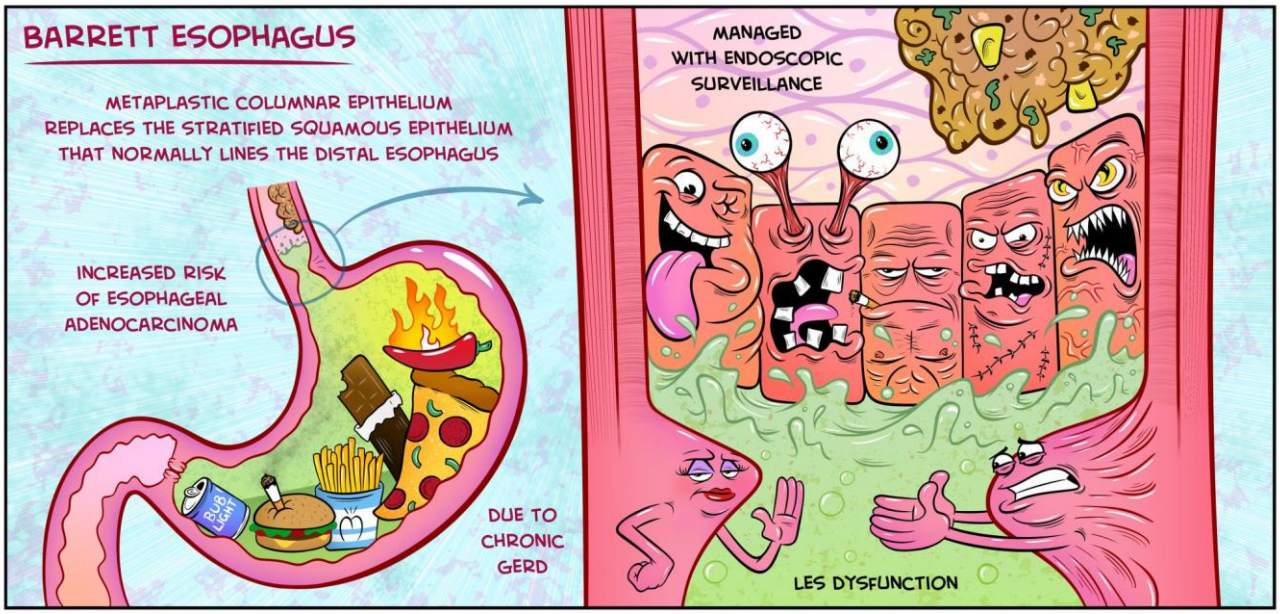


Upper Gastrointestinal Bleeding

Patients may present with the following:

- Haematemesis and/ or malaena
- Epigastric discomfort
- Sudden collapse

The extent to which these will occur will depend upon the source. Mortality is higher in patients presenting with haematemesis than malaena alone.

Oesophageal bleeding

Cause	Presenting features
Oesophagitis	Small volume of fresh blood, often streaking vomit. Malaena rare. Often ceases spontaneously. Usually history of antecedent GORD type symptoms.
Cancer	Usually small volume of blood, except as pre terminal event with erosion of major vessels. Often associated symptoms of dysphagia and constitutional symptoms such as weight loss. May be recurrent until malignancy managed.
Mallory Weiss Tear	Typically brisk small to moderate volume of bright red blood following bout of repeated vomiting. Malaena rare. Usually ceases spontaneously.
Varices	Usually large volume of fresh blood. Swallowed blood may cause malaena. Often associated with haemodynamic compromise. May stop spontaneously but re-bleeds are common until appropriately managed.

Gastric Bleeding

Cause	Presenting features
Gastric cancer	May be frank haematemesis or altered blood mixed with vomit. Usually prodromal features of dyspepsia and may have constitutional symptoms. Amount of bleeding variable but erosion of major vessel may produce considerable haemorrhage.
Dieulafoy Lesion	Often no prodromal features prior to haematemesis and malaena, but this arteriovenous malformation may produce quite considerable haemorrhage and may be difficult to detect endoscopically.
Diffuse erosive gastritis	Usually haematemesis and epigastric discomfort. Usually there is an underlying cause such as recent NSAID usage. Large volume haemorrhage may occur with considerable haemodynamic compromise.
Gastric ulcer	Small low volume bleeds more common so would tend to present as iron deficiency anaemia. Erosion into a significant vessel may produce considerable haemorrhage and haematemesis.

Duodenum

Most common cause of major haemorrhage is a posteriorly sited duodenal ulcer. However, ulcers at any site in the duodenum may present with haematemesis, malaena and epigastric discomfort. The pain of duodenal ulcer is slightly different to that of gastric ulcers and often occurs several hours after eating. Peri ampullary tumours may bleed but these are rare. In patients with previous abdominal aortic aneurysm surgery aorto-enteric fistulation remains a rare but important cause of major haemorrhage associated with high mortality.

Management

- Admission to hospital careful monitoring, cross match blood, check FBC, LFTs, U+E and Clotting (as a minimum)
- Patients with on-going bleeding and haemodynamic instability are likely to require O negative blood pending cross matched blood
- Early control of airway is vital (e.g. Drowsy patient with liver failure)
- Patients with suspected varices should receive terlipressin prior to endoscopy
- Ideally all patients admitted with upper gastrointestinal haemorrhage should undergo Upper GI endoscopy within 24 hours of admission. In those who are unstable this should occur immediately after resuscitation or in tandem with it. The endoscopy department is a potentially dangerous place for unstable patients and it may be safer to perform the endoscopy in theatre with an anaesthetist present.
- Varices should be banded or subjected to sclerotherapy. If this is not possible owing to active bleeding, then a Sengaksten- Blakemore tube (or Minnesota tube) should be inserted. This should be done with care; gastric balloon should be inflated first and oesophageal balloon second. Remember the balloon will need deflating after 12 hours (ideally sooner) to prevent necrosis. Portal pressure should be lowered by combination of medical therapy +/- TIPSS.
- Patients with erosive oesophagitis / gastritis should receive a proton pump inhibitor.
- Mallory Weiss tears will typically resolve spontaneously
- Identifiable bleeding points should receive combination therapy of **injection of adrenaline** and either a thermal or mechanical treatment. All who have received intervention should receive a continuous infusion of a proton pump inhibitor (IV omeprazole for 72 hours) to reduce the re-bleeding rate.
- Patients with diffuse erosive gastritis who cannot be managed endoscopically and continue to bleed may require gastrectomy
- Bleeding ulcers that cannot be controlled endoscopically may require **laparotomy and ulcer underruning**

Indications for surgery

- Patients > 60 years
- Continued bleeding despite endoscopic intervention
- Recurrent bleeding
- Known cardiovascular disease with poor response to hypotension

Surgery

Duodenal ulcer

Laparotomy, duodenotomy and under running of the ulcer. If bleeding is brisk then the ulcer is almost always posteriorly sited and will have invaded the gastroduodenal artery. Large bites using 0 Vicryl are taken above and below the ulcer base to occlude the vessel. The duodenotomy should be longitudinal but closed transversely to avoid stenosis.

For gastric ulcer

Under-running of the bleeding site
Partial gastrectomy-antral ulcer
Partial gastrectomy or under running the ulcer- lesser curve ulcer (involving left gastric artery)
Total gastrectomy if bleeding persists

Summary of Acute Upper GI bleeding recommendations:

The need for admission and timing of endoscopic intervention may be predicted by using the Blatchford score. This considers a patients Hb, serum urea, pulse rate and blood pressure. Those patients with a score of 0 are low risk, all others are considered high risk and require admission and endoscopy.

The requirement for pre endoscopic proton pump inhibition is contentious. In the UK the National Institute of Clinical Excellence guidelines suggest the pre endoscopic PPI therapy is unnecessary. Whilst it is accepted that such treatment has no impact on mortality or morbidity a Cochrane review of this practice in 2007 did suggest that it reduced the stigmata of recent haemorrhage at endoscopy. As a result, many will still administer PPI to patients prior to endoscopic intervention.

Following endoscopy, it is important to calculate the Rockall score for patients to determine their risk of rebleeding and mortality. A score of 3 or less is associated with a rebleeding rate of 4% and a very low risk of mortality and identifies a group of patients suitable for early discharge.

Rockall Score

A patients should have their Rockall score calculated following endoscopy for upper GI haemorrhage

Mnemonic for Rockall score

ABCDE

A: Age
B: Blood pressure drop (Shock)
C: Co-morbidity
D: Diagnosis
E: Evidence of bleeding

Applies to upper gastrointestinal bleeding

Variable	Score 0	Score 1	Score 2	Score 3
Age	<60	60-79	>80	
Shock	None	Pulse >100	Hypotension (systolic <100mmHg)	
Co-Morbidity	Nil or minimal		Major organ disease e.g. IHD, CCF	Renal or liver failure, metastatic cancer
Diagnosis	Mallory-Weiss	All	GI Cancer	
Evidence of Bleeding	None		Clot, Blood, spurting vessel	

Score <3 = Good prognosis (mortality approx. 2%)
Score >8= High mortality (Mortality approx. 40%)

Dysphagia

Extrinsic	• Mediastinal masses • Cervical spondylosis
Oesophageal wall	• Achalasia • Diffuse oesophageal spasm • Hypertensive lower oesophageal sphincter
Intrinsic	• Tumours • Strictures • Oesophageal web • Schatzki rings
Neurological	• CVA • Parkinson's disease • Multiple Sclerosis • Brainstem pathology • Myasthenia Gravis

Investigations

- All patients require an upper GI endoscopy unless there are compelling reasons for this not to be performed. Motility disorders may be best appreciated by undertaking fluoroscopic swallowing studies.
- A full blood count should be performed.
- Ambulatory oesophageal pH and manometry studies will be required to evaluate conditions such as achalasia and patients with GORD being considered for fundoplication surgery.

Bariatric Surgery

Obesity is a major health problem in the Western world. Surgical solutions to the problem have evolved dramatically over the past few years. Randomised controlled trials have shown that dramatic weight loss can be achieved following surgical interventions compared with standard medical therapy. The weight loss process is also more durable following surgery than with non-surgical interventions.

Case selection

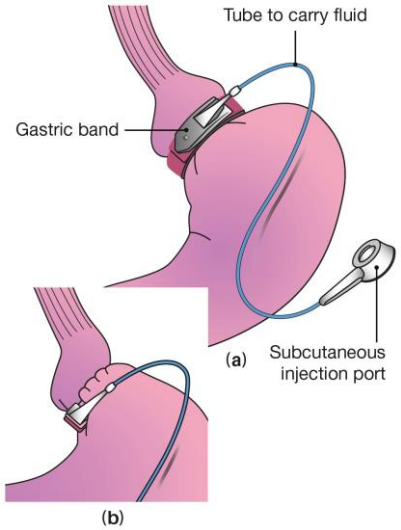
BMI >/= 40 kg/m^2 or between 35-40 kg/m^2 and other significant disease (for example, type 2 diabetes, hypertension) that could be improved with weight loss.

Pre-requisites to surgery (NICE UK Guidelines)

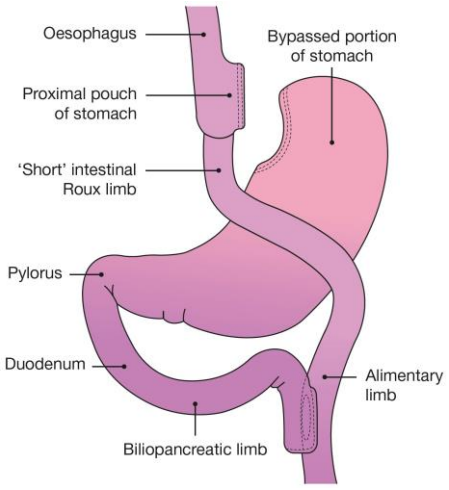
- All non-surgical measures have failed to achieve or maintain adequate clinically beneficial weight loss for at least 6 months.
- Will receive intensive specialist management
- They are generally fit for anaesthesia and surgery
- They commit to the need for long-term follow-up
- First-line option for adults with a BMI > 40 kg/m^2 in whom surgical intervention is considered appropriate; consider orlistat if there is a long waiting list.

Surgical options

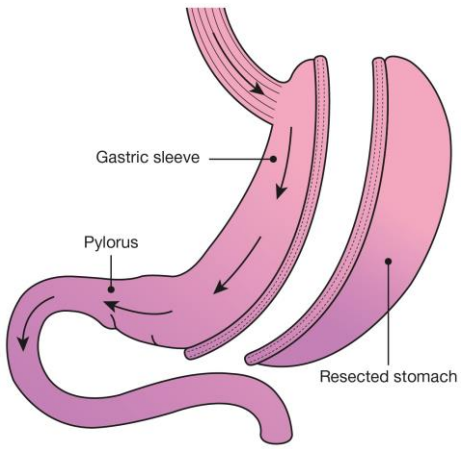
Adjustable gastric band	• Laparoscopic placement of adjustable band around proximal stomach. • Contains an adjustable filling port • Effective method for lifestyle control • Reversible • Takes longer to achieve target weight • Complications such as band erosion (rare), slippage or loss of efficacy may require re-intervention
Gastric bypass	• Combines changes to reservoir size with malabsorptive procedure for more enduring weight loss. • Technically more challenging • Risks related to anastomoses (2% leak rate) • Irreversible • Up to 50% may become B12 deficient
Sleeve gastrectomy	• Resection of stomach using stapling devices • Less popular now as initial promising results not sustained



Adjustable gastric band. Gastric band surgery showing (a) a small 'virtual' pouch of stomach below the gastro-



Gastric bypass showing a short vertical lesser curve-based gastric pouch with Roux-en-Y jejunojejunostomy reconstruction.



Sleeve gastrectomy.

Gastric Cancer

Overview

There are 700,000 new cases of gastric cancer worldwide each year. It is most common in Japan and less common in western countries. It is more common in men and incidence rises with increasing age. The exact cause of many sporadic cancer is not known, however, familial cases do occur in HNPCC families. In addition, smoking and smoked or preserved foods increase the risk. Japanese migrants retain their increased risk (decreased in subsequent generations). The distribution of the disease in western countries is changing towards a more proximal location (perhaps due to rising obesity).

Pathology

There is some evidence of support a stepwise progression of the disease through intestinal metaplasia progressing to atrophic gastritis and subsequent dysplasia, through to cancer. The favoured staging system is TNM. The risk of lymph node involvement is related to size and depth of invasion; early cancers confined to submucosa have a 20% incidence of lymph node metastasis. Tumours of the gastro-oesophageal junction are classified as below:

Type 1	True oesophageal cancers and may be associated with Barrett's oesophagus.
Type 2	Carcinoma of the cardia, arising from cardiac type epithelium or short segments with intestinal metaplasia at the oesophagogastric junction.
Type 3	Sub cardial cancers that spread across the junction. Involve similar nodal stations to gastric cancer.

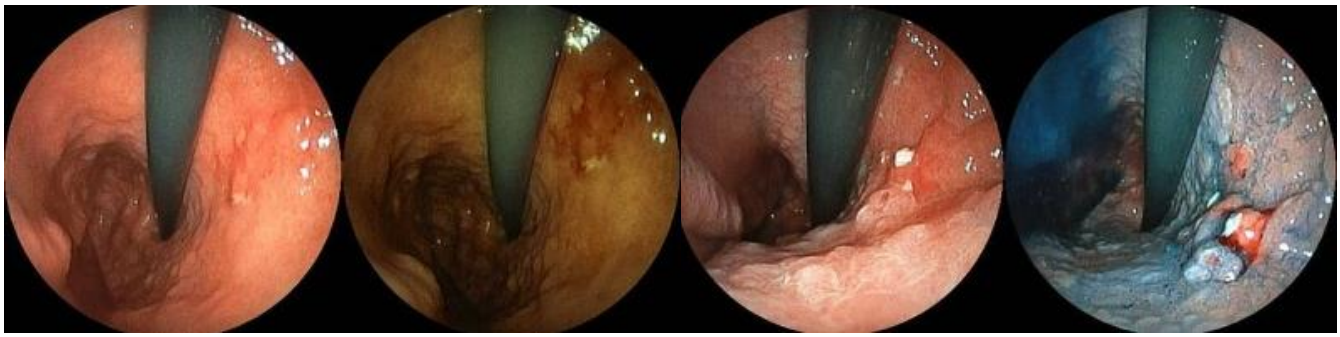
Groups for close endoscopic monitoring

- Intestinal metaplasia of columnar type
- Atrophic gastritis
- Low to medium grade dysplasia
- Patients who have previously undergone resections for benign peptic ulcer disease (except highly selective vagotomy).

Referral to endoscopy

Patients of any age with dyspepsia and any of the following	Patients without dyspepsia	Worsening dyspepsia
Chronic gastrointestinal bleeding	Dysphagia	Barretts oesophagus
Dysphagia	Unexplained abdominal pain or weight loss	Intestinal metaplasia
Weight loss	Vomiting	Dysplasia
Iron deficiency anaemia	Upper abdominal mass	Atrophic gastritis
Upper abdominal mass	Jaundice	Patient aged over 55 years with unexplained or persistent dyspepsia

Upper GI endoscopy performed for dyspepsia. The addition of dye spraying (as shown in the bottom right) may facilitate identification of smaller tumours



Staging

- CT scanning of the chest abdomen and pelvis is the routine first line staging investigation in most centres.
- Laparoscopy to identify occult peritoneal disease
- PET CT (particularly for junctional tumours)

Treatment

- **Proximally sited** disease greater than **5-10cm from the OG junction** may be treated by **subtotal gastrectomy**
- **Total gastrectomy if tumour is <5cm from OG junction**
- For type 2 junctional tumours (extending into oesophagus) oesophagogastrectomy is usual
- Endoscopic sub mucosal resection may play a role in early gastric cancer confined to the mucosa and perhaps the sub mucosa (this is debated)
- Lymphadenectomy should be performed. A D2 lymphadenectomy is widely advocated by the Japanese, the survival advantages of extended lymphadenectomy have been debated. However, the overall recommendation is that a D2 nodal dissection be undertaken.
- Most patients will receive chemotherapy either pre or post operatively.

Prognosis UK Data

Disease extent	Percentage 5 year survival
All RO resections	54%
Early gastric cancer	91%
Stage 1	87%
Stage 2	65%
Stage 3	18%

Operative procedure

Total Gastrectomy , lymphadenectomy and Roux en Y anastomosis

General anaesthesia
Prophylactic intravenous antibiotics
Incision: Rooftop.
Perform a thorough laparotomy to identify any occult disease.
Mobilise the left lobe of the liver off the diaphragm and place a large pack over it. Insert a large self-retaining retractor e.g. omnitract or Balfour (take time with this, the set up should be perfect). Pack the small bowel away.
Begin by mobilising the omentum off the transverse colon.
Proceed to detach the short gastric vessels.
Mobilise the pylorus and divide it at least 2cm distally using a linear cutter stapling device.
Continue the dissection into the lesser sac taking the lesser omentum and left gastric artery flush at its origin.
The lymph nodes should be removed en bloc with the specimen where possible.
Place 2 stay sutures either side of the distal oesophagus. Ask the anaesthetist to pull back on the nasogastric tube. Divide the distal oesophagus and remove the stomach.
The oesphago jejunal anastomosis should be constructed. Identify the DJ flexure and bring a loop of jejunum up to the oesophagus (to check it will reach). Divide the jejunum at this point. Bring the divided jejunum either retrocolic or antecolic to the oesophagus. Anastamose the oesophagus to the jejunum, using either interrupted 3/0 vicryl or a stapling device.
Then create the remainder of the Roux en Y reconstruction distally.
Place a jejunostomy feeding tube.
Wash out the abdomen and insert drains (usually the anastomosis and duodenal stump). Help the anaesthetist insert the nasogastric tube (carefully!)
Close the abdomen and skin.
Enteral feeding may commence on the first post-operative day. However, most surgeons will leave patients on free NG drainage for several days and keep them nil by mouth.

Gastric Emptying

- The stomach serves both a mechanical and immunological function. Solid and liquid are retained in the stomach during which time repeated peristaltic activity against a closed pyloric sphincter will cause fragmentation of food bolus material. Contact with gastric acid will help to neutralise any pathogens present.
- The amount of time material spends in the stomach is related to its composition and volume. For example, a glass of water will empty more quickly than a large meal. The presence of amino acids and fat will all serve to delay gastric emptying.

Controlling factors

Neuronal stimulation of the stomach is mediated via the vagus and the parasympathetic nervous system will tend to favor an increase in gastric motility. It is for this reason that individuals who have undergone truncal vagotomy will tend to routinely require either a pyloroplasty or gastro-enterostomy as they would otherwise have delayed gastric emptying.

The following hormonal factors are all involved:

Delay emptying	Increase emptying
Gastric inhibitory peptide	Gastrin
Cholecystokinin	
Enteroglucagon	

Diseases affecting gastric emptying

All diseases that affect gastric emptying may result in bacterial overgrowth, retained food and eventually the formation of bezoars that may occlude the pylorus and make gastric emptying even worse. Fermentation of food may cause dyspepsia, reflux and foul smelling belches of gas.

Iatrogenic

Gastric surgery can have profound effects on gastric emptying. As stated above any procedure that disrupts the vagus can cause delayed emptying. Whilst this is particularly true of vagotomy, this operation is now rarely performed. Surgeons are divided on the importance of vagal disruption that occurs during an oesophagectomy, some will routinely perform a pyloroplasty and others will not.

When a distal gastrectomy is performed, the type of anastomosis performed will impact on emptying. When a gastro-enterostomy is constructed, a posterior, retrocolic gastroenterostomy will empty better than an anterior one.

Diabetic gastroparesis

This is predominantly due to neuropathy affecting the vagus nerve. The stomach empties poorly and patients may have episodes of repeated and protracted vomiting. Diagnosis is made by upper GI endoscopy and contrast studies. In some cases, a radio nucleotide scan is needed to demonstrate the abnormality more clearly. In treating these conditions, drugs such as metoclopramide will be less effective as they exert their effect via the vagus nerve. One of the few prokinetic drugs that do not work in this way is the antibiotic **erythromycin**.

Malignancies

Obviously a distal gastric cancer may obstruct the pylorus and delay emptying. In addition, malignancies of the pancreas may cause extrinsic compression of the duodenum and delay emptying. Treatment in these cases is by gastric decompression using a wide bore nasogastric tube and insertion of a stent or, if that is not possible, by a surgical gastroenterostomy. As a general rule gastroenterostomies constructed for bypass of malignancy are usually placed on the anterior wall of the stomach (in spite of the fact that they empty less well). A Roux en Y bypass may also be undertaken, but the increased number of anastomoses for this, in malignant disease that is being palliated, is probably not justified.

Congenital Hypertrophic Pyloric Stenosis

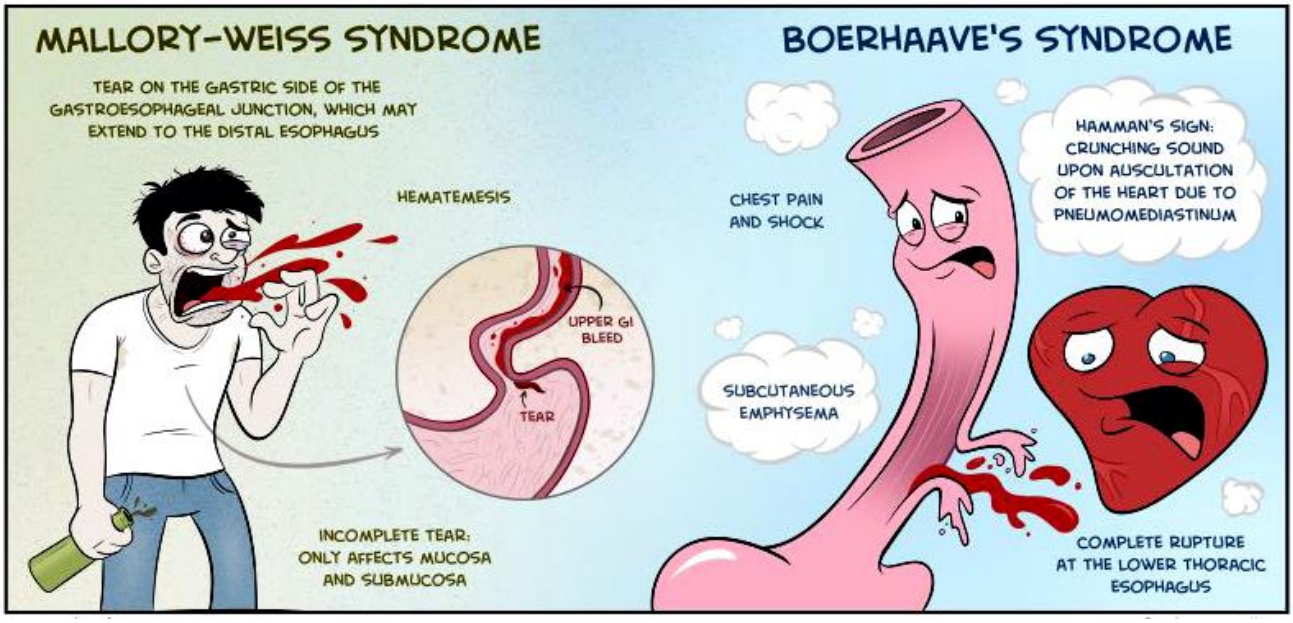
This is typically a disease of infancy. Most babies will present around 6 weeks of age with projectile non bile stained vomiting. It has an incidence of 2.4 per 1000 live births and is more common in males. Diagnosis is usually made by careful history and examination and a mass may be palpable in the epigastrium (often cited seldom felt!). The most important diagnostic test is an ultrasound that usually demonstrates the hypertrophied pylorus. Blood tests may reveal a hypochloraemic metabolic alkalosis if the vomiting is long standing. Once the diagnosis is made the infant is resuscitated and a pyloromyotomy is performed (either open or laparoscopically). Once treated there are no long term sequelae.

Oesophageal Disease

Disorder	Features
Mallory-Weiss Tear	Usually history of antecedent vomiting. This is then followed by the vomiting of a small amount of blood. There is usually little in the way of systemic disturbance or prior symptoms.
Hiatus hernia of gastric cardia	Often longstanding history of dyspepsia, patients are often overweight. Uncomplicated hiatus hernias should not be associated with dysphagia or haematemesis.
Oesophageal rupture	Complete disruption of the oesophageal wall in absence of pre-existing pathology. Left postero-lateral oesophageal is commonest site (2-3cm from OG junction). Suspect in patients with severe chest pain without cardiac diagnosis and signs suggestive of pneumonia without convincing history, where there is history of vomiting. Erect CXR shows infiltrate or effusion in 90% of cases.
Squamous cell carcinoma of the oesophagus	History of progressive dysphagia. Often signs of weight loss. Usually **little or no history** of previous GORD type symptoms. ***(↑ risk é achalasia)***
Adenocarcinoma of the oesophagus	Progressive dysphagia, may have previous symptoms of **GORD** or **Barrett's** oesophagus.
Peptic stricture	Longer history of dysphagia, often not progressive. Usually symptoms of GORD. Often lack systemic features seen with malignancy
Dysmotility disorder	May have dysphagia that is episodic and non-progressive. Retrosternal pain may accompany the episodes.

Diagnosis

Most of the differential diagnoses listed above can be accurately categorised by upper GI endoscopy (usually most patients). Where this fails to demonstrate a mechanical stricture the use of pH and manometry studies together with radiological contrast swallows will facilitate the diagnosis.

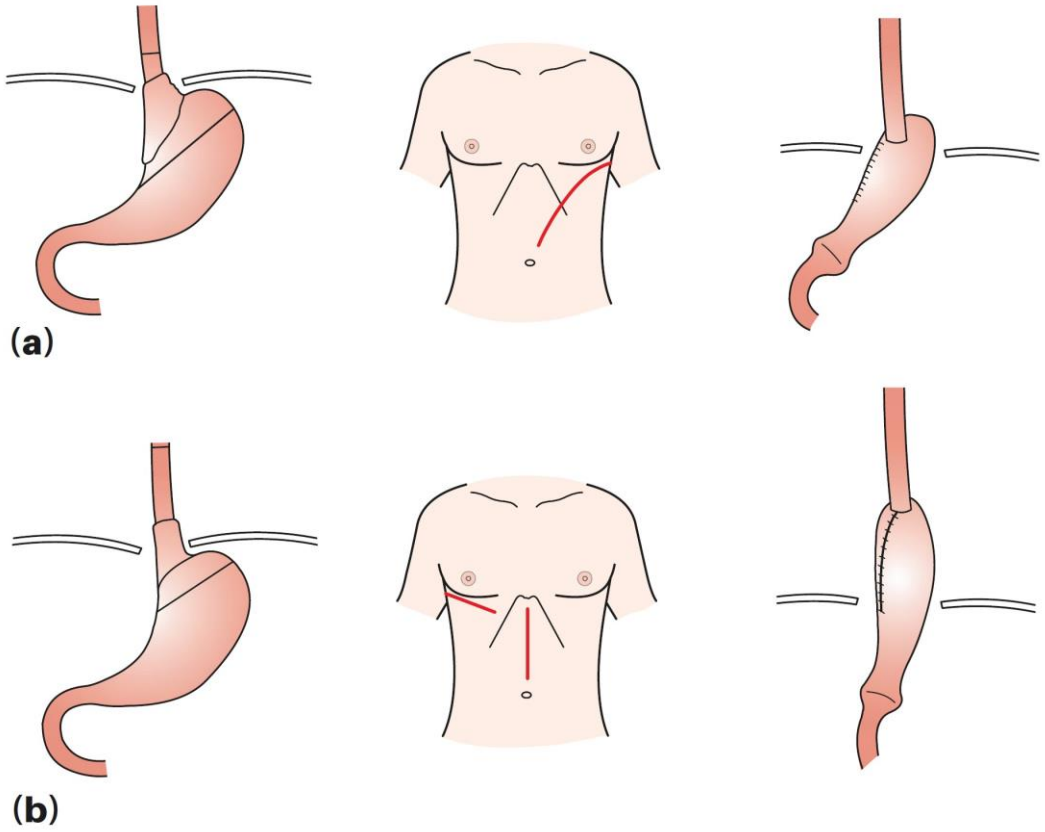


Oesophageal Cancer - Treatment

Treatments for SCC's and adenocarcinomas of the oesophagus differ. This is primarily due to the positive outcomes that are observed when localised SCC's (particularly of the **proximal oesophagus are treated with radical Chemoradiotherapy**, obviating the need for surgery).
Only those patients whose staging investigations are negative for metastatic disease should be considered for surgery.

Surgical options

Endoscopic mucosal resection	Treatment for early **localised** adenocarcinoma of the **distal** oesophagus. Survival mirrors that of surgical resection for Tis and **T1** disease
Transhiatal oeosphagectomy	Most commonly used for junctional (type II) tumours where limited thoracic oesophageal resection is required. Less morbidity than two field oesophagectomy
Ivor Lewis oesophagectomy	Two stage approach for **middle and distal tumours**. Very commonly performed, intrathoracic anastomosis will result in mediastinitis in event of anastomotic leak. Lower incidence of recurrent laryngeal nerve injury
McKeown oesophagectomy	Three field approach, may be useful for proximal tumours. Anastomotic leakage is less serious. Higher incidence of recurrent laryngeal nerve injury



The two usual approaches for surgery of the oesophagus

(a) the thoracoabdominal, which opens the abdominal and thoracic cavities together

(b) the two-stage Ivor Lewis approach, in which the abdomen is opened first, closed and then the thoracotomy is performed.

In the McKeown operation, a third incision in the neck is made to complete the cervical anastomosis.

Neoadjuvant and adjuvant treatment

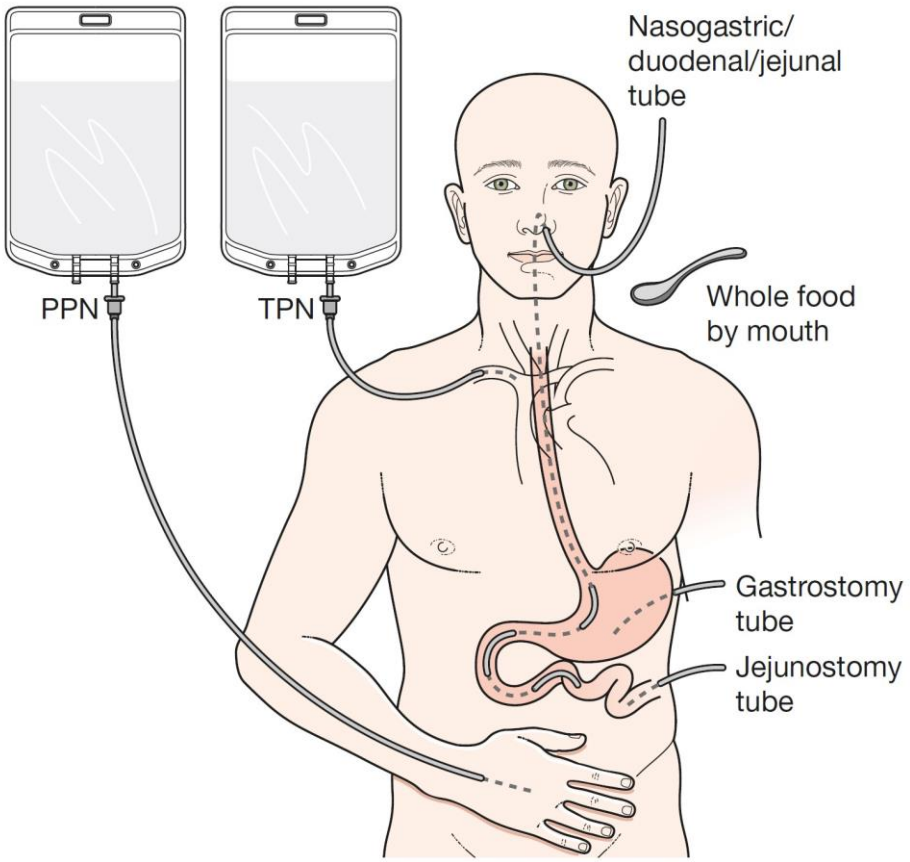
- Neoadjuvant radiotherapy alone prior to resection confers little benefit and is not routinely performed
- Preoperative chemotherapy is associated with a survival advantage (OE02 trial)
- Peri operative (pre and post-operative) chemotherapy confers a survival advantage in junctional tumours
- Post-operative chemotherapy is not generally recommended following oesophageal resections outside clinical trials

Palliation strategies

- Combination chemotherapy improves quality of life and survival in non-operable disease
- Trastuzumab may improve survival in patients with HER 2 positive tumours
- Oesophageal intubation with self-expanding metal stents is the treatment of choice in patients with occluding tumours >2cm from the cricopharyngeus
- Covered metal stents are useful in cases of malignant fistulas
- Laser therapy and argon plasma coagulation may be useful as therapies for tumour overgrowth and bleeding
- Photodynamic therapy and ethanol injections confer little benefit and should not be routinely used

Nutrition Options in Surgical Patients

Oral intake	• Easiest option • May be supplemented by calorie rich dietary supplements • May contra indicated following certain procedures
Naso gastric feeding (NGT)	• Usually administered via fine bore naso gastric feeding tube • Complications relate to aspiration of feed or misplaced tube • May be safe to use in patients with impaired swallow • Often contra indicated following head injury due to risks associated with tube insertion
Naso jejunal feeding	• Avoids problems of feed pooling in stomach (and risk of aspiration) • Insertion of feeding tube more technically complicated (easiest if done intra operatively) • Safe to use following oesophagogastric surgery
Feeding jejunostomy	• Surgically sited feeding tube • May be used for long term feeding • Low risk of aspiration and thus safe for long term feeding following upper GI surgery • Main risks are those of tube displacement and peritubal leakage immediately following insertion, which carries a risk of peritonitis
Percutaneous endoscopic gastrostomy (PEG)	• Combined endoscopic and percutaneous tube insertion • May not be technically possible in those patients who cannot undergo successful endoscopy • Risks include aspiration and leakage at the insertion site
Total parenteral nutrition (TPN)	• The definitive option in those patients in whom enteral feeding is contra indicated • Individualised prescribing and monitoring needed • Should be administered via a central vein as it is strongly phlebitic • Long term use is associated with fatty liver and deranged LFT's



Techniques used for adjuvant nutritional support. PPN, partial parenteral nutrition; TPN, total parenteral nutrition.