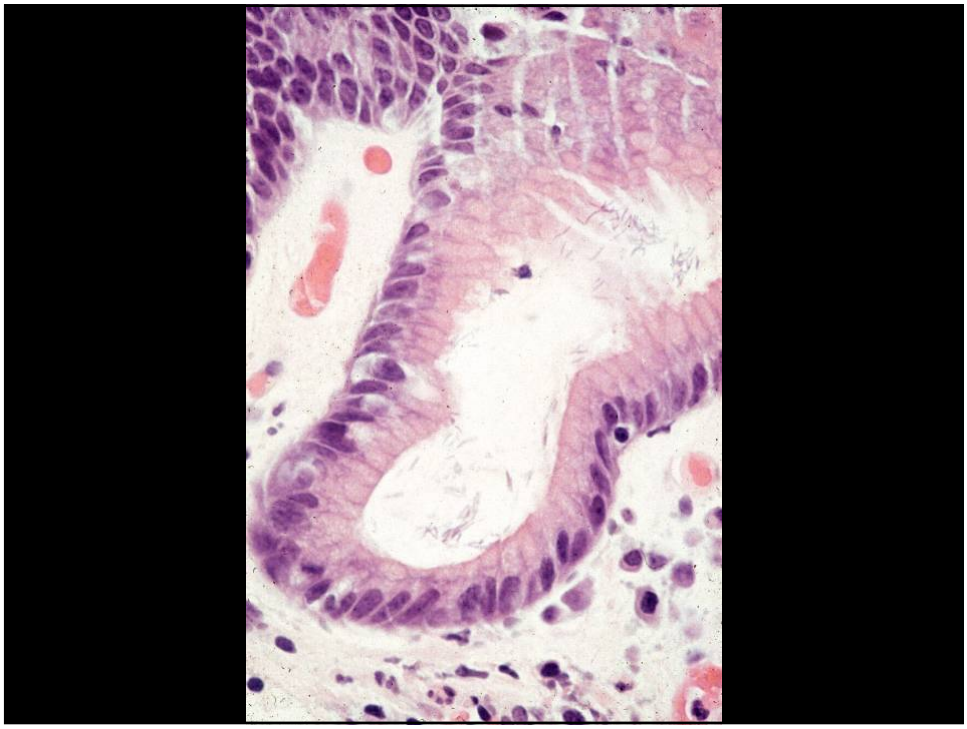



**Fig. 2.03** Pathogenetic pathways operative in the evolution of oesophageal and gastric carcinoma. Intestinal metaplasia is a common precursor lesion that may result from gastro-oesophageal reflux disease (GERD) or chronic *H. pylori* infection.





## *Helicobacter Heilmannii* Gastritis

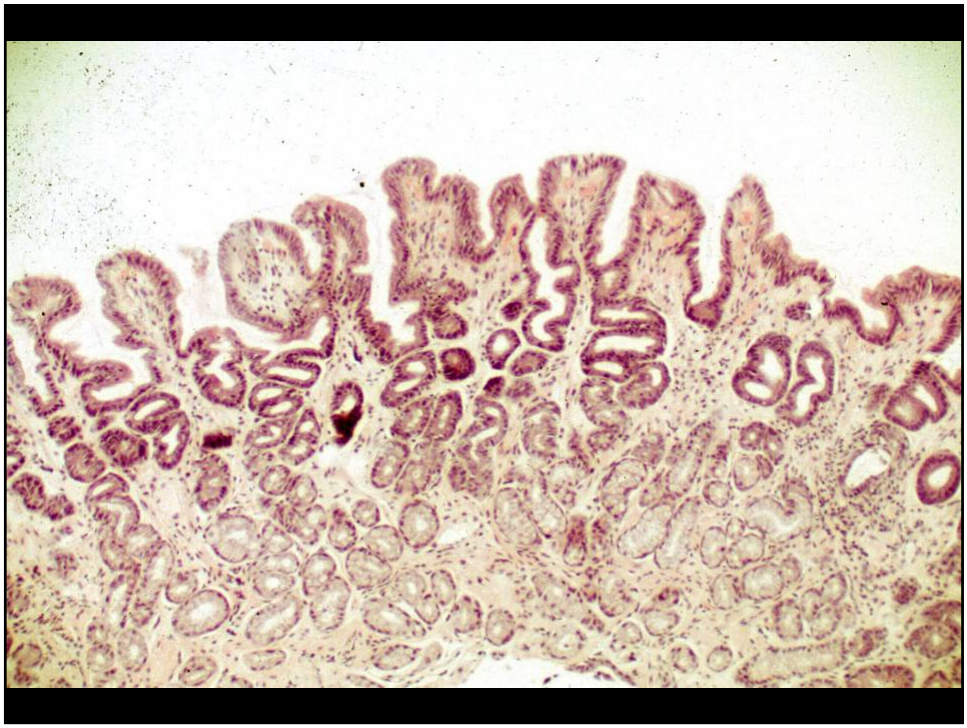
### Complications

- MALT lymphoma: 7/202
- Gastric cancer: 1/51 and 1/202
- Ulcers: 2/302 non *Hp* ulcers
- Coinfection with *H. pylori*: 1.6%

## "Chemical" Gastritis
## (type C gastritis, reactive gastropathy)

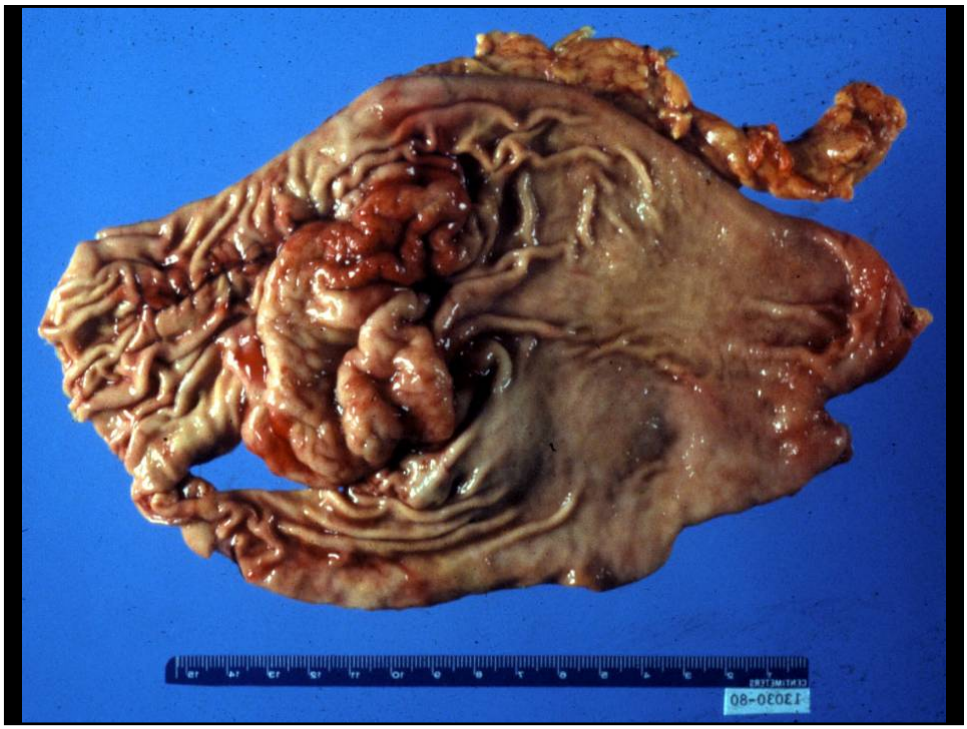
NSAIDS-related
Duodenal reflux-related

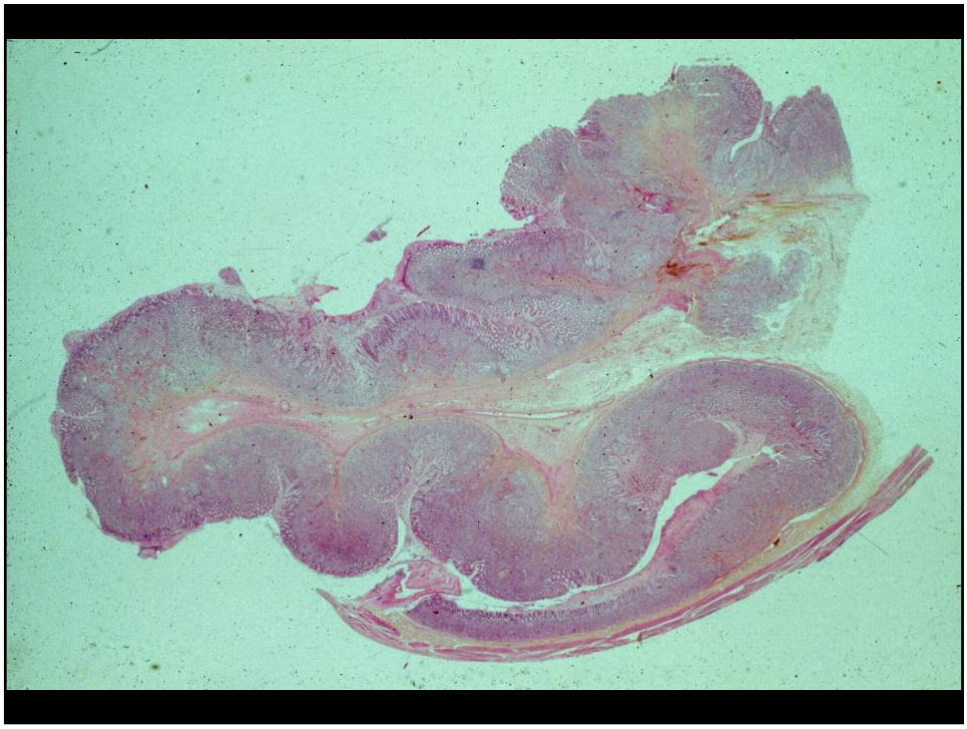
Foveolar hyperplasia
Mucosal edema and fibrosis
Mild chronic inflammation

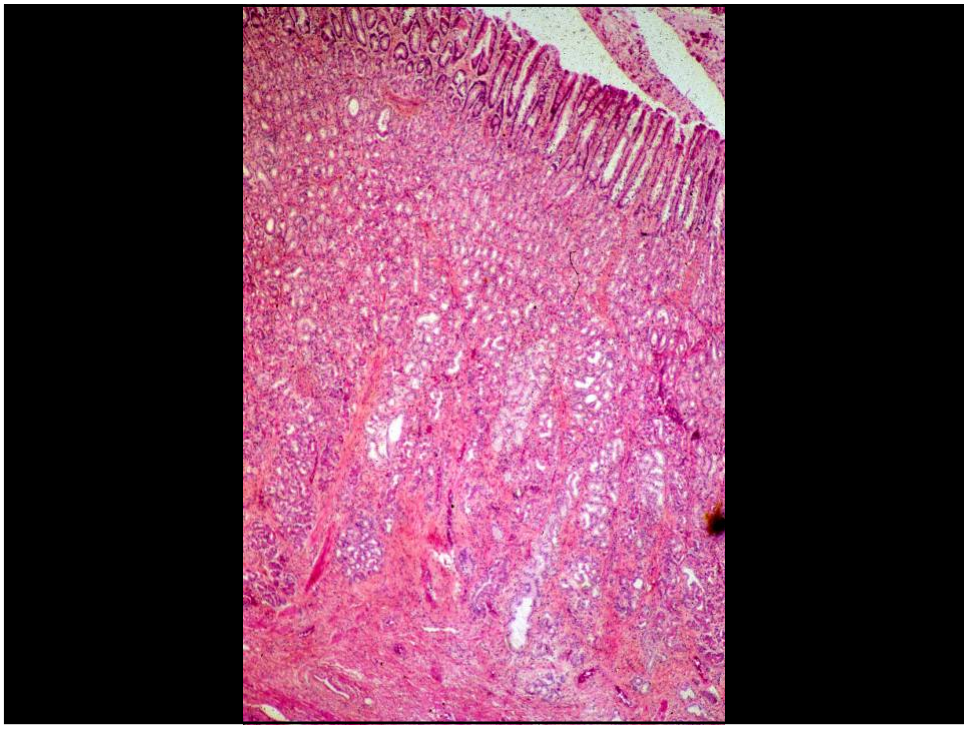


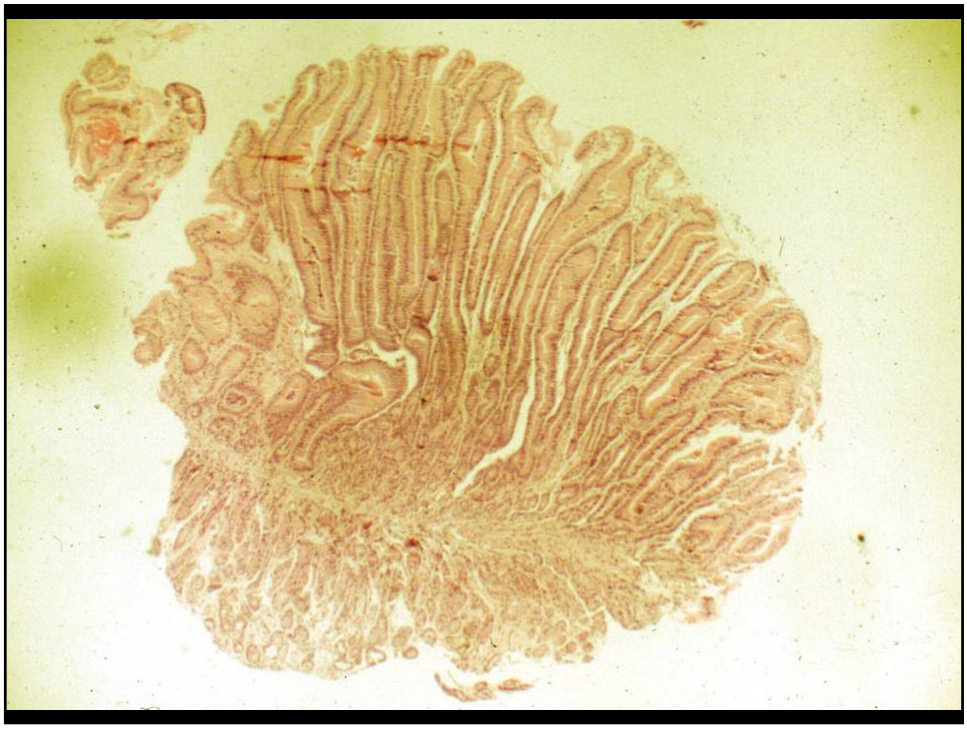
*Conditions which may demonstrate the changes of reactive gastropathy*

Duodenogastric reflux
Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs)
Alcohol
Vascular disturbances
shock, ischemia, stress,
Local trauma (nasogastric tubes)
Radiation and chemotherapy
Idiopathic



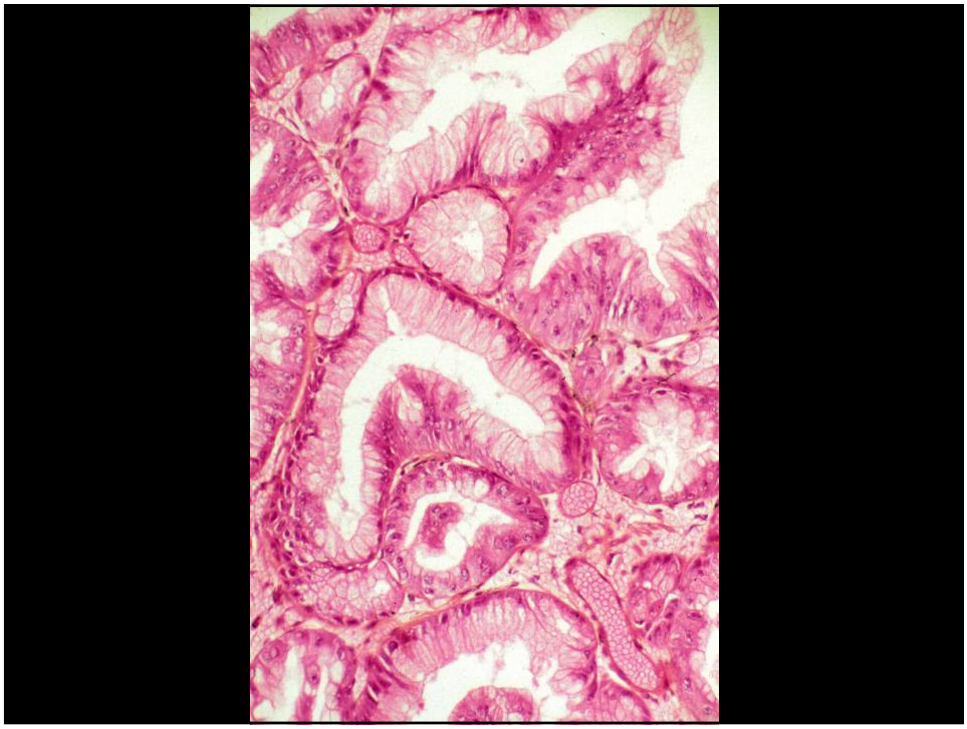


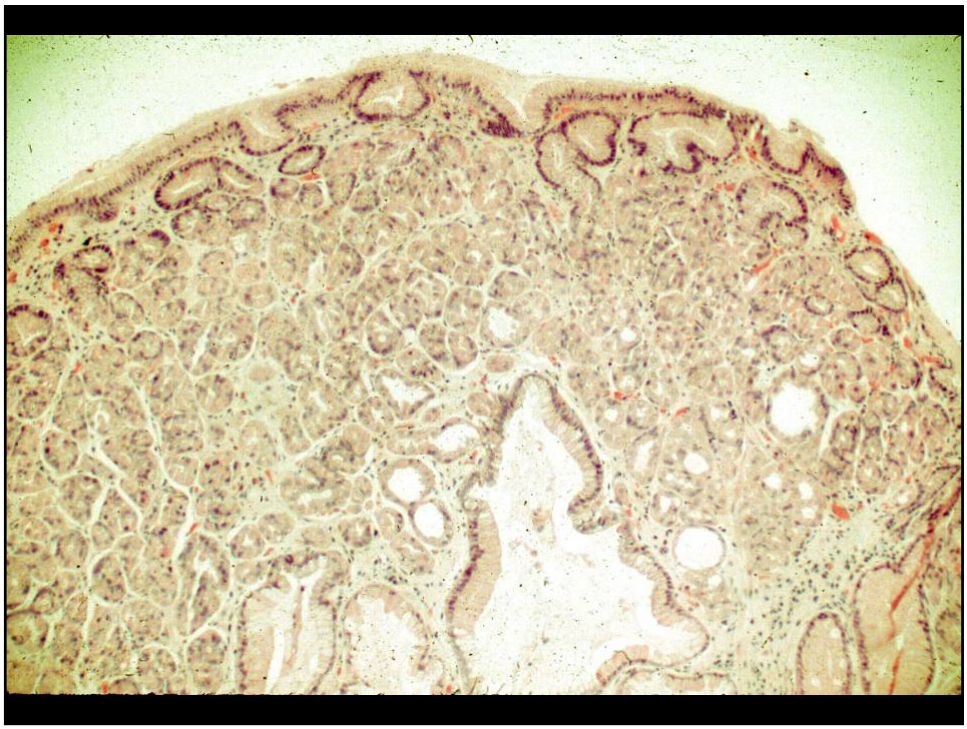


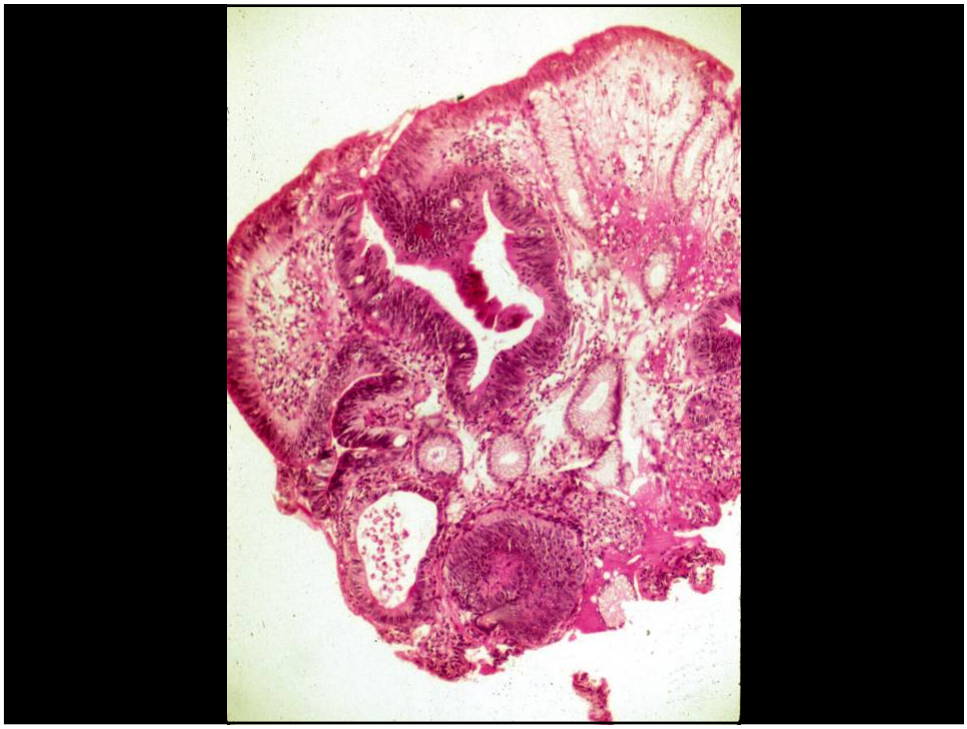


## Gastric Polyps

- Hyperplastic polyp
- Fundic gland polyp
- Adenomatous polyp
- Inflammatory fibroid polyp







**Epithelial tumours**

Intraepithelial neoplasia – Adenoma

Carcinoma

- Adenocarcinoma
  - intestinal type
  - diffuse type
- Papillary adenocarcinoma
- Tubular adenocarcinoma
- Mucinous adenocarcinoma
- Signet-ring cell carcinoma
- Adenosquamous carcinoma
- Squamous cell carcinoma
- Small cell carcinoma
- Undifferentiated carcinoma
- Others

Carcinoid (well differentiated endocrine neoplasm)

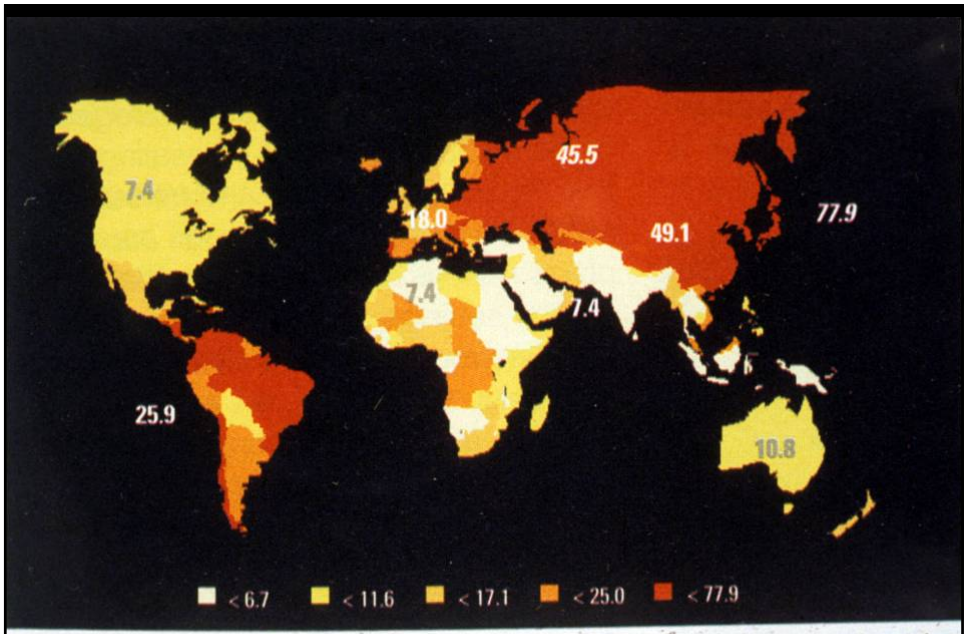



**Fig. 3.01** Worldwide annual incidence (per 100,000) of stomach cancer in males. Numbers on the map indicate regional average values.

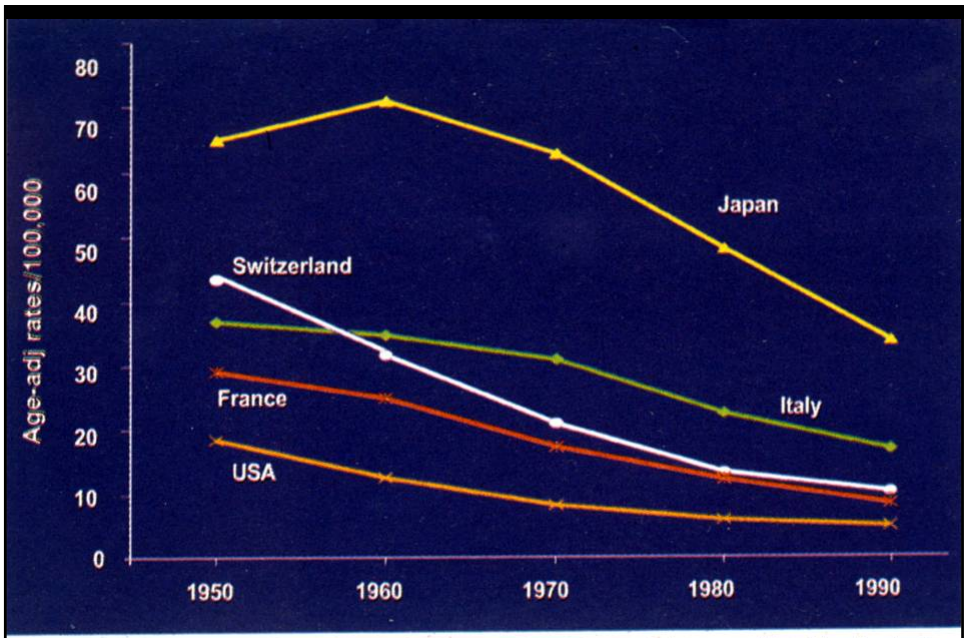



**Fig. 3.02** The mortality of stomach cancer is decreasing worldwide, including countries with a high disease burden.

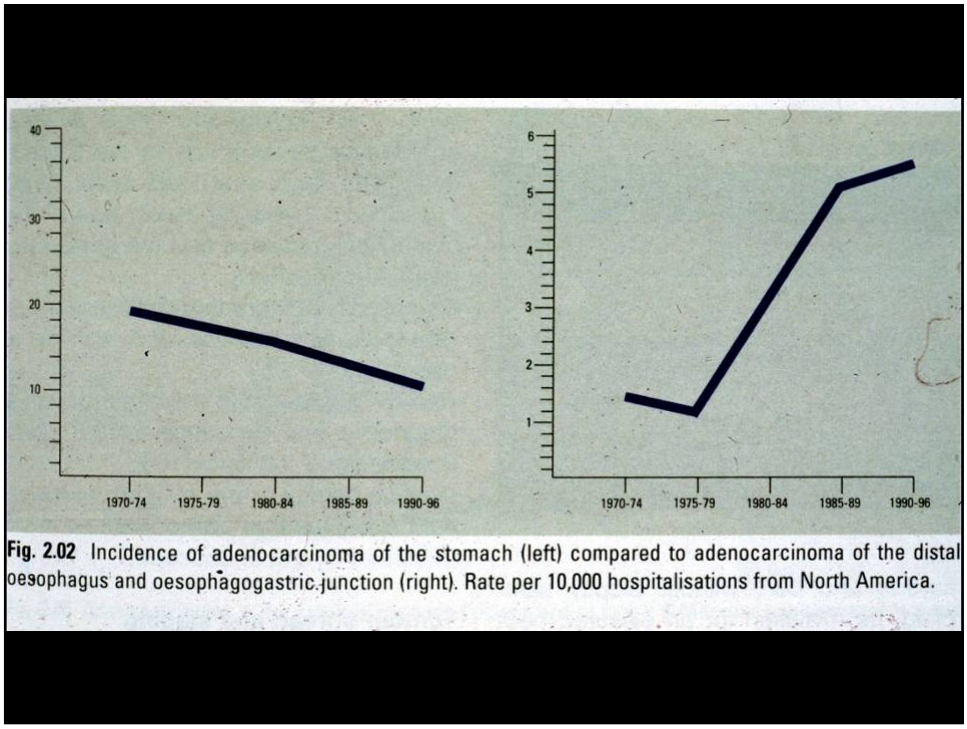
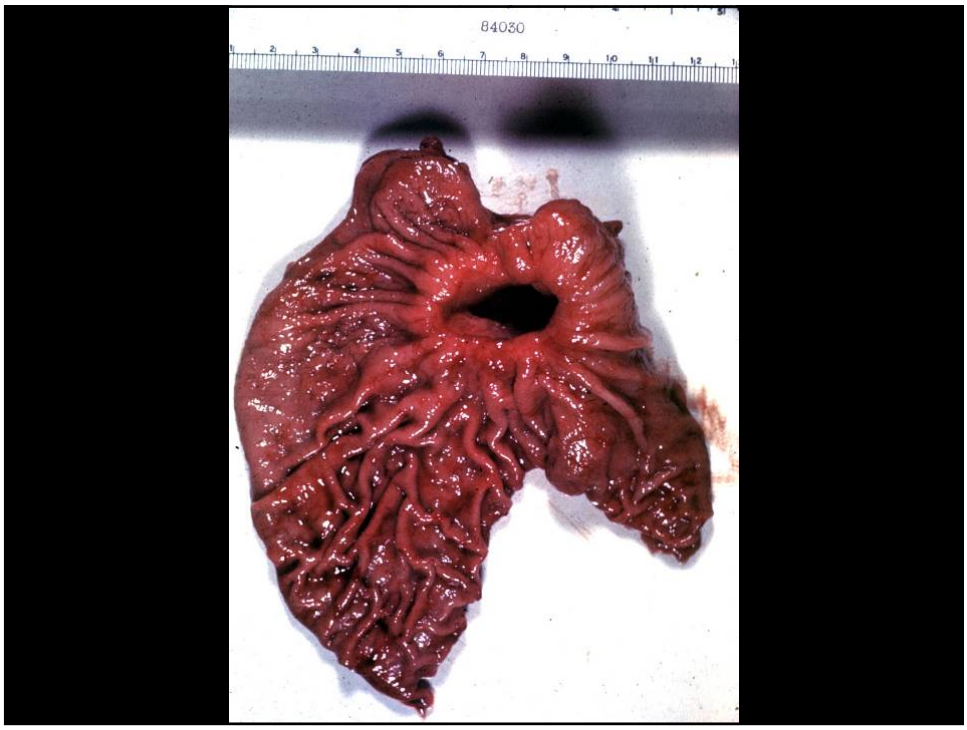
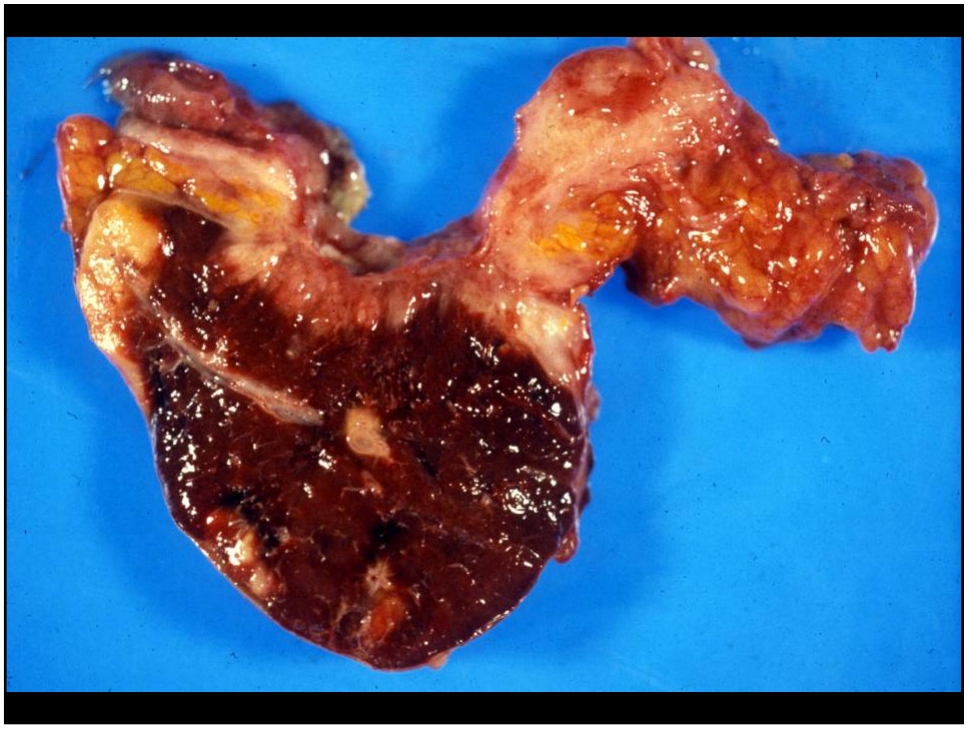
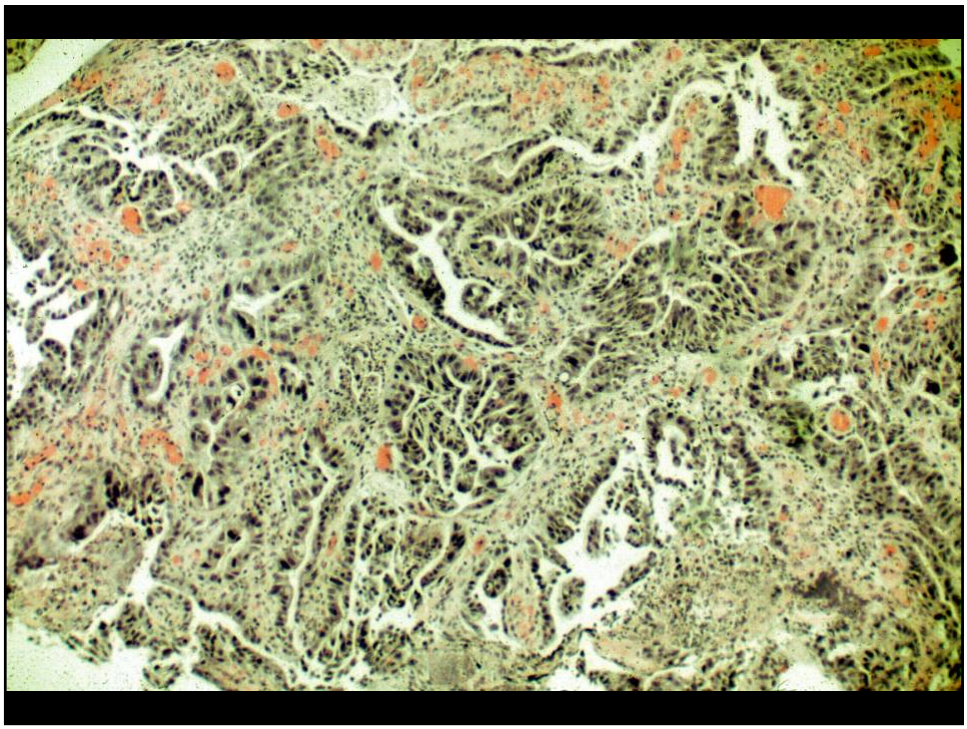
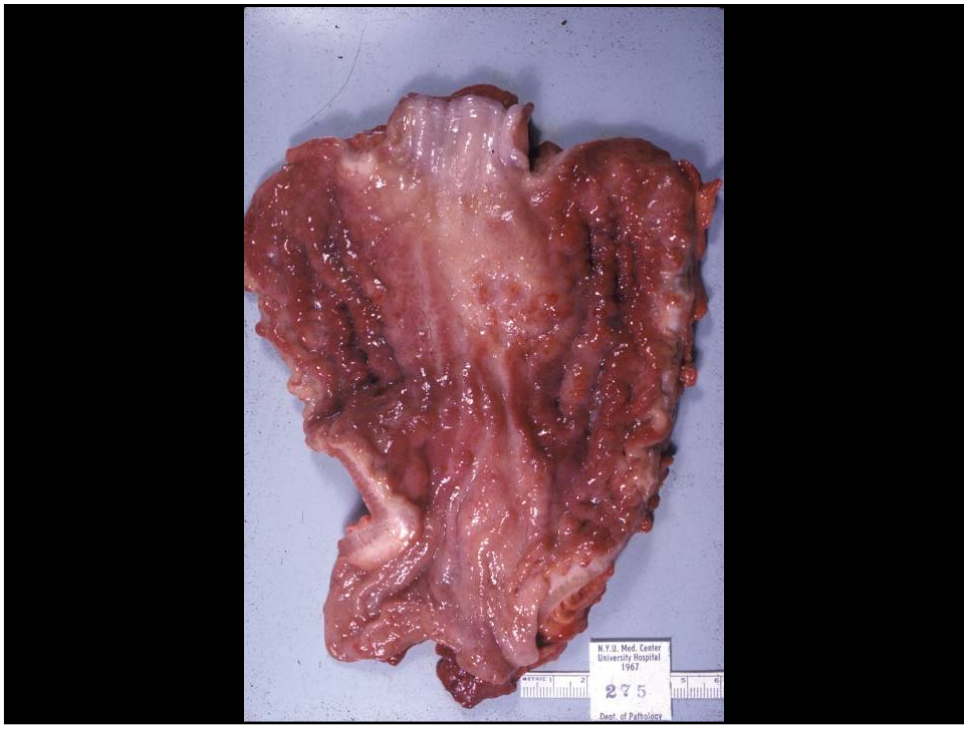



**Fig. 2.02** Incidence of adenocarcinoma of the stomach (left) compared to adenocarcinoma of the distal oesophagus and oesophagogastric junction (right). Rate per 10,000 hospitalisations from North America.








N.Y.U. Med. Center
University Hospital
1967
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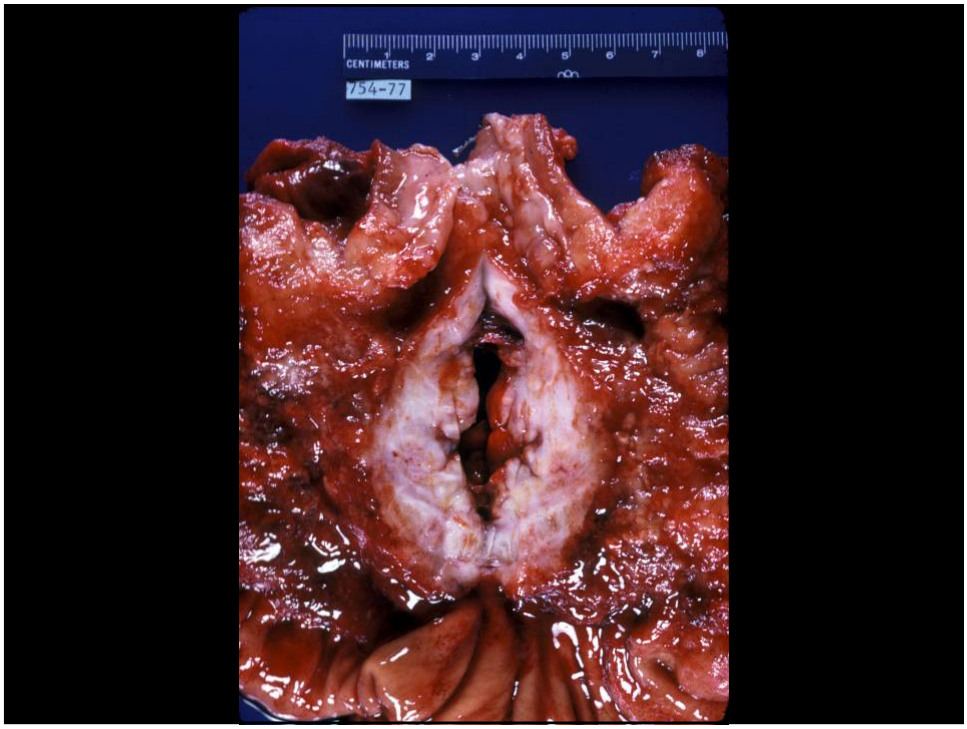
Dept. of Pathology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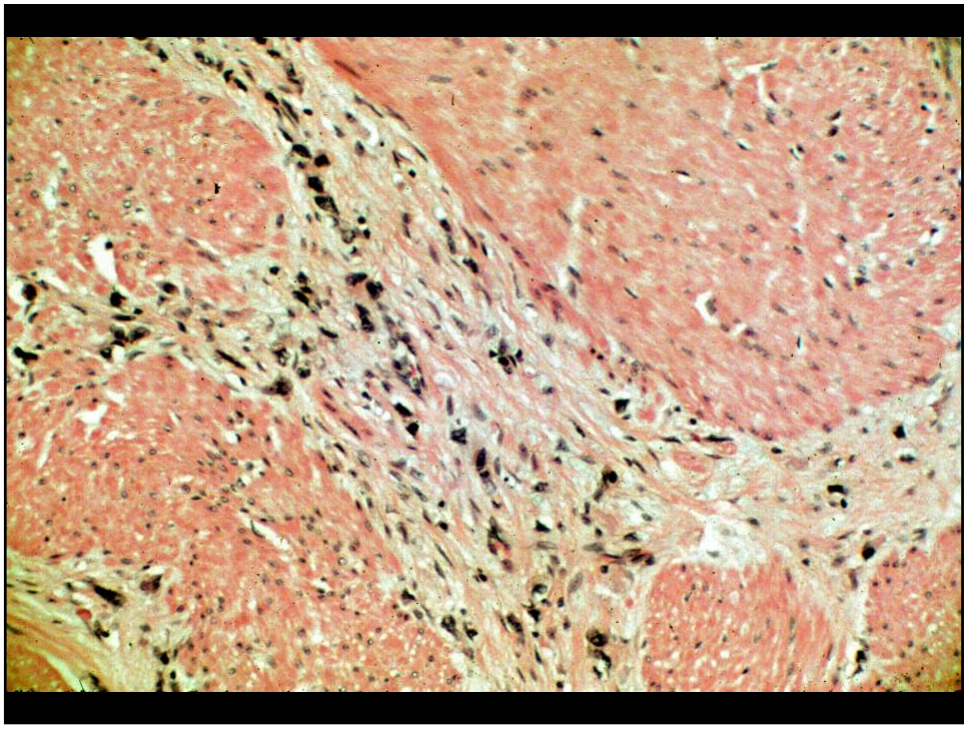


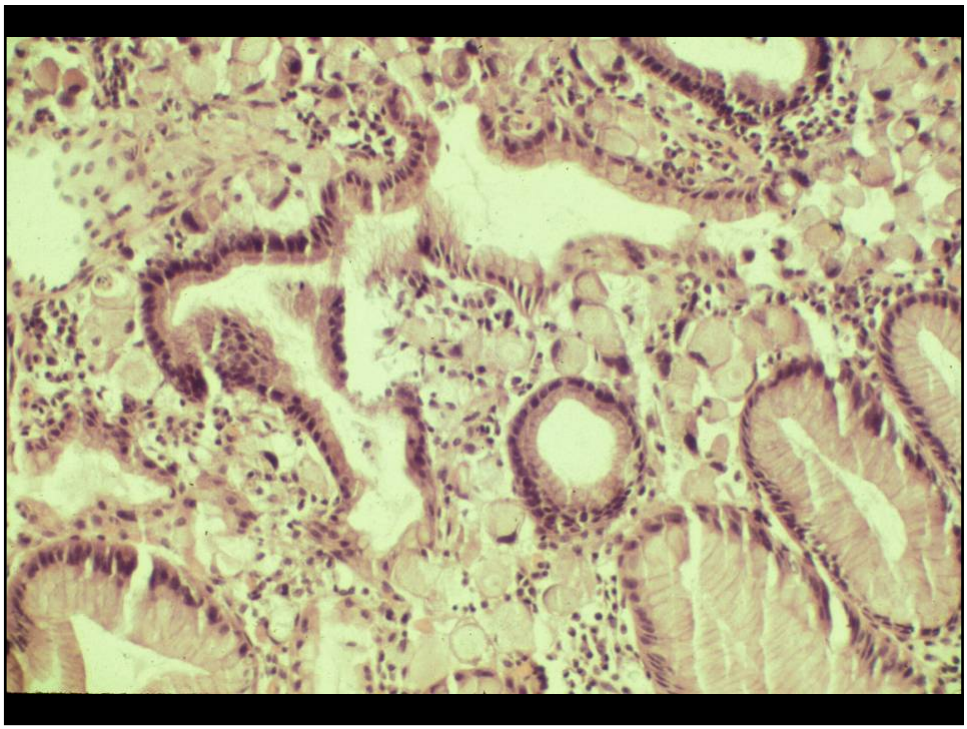


**Table 17–4.** MAJOR FEATURES OF LAURENS' CLASSIFICATION OF GASTRIC CARCINOMA

| FEATURE | TYPE OF CARCINOMA | |
|---|---|---|
| | ***Intestinal*** | ***Diffuse*** |
| Most common gross configuration | Polypoid; fungating | Ulcerative; infiltrating |
| Microscopic features | | |
| Differentiation | Well-differentiated; gland-forming | Poorly differentiated; signet-ring cells |
| Mucin production | Limited: confined to gland lumens | Extensive: may be prominent in stroma around glands ("colloid" carcinoma) |
| Growth pattern | Expansile; inflammation often prominent | Noncohesive; infiltrative |
| Association with intestinal metaplasia | Almost universal | Less frequent |
| Clinical features | | |
| Mean age (years) | 55 | 48 |
| Sex ratio (M:F) | 2:1 | approximately 1:1 |
| Decreasing incidence in Western countries | Yes | No |

Adapted from Antonioli, D.A.: Gastric carcinoma and its precursors. Monogr. Pathol. *31*:144, 1990.

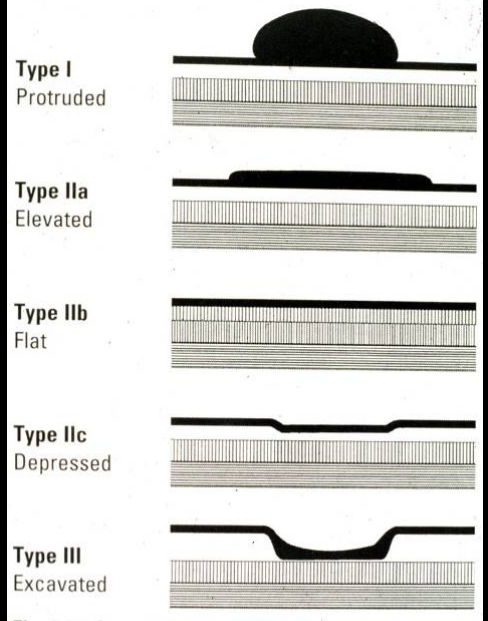
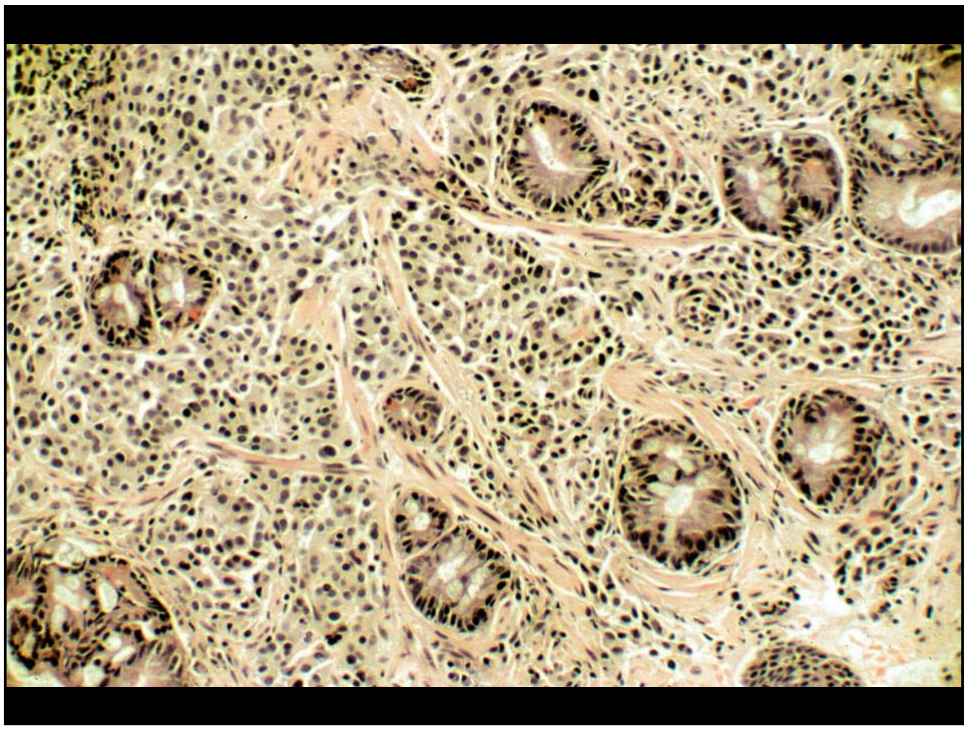


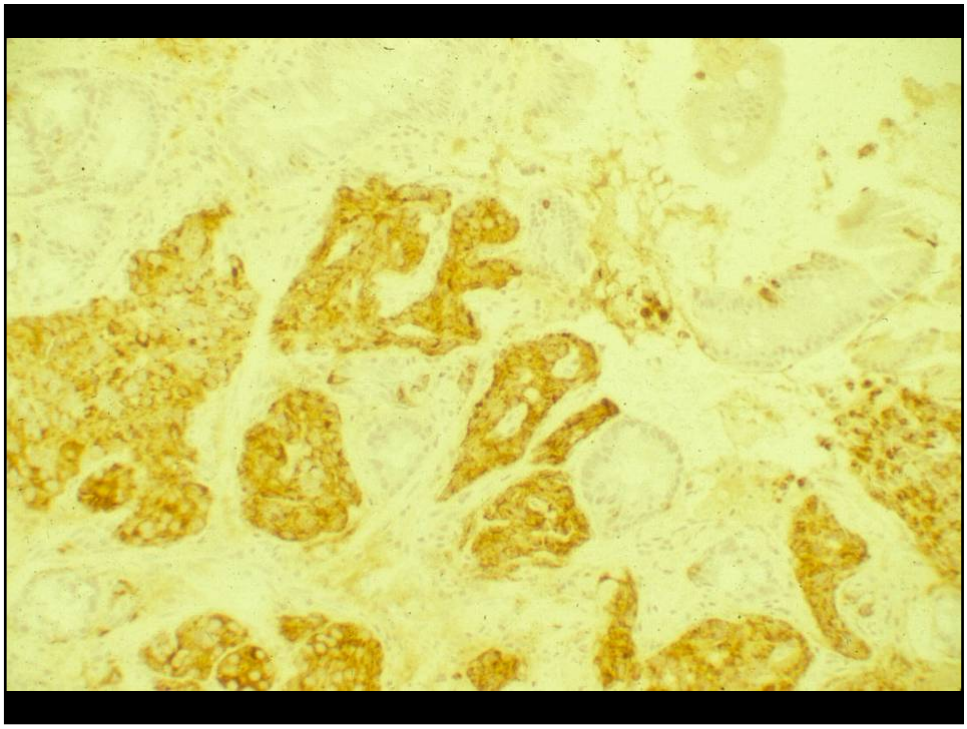

**Fig. 3.04** Growth features of early gastric carcinoma.

**Table 3.02.**
Histological classification of endocrine neoplasms of the stomach[1]

| |
|---|
| **1. Carcinoid – well differentiated endocrine neoplasm** |
| 1.1 ECL-cell carcinoid |
| 1.2 EC-cell, serotonin-producing carcinoid |
| 1.3 G-cell, gastrin-producing tumour |
| 1.4 Others |
| **2. Small cell carcinoma – poorly differentiated endocrine neoplasm** |
| **3. Tumour-like lesions** |
| Hyperplasia |
| Dysplasia |

[1] Benign behaviour of ECL-cell carcinoid is associated with the following: tumour confined to mucosa-submucosa, nonangioinvasive, < 1cm in size, nonfunctioning; occurring in CAG or MEN-1/ ZES. Aggressive behaviour of ECL-cell carcinoid is associated with the following: tumour invades muscularis propria or beyond, > 1cm in size, angioinvasive, functioning, and sporadic occurrence.





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|---|---|
| **Non-epithelial tumours** | |
| Leiomyoma | 8890/0 |
| Schwannoma | 9560/0 |
| Granular cell tumour | 9580/0 |
| Glomus tumour | 8711/0 |
| Leiomyosarcoma | 8890/3 |
| GI stromal tumour | 8936/1 |
| benign | 8936/0 |
| uncertain malignant potential | 8936/1 |
| malignant | 8936/3 |
| Kaposi sarcoma | 9140/3 |
| Others | |
| Malignant lymphomas | |
| Marginal zone B-cell lymphoma of MALT-type | 9699/3 |
| Mantle cell lymphoma | 9673/3 |
| Diffuse large B-cell lymphoma | 9680/3 |
| Others | |
| **Secondary tumours** | |