**MICROSCOPIC ANATOMY OF THE STOMACH AND DUODENUM**

**Parietal cells**  
These are in the bodacid-secreting portion) of the stomach and line the gastric crypts. They are responsible for the production of hydrogen ions to form hydrochloric acid.

**Chief cells**  
Proximally in the gastric crypts and produce pepsinogen. Two forms of pepsinogen are described: pepsinogen I and pepsinogen II.

**Endocrine cells**  
In the gastric antrum, the mucosa contains G cells, which produce gastrin. Throughout the body of the stomach, enterochromaffin-like (ECL) cells are abundant and produce Histamine, somatostatin-producing D cells throughout the stomach, and somatostatin has a negative regulatory role.
PHYSIOLOGY OF THE STOMACH AND DUODENUM
Flexible endoscopy
Contrast radiology
Ultrasonography
CT scanning and magnetic resonance imaging
CT/positron emission tomography
Laparoscopy
Gastric emptying studies
Angiography
PET Scan = advanced gastric cancer with lymph node spread
Investigation for H. pylori

13C and 14C Urea breath test

HISTOLOGICALLY Giemsa or the Ethin–Starey silver stains,

Serological test

Breath tests or faecal antigen tests are recommended for the pretreatment diagnosis of H. pylori infection in the community

It causes chronic gastritis, peptic ulcer and gastric cancer

eradication therapy is recommended for patients with duodenal ulcer disease, but not for patients with nonulcer dyspepsia or in asymptomatic patients who are infected

H. pylori is now classed by the World Health Organisation as a class 1 carcinogen
The spiral bacterium *H. pylori* is critical in the development of type B gastritis, peptic ulceration and gastric cancer. Infection appears to be acquired mainly in childhood and the infection rate is inversely associated with socioeconomic status.

**Eradication,** recommended specifically in patients with peptic ulcer disease, can be achieved in up to 90 per cent of patients with a combination of a proton pump inhibitor and antibiotics, and reinfection is uncommon (<0.5 percent).

**Erosive gastritis** is usually related to the use of NSAIDs. **Type A gastritis** is an autoimmune process and is associated with the development of pernicious anaemia and gastric cancer.
Autoimmune
Circulating antibodies to the parietal cell hypochlorhydria and ultimately achlorhydria pernicious anaemia
Production of high levels of gastrin from the antral G cells
Patients with type A gastritis are predisposed to the development of gastric cancer
Type B gastritis

Affect antrum
Association of this type of gastritis with *H. pylori*
Patients with pangastritis seem to be most prone to the development of gastric cancer.
This is caused by enterogastric reflux and is particularly common after gastric surgery have had a cholecystectomy. Bile chelating or prokinetic agents may be useful in treatment. Operation for the condition should be reserved for the most severe cases.
The NSAID-induced gastric lesion is associated with inhibition of the cyclo-oxygenase type 1 (COX-1) receptor enzyme, hence reducing the production of cytoprotective prostaglandins in the stomach. The use of specific COX-2 inhibitors reduces the incidence of these side effects.
Stress gastritis

common sequel of serious illness (follows cardiopulmonary bypass) or injury and is characterised by a reduction in the blood supply to superficial mucosa of the stomach.

the routine use of H2-antagonists with or without barrier agents, such as sucralfate, in patients who are on intensive care unit.
Ménétrier’s disease

Unusual condition
gross hypertrophy of the gastric mucosal folds, mucus production and hypochlorhydria.
Hypoproteinemia & anemia
Premalignant condition
Over expression of TGF-α
Treatment: gastrectomy
Rare type infiltration of the gastric mucosa by T cells and is probably associated with *H. pylori infection*. resembles pattern seen in coeliac disease or lymphocytic colitis.

Eosinophilic gastritis: allergy

Granulomatous gastritis: Crohns disease and TB

Acquired immunodeficiency syndrome (AIDS) gastritis

Phlegmonous gastritis: bacterial
Common sites for peptic ulcers are the first part of the duodenum and the lesser curve of the stomach.

They also occur on the stoma following gastric surgery, the oesophagus and even in a Meckel’s diverticulum infection with *H. pylori* and the consumption of NSAIDs are the most important factors in the development of peptic ulceration.

Cigarette smoking predisposes to peptic ulceration and increases the relapse rate after treatment, with either gastric antisecretory agents or, in the past, elective Surgery.
the peak incidence is now in a much older age group

More common in men,
Most occur in the first part of the duodenum chronic ulcer penetrates the mucosa and into the muscle coat, leading to fibrosis.

The fibrosis causes deformities such as pyloric stenosis ‘kissing ulcers’.

Anterior ulcer tend to perforate, Posterior ulcer tend to bleed

malignancy in this region is so uncommon that under normal circumstances surgeons can be confident that they are dealing with benign disease
Destruction of the muscular coat is observed and the base of the ulcer is covered with granulation tissue, the arteries in this region showing the typical changes of endarteritis Obliterans
As with duodenal ulceration, *H. pylori* and NSAIDs are the important aetiological factors.

Gastric ulceration is also associated with smoking. Gastric ulceration is substantially less common than duodenal ulceration.

The sex incidence is equal and the population with gastric ulcers tends to be older.

It is more prevalent in low socioeconomic groups.
Gastric ulcers tend to be larger.
Fibrosis rarely seen hourglass contraction of the stomach.

Large chronic ulcers may erode posteriorly into the pancreas and, on other occasions, into major vessels such as the splenic artery.

Chronic gastric ulcers are much more common on the lesser curve (especially at the incisura angularis).
Giant ulcers are those that are more than 3 cm in diameter. These ulcers have an increased association with cancer: 30% of those larger than 3 cm harbor malignant disease.

Earlier surgical intervention is generally warranted given this association.

Endoscopy with multiple biopsies (at least four with jumbo forceps and eight with regular) to include both the ulcer base and edge usually provide sufficient tissue for diagnosis to guide therapy, with treatment of nonmalignant ulcers adhering to guidelines as outlined previously, depending on the location.
Malignancy in gastric ulcer

It is fundamental that any gastric ulcer should be regarded as being malignant.

Multiple biopsies should always be taken, perhaps as many as ten well-targeted biopsies.

It is important that further biopsies are taken while the ulcer is healing and when healed.

At operation, even experienced surgeons may have difficulty distinguishing between the gastric cancer and a benign ulcer.
Clinical features of peptic ulcer

Pain
Periodicity
Vomiting: fibrosis
Alteration in weight
Bleeding: **microcytic anaemia** is not uncommon
Peptic ulcer and H. pylori then eradication Rx

NSAIDS and stomal ulcer

Patients with Zollinger–Ellison syndrome should be treated in the long term with proton pump inhibitors unless the tumour can be adequately managed by surgery.
<table>
<thead>
<tr>
<th>Length of treatment</th>
<th>Agent</th>
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| Orally, twice daily for 14 days | PPI (omeprazole 20 mg OR lansoprazole 30 mg)  
+ Amoxicillin 1000 mg  
+ Clarithromycin 500 mg |
| Orally, twice daily for 14 days | PPI (omeprazole 20 mg OR lansoprazole 30 mg)  
+ Metronidazole 500 mg  
+ Clarithromycin 500 mg |
| Orally, given as indicated for 14 days | Alternative regimen:  
Bismuth subsalicylate 525 mg qid  
+ Metronidazole 500 mg tid  
+ Tetracycline 500 mg qid  
+ PPI (omeprazole 20 mg OR lansoprazole 30 mg daily) |
Why some ulcers fail to heal

- persistent *H. pylori* Infection
- poor compliance
- ingestion of NSAIDs
- Zollinger–Ellison syndrome
Billroth II gastrectomy: Two-thirds of the stomach removed, the duodenal stump is closed and the stomach anastomosed to the jejunum.

Gastrojjenostomy

Truncal vagotomy with drainage (HM)

Highly Selective vagotomy with drainage

Truncal vagotomy with antrectomy
Pyloroplasty.
Operations for gastric ulcer

Billroth I
gastrectomy
Most peptic ulcers are caused by *H. pylori* or NSAIDs

Duodenal ulcers are more common than gastric ulcers, but the symptoms are indistinguishable

Gastric ulcers may become malignant and an ulcerated gastric cancer may mimic a benign ulcer

Gastric antisecretory agents and *H. pylori eradication* therapy are the mainstay of treatment, and elective surgery is very rarely performed

The long-term complications of peptic ulcer surgery may be difficult to treat

The common complications of peptic ulcers are perforation, bleeding and stenosis

The treatment of the perforated peptic ulcer is primarily surgical, although some patients may be managed conservatively
Sequelae of peptic ulcer surgery

1. Recurrent ulceration
2. Small stomach syndrome
3. Bile vomiting
4. Early and late dumping
5. Postvagotomy diarrhea
6. Malignant transformation
7. Nutritional consequences (B12, iron, bone)
8. Gall stones
Early dumping

The small bowel is filled with foodstuffs from the stomach, which have a high osmotic load, and this leads to the sequestration of fluid from circulation into the gastrointestinal tract..

The principal treatment is **dietary manipulation**. Small, dry meals are best, and avoiding fluids with a high carbohydrate content..

Surgery: **Roux en Y reconstruction**
This is reactive *hypoglycaemia*. The carbohydrate load in the small bowel causes a rise in the plasma glucose, which, in turn, causes insulin levels to rise, causing a secondary hypoglycaemia.

The treatment is essentially the same as for early dumping.

*Octreotide* is very effective in dealing with this problem.
<table>
<thead>
<tr>
<th></th>
<th>Early</th>
<th>late</th>
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<tbody>
<tr>
<td>incidence</td>
<td>5-10%</td>
<td>5%</td>
</tr>
<tr>
<td>Relation to meal</td>
<td>immediately</td>
<td>Second hour after meal</td>
</tr>
<tr>
<td>Duration of attack</td>
<td>30-40 min</td>
<td>same</td>
</tr>
<tr>
<td>relief</td>
<td>Lying down</td>
<td>food</td>
</tr>
<tr>
<td>Aggrevated by</td>
<td>More food</td>
<td>exercise</td>
</tr>
<tr>
<td>Precipitating factor</td>
<td>CHO</td>
<td>CHO</td>
</tr>
<tr>
<td>Major symptom</td>
<td>Fullness, sweating, tachycardia and sometimes diarrhea</td>
<td>Tremor faintness and prostration</td>
</tr>
</tbody>
</table>
Previously, most patients were middle aged, with a ratio of 2:1 of male:female.

With time, there has been a steady increase in the age of the patients suffering this complication and an increase in the numbers of females, such that perforations now occur most commonly in elderly female patients. NSAIDs appear to be responsible for most of these perforations.
sudden onset severe generalised abdominal pain

Initially, the patient may be shocked with a tachycardia but a pyrexia is not usually observed until some hours after the event.

Boardlike rigidity and the patient is disinclined to move because of the pain. The abdomen does not move with respiration.

The perforation may be self-limiting??how
Erect plain chest radiograph will reveal free gas under the diaphragm in an excess of 50 per cent of cases.

CT imaging is more accurate

serum amylase: WHY?
The treatment is principally surgical.

Systemic antibiotics resuscitation and analgesia

**Laparotomy** is performed, usually through an upper midline incision, thorough peritoneal toilet to remove all of the fluid and food debris.

If the perforation is in the duodenum it can usually be closed by several well-placed sutures, closing the ulcer in a transverse direction as with a pyloroplasty.

It is common to place an omental patch over the perforation.

Gastric ulcers should be excised and closed, so that malignancy can be excluded.

**Massive duodenal** or gastric perforation such that simple closure is impossible; in these patients a Billroth II gastrectomy or subtotal gastrectomy with Roux-en-Y reconstruction

**Stomach** is kept empty postoperatively by nasogastric suction, and that gastric antisecretory agents are commenced to promote healing of the residual ulcer.
FACTORS associated with poor outcome

- delay in diagnosis (>24 hours)
- medical comorbidities
- shock
- increasing age (>75).

Patients who have suffered one perforation may suffer another one
The two common causes of gastric outlet obstruction are:

1-gastric cancer

2-pyloric stenosis secondary to peptic ulceration.
Clinical features

**pain** may become unremitting and in other cases it may largely disappear.

The **vomitus** is characteristically unpleasant in nature and is totally lacking in bile.

It is possible to recognise foodstuff taken several days previously.

**losing weight**, and appears unwell and **dehydrated**.

**succussion splash** may be audible on shaking the patient’s abdomen.
vomiting of hydrochloric acid results in hypochloraemic alkalosis

Initially, the sodium and potassium may be relatively normal

Dehydration progresses lead to renal dysfunction

Initially, the urine has a low chloride and high bicarbonate content

This bicarbonate is excreted along with sodium, and so with time the patient becomes progressively hyponatraemic and more profoundly dehydrated

Because of the dehydration, a phase of sodium retention follows and potassium and hydrogen are excreted

The urine becoming paradoxically acidic and hypokalaemia

Alkalosis leads to a lowering in the circulating ionised calcium, and tetany can occur
Management

The patient should be rehydrated with intravenous isotonic saline with potassium supplementation.

Replacing the sodium chloride and water allows the kidney to correct the acid-base abnormality.

gastric antisecretory agent given intravenously

a wide-bore gastric Tube

endoscopy and contrast radiology

Biopsy of the area around the pylorus is essential to exclude malignancy.

Dilatation, stent, drainage procedure and surgery for CA
Other causes of gastric outlet obstruction

Adult pyloric stenosis

Pyloric mucosal diaphragm
Risak of CA
Biopsy is essential
Types:
- **Metaplastic**: most common associated with H.P and regress after eradication therapy
- **Inflammatory polyps**: common
- **Fundic gland polyps**: associated with PPI use and FAP
- **Adenomatous** polyp: pre malignant, 10% of polyps

Gastric carcinoids arising from the ECL cells are seen in patients with pernicious anaemia and usually appear as small polyps
major cause of cancer mortality Worldwide

prognosis tends to be poor, with cure rates little better than 5–10 per cent

Early diagnosis is the key to success

The only treatment modality able to cure the disease is resectional surgery
In the UK, it is approximately 15/100,000 per Year.

USA 10/100,000 per year

70/100,000 per Year in Japan

Men are more affected by the disease than women.

Increase the proximal stomach, particularly the oesophagogastric junction.

Proximal gastric cancer does not seem to be associated with *H. pylori* infection.

Proximal CA: high SE
Distal CA: Low SE
ETIOLOGY

H. pylori
gastric atrophy, intestinal metaplasia, Pernicious Anaemia
gastric polyps

Billroth II, gastroenterostomy or pyloroplasty, duodenogastric reflux
and reflux gastritis

Cigarette smoking and dust ingestion

DIETS: spirit, salt intake,
Obesity: proximal CA

Genetic factors:
Mutation APC gene (Tumor supressor gene) or b-catenin, E cadherin
hereditary non-polyposis colorectal (HNPCC) (Lynch syndrome)

Inactivation of p53
Clinical features

Dyspepsia
In advanced cancer, early satiety, bloating, distension

Vomiting
iron deficiency anaemia

Obstruction leads to dysphagia, epigastric Fullness
Gastric outlet obstruction

Thrombophlebitis (Trousseau’s sign) and deep venous thrombosis.
Upper oesophagus: 2%
Mid oesophagus: 6%
Lower oesophagus: 22%
GE: 18%
Cardia: 17%
Body: 15%
Antrum: 13%
Pylorus: 7%
Intestinal: polyp or ulcer

Diffuse: without mass but more involvement (worse Px)
Early gastric CA:
Mucosa and submucosa with or without LN involvement
T1 & any N
CURABLE
5yrs survival rate 90%
Type 0-I — Protruded type

Type 0-IIa — Superficial and elevated type

Type 0-IIb — Flat type

Type 0-IIc — Superficial and depressed type

Type 0-III — Excavated type
Advanced gastric CA:

involves the muscularis

Bormann classification from I to IV

III & IV are incurable
<table>
<thead>
<tr>
<th>Borrmann I</th>
<th>육기형</th>
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<tbody>
<tr>
<td>Borrmann II</td>
<td>궤양형</td>
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<tr>
<td>Borrmann III</td>
<td>궤양침윤형</td>
</tr>
<tr>
<td>Borrmann IV</td>
<td>미만형</td>
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</tbody>
</table>
TNM staging

T1: Tumour involves lamina propria, submucosa
T2: Tumour invades muscularis propria
T3: Tumour involves subserosa
T4a: Tumour perforates serosa
T4b: Tumour invades adjacent organs

N0: No lymph nodes
N1: Metastasis in 1–2 regional nodes
N2: Metastasis in 3–6 regional nodes
N3a: Metastasis in 7–15 regional nodes
N3b: Metastasis in more than 15 regional nodes

M0: No distant metastasis
M1: Distant metastasis (this includes peritoneum and distant lymph nodes)

Lymph node involvement can occur in stage I
No distant metastasis before stage IV disease
Spread of CA stomach

Direct spread pancreas, colon and liver

Lymphatic spread  (Troisier’s sign).

Blood-borne metastases liver, lung and bone

Transperitoneal spread: Krukenberg’s tumours and Sister Joseph’s nodule
* Hematogenous metastasis
* Involvement of distant peritoneum
* N4 nodal disease
* Fixation to structures that cannot be removed
* Signs of Inoperability

* Positive cytology in peritoneal wash
* Peritoneal deposits
* Posterior fixation
* Fixed celiac nodes
* Para-aortic nodes
* Liver metastasis
Total gastrectomy

Total resection of cancerous stomach

Esophagus
Diaphragm

Jejunum
Duodenum
For tumours distally placed in the stomach

This will lead to enterogastric reflux So Roux loop will solve the problem
Leakage of the oesophagojejunostomy fistula from the wound or drain site perform a water-soluble contrast swallow at 5–7 days after the operation to determine whether the anastomosis is intact leakage from the duodenal stump Paraduodenal collections can be drained radiologically Biliary peritonitis requires a laparotomy and peritoneal toilet secondary haemorrhage from the exposed or divided blood vessels
Palliative surgery, stenting and recanalization

obstruction

bleeding
Fig. 18.14  Palliative bypass procedure for gastric carcinoma.
Radiotherapy is controversial

Chemotherapy by: epirubacin, *cis-platinum* and *infusional 5-FU* or an oral analogue such as *capecitabine*
Gastric cancer is one of the most common causes of cancer death in the world

The outlook is generally poor, owing to the advanced stage of the tumour at presentation

Better results are obtained in Japan, which has a high population incidence, screening programmes and a high quality surgical treatment

The aetiology of gastric cancer is multifactorial, but *H. pylori* is an important factor for distal but not proximal gastric cancer

Early gastric cancer is associated with very high cure rates

Gastric cancer can be classified into intestinal and diffuse types, the latter having a worse prognosis
In the West, proximal gastric cancer is now more common than distal cancer and is usually of the diffuse type.

Spread may be by lymphatics, blood, transcoelomic or direct, but distant metastases are uncommon in the absence of lymph node involvement.

The treatment of curable cases is by radical surgery and removal of the second tier of nodes (around the principal arterial trunks) may be advantageous.

Gastric cancer is chemosensitive and chemotherapy improves survival in patients having surgery for the condition and in advanced disease.
50 per cent will be found in the Stomach
tumours of mesenchymal origin and are observed equally in males and females

mutation in the tyrosine kinase \textit{c-kit oncogene}.
sensitive to the tyrosine kinase antagonist imatinib, an 80 per cent objective response rate

size and mitotic figures index are the best predictors of metastasis.
GIST comprise 1–3 per cent of all gastrointestinal neoplasia.

The only ways that many stromal tumours are recognised are either that the mucosa overlying the tumour ulcerates leading to bleeding, or that they are noticed incidentally at endoscopy.

Targeted biopsy by endoscopic ultrasound is more helpful.

Tumours over 5 cm in diameter should be considered to have metastatic potential.

Surgery is the primary mode of treatment; lymphadenectomy is not required.

The prognosis of advanced metastatic GIST has been dramatically improved with imatinib chemotherapy but resection of metastases, especially from the liver, still has an important role.
It is first important to distinguish primary gastric lymphoma from involvement of the stomach in a generalised lymphomatous process.

Incidence of lymphoma increasing accounts for approximately 5 per cent of all gastric Neoplasms.

Common in 6th decade pain, weight loss and bleeding.

Acute presentations (POUB) obstruction, are not common.
B cell derived
the tumour arising from the mucosa-associated lymphoid tissue (MALT)

Diffuse mucosal thickening, which may ulcerate

Diagnosis is made as a result of the endoscopic biopsy
CT scans of the chest
abdomen and bone marrow aspirate are required, full blood count

early gastric lymphomas may regress and disappear when the *Helicobacter* infection is treated.

Treatment: surgery although some prefer chemoRx??
Douodenal tumors

Benign: villous adenoma (FAP), periampullary area and premalignant

Douodenal adenocarcinoma: uncommon, ass. With FAP most common site for adenocarcinoma arising in the small bowel.

Anaemia due to ulceration of the tumour or obstruction as the polypoid neoplasm begins to obstruct the duodenum.

Obstructive jaundice

70 per cent of the patients have resectable disease

The five-year survival rate is in the region of 20 per cent

Curative surgical treatment (Whiplle procedure)
Zollinger–Ellison syndrome

Found in doudenum and head of pancreas
It is a cause of persistent peptic ulceration
sporadic or associated with the autosomal dominantly
inherited multiple endocrine neoplasia (MEN) type I
PASSARO triangle

Dx:
Fasting serum gastrin
In case of moderate hypergastrinemia, a secretin
stimulation test can help in the diagnosis
Localization by somatostatin scintigraphy (octeriotide
scan)

Rx: PPI and surgery
Gastrinoma Triangle

90% of gastrinomas are located within this anatomic triangle

50% duodenum
50% pancreas
Doudenal obstruction

Doudenal causes

Pancreatic causes

Superior mesenteric artery syndrome

Metastasis from colorectal and gastric CA
Trichobezoar and Phytothecozoa
GASTRIC VOLVULUS

Rotation of the stomach usually occurs around the axis and between its two fixed points: cardia & pylorus horizontal (organoaxial) common vertical (mesenteroaxial) direction

Associated with a large diaphragmatic defect (paraoesophageal herniation)
The condition is commonly chronic, the patient presenting with difficulty in eating Ischemia when acutely presented contrast radiograph is superior to endoscopy in Dx
Diphragmatic defect repair with mesh

Separation of stomach from transverse colon

Anterior gastopexy