

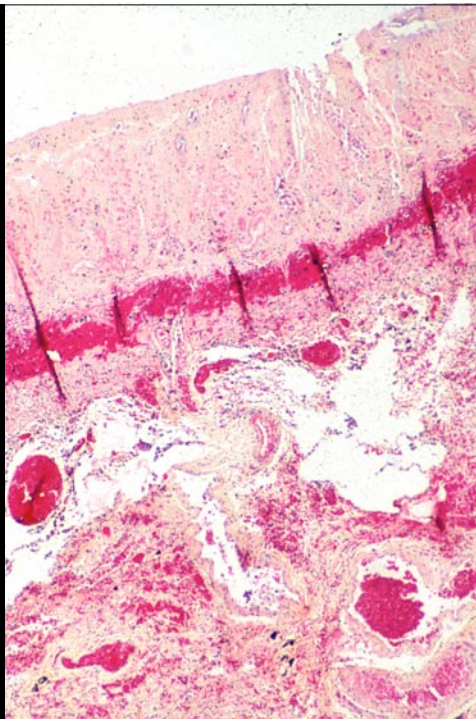
## Gastritis: Causes

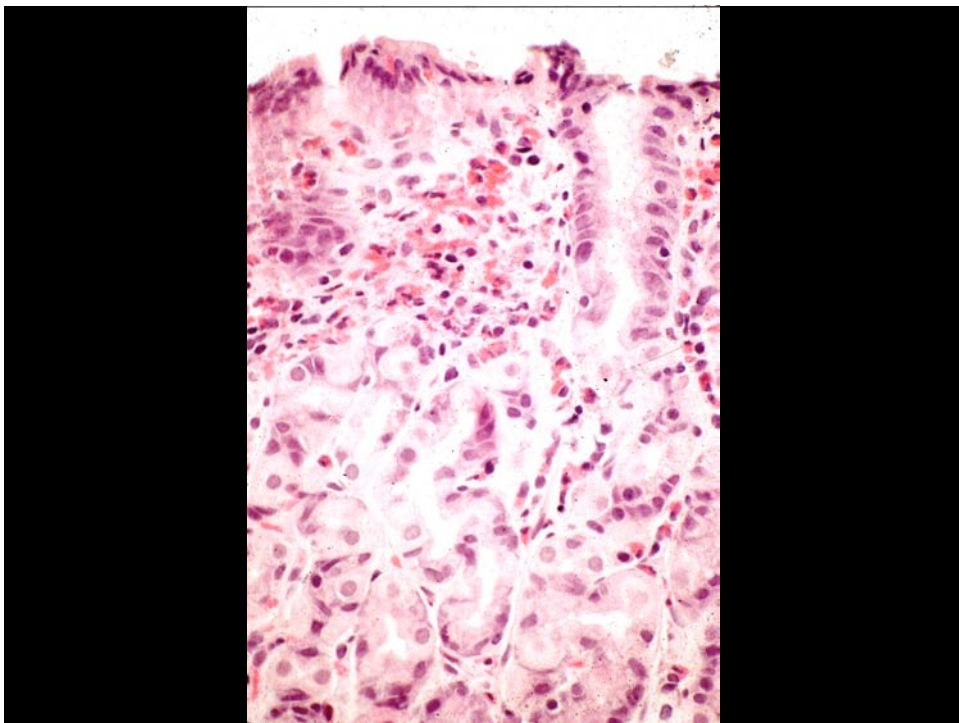
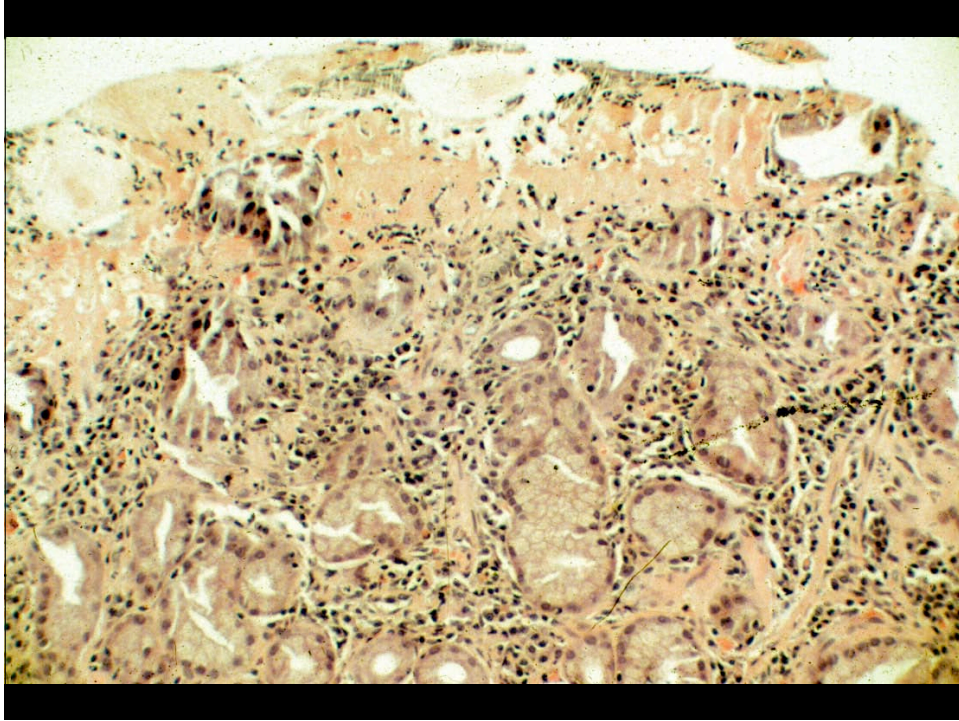
- *Helicobacter pylori* infection
- NSAID use
- Excessive alcohol consumption
- Heavy smoking
- Radiotherapy
- Cancer chemotherapy
- Systemic infections (*Salmonella*, CMV)
- Severe stress
- Ischemia and shock
- Suicide attempts with acids or alkali
- Mechanical trauma
- Distal gastrectomy

## 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)

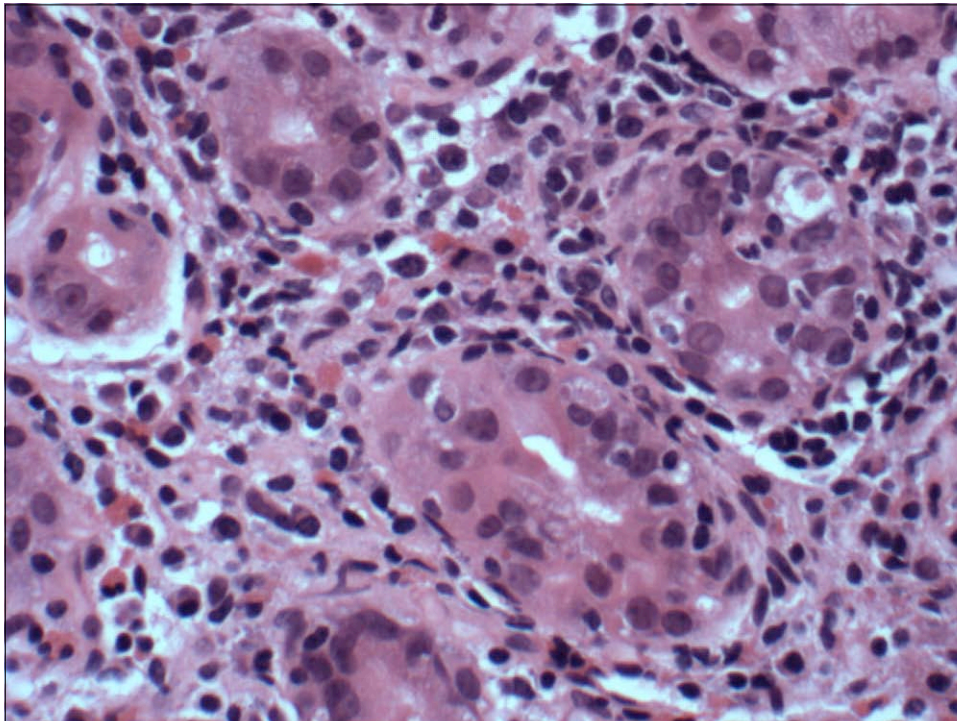


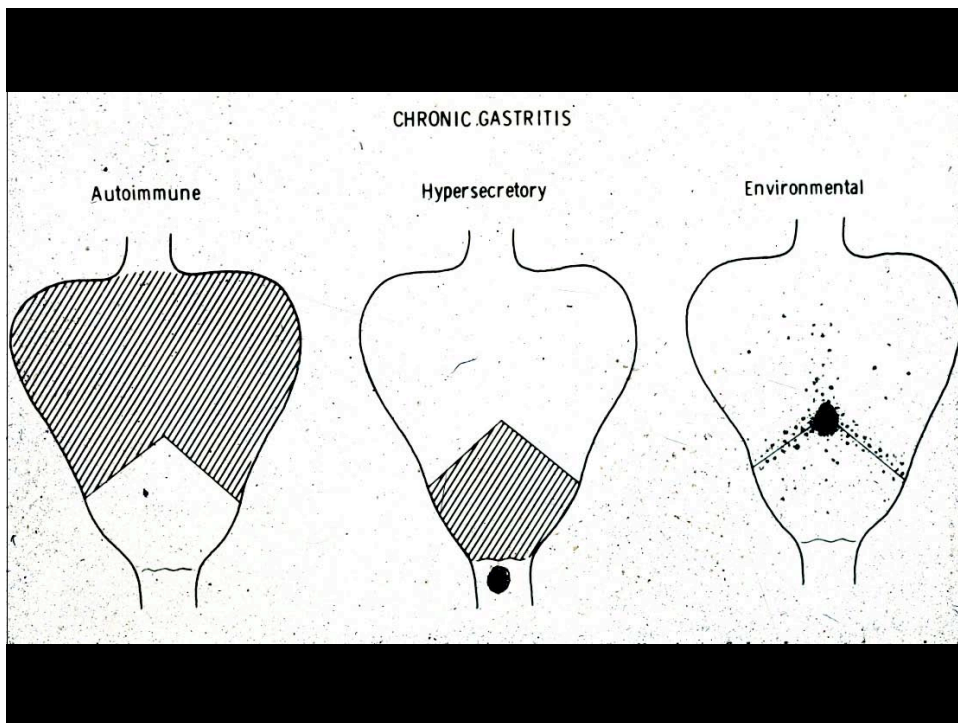
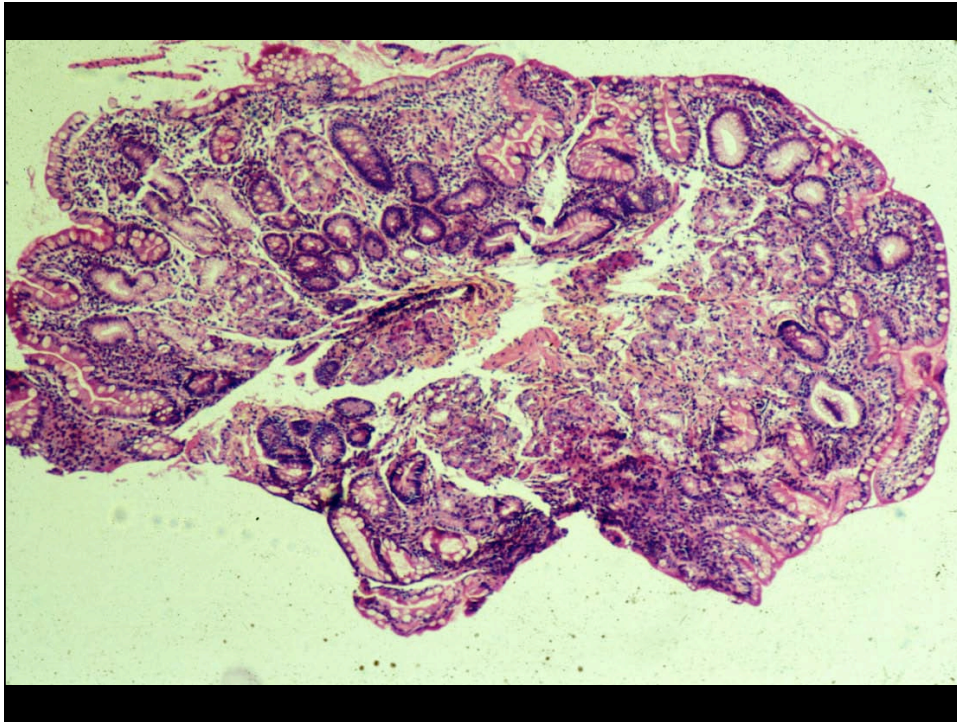




## Types of Chronic Gastritis

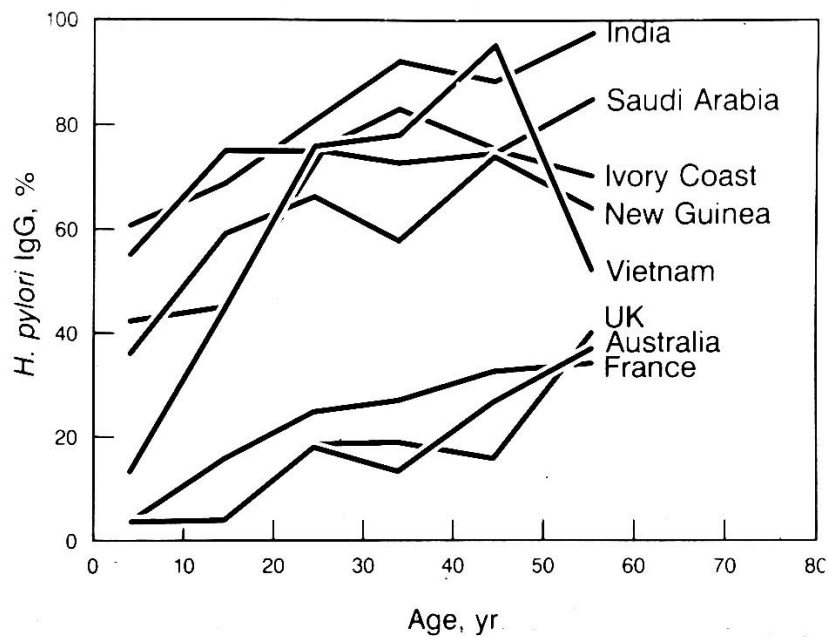
- **Autoimmune gastritis (type A gastritis):** diffuse gastritis of corpus; antibodies to parietal cells and intrinsic factor; low acid, pernicious anemia; associated with other autoimmune disorders; uncommon.
- ***Helicobacter pylori* gastritis (type B gastritis):** may affect all parts of the stomach, 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.





## 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**

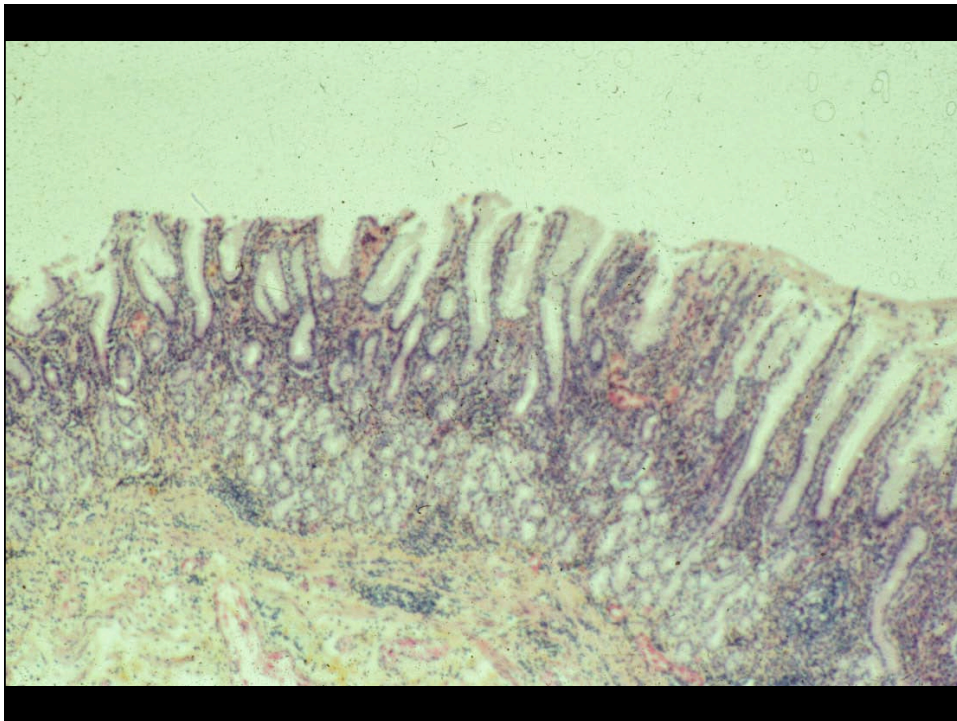
Chronic gastritis of antrum and corpus  
Chronic gastritis, antrum-predominant  
Chronic gastritis, corpus-predominant



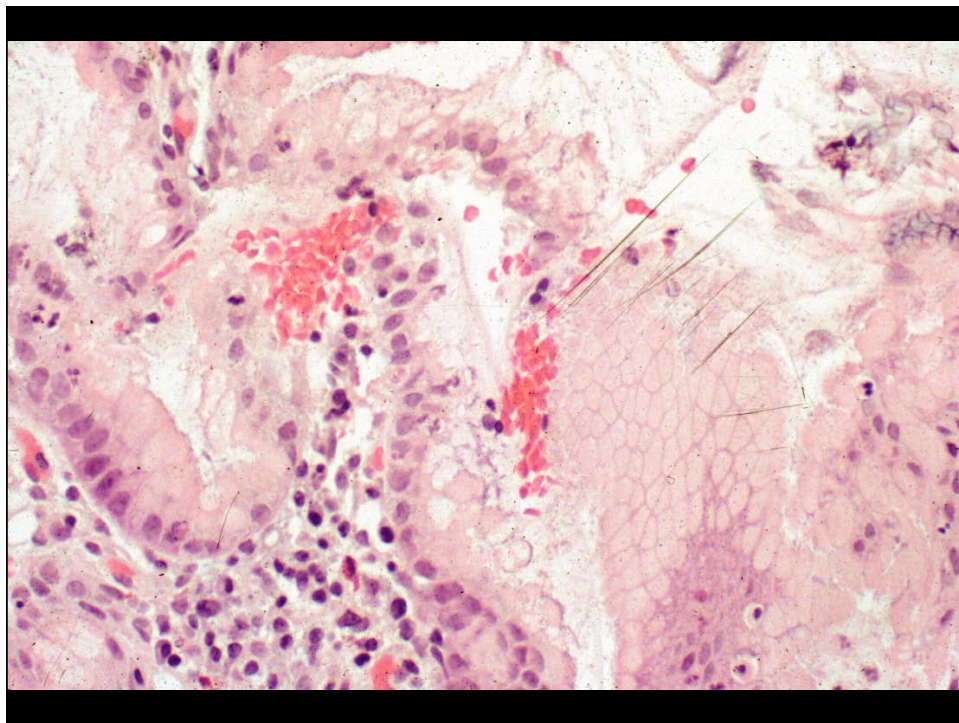
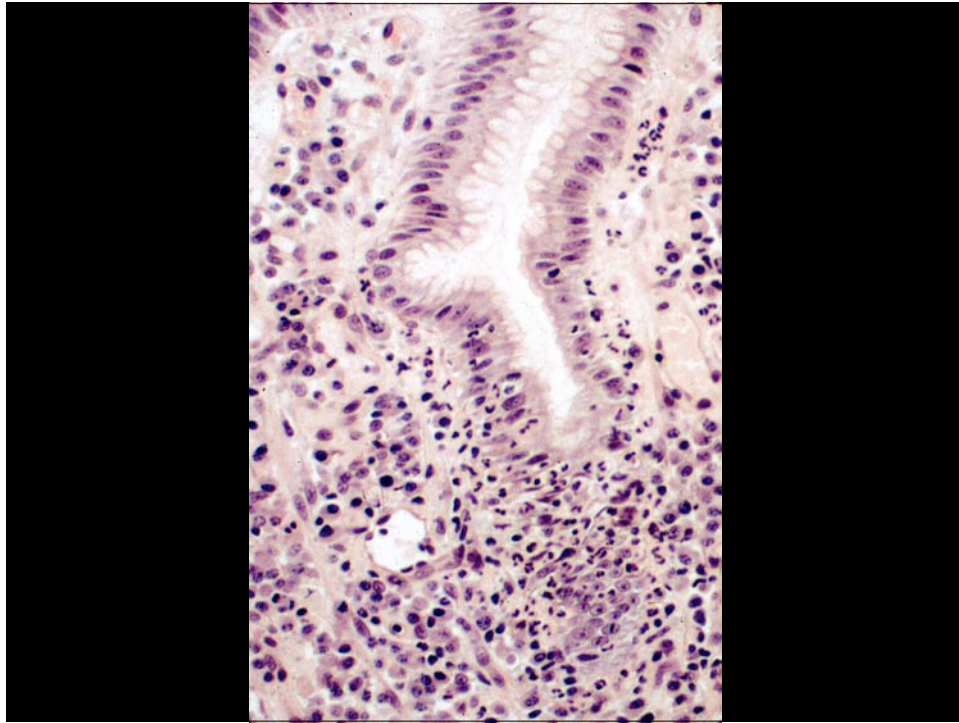
## **H. PYLORI UNUSUAL FEATURES**

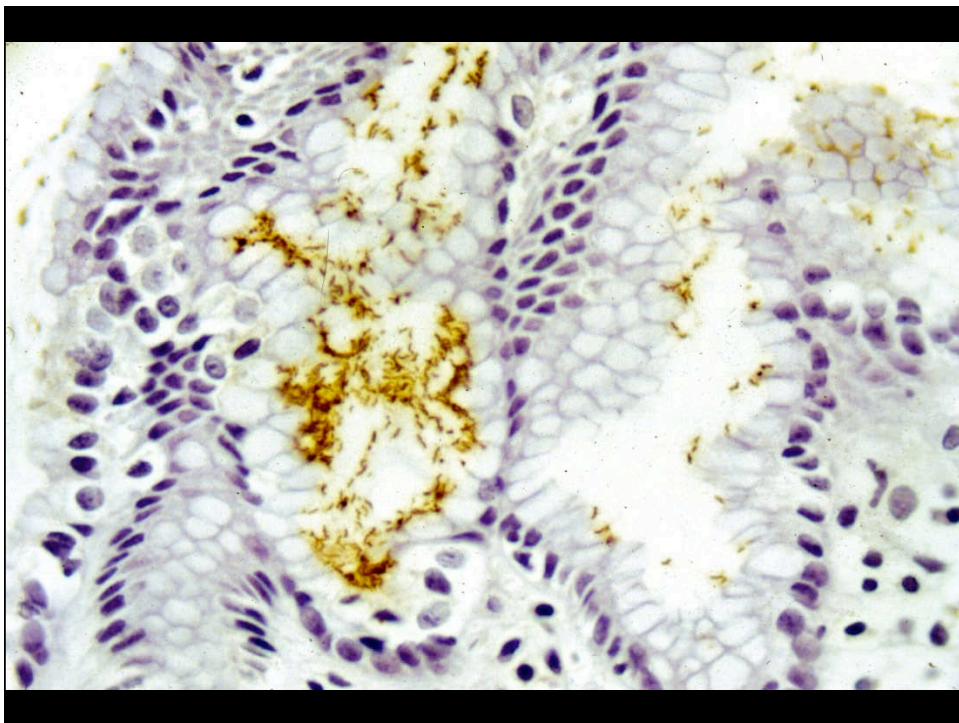
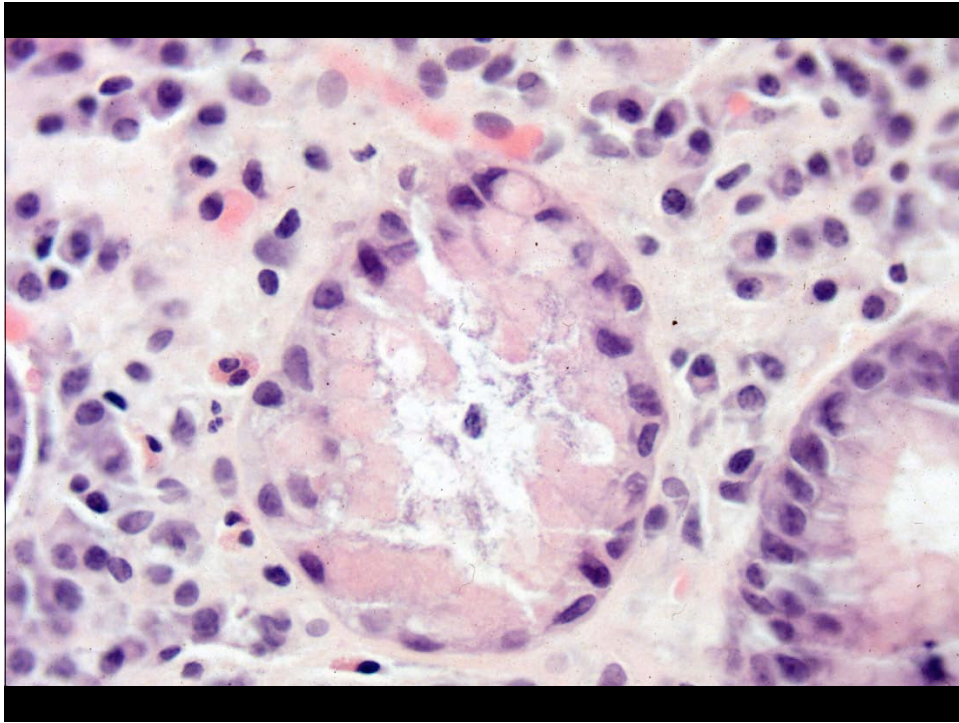
<b>First cultured in</b>	<b>1982 (April 14)</b>
<b>"Unidentified curved bacilli"</b>	<b>1983</b>
<b>Campylobacter pyloridis</b>	<b>1984</b>
<b>Campylobacter pylori</b>	<b>1987</b>
<b>HELICOBACTER pylori</b>	<b>1989</b>

**Natural habitat: human stomach**

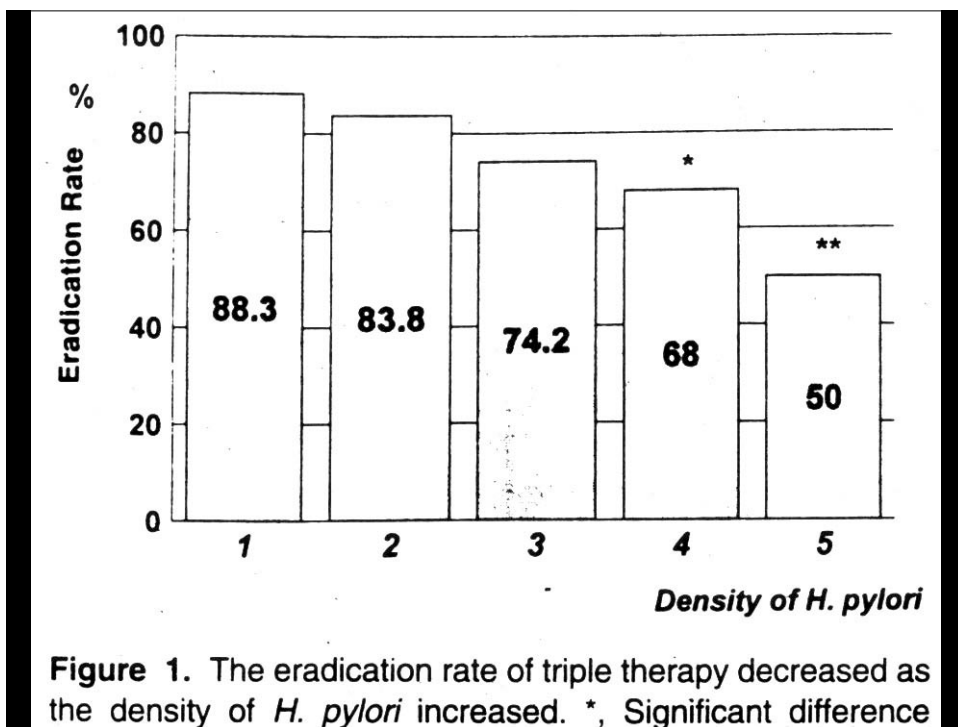
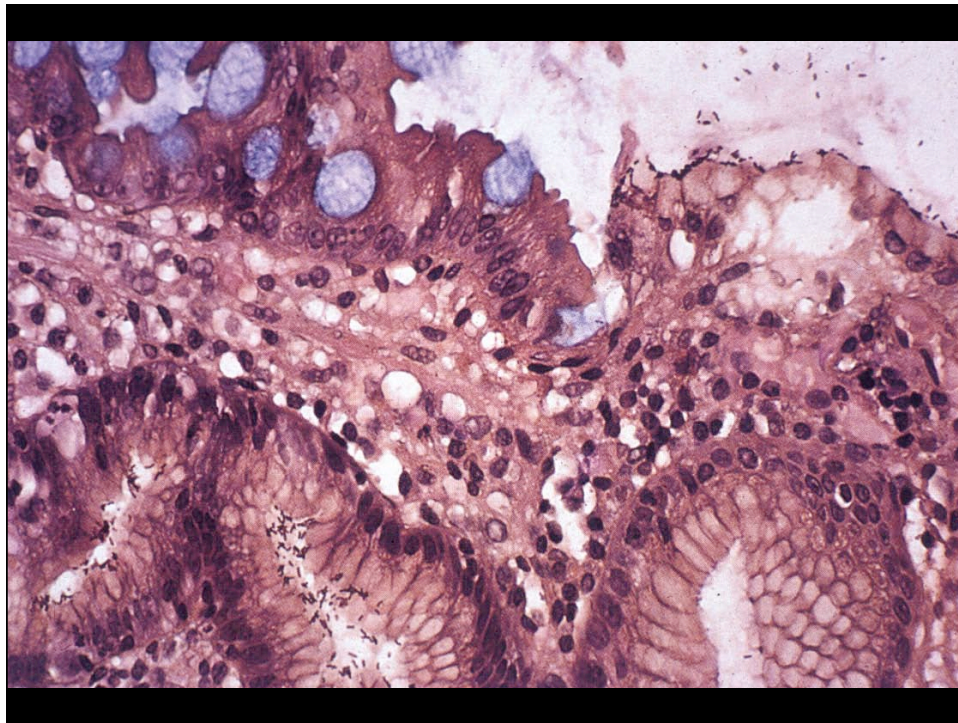




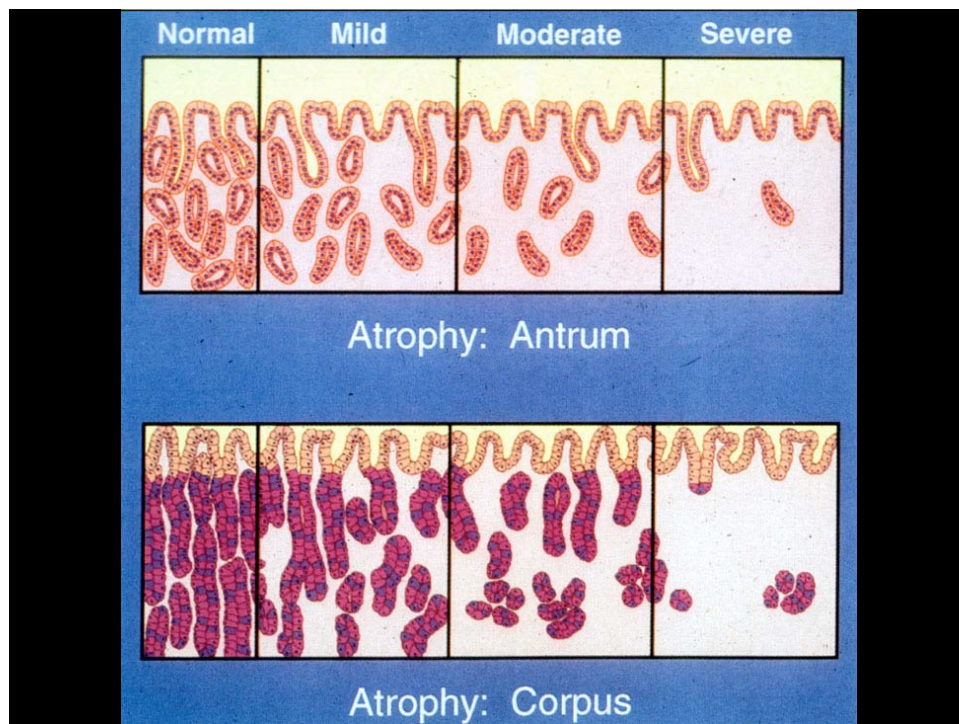






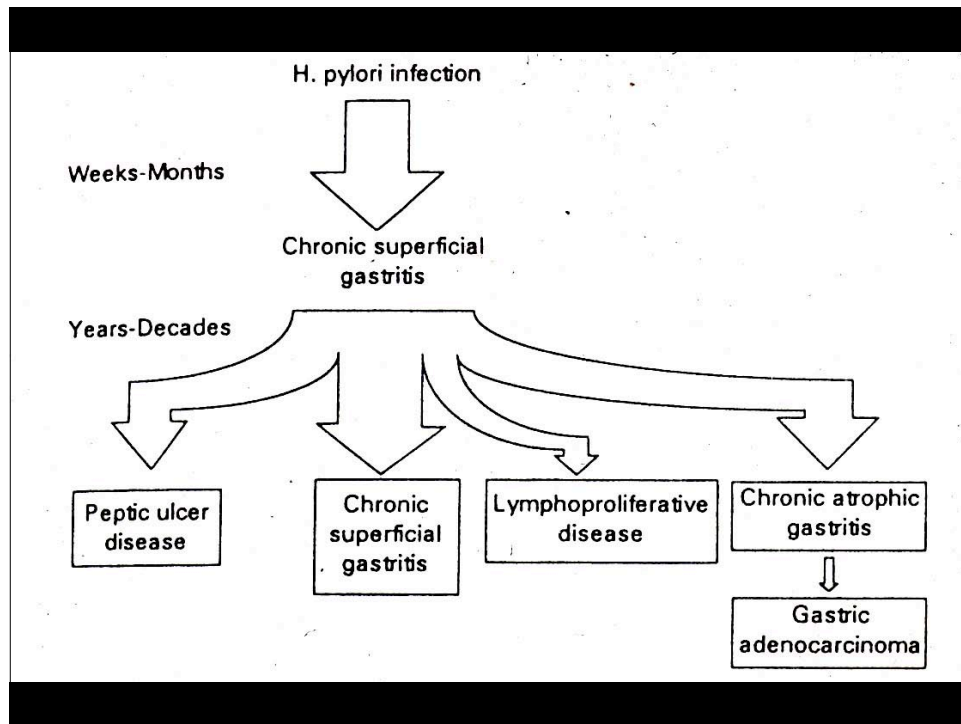






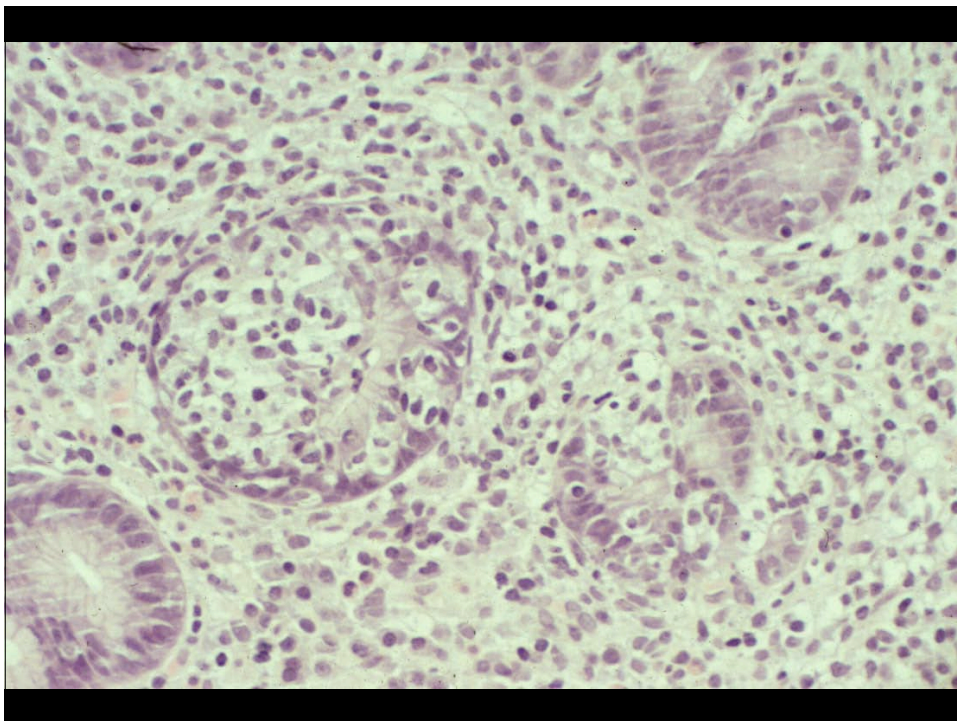
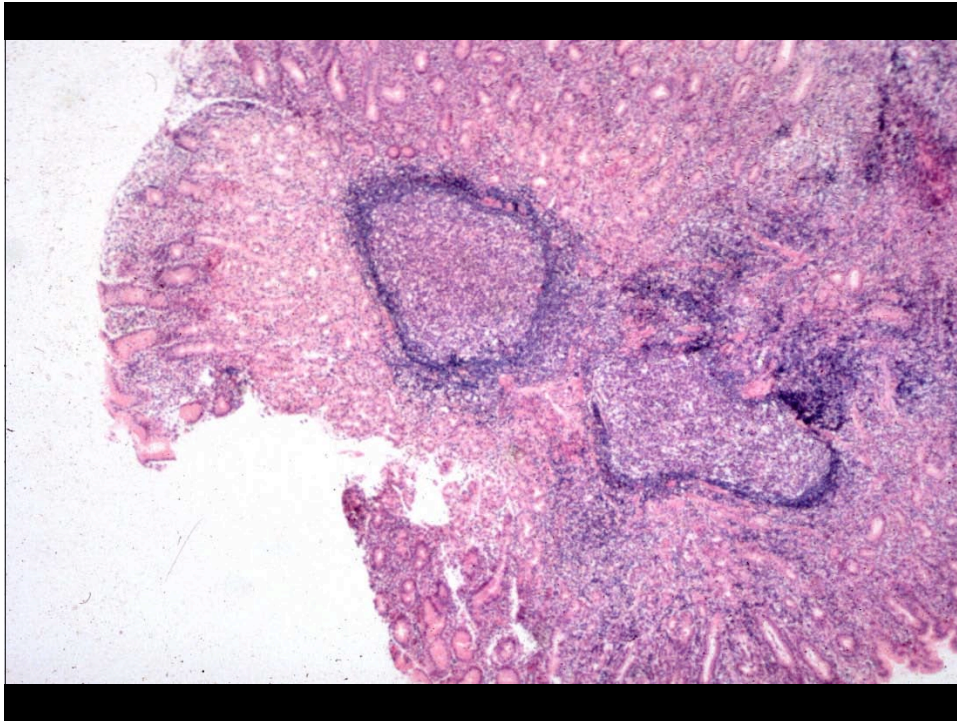
### Some Characteristics and Outcomes of the Chronic Gastritides

Parameter	Topography of the Atrophy			
	Severe antral atrophy	None	Severe panatropy	Severe corpus atrophy
HP related	Yes <sup>a</sup>	Yes	Yes <sup>a</sup>	No
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%





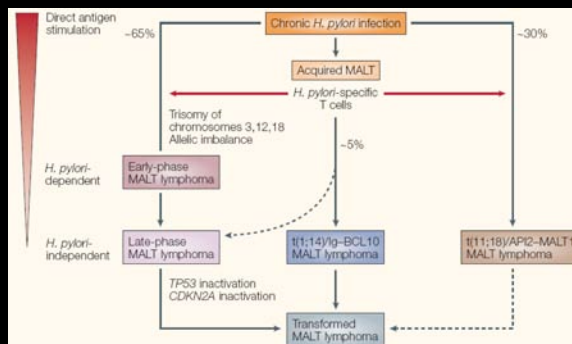
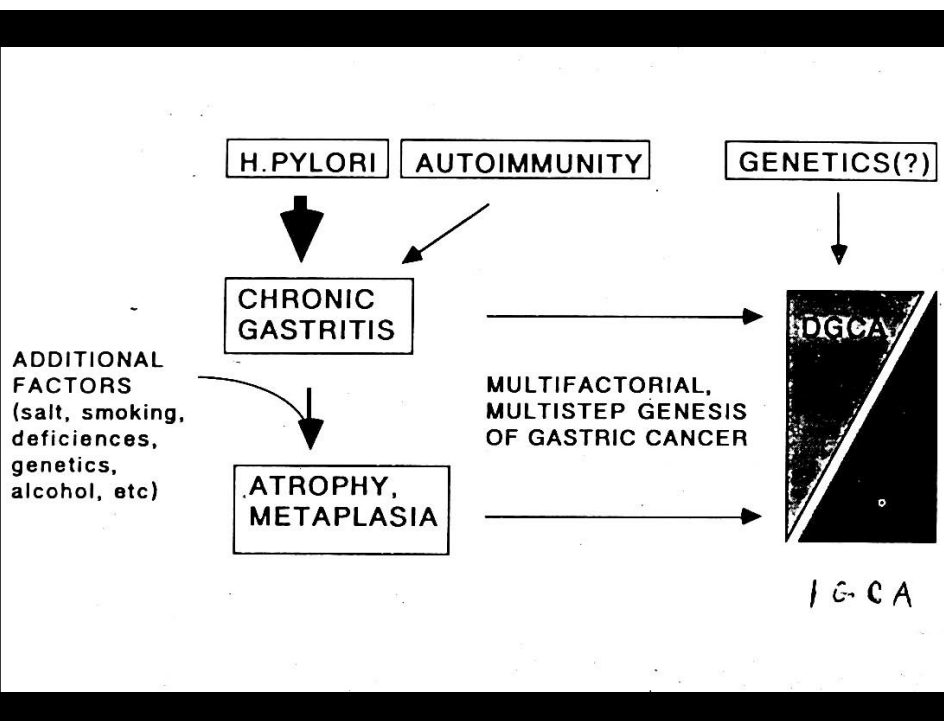
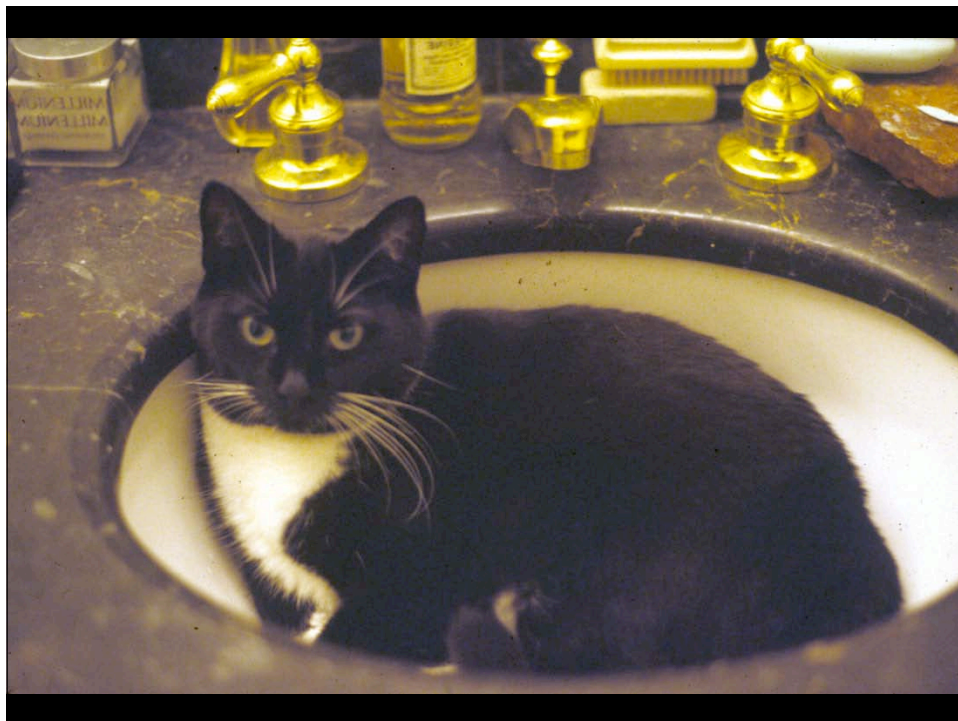
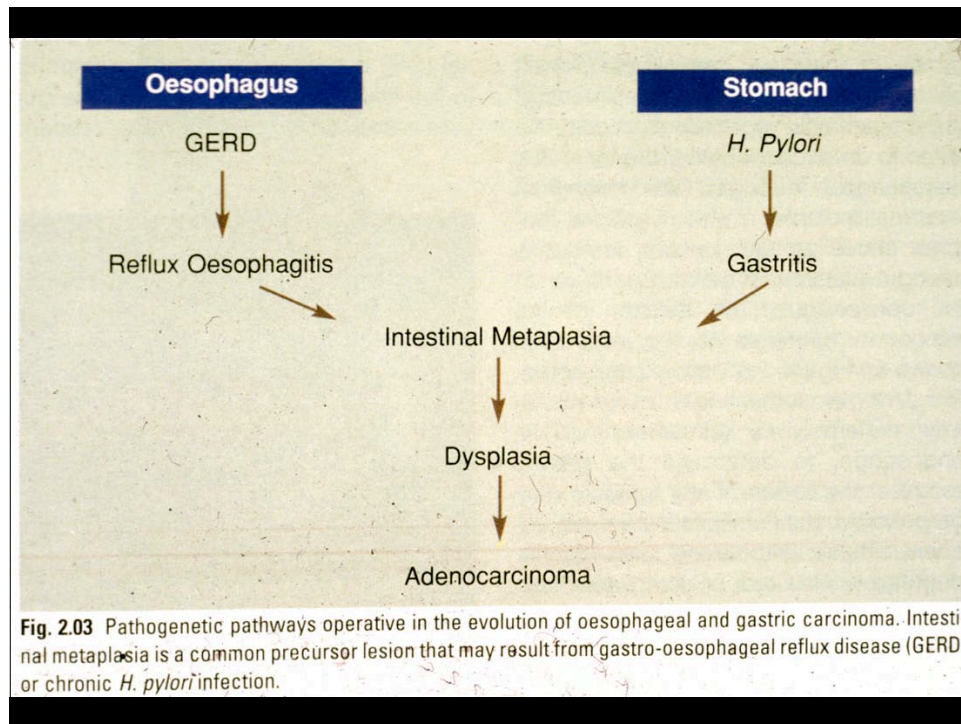
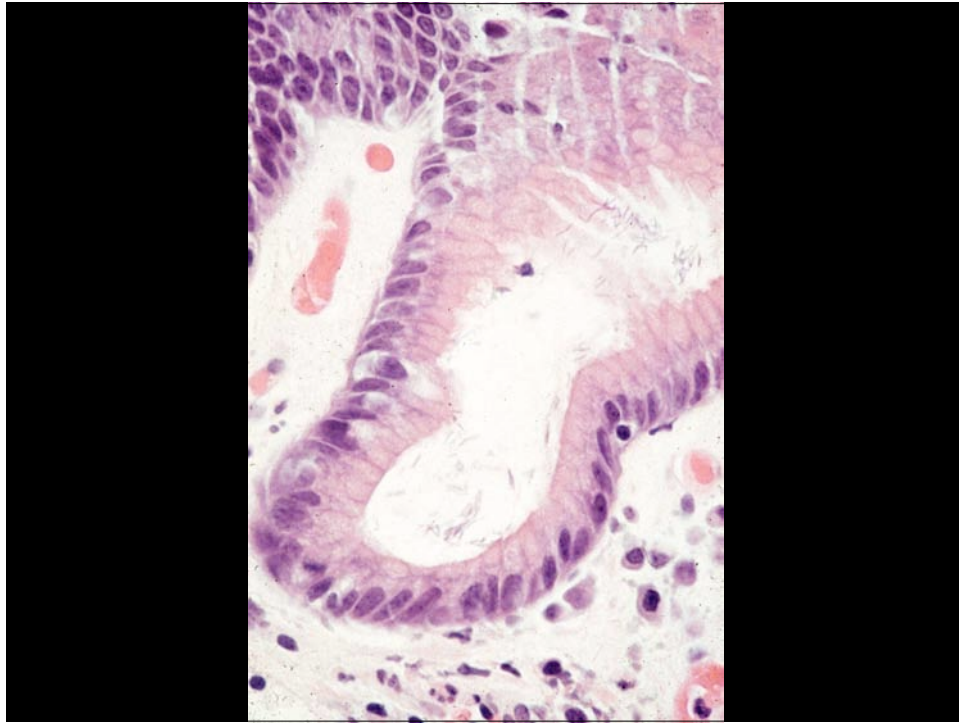


Figure 1 | Multistage development of gastric MALT lymphoma. *H. pylori* infection stimulates the production of lymphoid infiltrates, which leads to the formation of acquired mucosa-associated lymphoid tissue (MALT) in the gastric mucosa. As a result of both direct and indirect immunological stimulation (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)(p11;p11)/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)(q21;q21)/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*.







## *Helicobacter Heilmannii* Gastritis

### Complications

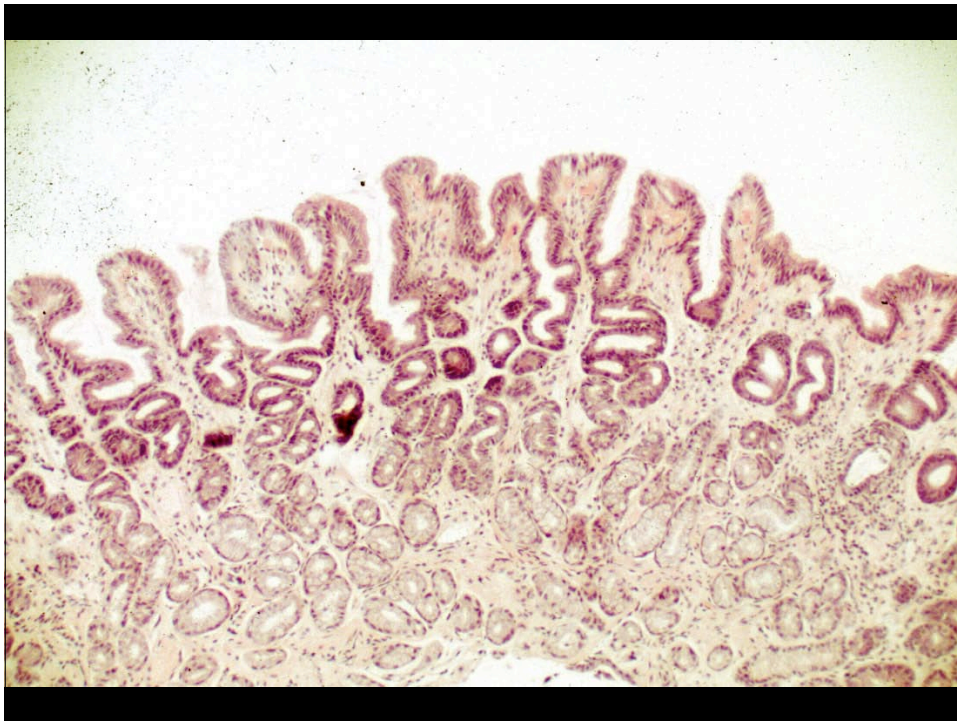
- MALT lymphoma: 7/202
- Gastric cancer: 1/51 and 1/202
- Ulcers: 2/302 non *Hp* ulcers
- Coinfection with *H. pylori*: 1.6%



**“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

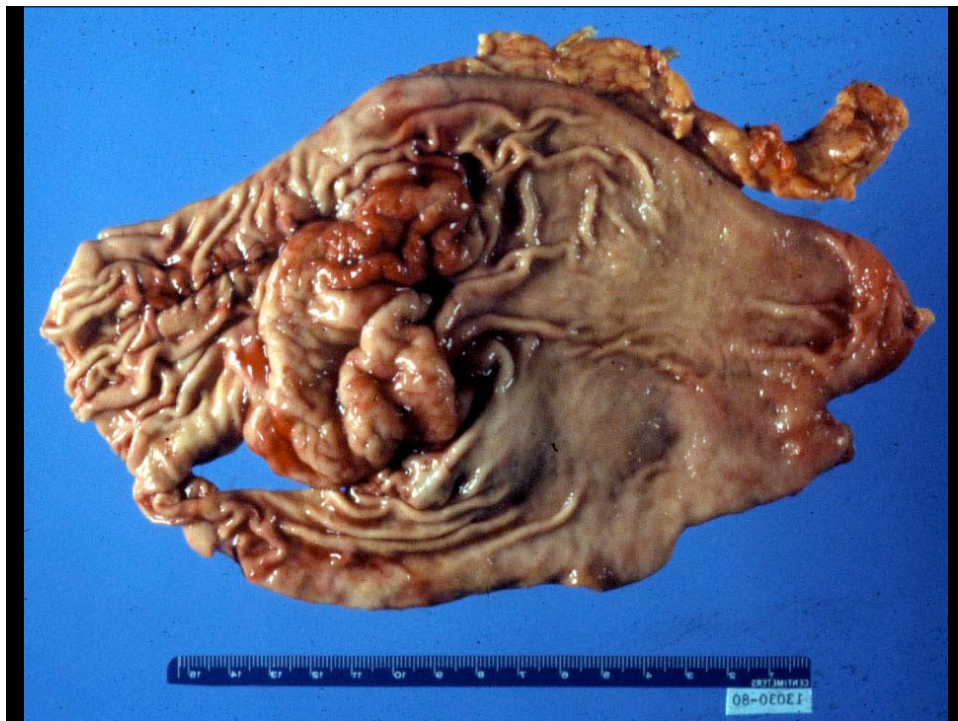
Vascular disturbances

shock, ischemia, stress,

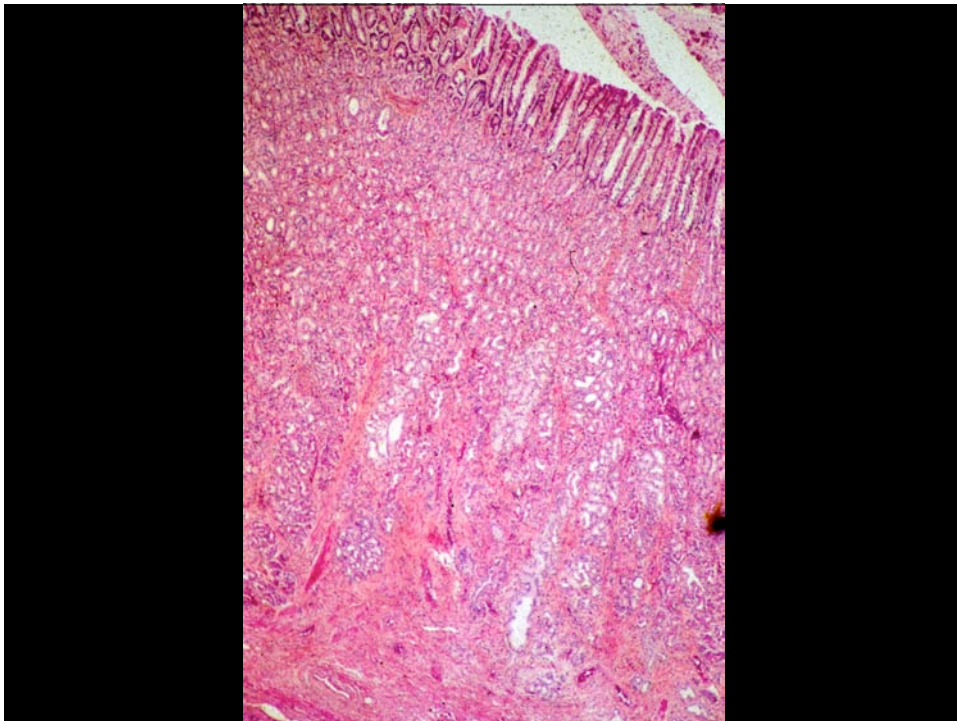
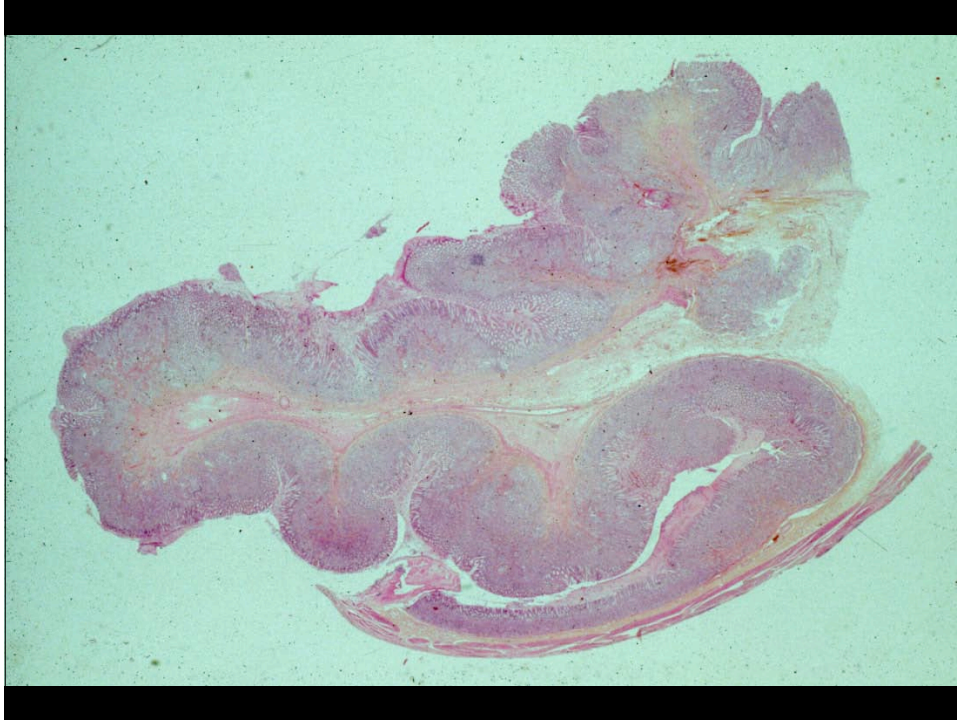
Local trauma (nasogastric tubes)

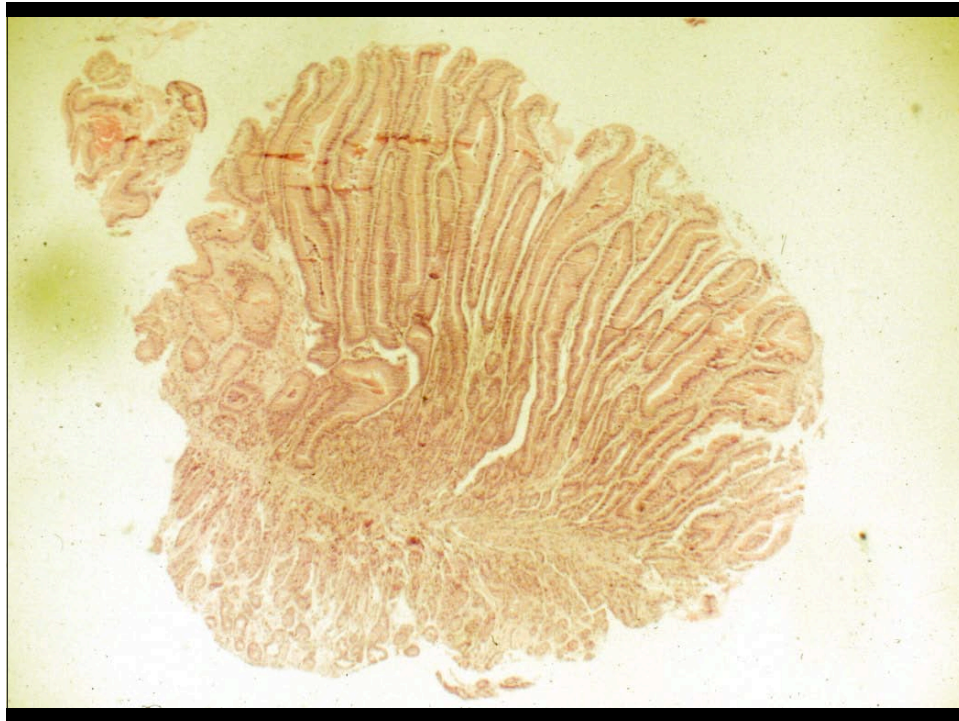
Radiation and chemotherapy

Idiopathic





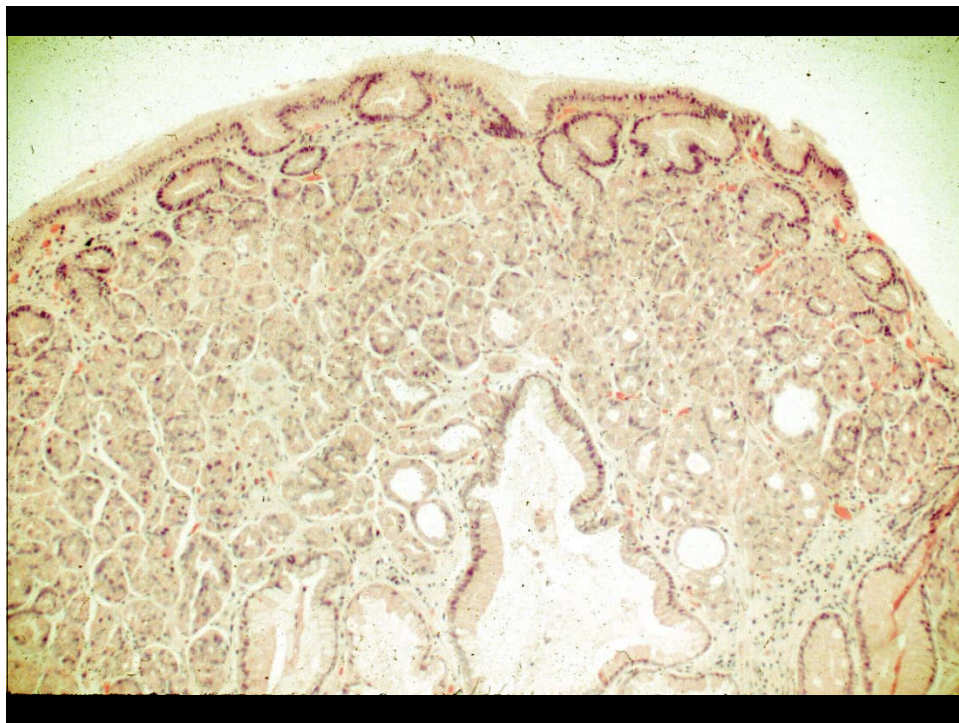




## Gastric Polyps

- 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

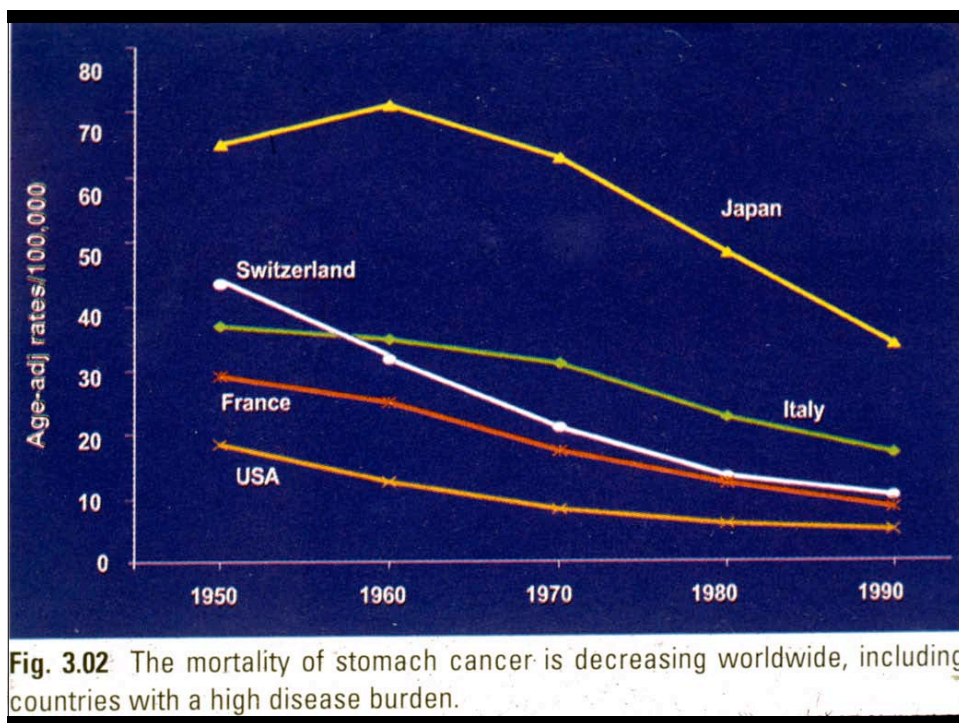
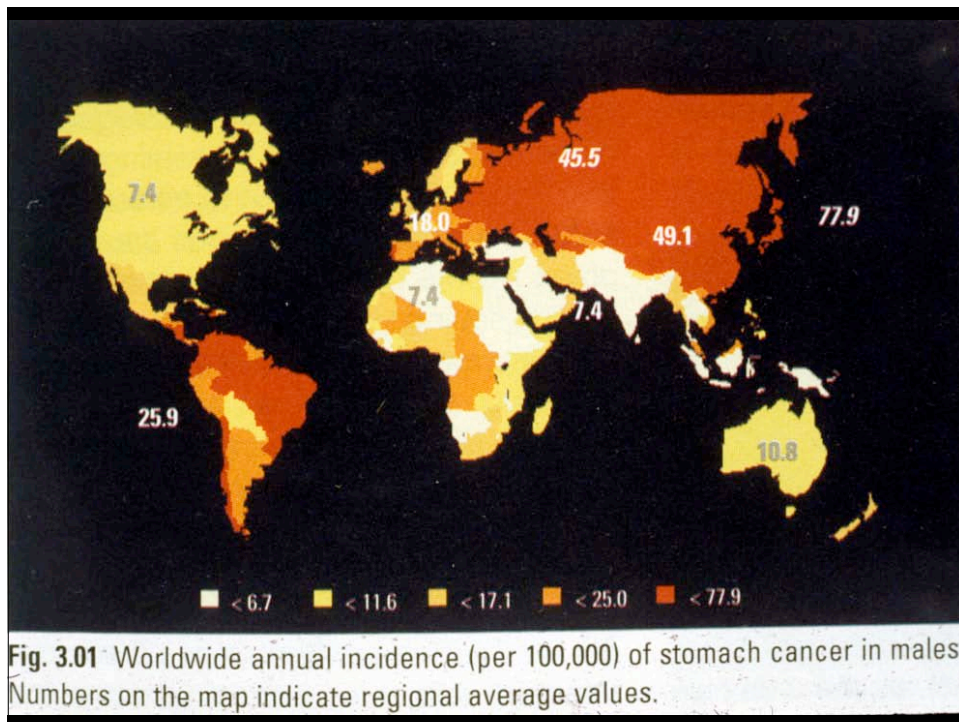
- Small cell carcinoma

- Undifferentiated carcinoma

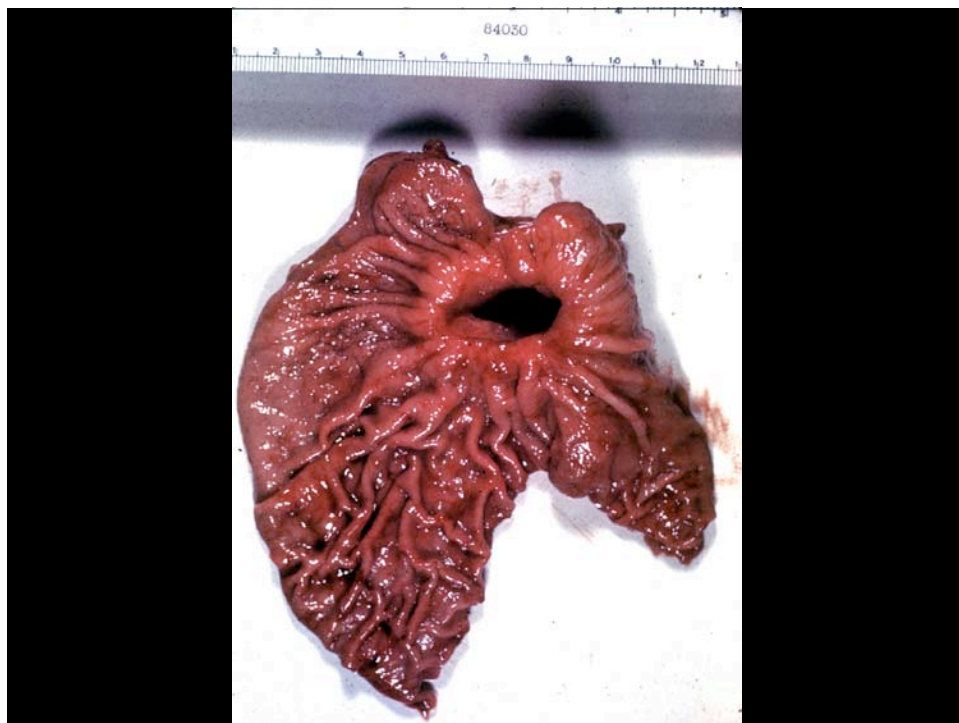
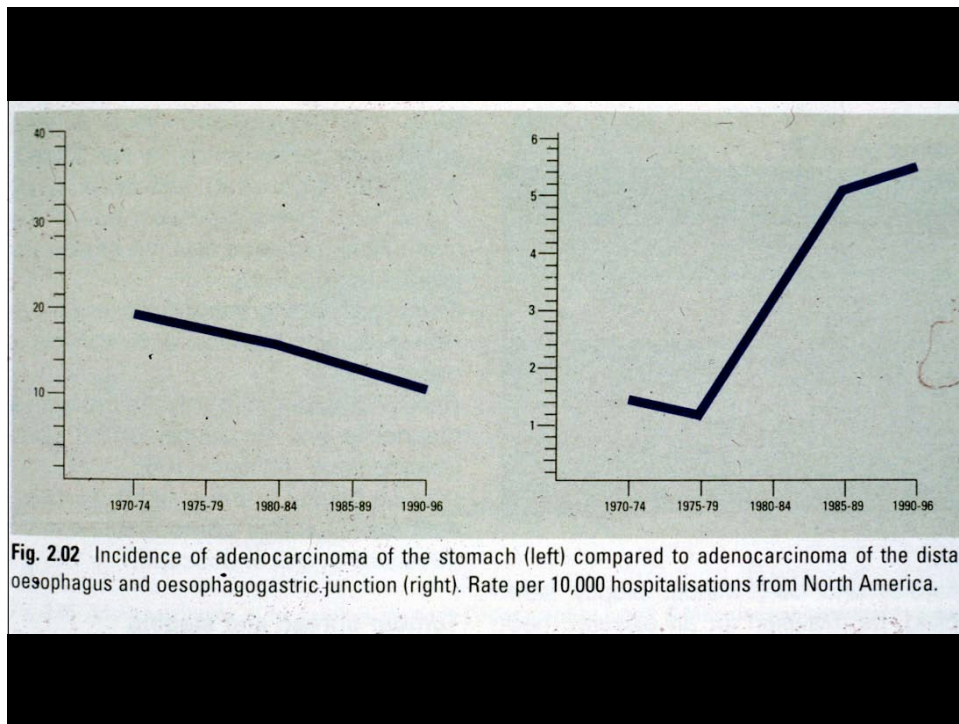
- Others

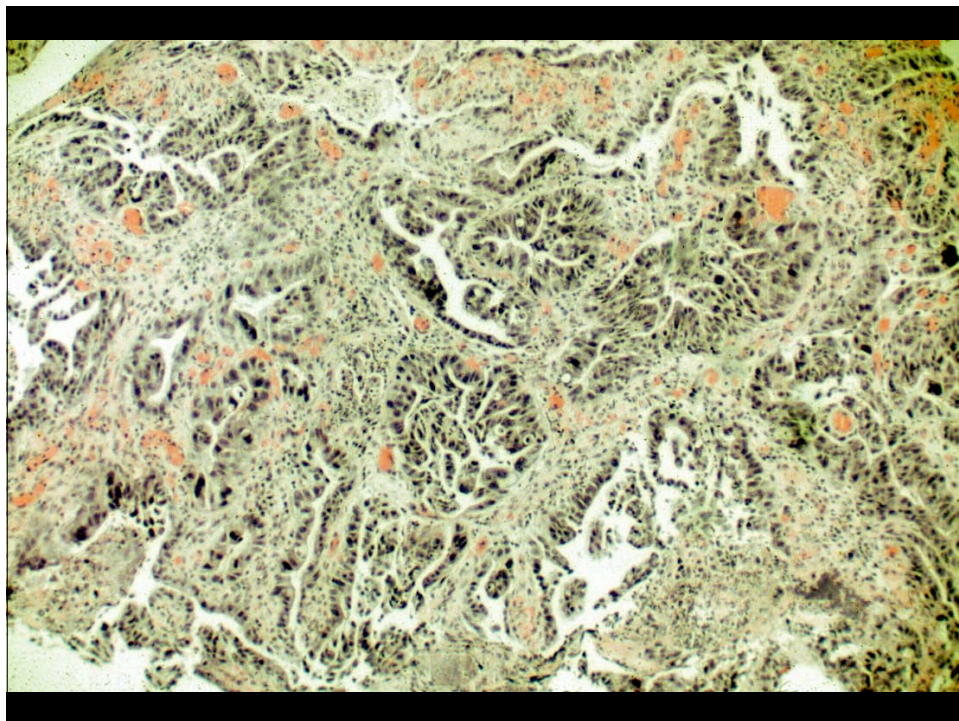
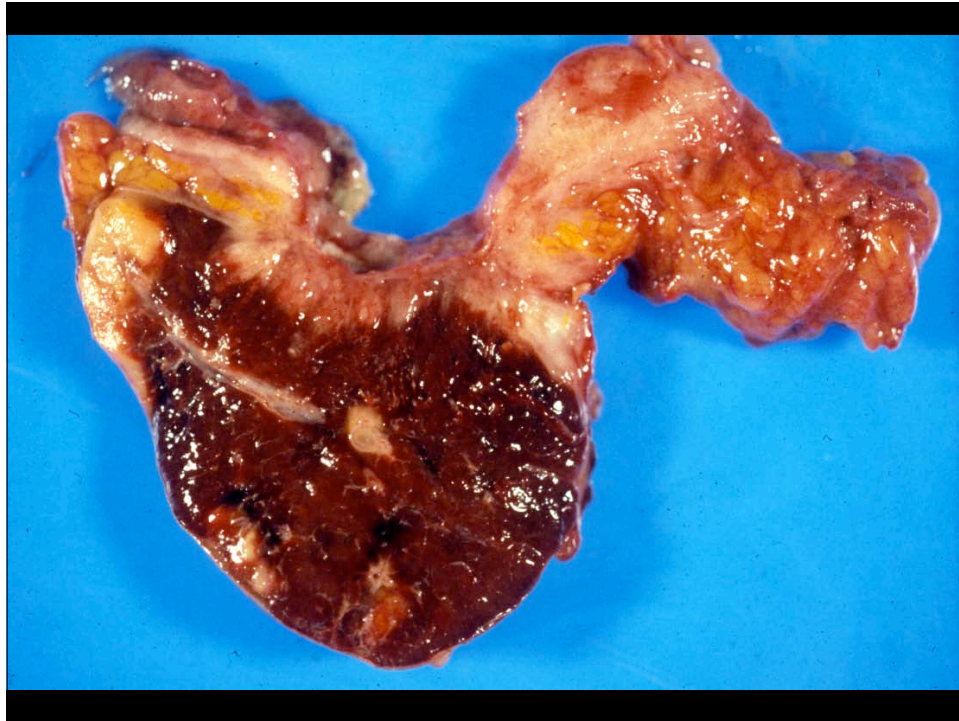
Carcinoid (well differentiated endocrine neoplasm)



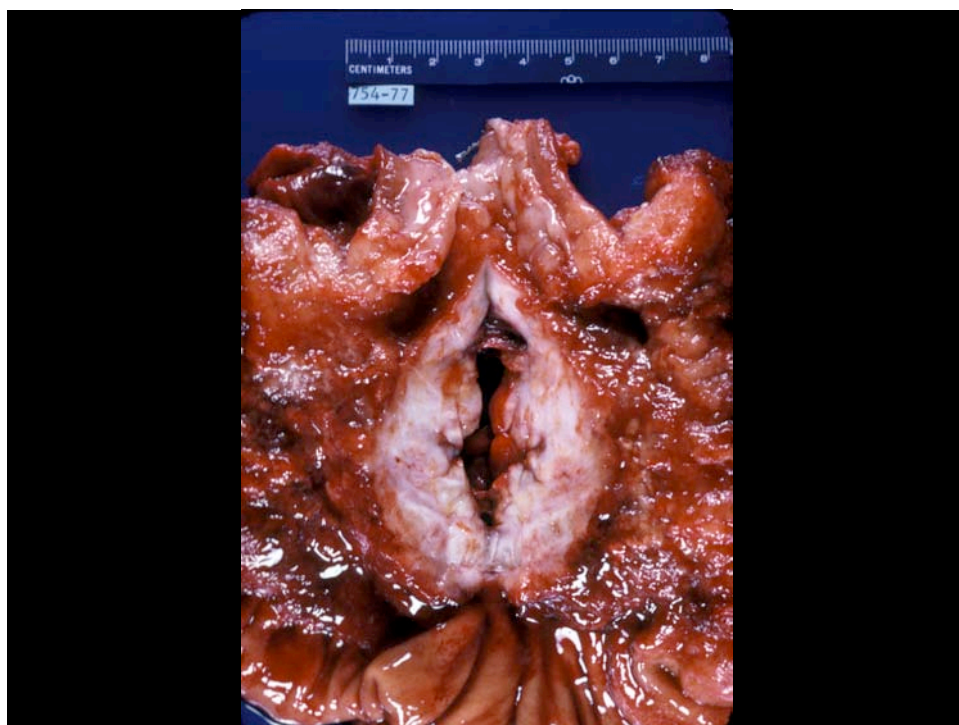
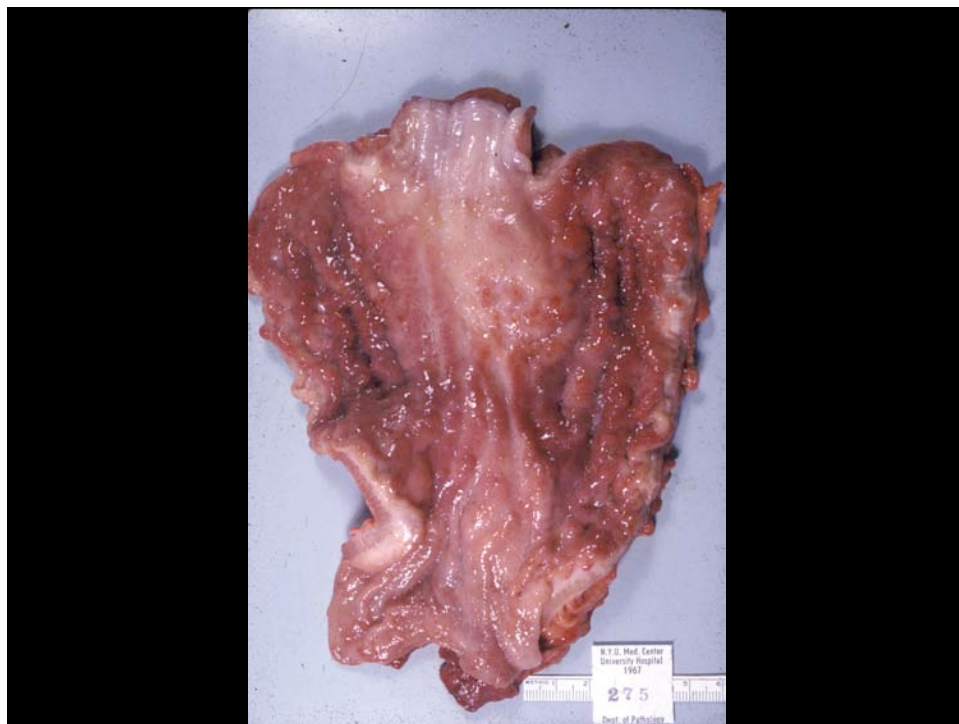




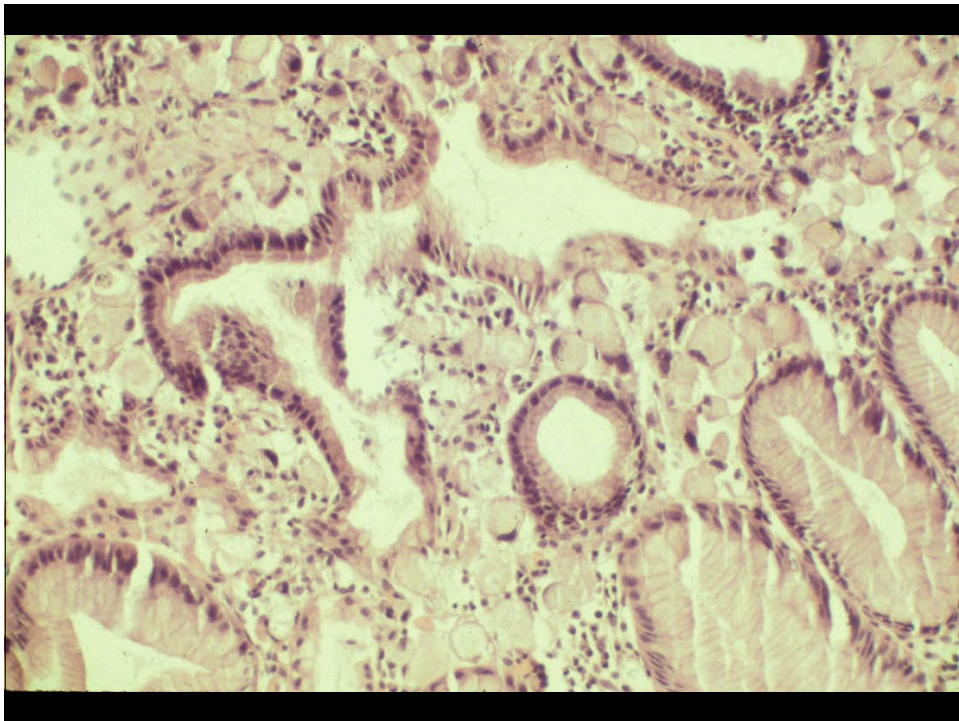
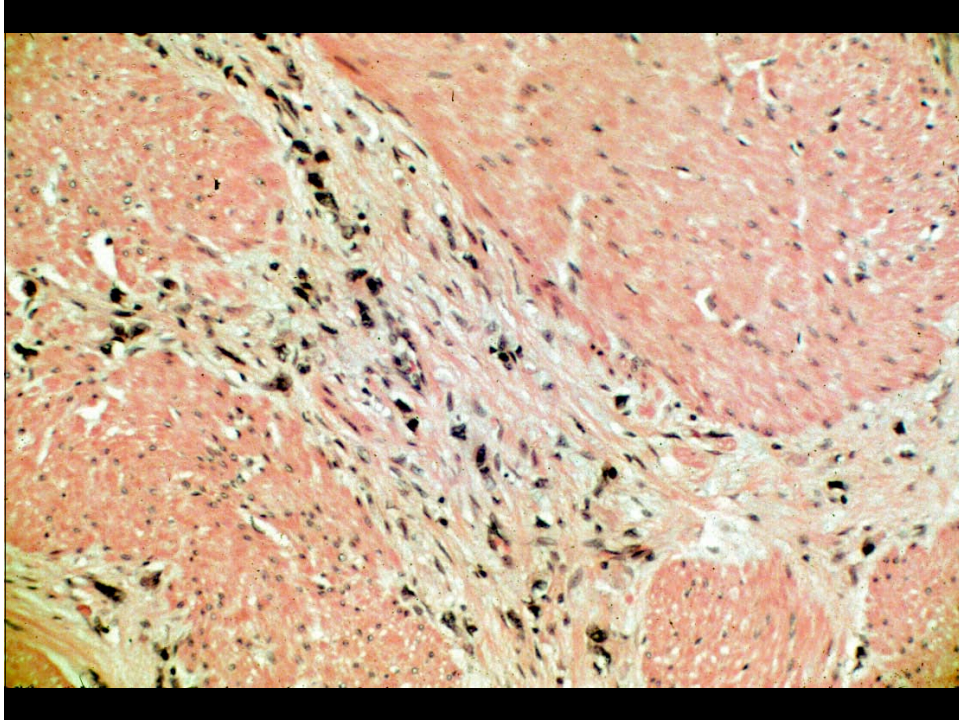












**Table 17-4. MAJOR FEATURES OF LAURENS' CLASSIFICATION OF GASTRIC CARCINOMA**

FEATURE	TYPE OF CARCINOMA	
	<i>Intestinal</i>	<i>Diffuse</i>
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.

**Type I**  
Protruded



**Type IIa**  
Elevated



**Type IIb**  
Flat



**Type IIc**  
Depressed



**Type III**  
Excavated



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



**Table 3.02.**

Histological classification of endocrine neoplasms of the stomach<sup>1</sup>

**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 –**

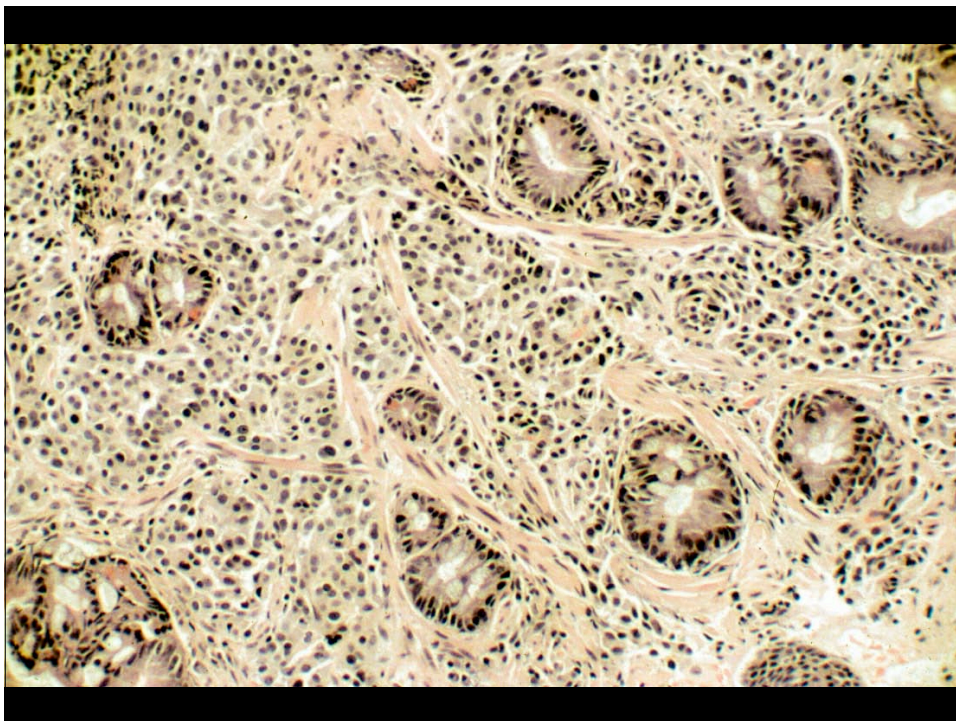
**poorly differentiated endocrine neoplasm**

**3. Tumour-like lesions**

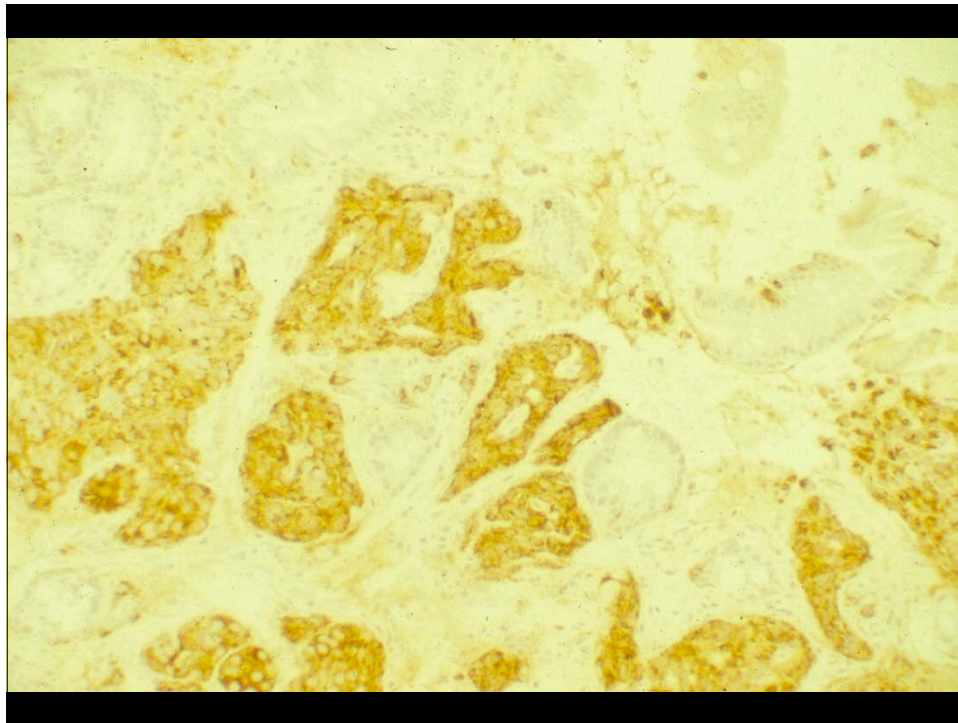
Hyperplasia

Dysplasia

<sup>1</sup>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.







<b>Non-epithelial tumours</b>	
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	
<b>Malignant lymphomas</b>	
Marginal zone B-cell lymphoma of MALT-type	9699/3
Mantle cell lymphoma	9673/3
Diffuse large B-cell lymphoma	9680/3
Others	
<b>Secondary tumours</b>	

