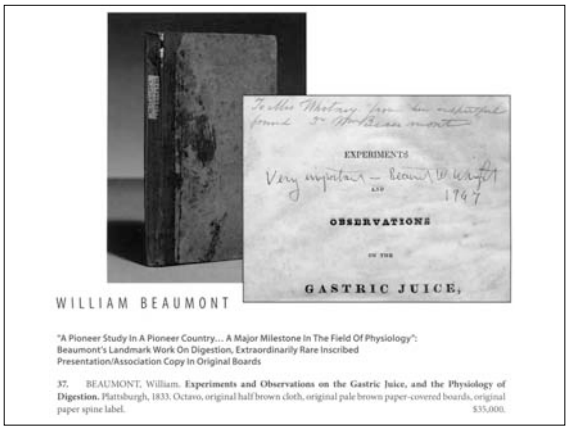


"A Pioneer Study In A Pioneer Country... A Major Milestone In The Field Of Physiology": Beaumont's Landmark Work On Digestion, Extraordinarily Rare Inscribed Presentation/Association Copy In Original Boards

37. BEAUMONT, William. **Experiments and Observations on the Gastric Juice, and the Physiology of Digestion.** Plattsburgh, 1833. Octavo, original half brown cloth, original pale brown paper-covered boards, original paper spine label. $35,000.

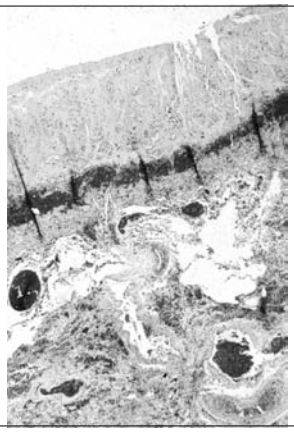
Gastritis: Causes

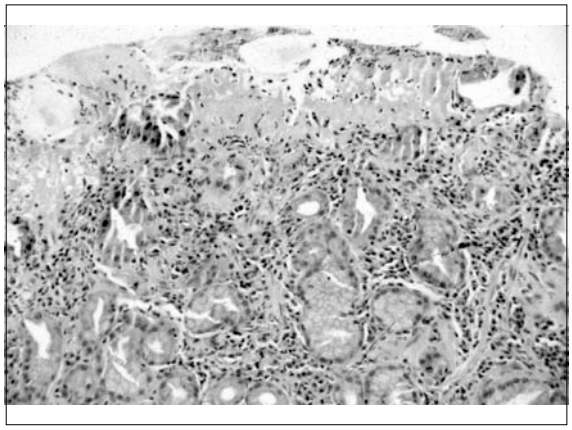
- ***Helicobacter pylori* infection**
- **NSAID use**
- Excessive alcohol consumption
- Heavy smoking
- Radiotherapy
- Cancer chemotherapy
- Systemic infections (*Salmonella*, CMV)
- Severe stress
- Ischemia and shock
- Suicide attempts with acids or alkali
- Mechanical trauma
- Distal gastrectomy

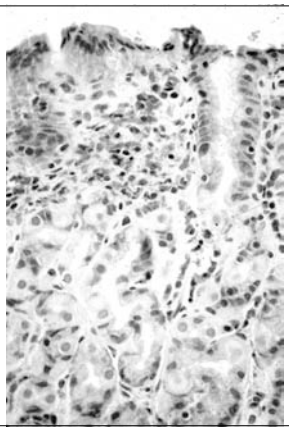
GASTRITIS

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)

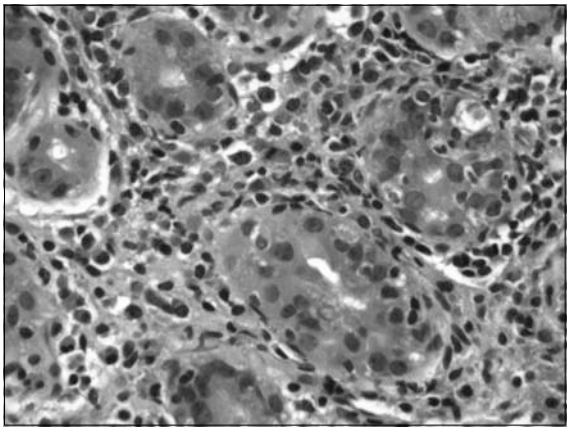


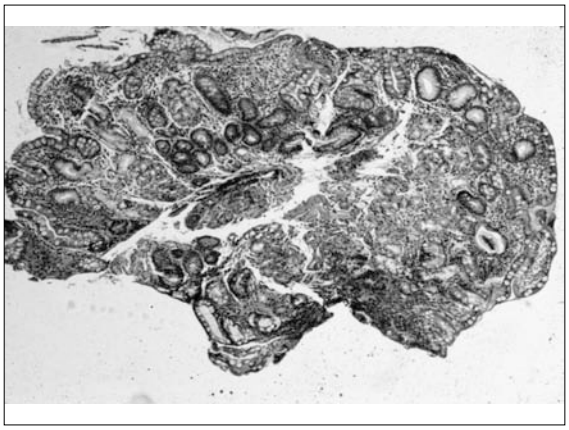


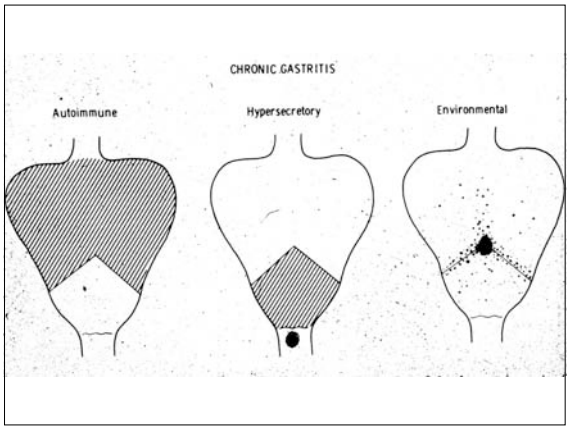


Types of Chronic Gastritis

- **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.

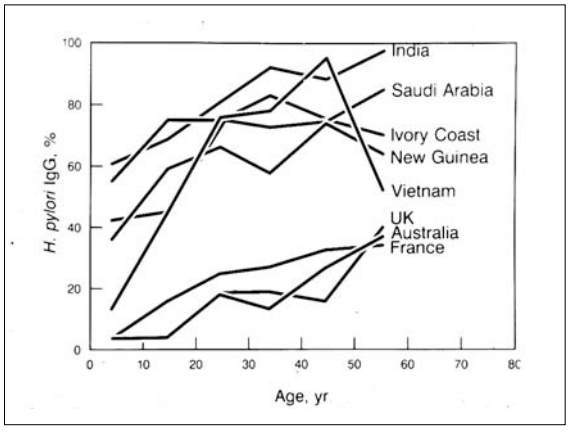






Prevalence of Biopsy-proven *H. pylori* Gastritis

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

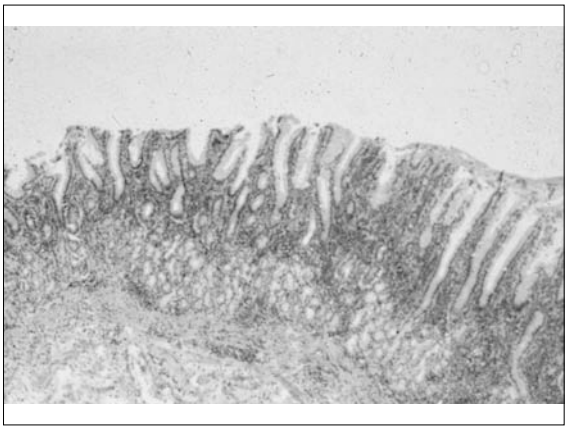
Topographic Types

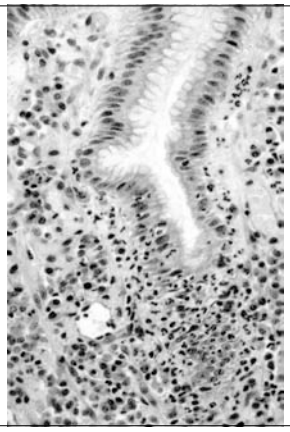
Chronic gastritis of antrum and corpus
Chronic gastritis, antrum-predominant
Chronic gastritis, corpus-predominant

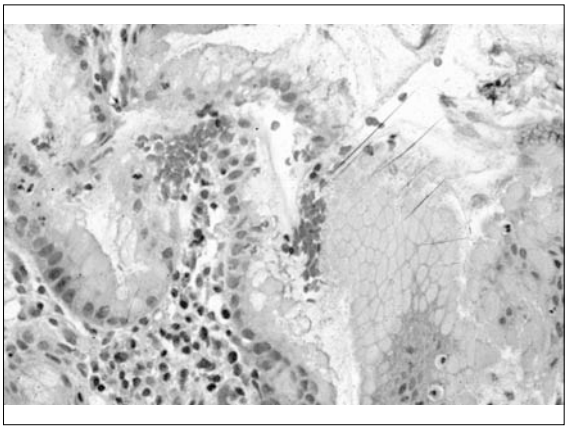
H. PYLORI UNUSUAL FEATURES

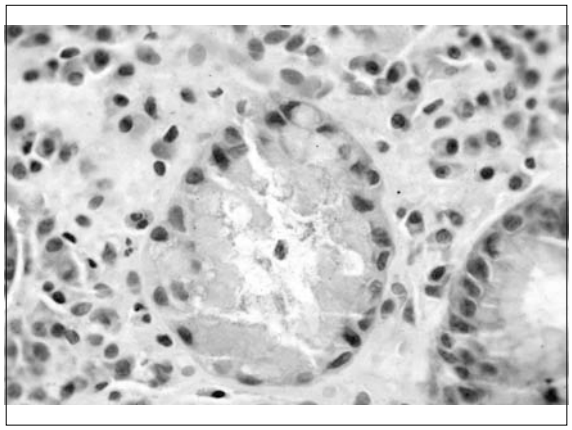
First cultured in	1982 (April 14)
"Unidentified curved bacilli"	1983
Campylobacter pyloridis	1984
Campylobacter pylori	1987
HELICOBACTER pylori	1989

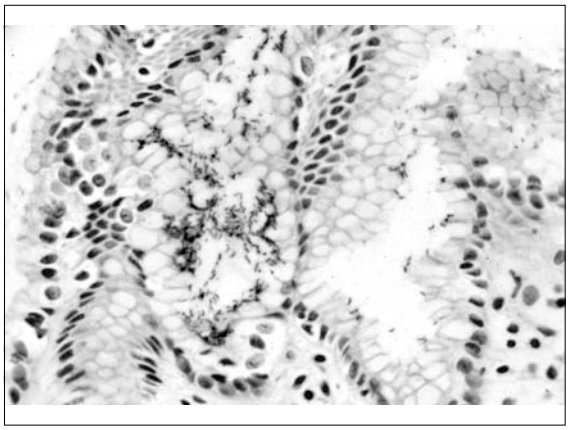
Natural habitat: human stomach

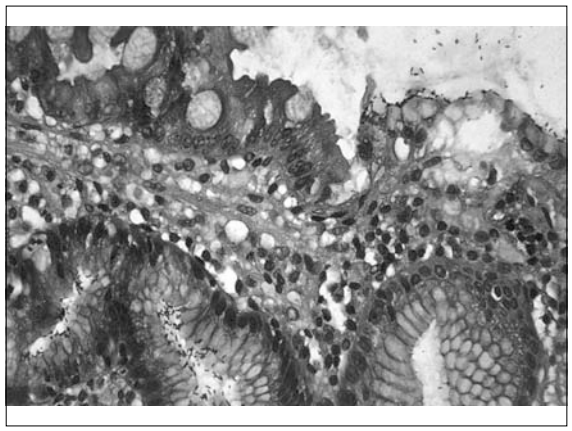












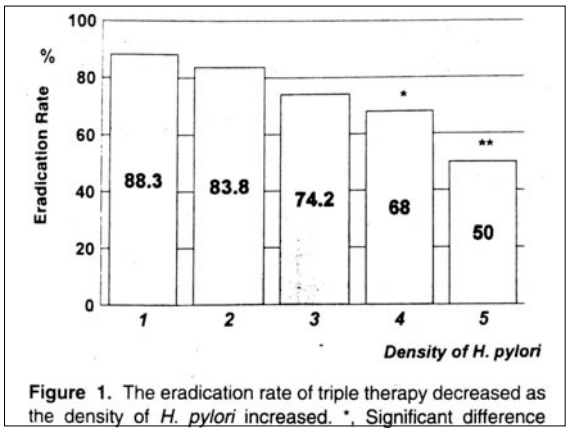
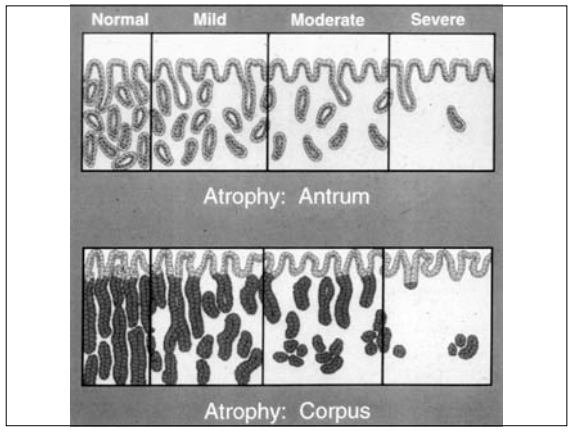
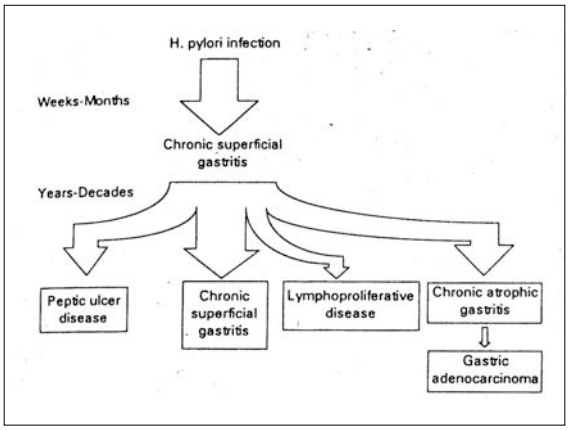


Figure 1. The eradication rate of triple therapy decreased as the density of *H. pylori* increased. *, Significant difference



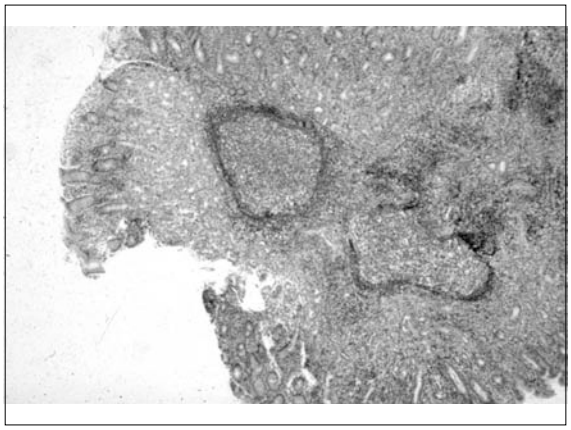
Some Characteristics and Outcomes of the Chronic Gastritides

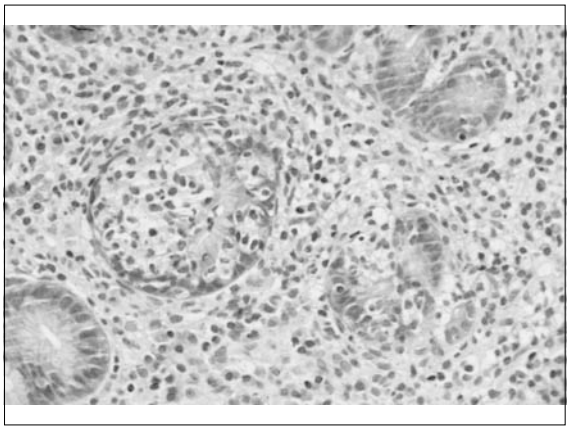
	Topography of the Atrophy			
Parameter	**Severe antral atrophy**	**None**	**Severe panatrophy**	**Severe corpus atrophy**
HP related	Yes[a]	Yes	Yes[a]	No Autoimmune pathogenesis
Gastrin output	Impaired	Mild increase	Low	High
Acid output	Normal	Normal	Low	Achlorhydria
Peptic ulcer	Increased	Increased	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%**





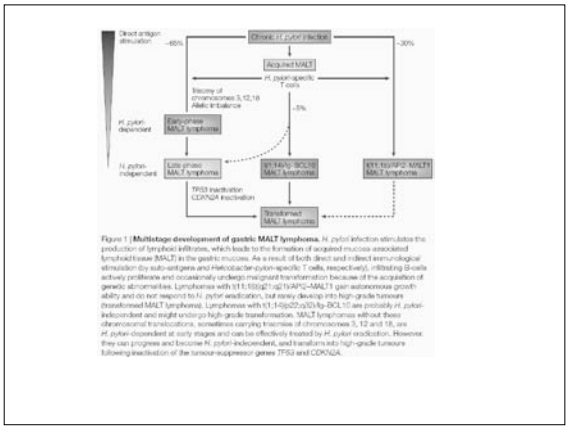
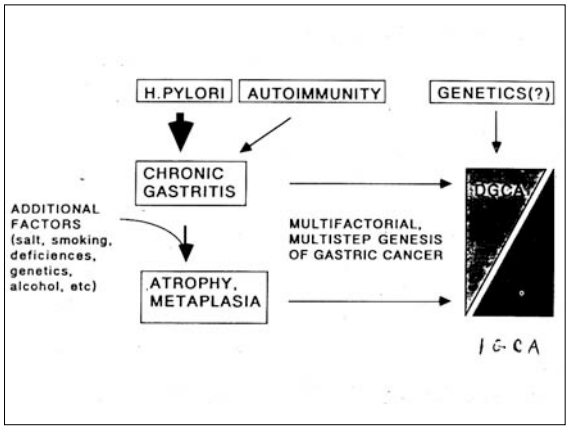


Figure 1 | **Multistage development of gastric MALT lymphoma.** *H. pylori* infection stimulates the production of lymphoid infiltrates, which leads to the formation of acquired mucosa-associated lymphoid tissue (MALT) in the gastric mucosa. As a result of both direct and indirect immunological stimulation 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)/*Bcl-10*–IGH or t(14;18)(q32;q21)/IGH–*MALT1* 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*.



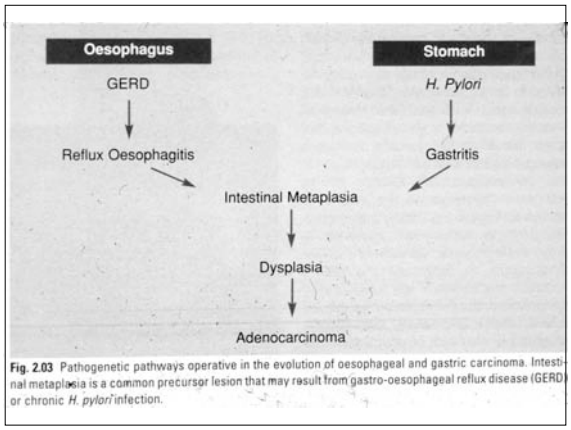
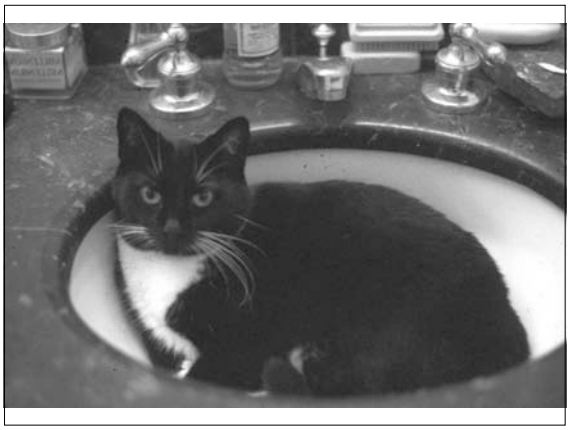
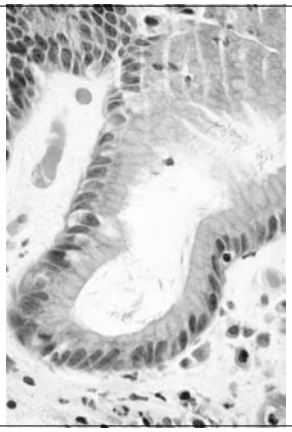


Fig. 2.03 Pathogenetic pathways operative in the evolution of oesophageal and gastric carcinoma. Intestinal metaplasia is a common precursor lesion that may result from gastro-oesophageal reflux disease (GERD) or chronic *H. pylori* infection.





Helicobacter Heilmannii Gastritis

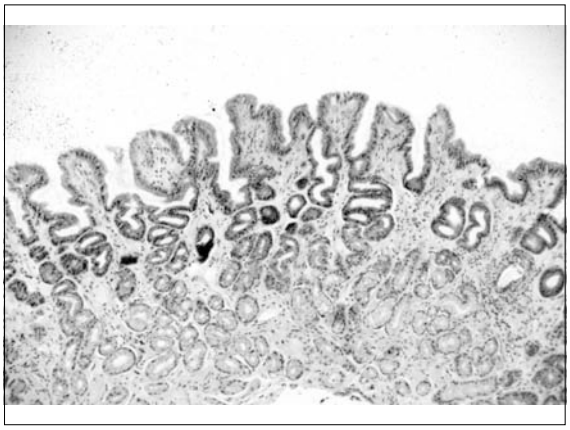
Complications

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

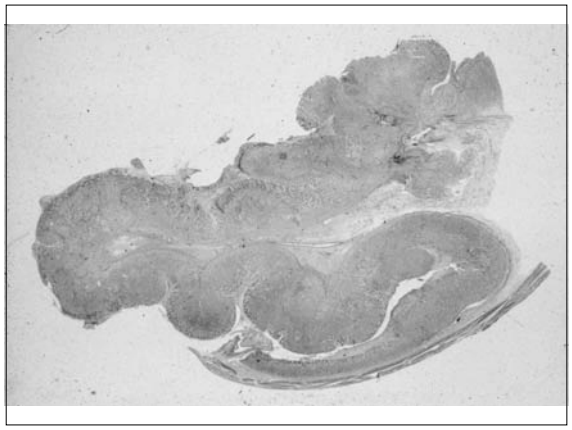
"Chemical" Gastritis
(type C gastritis, reactive gastropathy)

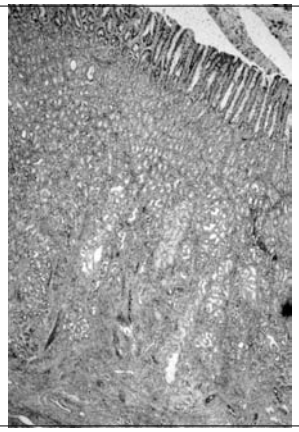
NSAIDS-related
Duodenal reflux-related

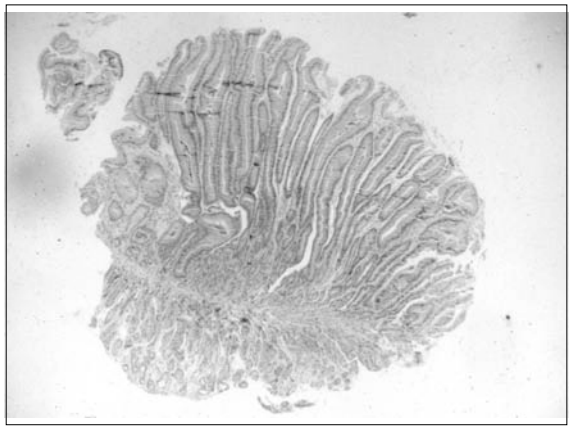
Foveolar hyperplasia
Mucosal edema and fibrosis
Mild chronic inflammation



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

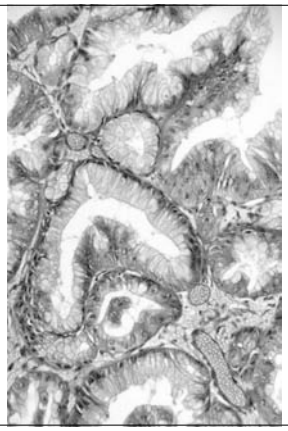


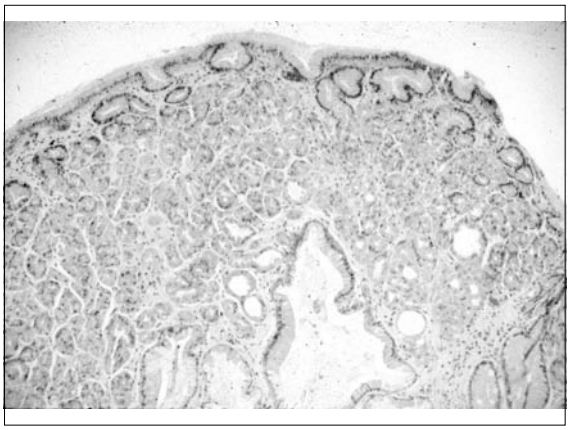


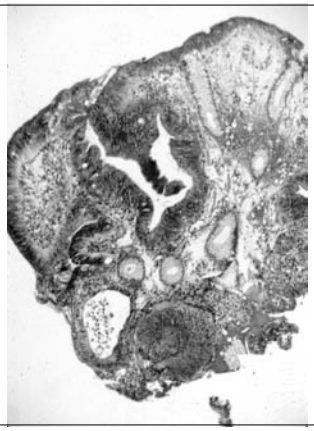


Gastric Polyps

- 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)

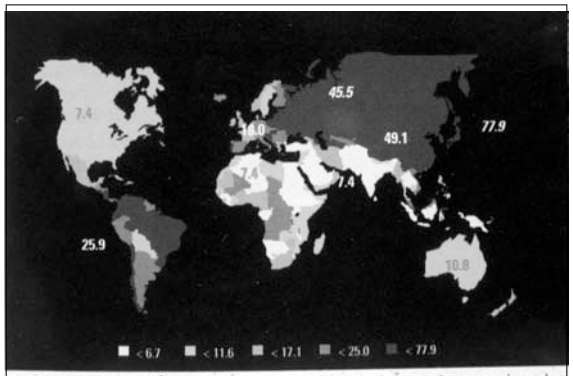


Fig. 3.01 Worldwide annual incidence (per 100,000) of stomach cancer in males. Numbers on the map indicate regional average values.

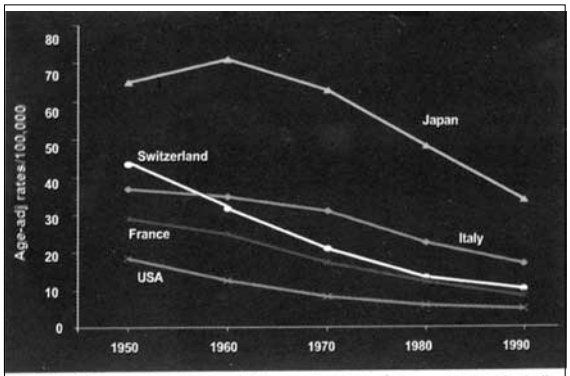


Fig. 3.02 The mortality of stomach cancer is decreasing worldwide, including countries with a high disease burden.

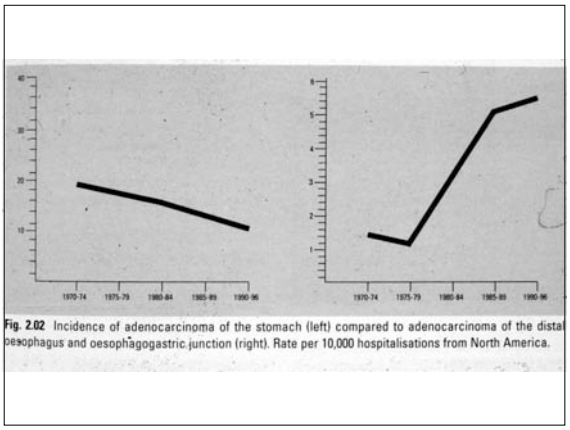
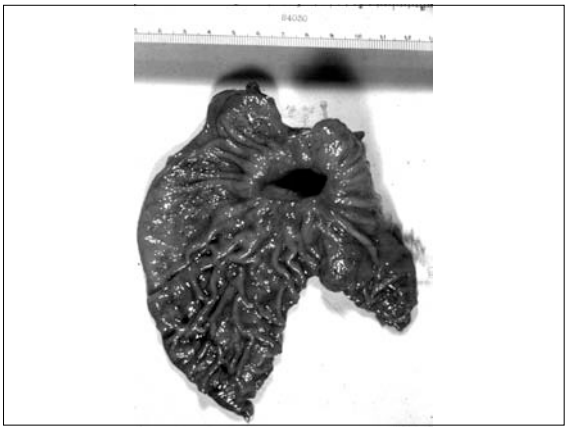
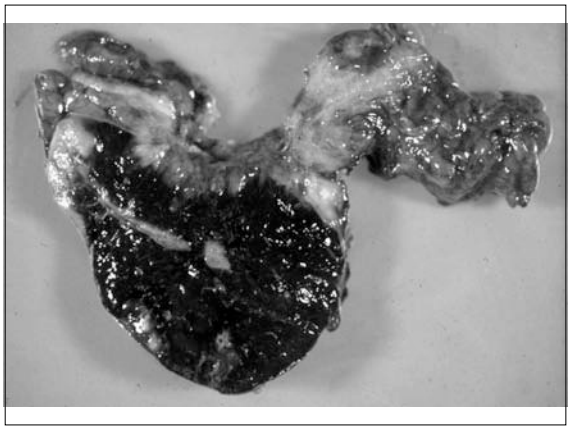
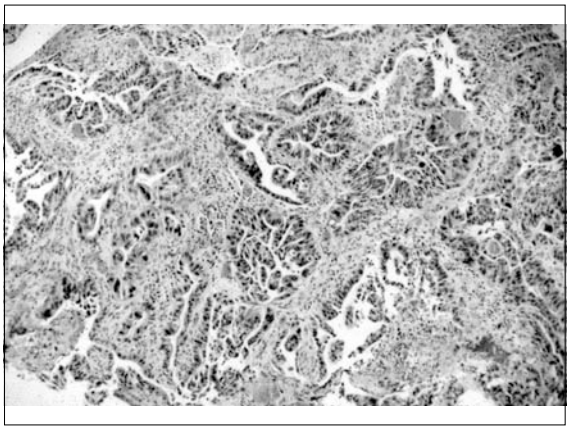
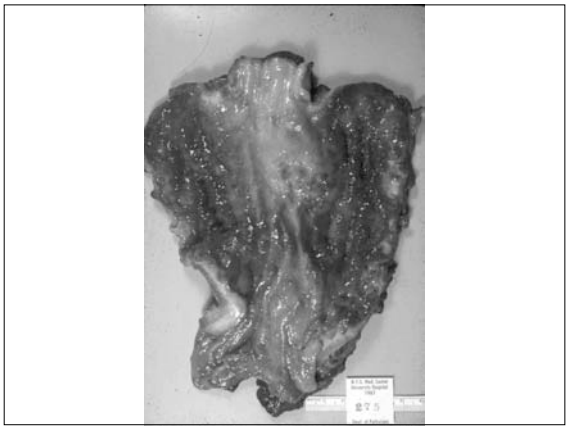


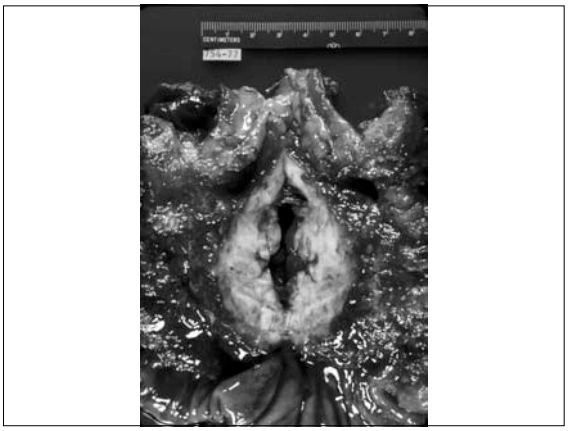
Fig. 2.02 Incidence of adenocarcinoma of the stomach (left) compared to adenocarcinoma of the distal oesophagus and oesophagogastric junction (right). Rate per 10,000 hospitalisations from North America.

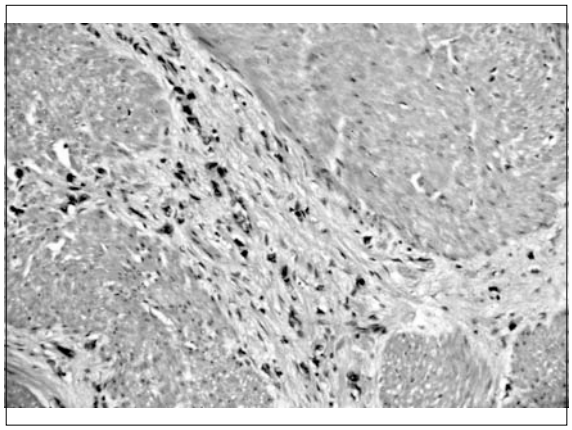












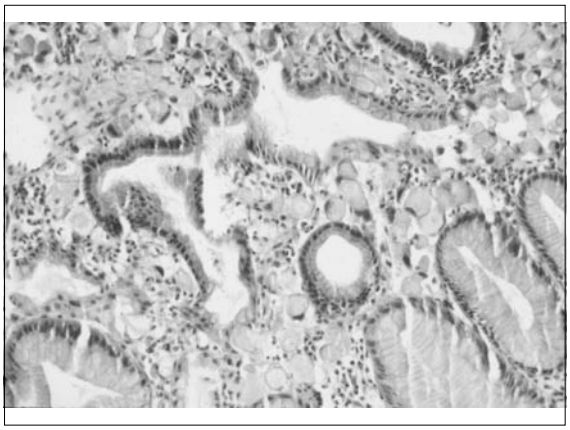


Table 17–4. MAJOR FEATURES OF LAURENS' CLASSIFICATION OF GASTRIC CARCINOMA

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.

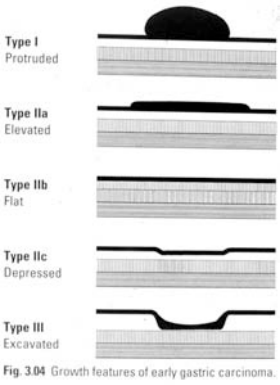


Fig. 3.04 Growth features of early gastric carcinoma.

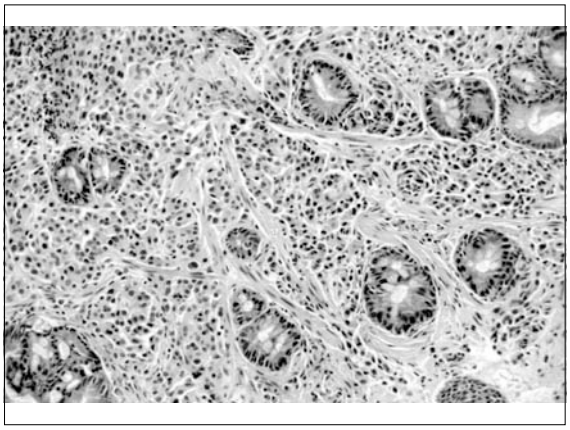
Table 3.02.
Histological classification of endocrine neoplasm of the stomach[1]

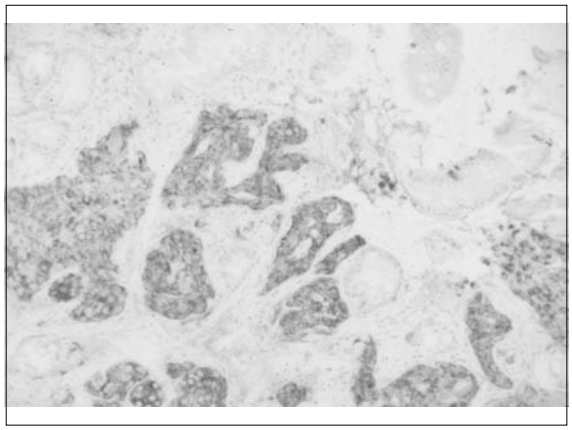
1. Carcinoid –
well differentiated endocrine neoplasm
1.1 ECL-cell carcinoid
1.2 EC-cell, serotonin-producing carcinoid
1.3 G-cell, gastrin-producing tumour
1.4 Others

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.





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