STOMACH AND DUODENUM

The stomach has important **digestive**, **nutritional**, and **endocrine** functions. It is to act as a reservoir for ingested food. It also serves to break down foodstuffs mechanically and commence the processes of digestion before these products are passed on into the duodenum.

ANATOMY

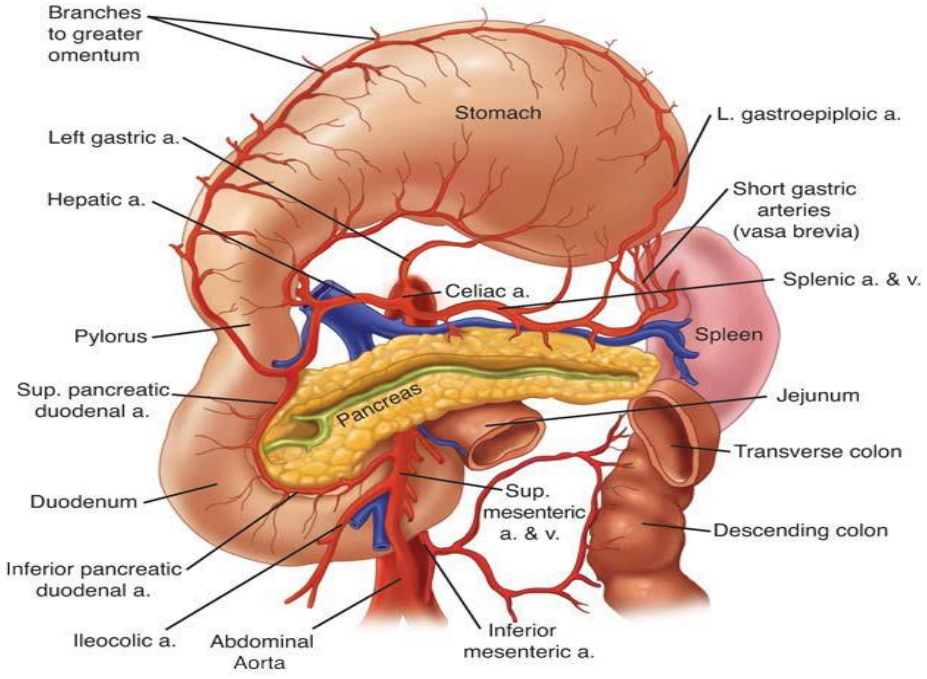
Blood supply

The stomach is the ***most richly*** vascularized portion of the GIT.

On the lesser curve, the *left gastric artery*, a branch of the coeliac axis, the *right gastric artery*, which arises from the common hepatic artery.

The gastroduodenal artery, which is also a branch of the hepatic artery, passes behind the first part of the duodenum, highly relevant with respect to the bleeding duodenal ulcer, it divides into the superior pancreaticoduodenal artery (supplies the duodenum and pancreatic head, in an anastomosis with the inferior pancreaticoduodenal artery, a branch of the superior mesenteric artery.) and the *right gastroepiploic* artery (runs along the greater curvature, and anastomoses with the *left gastroepiploic* artery, a branch of the splenic artery)

The fundus of the stomach is supplied by the vasa brevia (*short gastric arteries*) branches from the splenic artery.



Veins

Equivalent to the arteries. those along the lesser curve ending in the **portal vein** and those on the greater curve joining via the splenic vein.The left gastric (coronary vein, which is dilated in portal hypertension)

and right gastric veins usually drain into the portal vein. The right gastroepiploic vein drains into the superior mesenteric vein, and the left gastroepiploic vein drains into the splenic vein.

Lymphatics

These are of considerable **importance in the surgery** of gastric cancer.

The gastric lymphatics **parallel** the blood vessels . They have a rich anastomotic network.

The cardia and medial half of the corpus commonly drain to nodes along the left gastric and celiac axis. The lesser curvature side of the antrum usually drains to the right gastric and pyloric nodes, while the greater curvature half of the distal stomach drains to the nodes along the right gastroepiploic chain. The proximal greater curvature side of the stomach usually drains into nodes along the left gastroepiploic or splenic hilum. The nodes along both the greater and lesser curvature commonly drain into the celiac nodal basin.

Nerves

As with the entire GIT, the stomach and duodenum possess both **intrinsic** and **extrinsic** nerve supplies. The intrinsic nerves exist principally in two plexuses, the myenteric plexus of Auerbach and the submucosal plexus of Meissner.

The extrinsic supply is derived mainly from the vagus nerves. Vagal fibres condense into a posterior & anterior trunks, they are both afferent (sensory) and efferent(secreto-motor). The sympathetic supply is derived mainly from the coeliac ganglia.

HISTOLOGY

The gastric epithelial cells are mucus producing and are turned over rapidly. It has numerous endocrine cells.

Parietal cells

These are in the body (HCl acid-secreting) of the stomach and line the gastric crypts.

Chief cells

These lie principally proximally and produce pepsinogen, which is activated in the stomach to produce the digestive protease, pepsin.

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, a key factor in driving gastric acid secretion. There are large numbers of somatostatin-producing D cells throughout the stomach, and somatostatin has a negative regulatory role.

Duodenum

It is lined by a mucus-secreting columnar epithelium.

Brunner's glands lie beneath the mucosa and are similar to the pyloric glands in the pyloric part of the stomach. Endocrine cells in the duodenum produce cholecystokinin and secretin.

Source

Stimulate secretion

Gastrin G cells

Histamine ECL cells

Acetylcholine Neurones

Gastrin-releasing peptide Neurones and mucosa

Cholecystokinin (CCK) Duodenal endocrine cells

Inhibit secretion

Somatostatin D cells and neurones
Secretin Duodenal endocrine cells
Enteroglucagon Small intestinal endocrine cells
Prostaglandins Mucosa
Neurotensin Neurones
GIP Duodenal and jejunal endocrine cells
PYY Small intestinal endocrine cells

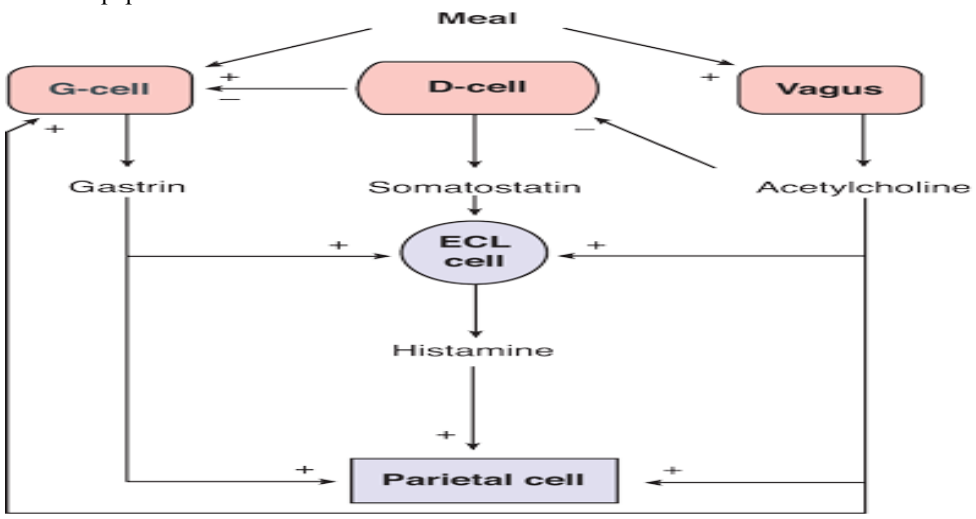
Stimulate motility

Acetylcholine Neurones
5-HT Neurones
Histamine ECL cell
Substance P Neurones
Substance K Neurones
Motilin Neurones
Gastrin G cells
Angiotensin

Inhibit motility

Somatostatin D cells and neurones
VIP Neurones
Nitric oxide Neurones and smooth muscle
Noradrenaline Neurones
Encephalin Neurones
Dopamine Neurones

ECL, entrochromaffin-like cells; GIP, gastric inhibitory polypeptide; PYY, peptide YY; VIP, vasoactive intestinal peptide..



The investigation of gastric disorders

_ Flexible endoscopy is the most commonly used and sensitive technique for investigating the stomach and duodenum

_ Axial imaging, particularly multislice CT, is useful in the staging of gastric cancer

_ CT/PET is useful in staging gastric cancer

_ Endoscopic ultrasound is the most sensitive technique in the evaluation of the 'T' stage of gastric cancer and in the
assessment of duodenal tumours
_ Laparoscopy is very sensitive in detecting peritoneal metastases, and laparoscopic ultrasound provides an accurate evaluation of lymph node and liver metastases.
-Gastric emptying studies are useful in the study of gastric dysmotility problems**.**

HELICOBACTER PYLORI

It has proved to be of importance in the aetiology of a number of common gastroduodenal diseases such as chronic gastritis, peptic ulceration and gastric cancer(ca & lymphoma).

The organism is spiral shaped**, Gram N-ve**, **micro-aerophillic**, and is fastidious in its requirements, being difficult to culture outside the
mucous layer of the stomach. Infection with *H. pylori* may be the most common human infection.. Up to **50 %** of the world's population may be infected with helicobacter. Most infection is acquired in childhood and the possibility of infection is inversely related to socioeconomic group. The faecal–oral spread seems most likely

It has the ability to hydrolyse urea (by **urease**) , resulting in the production of ammonia, a strong alkali. The effect of ammonia on the antral G cells is to cause the release of gastrin , which, in turn, may result in gastric acid hypersecretion.

Infection with *H. pylori* leads to the disruption of the gastric mucous barrier by its enzymes, and the inflammation induced in the gastric epithelium is the basis of many of the associated disease processes e.g. chronic (type B) gastritis. That involves the migration and degranulation of acute inflammatory cells, such as neutrophils, and also the accumulation of chronic inflammatory cells, such as macrophages and lymphocytes.

Some strains of *H. pylori* produce cytotoxins, notably the *Cag A*and *Vac A* products.

Although normal duodenal mucosa cannot be infected with *H. pylori*, gastric metaplasia (in response to excessive acid exposure) in the duodenum is commonly infected and this infection results in the same inflammatory process that is observed in the gastric mucosa. The result is duodenitis, which is almost certainly the precursor of duodenal ulceration.

Commonly used eradication regimes include a proton pump inhibitor and two antibiotics, such as metronidazole and amoxycillin. Very high eradication rates,(**90%**), can be achieved with combinations that include the antibiotic clarithromycin,

. Reinfection following successful eradication appears rare (<0.5 %) but incomplete eradication is a more important clinical problem.

Investigations of H.pylori

1-Breath Test (Carbon Isotope-urea Breath Test, or UBT) screening or completion treatment.
2- Serology antibodies to H pylori
3- Stool Test. A stool test can detect Ag of H pylori in the feces.
4-Endoscopy & Biopsy: Invasive .

GASTRITIS

Type A gastritis

This is an **autoimmune** condition in which there are circulating antibodies to the **parietal** cell. This results in the atrophy of the parietal cell mass, hence **hypochlorhydria** and ultimately achlorhydria. As intrinsic factor is also produced by the parietal cell there is malabsorption of vitamin B12, which, if untreated, may result in **pernicious anaemia**. The hypochlorhydria leads to the production of high levels of gastrin & this results in chronic hypergastrinaemia. This, in turn, results in hypertrophy & that microadenomas of the ECL cells in the body of the stomach. Very rarely, these tumours can become malignant. Patients with type

A gastritis are predisposed to the development of **gastric cancer**, and screening such patients endoscopically may be appropriate.

Type B gastritis

It is associated with ***H. pylori***. Most commonly, type B gastritis affects the antrum, and it is these patients who are prone to peptic ulcer disease. *Helicobacter*-associated pangastritis is also a very common manifestation of infection. Patients with pangastritis seem to be most prone to the development of gastric cancer.

Intestinal **metaplasia** is associated with chronic pangastritis with atrophy, but if associated with dysplasia has significant malignant potential and requires endoscopic screening.

Reflux gastritis

This is caused by **enterogastric reflux** and is particularly common after gastric surgery, a cholecystectomy or occasionally with no previous surgery. Its histological features are distinct from other types of gastritis.

Bile chelating or prokinetic agents may be useful in treatment and as a temporising measure to avoid consideration of **revisional surgery**, which is reserved for the most severe cases.

Erosive gastritis

This is caused by agents that disturb the gastric **mucosal barrier**; **NSAIDs** and alcohol are common causes. NSAIDs inhibits the cyclo-oxygenase type 1 (COX-1) receptor enzyme, reducing the production of cytoprotective prostaglandins (mucus) in the stomach. The use of specific COX-2 inhibitors reduces the incidence of these side effects. However, cardiovascular S/E in long term use.

Stress gastritis

This is a common sequel of **serious illness** ,e.g.burn or injury, & s.t after cardiopulmonary bypass. Its d.t reduction in the blood supply **(ischemia)** to superficial mucosa of the stomach.

Prevention of it is much easier than treatment, and hence the routine use of H2-antagonists with or without barrier agents, such as sucralfate, in patients who are on intensive care.

Ménétrier's disease

This is an unusual condition characterised by gross hypertrophy of the gastric mucosal folds, mucus production and **hypochlorhydria**.

The condition is **premalignant** and may present with **hypoproteinaemia** and anaemia. There is no treatment other than a gastrectomy

Lymphocytic gastritis

Rare. Infiltration of the gastric mucosa by **T cells** and is probably associated with *H. pylori* infection. The pattern of inflammation resembles that seen in coeliac disease or lymphocytic colitis.

Other forms of gastritis

Eosinophilic gastritis (allergic). Granulomatous gastritis(Crohn's disease , tuberculosis). Acquired immunodeficiency syndrome(AIDS) gastritis (infection with cryptosporidiosis). Phlegmonous gastritis (rare bacterial infection in patients with severe intercurrent illness).

PEPTIC ULCER

Although the name 'peptic' ulcer suggests an association with pepsin, this is essentially unimportant as in the absence of acid.

Common sites are1- the first part of the duodenum, 2-the lesser curve of the stomach, on 3-the stoma following gastric surgery, 4-the oesophagus, & 5- Meckel's diverticulum (ectopic gastric epithelium). In general, the ulcer occurs at a junction between different types of epithelia, the ulcer occurring in the epithelium least resistant to acid damage.

H.pylori and NSAIDs act synergistically to promote ulcer development and ulcer bleeding. Cigarette smoking predisposes to peptic ulceration and increases the relapse rate after treatment.

Duodenal ulceration

Incidence

First, even before the introduction of H2-receptor antagonists, the incidence of duodenal ulceration and the frequency of elective surgery for the condition were **falling**. In part, this may relate to the widespread use of gastric antisecretory agents and *H. pylori* eradication therapy.

Second, the peak incidence is now in a much **older age** group than previously and, & still more common in men.

Pathology

Most occur in the **first part** of the duodenum . A chronic ulcer penetrates the mucosa and into the muscle coat, leading to fibrosis. The fibrosis causes deformities such as pyloric stenosis.

The situation in which there is both a posterior and an anterior duodenal ulcer is referred to as '**kissing** ulcers'.

Anteriorly placed ulcers tend to **perforate** ,while **posterior** duodenal ulcers tend to **bleed**, by eroding into the **gastroduodenal** artery. Unlike gastric ulcers, malignant change is very unlikely.

Histopathology

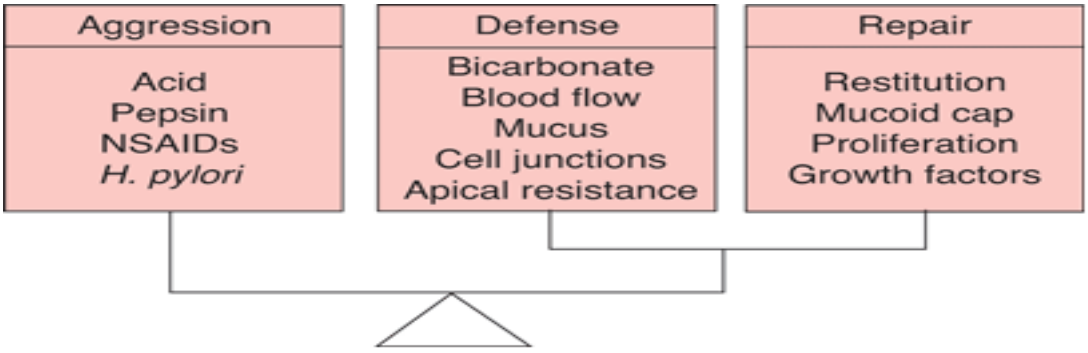
Destruction of the muscular coat , and the base of the ulcer is covered with granulation tissue.

Gastric ulcers

Incidence

As with duodenal ulceration, *H. pylori* and NSAIDs are the important aetiological factors. Gastric ulceration is also associated with smoking.

Differences between gastric & duodenal ulceration. First, gastric ulceration is **less** common. The sex incidence is **equal** and the population with gastric ulcers is **older**. It is more prevalent in **low** socioeconomic groups and more common in the **developing** world than in the West.



Pathology

This is essentially similar to that of a duodenal ulcer, except that gastric ulcers tend to be **larger**. Fibrosis, may result in the now rarely seen hourglass contraction of the stomach.

Large chronic ulcers may erode posteriorly into the, into major vessels such as the splenic artery, or into other organs such as the transverse colon. Chronic gastric ulcers are much more common on the lesser curve.

Malignancy in gastric ulcers

Chronic duodenal ulcers are not associated with malignancy, in contrast, gastric ulcers are.
Two clinical extremes must be distinguished to understand this problem properly. **First**, sometimes a benign chronic gastric ulcer undergoes **malignant transformation** (rare). **The 2nd** when a patient presented with an ulcerated gastric lesion but **biopsies reveal malignancy** (common).
It is fundamental that any gastric ulcer should be regarded as **being malignant**. Multiple **biopsies** should always be taken, as many as ten well-targeted biopsies, before an ulcer can be accepted as being benign.
Modern antisecretory agents can frequently heal the ulceration associated with gastric cancer but, are ineffective in treating the malignancy itself.

Other peptic ulcers

The prepyloric gastric ulcer is usually treated with proton pump inhibitors. Pyloric channel ulcers are similar to duodenal ulcers. Both prepyloric and pyloric ulcers may be malignant, and biopsy is essential.
Stomal ulcers occur after a gastroenterostomy, usually found on the jejunal side of the stoma.

Clinical features of peptic ulcers

Gastric and duodenal ulceration, cannot be differentiated on the basis of symptoms.

Pain

Epigastric, often described as gnawing and may radiate
to the back. Eating may sometimes relieve the discomfort.

Periodicity

Symptoms may disappear for weeks or months to return again. This may be related to the spontaneous healing of the ulcer.

Vomiting

It is not a notable feature unless stenosis has occurred.

Alteration in weight

Weight loss (GU) or, sometimes, weight gain (DU) may occur

Bleeding

The bleeding may be chronic and presentation with microcytic anaemia is not uncommon, or acute presentation with haematemesis and melaena.

Investigation of the patient with suspected peptic ulcer

Gastroduodenoscopy

The investigation of choice, & if a well-trained operator is highly sensitive and specific.
In the stomach, any abnormal lesion should be multiple biopsied,
Biopsies of the antrum usually taken to exclude gastritis and a CLO test for the presence
of *H. pylori*.
In the duodenum, care must be taken to view all of the first part.
Other investigations not routinely done: Barium meal, tests for H. pylori, & baseline serum gastrin (for suspected gastrinoma).

Treatment of peptic ulceration

The vast majority of uncomplicated cases are treated **medically**. Surgical treatment of uncomplicated cases is now seldom performed.

Medical treatment

Modifications to the patient's lifestyle, as physiologic and psychologic stress play a role in the development of peptic ulcer in some patients . advice patient about cessation of cigarette smoking, & avoid NSAIDs as possible.

H2-receptor antagonists and proton pump inhibitors

H2-antagonists revolutionised the management of peptic ulceration. Most duodenal ulcers and gastric ulcers can be healed by a few weeks of treatment with these drugs.

If the patient does not respond, then proton pump inhibitors can effectively render a patient achlorhydric and all benign ulcers will heal using these drugs, the majority within 2 weeks. Symptom relief is rapid, most patients being asymptomatic within a few days.

Eradication therapy

It is the mainstay of treatment for peptic ulceration,& now routinely given to any patient has a peptic ulcer and *H. pylori* is the principal aetiological factor (not taking NSAIDs), then complete eradication of the organism will cure the disease and reinfection as an adult is uncommon.

Medications/Dose/Frequency Duration

PPI + clarithromycin 500 mg bid + amoxicillin 1000 mg bid 10–14 d

PPI + clarithromycin 500 bid + metronidazole 500 bid 10–14 d

PPI + amoxicillin 1000 mg bid, then 5 d

PPI + clarithromycin 500 mg bid + tinidazole 500 mg bid 5 d

Salvage regimens for patients who fail one of the above initial regimens:

Bismuth subsalicylate 525 mg qid + metronidazole 250 mg qid + tetracycline 500 mg qid + PPI 10–14 d

PPI + amoxicillin 1000 mg bid + levofloxacin 500 mg daily 10 d

PPI = proton pump inhibitor.

Surgical treatment of uncomplicated peptic ulceration

Surgery for uncomplicated peptic ulceration is now of **historical** interest. A description of operations used in the treatment, but still occasionally employed for the complicated ulcer.

Operations for duodenal ulceration

Duodenal ulcer surgery (rationale)

This has been achieved by diversion of the acid away from the duodenum, reducing the secretory potential of the stomach, or both (in historical sequence).

Billroth II gastrectomy

Distal gastrectomy with gastrojejunal anastomosis.

Gastrojejunostomy

Truncal vagotomy and drainage

Denervation of the antropyloroduodenal segment results in gastric stasis (in truncal vagotomy alone), so they added a drainage procedure either pyloroplasty or Gastrojejunostomy.

Highly selective vagotomy

Only the parietal cell mass of the stomach is denervated. This proved to be the most satisfactory operation for duodenal ulceration, with a low incidence of side effects and acceptable recurrence rates.

Truncal vagotomy and antrectomy

Truncal vagotomy, the antrum (the source of gastrin) is removed, and the gastric remnant is joined to the duodenum.

Operations for gastric ulcer

In gastric ulceration the diseased tissue is usually **removed**. That malignancy can then be confidently excluded. As with duodenal ulceration, such surgery is not now performed except for complications.

Billroth I gastrectomy

The distal stomach is resected including the ulcer that is usually situated on the lesser curve. The remnant is anastomsed to the duodenum.

Sequelae of peptic ulcer surgery

These sequelae principally follow from the more destructive operations that are now seldom performed.

1-Recurrent ulceration

Most recurrent ulcers will heal with potent antisecretory

Agents. As with other peptic ulcers, recurrent ulcers may present with complications.

2-Small stomach syndrome

Early satiety due to the loss of receptive relaxation.

3-Bile vomiting

It can occur after vagotomy with drainage or gastrectomy. Bile chelating agents can be tried but if ineffective, then revisional surgery may be indicated.

4-Early and late dumping

	Early dumping	Late dumping
Incidence	5–10%	5%
Relation to meals	Almost immediate	2 hrs after meal
Durations of attack	30–40 minutes	Same
Relief	Lying down	Food
Aggravated by	More food	Exercise
Precipitating factor	Food, especially carbohydrate-rich and liquids.	Same
Major symptoms	Epigastric fullness, sweating, lightheadedness, tachycardia, colic, diarrhoea	Tremor, faintness, prostration (d.t. 2nd ary hypoglycemia)
Treatment	Dietary manipulation: small, dry meals. Somatostatin analogue (octreotide). Revisional surgery	Same

5-Postvagotomy diarrhoea

This can be the most devastating symptom to afflict patients having peptic ulcer surgery.

(except in highly selective vagotomy) due to rapid gastric emptying. It is difficult to treat.

6-Malignant transformation

Gastrectomy or vagotomy and drainage are risk factors for gastric cancer, as bile reflux gastritis, intestinal metaplasia and gastric cancer are linked.

7-Nutritional consequences

They are more common after gastrectomy than after vagotomy and drainage. Weight loss, anaemia may be due to either iron or vitamin B12 deficiency.

Bone disease mainly in women, treated with dietary supplementation, calcium and vitamin D, and exercise.

8-Gallstones
This strongly associated with truncal vagotomy (bile stasis).

The complications of peptic ulceration
The common complications 1-perforation, 2-bleeding, & 3-stenosis.

Perforated peptic ulcer

NSAIDs appear to be responsible for most of these perforations. The most common site of perforation is **the anterior** aspect of the duodenum.

Clinical features

The patient, who may have **a history of peptic** ulceration, develops sudden onset severe generalised abdominal pain due to the irritant effect of gastric acid on the peritoneum. Bacterial peritonitis supervenes over a few hours, with deterioration in the patient's condition. Initially, the patient may be shocked with a tachycardia but a pyrexia is not usually observed until some hours after the event. The abdomen exhibits a **board-like** rigidity and the patient is disinclined to move because of the pain. The abdomen does not move with respiration.

Investigations
1-Erect plain chest radiograph will reveal free gas under the diaphragm in an excess of 50 % of cases.
2- CT imaging is more accurate
3-Serum amylase to distinguish between peptic ulcer perforation and pancreatitis as its levels are not usually as high as these commonly seen in acute pancreatitis.

Treatment
The initial priorities are resuscitation and analgesia (titrate the analgesic dose). Then, treatment is principally **surgical**. Laparotomy is performed, or laparoscopy if the diagnosis is uncertain. The aim is thorough peritoneal **toilet** to remove all of the fluid and food debris.
If perforation is in the duodenum it can usually be **closed** by several well-placed sutures, then place an **omental patch** over the perforation.
Gastric ulcers should be **excised** and closed, so that **malignancy** can be excluded.
All patients should be treated with systemic antibiotics, gastric antisecretory agents should be started immediately to promote healing of the residual ulcer. In patients with *Helicobacter* associated ulcers, eradication therapy is appropriate. Stomach is kept empty postoperatively by nasogastric suction.
Life-long treatment with proton pump inhibitors is a reasonable option.

Upper GIT bleeding

It is strongly associated with **NSAID** use. Incidence in west 1/1000, with a mortality rate of (5-10%).

Causes of upper gastrointestinal bleeding.

Ulcers 60%
Oesophageal 6%
Gastric 21%
Duodenal 33%
Erosions 26%
Oesophageal 13%

Gastric 9%
Duodenal 4%
Mallory–Weiss tear 4%
Oesophageal varices 4%
Tumour 0.5 %
Vascular lesions, e.g. Dieulafoy's disease 0.5 %
Others 5 %

Whatever the cause, the principles of management are identical.
First, the patient should be adequately **resuscitated** and, then investigated urgently to determine the cause of the bleeding.
For any significant gastrointestinal bleed, intravenous access should be established and, for those with severe bleeding, central venous pressure monitoring should be set up and bladder catheterisation performed. Blood should be cross-matched and the patient transfused as clinically indicated.
Sometimes, lifesaving manoeuvres have to be undertaken accordingly. For instance, in patients with known oesophageal varices and uncontrollable bleeding, a **Sengstaken– Blakemore** tube may be inserted before an endoscopy. In some patients, bleeding is secondary to a coagulopathy, which should be corrected, if possible, with fresh-frozen plasma or concentrated clotting factors.
Upper gastrointestinal endoscopy should be carried out by an experienced operator as soon as practicable after the patient has been stabilized.
Rockall score can be used in a pre-endoscopy format to stratify patients to safe early discharge and postendoscopy it can relatively accurately predict rebleeding and death.

Bleeding peptic ulcers

Recently, the population affected has become much older and the bleeding is commonly associated with the ingestion of **NSAIDs.** Diagnosis can normally be made endoscopically

A- Medical and minimally interventional treatments
Medical treatment has limited efficacy. Start with an antacid :either an H2-antagonist or a proton pump antagonist (the latter prevents rebleeding after endoscopy). Use tranexamic acid, an inhibitor of fibrinolysis.
Therapeutic endoscopy (adrenaline injection with heater probe and/or clips) can achieve haemostasis in 70 % of cases. This may be ineffective inpatients who are bleeding from large vessels and with which the majority of the mortality is associated.
Angiography with transcatheter embolisation in cases with unknown source of bleeding, or who rebleed after endoscopy.
Once these procedures failed , then proceed to surgery.

B-Surgical treatment
Criteria for surgery 1-A patient who continues to bleed, 2-Significant rebleeding, 3-Visible vessel in the ulcer base, 4-Spurting vessel, 5-Ulcer with a clot in the base, 6-A patient who has required more than 6 units of blood. Elderly and unfit patients are more likely to die as a result of bleeding than younger patients.
The aim of the operation is to stop the bleeding. The most common site of bleeding from a peptic ulcer is the duodenum (**the gastroduodenal** artery is the source of bleeding).
If the stomach is opened & the ulcer is not excised then a biopsy of the edge needs to be taken to exclude malignant transformation.
The minimum surgery that stops the bleeding is optimal. Acid can be inhibited by pharmacological means and appropriate eradication therapy will prevent ulcer recurrence.

Stress ulceration

This commonly occurs in patients with **major injury** or illness, who have undergone major surgery or who have major comorbidity. Many such patients are found in **ICU**.

The incidence has reduced , due to the prophylaxis (Acid inhibition and sucralfate)

It is better to **prevent** this condition than to try to treat it once it occurs.

Gastric erosions

Erosive gastritis has a variety of causes, especially NSAIDs.

Mallory–Weiss tear

This is a longitudinal tear at the gastro-oesophageal junction, which is induced by repetitive and strenuous vomiting, usually in alcoholics.

Occasionally, these lesions continue to bleed and require surgical treatment.

Dieulafoy's disease

Gastric arterial venous malformation.

The lesion itself is covered by normal mucosa and, when not bleeding, it may be invisible, so difficult to diagnose & treat endoscopically.

Tumours

All of the gastric tumours may present with chronic or acute upper gastrointestinal bleeding.

Portal hypertension (bleeding varices)

The management of bleeding gastric varices is very challenging.

Fortunately, most bleeding from varices is oesophageal and this is much more amenable to sclerotherapy, banding and balloon tamponade. Gastric varices may also be injected, or banned but it is more difficult.

The gastric balloon of the Sengastaken–Blakemore tube can be used to arrest the haemorrhage if it is occurring from the fundus of the stomach or gastro-oesophageal junction.

Octreotide is a somatostatin analogue that reduces portal pressure.

Glypressin can also be used.

TIPSS procedure (transjugular intrahepatic portosystemic shunt) may be useful.

Portal gastropathy

Portal gastropathy is the same disease process as Portal hypertension. The mucosa is affected by the increased portal pressure and may bleed, The treatment is as above.

Aortic enteric fistula

This diagnosis is suspected in any patient who had an aortic graft, develops unexplained haematemesis and melaena. The bleeding is not always massive. Dx : CT, Rx: surgery, with high mortality rate.

GASTRIC OUTLET OBSTRUCTION

The two common causes of gastric outlet obstruction are gastric **cancer** and pyloric stenosis secondary to **peptic** ulceration. Now, gastric outlet obstruction should be considered **malignant** until proven otherwise.

The term 'pyloric stenosis' is a misnomer, as the stenosis is seldom at the pylorus, but often at the first part of the duodenum the most common site for a peptic ulcer).

Recently, the most common cause of gastric outlet obstruction has been gastric cancer. In this circumstance the metabolic consequences may be different from those of benign pyloric stenosis because of the relative hypochlorhydria found in patients with gastric ca.

Clinical features

In benign gastric outlet obstruction there is usually a long history of peptic ulcer disease. Nowadays, the condition is becoming much less common. The vomitus is unpleasant in nature and is totally **lacking in bile** & contains foodstuff taken several days previously. Weight loss.

The patient appears unwell and dehydrated, with distended stomach and a succussion splash may be audible.

Metabolic effects

vomiting of HCl results in **hypochloraemic alkalosis**.

Initially, the urine has a low chloride and high bicarbonate content, reflecting the primary metabolic abnormality. 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 in preference. This results in the urine becoming paradoxically acidic and hypokalaemia ensues.

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

Management

Treating the patient involves **correcting the metabolic** abnormality and dealing with the mechanical problem. The patient should be rehydrated with intravenous isotonic saline (0.9 %NaCl) with potassium supplementation.

A large nasogastric, (or orogastric) tube to lavage the stomach until it is completely emptied.

Investigation of the patient with endoscopy and contrast radiology. Biopsy of the area around the pylorus is essential to exclude malignancy. The patient should also have a gastric antisecretory agent.

Severe cases are treated surgically, usually with a gastroenterostomy.

Endoscopic balloon dilatation, or a duodenal stent may be tried.

Other causes of gastric outlet obstruction

Adult pyloric stenosis

Rare, is commonly treated by pyloroplasty rather than pyloromyotomy.

Pyloric mucosal diaphragm

Rare present at middle age, treated by simple excision.

GASTRIC POLYPS

They may represent early gastric cancer. Biopsy is essential. 1- Metaplastic :the most common, associated with *H. pylori* infection and regress following eradication therapy. 2-Inflammatory polyps; common. 3-Fundic gland polyps ; associated with the use of proton pump inhibitors & in patients with FAP.

None of the above polyps has malignant potential. But adenomas have and should be removed, but they account for only 10 :% of polypoid lesions. 4- Gastric carcinoids arising from the ECL cells are seen in patients with pernicious anaemia and usually appear as small polyps.

GASTRIC CANCER

Carcinoma of the stomach is a major cause of cancer mortality worldwide. Its prognosis is poor, with cure rates (5–10) %, although better results are obtained in Japan, where the disease is common.

Early diagnosis is the key to success with this disease, & the only treatment modality able to cure the disease is resectional surgery.

Incidence

There are variations in the incidence worldwide. In the USA 10/100 000 per year. In Japan, the disease is much more common, with a 70/100 000 per year. That data make it clear that this is an **environmental** disease.

Men are more affected than women, & the incidence increases with age.

1-The incidence of distal gastric cancer is continuing to fall at 1 % per year. In contrast, there is increase in the incidence of carcinoma in the proximal stomach, (the oesophagogastric junction).

2-Carcinoma of the distal stomach and its body is most common in low socioeconomic groups, whereas, proximal gastric cancer seems to affect higher socioeconomic groups.

3-Proximal gastric cancer is not associated with *H. pylori* infection, in contrast with carcinoma of the body and distal stomach.

Aetiology

It is a ***multifactorial*** disease. 1- *H. pylori* seems to be principally associated with carcinoma of the body and distal stomach, as a result of gastritis, gastric atrophy, intestinal metaplasia, the dysplasia.

2-Pernicious anaemia and gastric atrophy.

3-Gastric polyps.

4- Peptic ulcer surgery, particularly those who have had drainage procedures such as Billroth II or pyloroplasty (4 X the risk), may be d.t. reflux gastritis & intestinal metaplasia.

5- Cigarette smoking and industrial dust ingestion.

6-Diet is important, change in the incidence of gastric cancer in Japanese families living in the USA. Excessive salt intake, spirits, deficiency of antioxidants and exposure to *N*-nitroso compounds. It is also associated with obesity and higher socioeconomic

7- Genetic factors.

Clinical features

The key to improve the outcome of gastric cancer is **early diagnosis**. So, a high index of suspicion is necessary.

Gastric antisecretory agents will improve the symptoms of gastric cancer so the disease should be excluded preferably before therapy is started.

In advanced cancer, early satiety, bloating, distension, iron deficiency anaemia and vomiting may occur. Obstruction leads to dysphagia, epigastric fullness or vomiting. With pyloric involvement the presentation may be of gastric outlet obstruction, although the alkalosis is usually mild or absent. Now, gastric outlet obstruction is more commonly associated with malignancy than benign disease. Non-metastatic effects of malignancy are seen, particularly thrombophlebitis (Trousseau's sign) and deep venous thrombosis.

Site

The proximal stomach is now the most common site for gastric cancer in the West. About **60 %** of all of the malignancies of oesophagus and stomach occur in proximity to the oesophagogastric junction. In Japan distal cancer still predominates, as it does in most of the rest of the world.

Pathology

According to **Lauren** classification, there are two forms of gastric cancer: **intestinal** gastric cancer and **diffuse** gastric cancer. Intestinal type, arises in areas of intestinal metaplasia, the tumour resembles a ca elsewhere in the GIT & forms polypoid tumours or ulcers. In contrast, diffuse gastric cancer (poor prognosis) infiltrates through the gastric wall without forming obvious mass lesions. Sometimes, a mixed form seen.

Gastric cancer can also be divided into **early** gastric cancer and **advanced** gastric cancer. Early gastric cancer is defined as cancer limited to the mucosa and submucosa with or without lymph node involvement. This type of cancer is eminently curable. Advanced gastric cancer involves the muscularis. Its macroscopic appearances have been classified by **Bormann** into **4** types.

International Union Against Cancer (UICC) staging of
gastric cancer.
T1 Tumour involves lamina propria, submucosa
T1a lamina propria
T1b 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)

Spread of carcinoma of the stomach

Distant metastases are uncommon in the absence of lymph node metastases. The diffuse type spreads via the submucosal and subserosal lymphatic plexus and it penetrates the gastric wall at an early stage.

Direct spread

The tumour penetrates the muscularis, serosa and ultimately adjacent organs such as the pancreas, colon and liver.

Lymphatic spread

This may be extensive, the tumour even appearing in the supraclavicular nodes (Troisier's sign). Unlike in most cancers, nodal involvement does not imply systemic dissemination.

Blood-borne metastases

These occur first to the liver and subsequently to other organs, including lung and bone. This is uncommon in the absence of nodal disease.

Transperitoneal spread

This is a common mode of spread once the tumour has reached the serosa of the stomach and indicates incurability. Tumours give rise to ascites. The ovaries may sometimes be the sole site of transcoelomic spread (Krukenberg's tumours). Tumour may spread via the abdominal cavity to the umbilicus (Sister Joseph's nodule).

Lymphatic drainage of the stomach

The efferent lymphatics from suprapyloric lymph nodes converge on the para-aortic nodes around the coeliac axis, whereas the efferent lymphatics from the subpyloric lymph nodes pass up to the main superior mesenteric lymph nodes. The lymphatic vessels related to the cardiac orifice of the stomach communicate freely with those of the oesophagus.

The prognosis depends on whether or not there is histological evidence of regional lymph node involvement.

Management

Surgical :

Total gastrectomy

The stomach is removed en bloc, including the tissues of the entire greater omentum and lesser omentum . GIT continuity is reconstituted by means of a oesophagojejunostomy Roux-en-Y.

The results of surgical treatment in Japan are much better than in the West.

Subtotal gastrectomy

For tumours distally placed in the stomach, it appears unnecessary to remove the whole stomach. Then anastomosis of the greater curve to the jejunum.

Palliative surgery

In patients suffering from significant obstruction or bleeding, palliative resection is appropriate. A palliative gastrectomy need not be radical and it is sufficient to remove the tumour and reconstruct the gastrointestinal tract.

Gastroenterostomy with a Roux loop, oesophagojejunostomy, palliative intubation, stenting or another form of recanalisation can be used.

Postoperative complications of gastrectomy

Radical gastrectomy is complex major surgery and has potential complications. 1-Leakage of the oesophagojejunostomy, or from a from the duodenal stump leading to paraduodenal collections, or biliary peritonitis 2-Fistula from the wound or drain site

It is common to perform a water-soluble contrast swallow at 5–7 days after the operation to determine whether the anastomosis is intact. 3- Nutritional deficiencies, 4-GIT functional & metabolic disorders.

Other treatment modalities

Radiotherapy

There are a number of radiosensitive tissues in the gastric bed, which limits the dose that can be given. Radiotherapy has a role in the palliative treatment of painful bony metastases.

Chemotherapy

Gastric cancer may respond well to combination cytotoxic chemotherapy and neoadjuvant chemotherapy improves the outcome following surgery. The best results are obtained using a combination of epirubacin, *cis*-platinum and infusional 5-FU or an oral analogue such as capecitabine.

GASTROINTESTINAL STROMAL TUMOURS (GIST**)**

GIST may arise in any part of the GIT but 50 % is found in the stomach. Previously named leiomyoma and leiomyosarcoma. These tumours are sensitive to the tyrosine kinase antagonist imatinib.

Peritoneal and liver metastases are most common but spread to lymph nodes extremely rare.
Endoscopic biopsy can be uninformative as the mucosa overlying the tumour is normal, unless the tumour has ulcerated.
Surgery is the primary mode of treatment by exision. Larger tumours may require a gastrectomy or duodenectomy, +\- imatinib chemotherapy.

GASTRIC LYMPHOMA

It is either a primary gastric lymphoma, or its involvement in a generalised lymphomatous process (more common). The incidence is increasing.
The presentation is no different from gastric cancer, the common symptoms being pain, weight loss and bleeding. Primary gastric lymphomas are B cell derived, the tumour arising from the mucosa-associated lymphoid tissue (MALT). Diagnosis is made by endoscopic biopsy. Surgery alone for the localised disease. Chemotherapy for systemic disease.
There is a role of *H. pylori*., so must be also treated.

OTHER GASTRIC CONDITIONS

Acute gastric dilatation

It is associated with pyloroduodenal disorders or postsurgery without nasogastric suction. The stomach, dilates enormously. Often the patient is also dehydrated and has electrolyte disturbances.
The treatment is nasogastric suction, with a large-bore tube, fluid replacement and treatment of the underlying condition.

Trichobezoar and phytobezoar

Trichobezoar (hair balls) unusual and are virtually exclusively found in female psychiatric patients, often young. It is caused by the pathological ingestion of hair, which remains undigested in the stomach. The hair ball can lead to ulceration and gastrointestinal bleeding, perforation or obstruction.
Phytobezoars are made of vegetable matter & found in patients with gastric stasis & follows gastric surgery.
The diagnosis by endoscopy or, a plain radiograph. Treatment consists of removal of the bezoar, which may require open surgical treatment.

Foreign bodies in the stomach

Usually these can be seen on a plain radiograph. If possible, they should be removed endoscopically but, if not, most can be left to pass normally. In general, an object which leaves the stomach will pass spontaneously.
Most adults who swallow foreign bodies have illdefined psychiatric problems
Intervention is reserved for symptomatic patients, in whom the foreign body is failing to progress.

Volvulus of the stomach

Rotation of the stomach usually occurs around the axis and between its two fixed points, i.e. the cardia and the pylorus. It can occur in the horizontal (organoaxial) or vertical (mesenteroaxial) direction but, commonly, it is the former which occurs. This condition is usually associated with a (paraoesophageal herniation).
The condition is commonly chronic, presenting with difficulty in eating. An acute presentation with ischaemia may occur. Endoscopically, it can be difficult to diagnose, so contrast radiograph is superior.

Treatment: If the problem is causing symptoms then surgical treatment is the only satisfactory approach. Via open or laparoscopic approach.

DUODENAL TUMOURS

Benign duodenal tumours

Duodenal villous adenomas occur in periampullary region. They are found in patients with FAP, & have malignant potential.

Summary box 63.7

Duodenal tumours

_ Duodenal villous adenomas are commonly found around the ampulla of Vater and are premalignant

_ Duodenal carcinoma is uncommon, but the most common site for adenocarcinoma is in the small intestine

_ Both adenoma and carcinoma occur commonly in patients with familial polyposis and screening these patients is advised

_ Pancreatic cancer is the most common cause of duodenal obstruction

Duodenal adenocarcinoma

Uncommon, but it is the most common site for adenocarcinoma of the small bowel. Most tumours originate in the periampullary region from villous adenomas. Features :anaemia , obstruction, obstructive jaundice.

Metastases are commonly to regional lymph nodes and the liver.

Curative surgical treatment will normally involve a pancreaticoduodenectomy (Whipple's procedure). Patients with FAP, which is due to a mutation in the *APC* gene on chromosome 5, are predisposed to periampullary cancer. Other duodenal malignancies include GISTs (see above) and neuroendocrine tumours.

Neuroendocrine tumours

. It is a common site for primary gastrinoma (Zollinger– Ellison syndrome). Non-functioning neuroendocrine tumour (carcinoid tumours) also occur but uncommon.

Zollinger–Ellison syndrome

A gastrin-producing endocrine tumour is often found in the duodenal loop, & in head of the pancreas. Dx: 1-S. level of gastrin 2-Pentagastrin stimulation test, 3- Localisation of the tumor mass by endoscopic US, CT, MRI. Gastrinomas may be either sporadic or associated with the autosomal dominantly inherited multiple endocrine neoplasia (MEN) type I. Some lesions are malignant with hepatic & lymphatic metastases. Treatment : surgical & PPI.

DUODENAL OBSTRUCTION

Duodenal obstruction in the adult is usually due to malignancy, and 1-cancer of the pancreas is the most common cause. Treatment is usually by gastroenterostomy but duodenal stenting is increasingly being used. Other malignancies that cause duodenal obstruction, including 2-metastases from colorectal and gastric cancer. 3-Primary duodenal cancer is less common. 4-Annular pancreas (rare). 5-attack of pancreatitis, 6-Arteriomesenteric compression.

Good Luck