Peptic ulcer

Peptic **ulcers** are **open sores** that develop on the inside **lining** of your **stomach and the upper portion GIT**, of your **small intestine**. The most common **symptom** of a peptic ulcer is **stomach pain**.

Peptic ulcers include:

- **Gastric ulcers** that occur on the inside of the stomach
- **Duodenal ulcers** that occur on the inside of the upper portion of your small intestine (duodenum)

The most common causes of peptic ulcers are **infection** with the bacterium **Helicobacter pylori (H. pylori)** and **long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs)** such as ibuprofen (Advil, Motrin IB, others) and **naproxen sodium** (Aleve). **Stress** and **spicy foods** do not cause peptic ulcers. However, these are aggregative factors they can make your symptoms **worse**.

Symptoms

1) Burning stomach pain
2) Feeling of fullness, bloating or belching
3) Intolerance to fatty foods
4) Heartburn
5) Nausea

The **most common peptic ulcer symptom** is **burning stomach pain**. **Stomach acid** makes the **pain worse**, as does having **an empty stomach**. The **pain** can often **be relieved** by eating certain foods that buffer stomach acid or by taking an acid-reducing medication, but then it may come back. The **pain** may be worse between meals and at night.

Many people with peptic ulcers don't even have symptoms.

Less often, ulcers may cause severe signs or symptoms such as:

1) Vomiting or vomiting blood — which may appear red or black
2) Dark blood in stools, or stools that are black or tarry
3) Trouble breathing
4) Feeling faint
5) Nausea or vomiting
6) Unexplained weight loss
7) Appetite changes

Causes

Peptic ulcers occur when **acid** in the digestive tract **eats** away at **the inner surface of the stomach or small intestine**. The **acid** can create a **painful open sore that may bleed**.

Your digestive tract is coated with a **mucous layer** that **normally protects against acid**. But **if** the amount of acid is increased **or** the amount of mucus is decreased, you could develop an **ulcer**.

Common causes include:

1) **A bacterium.** Helicobacter pylori bacteria commonly live in the mucous layer that covers and protects tissues that line the stomach and small intestine. Often, the H. pylori bacterium causes no problems, but it can cause inflammation of the stomach's inner layer, **producing an ulcer**.

   It's not clear how H. pylori infection spreads. It may be transmitted from **person to person** by close contact, such as kissing. People may also contract H. pylori **through food and water**.

2) **Regular use of certain pain relievers.** Taking aspirin, as well as certain over-the-counter and prescription pain medications called non-steroidal anti-inflammatory drugs (NSAIDs), can irritate or inflame the **lining** of your **stomach** and **small intestine**. These medications include ibuprofen (Advil, Motrin IB, others), naproxen sodium (Aleve, Anaprox DS, others), ketoprofen and others. They do not include acetaminophen (Tylenol, others).

3) **Other medications.** Taking certain other medications along with NSAIDs, such as **steroids**, **anticoagulants**, **low-dose aspirin**, selective **serotonin reuptake inhibitors (SSRIs), alendronate** (Fosamax) and **risedronate** (Actonel), can greatly increase the chance of developing **ulcers**.

Risk factors

In addition to having risks related to taking NSAIDs, you may have an increased risk of peptic ulcers if you:

1) **Smoke.** Smoking may **increase** the risk of peptic ulcers in people who are infected with H. pylori.
2) **Drink alcohol.** Alcohol can **irritate** and **erode** the mucous lining of your stomach, and it increases the amount of stomach acid that's produced.
3) **Have untreated stress.**
4) **Eat spicy foods.**

Alone, these factors do not cause ulcers, but they can make ulcers worse and more difficult to heal.

Complications

Left untreated, peptic ulcers can result in:

1) **Internal bleeding.** Bleeding can occur as slow blood loss that leads to anemia or as severe blood loss that may require hospitalization or a blood transfusion. Severe blood loss may cause black or bloody vomit or black or bloody stools.
2) **A hole (perforation) in your stomach wall.** Peptic ulcers can eat a hole through (perforate) the wall of your stomach or small intestine, putting you at risk of serious infection of your abdominal cavity (peritonitis).
3) **Obstruction.** Peptic ulcers can block passage of food through the digestive tract, causing you to become full easily, to vomit and to lose weight either through swelling from inflammation or through scarring.
4) **Gastric cancer.** Studies have shown that people infected with H. pylori have an increased risk of gastric cancer.

Perforation peritonitis is a frequently encountered **surgical emergency** in tropical countries like India, most commonly affecting young men in their prime of life. Despite advances in *surgical techniques, *antimicrobial therapy and *intensive care support, **management of peritonitis** continues to be **highly demanding**, **difficult** and **complex**.

A majority of patients present **late**, with **septicemia**, **thus increasing** the **incidence of *morbidity and *mortality** thereby **complicating** the **task** of anesthesiologist in the **perioperative period**.

Perforation peritonitis is a common surgical emergency. **Anesthesia** in patients with perforation peritonitis can be challenging. ***Delayed presentations**, ***old age**, ***hemodynamic instability**, ***presence of sepsis** and ***organ dysfunction** are some of the **predictors** of **poor outcome** in such patients.

Pre-operative optimization can **reduce** ***intraoperative and *post-operative morbidity and mortality,** but surgery **should not** be unnecessarily **delayed**. **Intensive care** in critical care settings may be essential.

Prevention

You may reduce your risk of peptic ulcer if you follow the same strategies recommended as home remedies to treat ulcers. It also may be helpful to:

1) **Protect yourself from infections.** It's not clear just how H. pylori spreads, but there's some evidence that it could be transmitted from person to person or through food and water.
   You can take steps to protect yourself from infections, such as H. pylori, by frequently washing your hands with soap and water and by eating foods that have been cooked completely.
2) **Use caution with pain relievers.** If you regularly use pain relievers that increase your risk of peptic ulcer, take steps to reduce your risk of stomach problems. For instance, **take your medication with meals**.
   Work with your doctor **to find the lowest dose possible that still gives you pain relief**.
3) **Avoid drinking alcohol when taking your medication,** since the two can combine to increase your risk of stomach upset.

If you need an NSAID, you may need to also **take additional medications such as an antacid, a proton pump inhibitor, an acid blocker or cytoprotective agent**. A class of NSAIDs called **COX-2 inhibitors** may be less likely to cause peptic ulcers, but may increase the risk of heart attack.

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The stomach

The stomach is an organ of the **digestive system**, **located** in the abdomen just below the ribs and on the left.

Swallowed food is **squeezed** down the oesophagus and **pushed** through a **sphincter** (small muscle ring) into the stomach, where it is mixed with "powerful gastric juices" containing **enzymes** and **hydrochloric acid**. The stomach is a **muscular bag**, so it can **churn the food** خض الطعام and **break it down mechanically** as well as **chemically**.

Once the food is the consistency of **smooth paste**, it **is squeezed through a second sphincter** into the **first part of the small intestine (duodenum)**. The lining of the stomach – the mucosa or gastric epithelium – is **layered** with multiple **folds**. Ulcers occur in this lining.

Causes of stomach ulcers

A **stomach ulcer** can be caused by a variety of **factors**, including:

1) ***Helicobacter pylori*** – bacteria is thought to be responsible for around **60**% of **stomach** ulcers and at least 90% of duodenal ulcers.
2) **Certain medications** – which include aspirin or clopidogrel (Clopidogrel is an antiplatelet medicine.), taken regularly to help prevent **heart attack** or **stroke**, and drugs for **arthritis**. Anti-inflammatory medications (NSAIDS) are thought to cause around two fifths of stomach ulcers.
3) **Cancer** – **stomach cancer** can present as an ulcer, particularly in older people.

Helicobacter pylori

The *Helicobacter pylori* bacterium (*H. pylori*) is the main cause of peptic ulcers. The discovery of this micro-organism in 1983 **revolutionized** many aspects of gastroenterology, including the treatment of stomach ulcers.

H. pylori **infection** is unusual in young Australians and occurs in **15 to 20%** of Australians **aged over 25**.

The bacteria reside on the **surface cells of the stomach under a layer of mucus**. They **produce** irritation by invading the surface cells, which triggers the cells to **produce chemicals (cytokines)** that **promote inflammation**.

H. pylori **directly causes** one-third of stomach ulcers and is a contributing factor in around three-fifths of cases. Other **disorders** caused by this infection include inflammation of the stomach (**gastritis**) and dyspepsia (indigestion).

Transmission may be caused by **sharing** food or utensils, coming into **contact** with infected vomit, and sharing water (such as well water).

H. pylori is the main environmental cause of stomach cancer.

Ulcer bleeding

Ulcer bleeding is a **serious complication** of ulcer disease and is particularly **deadly** in the elderly or those with multiple medical problems.

Bleeding from stomach ulcers is **more common** in people treated with **blood thinning agents**, such as warfarin, aspirin or clopidogrel (**Plavix**) and **those people should** also consider using regular anti-ulcer medication to prevent this complication.

Perforated ulcer

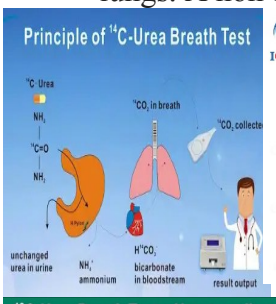
A **severe**, **untreated** ulcer **can** sometimes **burn** through the wall of the stomach, **allowing** digestive juices and food to leak into the abdominal cavity. This **medical emergency** is known as a **perforated ulcer**.

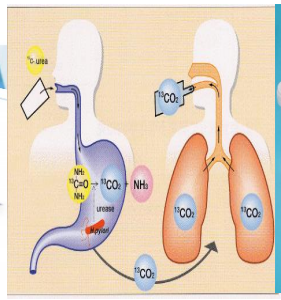
Treatment generally requires immediate surgery.

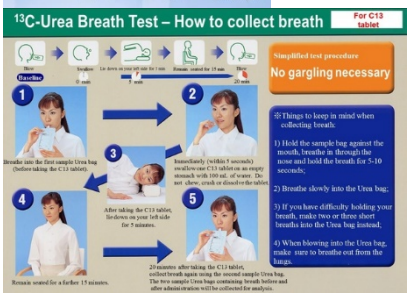
Diagnosis of a stomach ulcer

Diagnosing a stomach ulcer is done using a range of **methods**, including:

- **Endoscopy** – a thin flexible tube is threaded down the oesophagus into the stomach under light **anaesthesia**. The endoscope is fitted with a video capture device and highly detailed images of the stomach lining can be obtained. If a gastric ulcer has been found, the endoscopy must be **repeated** after treatment to ***ensure healing** and ***exclude the possibility of cancer.**
- **Biopsy** – a small tissue sample is taken during an endoscopy and tested in a laboratory. This biopsy should always be done if a gastric ulcer is found.
- **C14 breath test** – checks for the presence of *H. pylori*. The bacteria convert urea into carbon dioxide. The test involves swallowing an amount of radioactive carbon (C14) and testing the air exhaled from the lungs. A non-radioactive test can be used for children and pregnant women.







Treatment for a stomach ulcer

Special **diets** are now known to have **very little impact** on the **prevention or treatment of stomach ulcers**.

Treatment options can include:

1) **Medication** – including antibiotics, to **destroy** the *H. pylori* **colony**, and **drugs** to help **speed the healing** process. Different drugs need to be used in **combination**; some of the side effects can include **diarrhea** and **rashes**. Resistance to some of these antibiotics is becoming more common, however **80% of treatment courses are successful.**
    a. **Subsequent breath tests** – used to make sure the *H. pylori* infection has been **treated successfully**.
2) **Changes to existing medication** – the doses of arthritis medication, aspirin or other anti-inflammatory medication can be altered slightly to reduce their contributing effects on the stomach ulcer.
3) **Reducing acid** – tablets are available to **reduce** the acid content in the gastric juices.

4) **Lifestyle modifications** – including **quitting cigarettes**, since smoking reduces the natural defences in the stomach and impairs the healing process.

Management of Acute Upper Gastrointestinal Haemorrhage (UGIH)

Emergency upper gastrointestinal bleeding is a **common condition with high mortality**. Most patients undergo oesophagogastroduodenoscopy (OGD), but no universally agreed approach exists to the type of airway management required during the procedure. We **anaesthesia care** with tracheal intubation (TI group) and without airway instrumentation (monitored anaesthesia care, MAC group) during emergency OGD.

Acute upper haemorrhage (UGIH) is a **common medical emergency**, with an incidence of up to **150 per 100,000.** The incidence is **highest** in those from the lowest socio-economic groups. Although its **incidence** is declining, the **mortality remains high**.

Depending on the **site** and **rapidity of bleeding**, patients can present with *haematemesis, *malaena, *haematochezia (passage of blood PR) or ***syncope**.

There is a large range of clinical presentation, from **minor** UGIH that can be managed safely in the community to **catastrophic** exsanguination. All patients who have suspected UGIH should be **assessed in hospital**.

Pre-operative: initial assessment

I-History

The **presenting complaint**, **past history** and **medication history** are all important in determining the **Aetiology** of the UGIH.

For example, **(1)** a history of persistent vomiting followed by haematemesis in a young patient is strongly suggestive of a **Mallory Weiss tear**. **What is Mallory-Weiss syndrome associated with?**

A Mallory-Weiss tear **results** from **prolonged** and **forceful** vomiting, coughing or convulsions. Typically the **mucous membrane at the junction of the esophagus and the stomach develops lacerations which bleed**, **evident** by bright red blood in vomitus, or bloody stools. It may occur as a result of **excessive alcohol ingestion**

(2) There are a **number of drugs** (listed below) that increases a patient's risk of UGIB. When taking the history it is important to ascertain if the patient has any past history of (or risk factors for) **chronic liver disease**, as **bleeding** from oesophageal or **gastric varices** can be **significant** and **rapid**.

Drugs	Mechanism
Non steroidal anti-inflammatory e.g. aspirin, ibuprofen	Mucosal toxicity
COX-II inhibitors	
Prednisolone	
Warfarin	Impaired haemostasis
Anti-platelets e.g. Clopidogrel	
Aspirin	
Low molecular weight heparin and unfractionated heparin	
Selective serotonin reuptake inhibitors	

Table 1. *Drugs associated with Upper GI haemorrhage*

II-Examination

It is important to **carefully assess** any patient presenting with UGIH for *"**haemodynamic** instability" **or** *"**on-going bleeding"**. The assessment should include **pulse** and **blood pressure** (*lying and *standing), as well as **a rectal examination to look for malaena**. In addition, patients should be examined carefully for **stigmata of chronic liver disease.**

III- Investigations

At presentation **blood** should be taken for:

1) Full Blood Count
2) Urea and Creatinine (an elevated urea:creatinine ratio is a common finding in patients with significant UGIH)
3) Liver function tests
4) Clotting
5) Cross match

IV- Risk assessment

It is important to identify those patients who are at **risk** of "**ongoing bleeding" and "death"**. There have been several "**scoring systems"** developed to **help** in this process.

(1) "**Rockall scoring system**"
(2) **Glasgow by Blatchford et al.**

The "**Rockall scoring system**" is a validated **scoring system** used for **risk categorization** based on **simple clinical parameters**. Rockall Scores can be **calculated** both **(before) and (after) endoscopy,** but the post endoscopy Rockall Score provides a **more** accurate risk assessment. It comprises independent "risk factors" which have been shown to **accurately predict** both ***re-bleeding and *mortality** (Table 2).

	Score			
Variable	0	1	2	3
Age	<60	60-79	≥80	
Shock	none	pulse >100	pulse >100 BP <100	
Co morbidity	nil		cardiac failure/IHD Any major comorbidity	renal or liver failure metastatic disease
Diagnosis	Mallory Weiss tear No lesion	All other diagnoses	Malignancy of Upper GI tract	
Major SRH*	None or dark spot		Blood in upper GI tract Adherent clot or visible spurting vessel	

*Stigmata of recent haemorrhage
IHD – ischaemic heart disease

Table 2. ***Rockall scoring system (post endoscopy) for risk assessment of rebleedingand death in UGIH***

With increasing age there is an increased risk of death. **Mortality** in those aged **below 40** is negligible. Mortality increases to **30% in those aged over 90.**

Patients who have evidence of active bleeding and signs of shock have an 80% risk of death. Those with a **non-bleeding visible vessel** at endoscopy have a **50% chance of re-bleeding**.

Patients who have a **normal oesophagogastroduodenoscopy**, ***Mallory Weiss tear** or ***an ulcer with a clean base** have a **very low risk of re-bleeding** (fresh hematemesis +/- melaena associated with shock 24 hours after the initial event).

Low risk patients are defined as those with a post endoscopy score of ≤2. These patients have a 4% risk of re-bleeding and 0.1% mortality.

However, the best risk assessment tool for identifying low risk UGIH was developed in **Glasgow by Blatchford et al.** The "Blatchford score" is calculated **prior** to endoscopy and is based on simple *clinical and *laboratory parameters (table 3). Its principle use is to identify **low risk patients** who do not require any intervention (blood transfusion, endoscopic therapy, surgery). Approximately **20% of patients presenting with UGIH** have a Blatchford score of zero. Such patients can largely be managed **safely** in the community, as the **mortality** in this group is **nil**.

Admission risk marker	Score
Blood Urea mmol/L	
≥6.5 -7.9	2
8-9.9	3
10-24.9	4
≥25	6
Haemoglobin g/dL (men)	
≥12 -13	1
10-11.9	3
<10	6
Haemoglobin g/dL (women)	
≥10-12	1
<10	6
Systolic blood pressure mmHg	
100-109	1
90-99	2
<90	3
Other markers	
Pulse ≥100	1
Presentation with malaena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

Table 3. ***Blatchford Score for assessing the severity of UGIH***

Causes of upper GI haemorrhage

The cause for upper GI haemorrhage is **identified in 80% of cases**. The causes are shown in Table 4.

Diagnosis	% (approx)
Peptic Ulcer	35-50
Gastroduodenal erosions	8-15
Oesophagitis	5-15
Varices	5-10
Mallory Weiss tear	15
Upper GI malignancy	1-2
Vascular malformations	5
Miscellaneous	5

Table 4. *Causes of upper gastrointestinal haemorrhage*

Non-variceal upper GI haemorrhage الجزء العلوي غير الدوالي : management

1)Resuscitation

All patients should be managed by the system of ***Airway**, ***Breathing**, ***Circulation**.

All patients should receive 100% oxygen (unless contraindicated) and intravenous access obtained.

This entails the placement of at least **one 18 gauge cannula** and, if bleeding is significant, **2 large bore** (16 or 14 gauge) **cannulae** should be **sited**.

Either **colloid** or **crystalloid** can be used for **volume replacement**, aiming for a **systolic BP >100mmHg**.

If, after 2 liters have been **given**, the patient still has signs of shock this implies that **40% or more of the patients circulating volume has been lost. Transfusion is necessary in if:**

- (a) Bleeding is extreme i.e. active haematemesis + shock
  Or
- (b) When Hb <10g/L in the presence of an acute bleed

If bleeding is severe patients with UGIH should be managed on a high dependency **unit** where fluid resuscitation should continue. **A** catheter should be inserted and hourly urine volumes measured. **A** central venous catheter should be inserted in those patients with a significant cardiac history to guide fluid management.

The patient should be kept **fasted** صيام until there is haemodynamic stability and an endoscopy performed.

2)Endoscopy

Once **resuscitated**, patients with UGIH should have an **upper gastrointestinal endoscopy** by an experienced endoscopist.

a) In patients who are "**haemodynamically stable"** endoscopy can **safely be performed** in a **semi-elective manner**, ideally the **morning after admission** but certainly **within the first 24 hours of admission**. Not only does this aid diagnosis but it also identifies those patients who are suitable for early discharge.

Only a very small number of patients require endoscopy 'out of hours'.

b) **Therapeutic endoscopy** has been shown to improve prognosis in those who present with **severe UGIH** i.e. "**haemodynamic instability"** despite fluid resuscitation. It is these patients who benefit from an 'out of hours' service. Endoscopy in these patients should only be undertaken by a **skilled endoscopist** who has experience in the **therapeutic endoscopic** management of upper GI haemorrhage.

Principles of management

The major objectives of **therapy** are to speed healing, reduce pain, and prevent complications and recurrences while minimizing the costs and side effects of therapy. Although peptic ulcer disease represents a heterogeneous set of disorders, the overall approach to **medical therapy** is similar and centers on (a) **limiting precipitants** such as **NSAIDs**, stress, and **smoking**; (b) **eradicating *Helicobacter* infection**; (c) **reducing gastric acidity**; and (d) **protecting** the **mucosal barrier**. Combination programs are often used, particularly in instances of *Helicobacter*-induced disease.

Antibiotics are prescribed to treat the underlying infection, and **acid suppression** is given to speed ulcer healing and promote the relief of symptoms.

Anaesthetic input is often required to ***protect the airway from aspiration of blood** from the **GI tract during endoscopy**.

3) Haemostasis

Ideally all patients who show active bleeding, or who have endoscopic stigmata of recent haemorrhage, should receive **endoscopic therapy**. In general this **comprises**:

1) **Injection of adrenaline – 1:10,000 adrenaline injected** into 4 quadrants around the bleeding point, up to a maximum of 16mls. This will achieve haemostasis in 95% of patients; however bleeding recurs in up to 20%.
2) **Application of heat** – by **means of a heater probe**, *Argon plasma coagulation or *multipolar probe.

i. The **heater probe** has a thermocouple at the tip of the probe and can heat up instantaneously to achieve tissue coagulation.

ii. The **Argon plasma coagulator (APC)** is as effective as the heater probe. It uses argon gas to deliver evenly distributed thermal energy to a field of tissue adjacent to the probe. A high voltage spark is delivered at the tip of the probe, which ionizes the argon gas as it is sprayed from the probe tip

iii. **Multipolar probes** achieve haemostasis by heating the tissue with electricity that passes through the tip of the probe rapidly from positive and negative electrodes.

3) **Mechanical clips** – Endoscopically applied vascular clips are applied to bleeding vessels, however they can be technically difficult to apply, particularly in awkwardly placed ulcers.

When bleeding is seen it is recommended to apply dual modalities to stop bleeding i.e. adrenaline and thermal coagulation or adrenaline and clips. This is to decrease the risk of re-bleeding from use of adrenaline alone. All patients should be monitored for 4-6 hours post endoscopy. If haemodynamically stable after this period they should be allowed to eat and drink

All patients with ulcer disease should have a *H. pylori* (CLO) test taken at the time of endoscopy.

4)Medical management

1. **Proton Pump Inhibitors**

a. In patients with UGIH there is increased mucosal fibrinolytic activity, thus impairing haemostasis. Suppressing acid secretion **blunts** يضعف this response and makes any clot formation more stable. Clot lysis has been shown to occur at a pH <6. Proton pump inhibitors (PPI's) are the **only** class of **drug** that consistently increase gastric pH to >6. **Omeprazole** has been shown to be of benefit in acute UGIH, in terms of incidence of re-bleeding, reduced occurrence of persistent bleeding, decreased need for blood transfusion and decreased duration of hospital stay. Therefore, a PPI is recommended for empirical treatment of UGIB prior to endoscopy. This can either be parenteral or oral. If the bleeding is severe the parenteral route should be considered.

The role of high dose omeprazole (80mg stat followed by and infusion of 8mg/hr for 72hours) is used in those patients who have had successful endoscopic therapy of major ulcer bleeding and remain at high risk of re-bleeding.

2. **Tranexamic acid**

a. A meta-analysis has shown that the use of tranexamic acid in UGIH may **reduce** the need for surgical intervention, although it has **no effect** on the ulcer re-bleeding rate. Tranexamic acid is **not recommended** as routine therapy and its role in UGIH requires further evaluation.

5)Repeat upper GI endoscopy

Repeat endoscopy is performed if:

1) There is clinical evidence of **re-bleeding**, suggested by ongoing melaena or haematemesis, or haemodynamic evidence of blood loss. Most patients who re-bleed should have a repeat endoscopy not only to confirm the presence of blood prior to surgical intervention but also to **attempt** further endoscopic **control** of the haemorrhage.
2) If there were **suboptimal endoscopic views** at the initial endoscopy. The reason for suboptimal views is often due to large amounts of blood in the upper gastrointestinal tract following severe UGIH. In such patients it is prudent to repeat the endoscopy 12-24 hours after the initial intervention.

6)Surgery

Active non variceal UGIH that is uncontrollable by endoscopic therapy **needs** surgical intervention.

Only one clinical trial has looked at the different surgical procedures for **bleeding duodenal ulcers.**

Re-bleeding was lowest in those having a gastrectomy to include the ulcer with either a Billroth I or Billroth II reconstruction compared to more conservative measures.
However, the study **suggests** that when a bleeding duodenal ulcer is under run with specific ligation of the gastroduodenal and right gastroepiploic arteries the re-bleeding rate is reduced to that of those in the gastrectomy group. Most patients are now treated with this more conservative approach wherever possible.
Gastric ulcers are best excised or treated by partial gastrectomy. There is no clinical evidence that favours either intervention however if there is a suspicion of a malignant ulcer it is best treated with partial gastrectomy.

7)The role of interventional radiology

There is **a small group** of patients with **major non-variceal UGIH** who continue to bleed despite endoscopic intervention **who are unfit for operative surgery. Such patients** are often the **very elderly** with major co-morbidities who would not survive major surgery. Such patients should be considered **for mesenteric angiography**. This allows **the bleeding vessel to be identified and embolised** with coils. This will often successfully arrest on-going UGIH, but the mortality of this group remains high.

8)Follow up

In patients with UGIH it is important that a management plan be put in place upon discharge from hospital so that the chances of recurrence of any further bleeding is minimised. The following guidelines should be considered prior to discharge:

1) All patients who have peptic ulcer disease and who are **H.pylori positive** should **receive** standard eradication therapy.
2) Ulcers should be treated with a proton pump inhibitors (**PPI** for an initial period of 6-8 weeks.
3) Patients with ulcers associated with NSAID use should **stop the use** of these drugs.
4) If a patient needs to continue taking a NSAID then the least gastro-toxic (ibuprofen) should be used with concomitant administration of a PPI.
5) If a patient needs to continue taking an anti-inflammatory, consider a **cyclooxygenase II inhibitor**.
6) If a patient needs long term therapy with aspirin then a PPI prescribed concomitantly reduces the risk of recurrent bleeding.
7) All gastric ulcers need **repeat endoscopy** in 6-8/52 in order to *confirm **healing** and *exclude malignancy.

Variceal bleeding الدوالي : management

Increased **portal venous pressure** (usually in the context of chronic liver disease) causes the development of a portosystemic collateral circulation, with resultant portosystemic **shunts**. Such shunts manifest clinically as oesophageal and gastric **varices**. Patients with varices have a **30% lifetime risk of variceal bleeding**. The **mortality** for the first presentation of variceal bleeding is **50%**.
If a patient has **bled** once they have a 70% risk of recurrent bleeding. The **mortality** from subsequent bleeds is dependent on the severity of the underlying chronic liver disease (Table 5).

Child's-Pugh classification	Mortality (%)
A	5
B	25
C	50

Table 5. *Mortality from variceal bleeding in patients with differing severities of underlying chronic liver disease (Child-Pugh classification).*

I-Resuscitation

The general principles of resuscitation are the same as non variceal UGIH (see above). In addition, the following need to be considered:

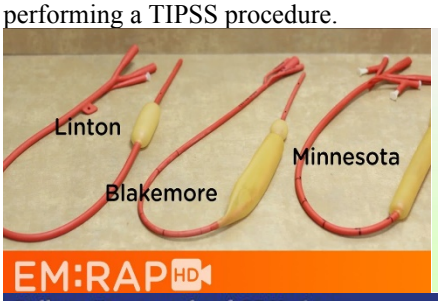
1) Bilateral large bore **intravenous** access should be established in all cases of suspected variceal bleeding. Initial fluid resuscitation should begin with colloid or crystalloid (aim to avoid sodium chloride). Bleeding can be **torrential** غزيرة . In such patients it is imperative to act quickly if **bleeding varices** are suspected. Cross match 6 units and transfuse as clinically required. The patient may require O-negative blood
2) Ideally, **central venous** access should be established to **guide fluid therapy**
3) Correct **thrombocytopenia** by platelet infusion if platelets <50x 10^9/L
4) Correct **clotting** with;
   i. Vitamin K iv 10mg
   ii. FFP
   iii. Cryoprecipitate (only if fibrinogen <75mg/dL)
   iv. and/or prothrombin complex concentrate (Octaplex)
5) **Broad spectrum antibiotics** (e.g. 3rd generation cephalosporins) should be given as they have been shown to reduce in hospital mortality by 20%
6) **Protect the airway** by elective endotracheal **intubation** of any patient where there is:
   i. Severe uncontrollable variceal bleeding
   ii. Severe encephalopathy
   iii. Inability to maintain oxygen saturations >90%
   iv. Aspiration (pneumonia)

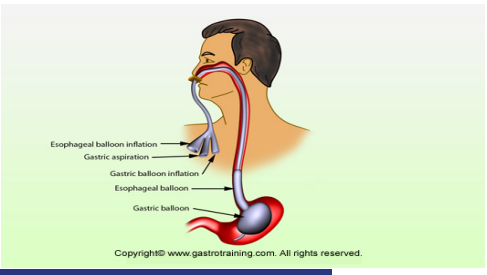
2- Endoscopy

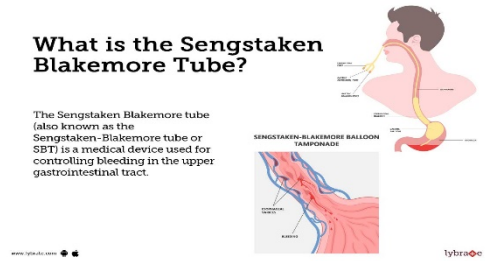
Endoscopy should be performed at the **earliest possible time** when the patient is **haemodynamically stable**. There are a number of methods of **treating varices**. The choice of therapy depends on the **site of the varices**.

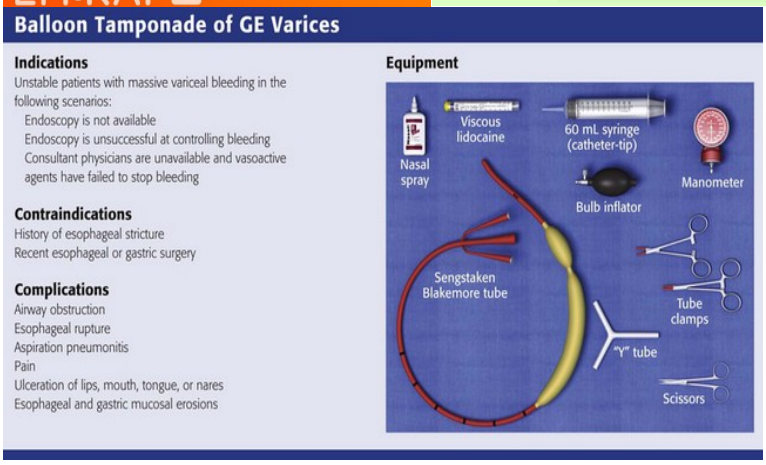
In patients with bleeding **oesophageal varices** "band ligation" is the method of choice as it has the greatest chance of controlling bleeding. However, in patients who are **actively bleeding** views of the lower oesophagus can be so poor that it is impossible to place the bands endoscopically. In such patients, injection "sclera-therapy" may be easier to apply. If **haemostasis** is not achieved by these therapies "balloon tamponade" using the "Sengstaken-Blakemore tube" is used (see below). This tends to be a temporary measure prior to either TIPSS or surgery.

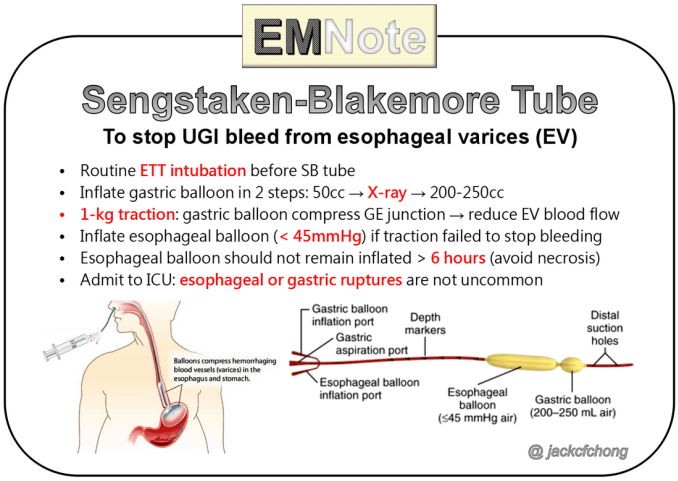
In patients with gastric varices, if varices are present at or around the gastro-oesophageal junction they are treated as oesophageal varices and either banded or sclerosed. However, if gastric varices are present in isolation initial therapy should be by the injection of cyanoacrylate (tissue glue). If this fails to control bleeding balloon tamponade is used as a temporary measure with a view to performing a TIPSS procedure.











3- Balloon Tamponade

Balloon tamponade is **only used** in **intubated patients** who **continue to bleed despite attempted endoscopic therapy**. The most widely used technique is by the deployment of a **Sengstaken-Blakemore tube**. This is a temporary orogastric tube consisting of 2 inflatable **balloons** (gastric and oesophageal) and **2 lumens** (*gastric and *proximal oesophagus). Once inserted the gastric balloon is inflated and the tube pulled back to achieve traction/compression of the feeding vessels at the gastro-oesophageal junction and gastric fundus. This controls bleeding in 90% of patients. The oesophageal balloon is rarely used and can cause ischaemic necrosis of the lower oesophageal mucosa, particularly if left inflated for protracted periods.

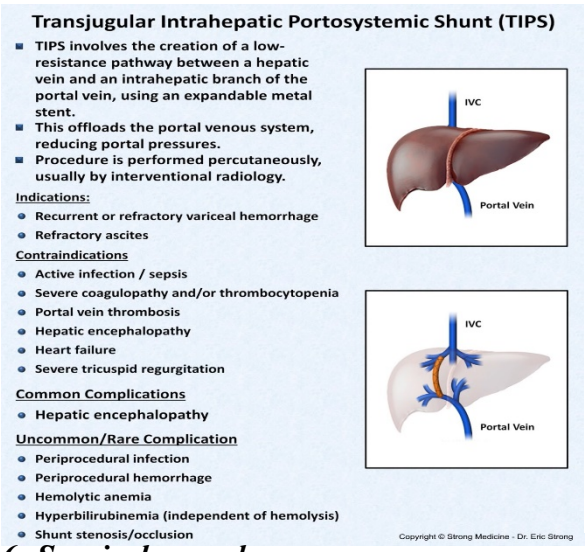
4- Lowering portal pressures by the use of drugs

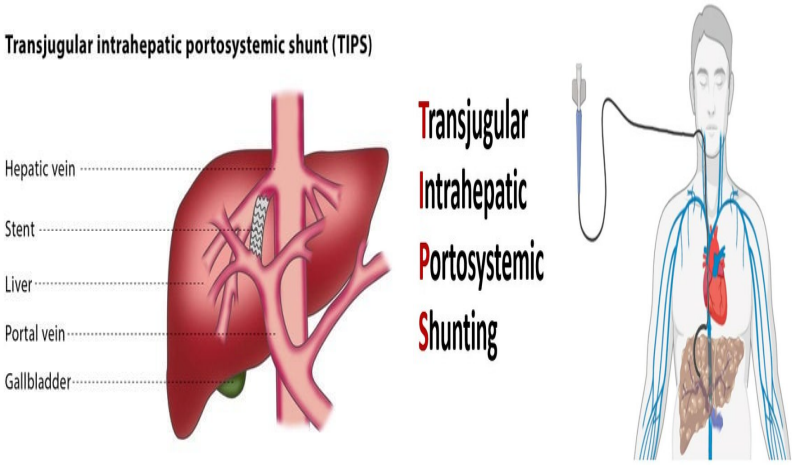
Vasopressin or its derivative, *terlipressin (either alone or in combination with *nitro-glycerine) are used in patients with **variceal bleeding**. These drugs *reduce portal blood flow and *variceal pressures. However, these agents also **increase systemic vascular resistance** and therefore **decrease cardiac output** and **coronary blood flow**. **Nitro-glycerine** is therefore sometimes used to **offset** these negative cardiovascular effects. Terlipressin has been shown to be as effective as balloon tamponade in stopping bleeding.

Alternatively, *somatostatin or its analogue *octreotide can be used as they cause selective splanchnic vasoconstriction and hence reduce portal blood flow. Neither has been shown to reduce mortality.

5- Transjugular intrahepatic portosystemic shunt (TIPSS)

This **procedure** is used when **variceal bleeding is not adequately controlled by endoscopy and/or balloon tamponade and terlipressin**. It is performed by an "**interventional radiologist**" under *local anaesthesia and *sedation. The jugular vein is cannulated and from there the hepatic vein. Once in the hepatic vein, a tract is created through the liver parenchyma into the portal vein under fluoroscopic guidance. This tract is then dilated and an expandable **metal stent** inserted to create a shunt between the portal and systemic system. **This causes a reduction in portal pressure**. TIPSS is an effective means of achieving haemostasis in uncontrolled variceal haemorrhage. However, 25% of patients become encephalopathic post procedure and 50% of stents block after 1 year. TIPSS is therefore regarded by many as a rescue therapy when endoscopic therapy fails and a bridge to transplantation.





6- Surgical procedures

Surgical shunt procedures are an alternative to TIPSS and perform essentially the same task. They create a shunt between the portal and systemic system by surgical means (e.g. **spleno-renal shunt**) and are occasionally used in an acute situation to control variceal bleeding. Oesophageal transection and gastric devascularisation are rapidly becoming obsolete, but can be of benefit in those with both portal and splenic vein thrombosis who are not therefore candidates for a surgical shunt procedure.

Liver transplantation is the treatment of choice for patients with decompensated chronic liver disease who have had a life-threatening bleed. This rapidly reverses any portal hypertension and other consequences of liver failure.

7- Long term follow up

This is aimed at **decreasing the patient's risk** of sustaining any future variceal bleeds.

All patients should be started on a beta blocker after the initial bleed. **Beta blockers** decrease portal pressure and have been shown to reduce mortality by decreasing the risk of re-bleeding. **Propranolol** is commonly used. Low doses should be used in the first instance, as propranolol normally has a high 'first pass' metabolism.

In patients with **chronic liver disease** this is lost and such patients are often sensitive to even paediatric doses. The **dose** is gradually titrated upwards with the aim of reducing the resting pulse rate by 25% (this is a good surrogate marker for effective reduction in portal pressure).

All patients who have **bled** should have repeated endoscopic treatment of their **varices**, ideally using **band ligation**. It is recommended that each varix be banded with a single band at 1 week intervals until eradicated. Patients should then be endoscoped at least every year to check for recurrent varices.

How should I prepare for anesthesia?

Make sure your healthcare provider has a current list of the medications, vitamins and other supplements you take. Certain drugs can interact with anesthesia or increase the risk of complications. You should also:

- **Avoid food and drinks for eight hours** before you go to the hospital unless directed otherwise.
- **Quit smoking,** even if it's just for one day before the procedure, to improve heart and lung health. For best results, stop smoking two weeks before your appointment.
- **Stop taking herbal supplements** for one to two weeks before the procedure as directed by your provider.
- **Stop taking Viagra® or other medications** for erectile dysfunction at least 24 hours before the procedure.
- **Take certain (but not all) blood pressure medications** with a sip of water as instructed by your healthcare provider.

What happens during anesthesia?

During anesthesia, a provider:

- Administers one or more types of anesthesia. They may also give you **anti-nausea medications**.
- Monitors vital signs, including blood pressure, **blood oxygen level**, pulse and heart rate.
- **Identifies and manages** issues like an allergic reaction or change in vital signs.
- Provides **guidelines for managing pain** after surgery.

Risks / Benefits

What are the potential side effects of anesthesia?

Most anesthesia side effects are temporary and go away within 24 hours, often sooner. Depending on the anesthesia type and how providers administer it, you may experience:

- Back pain or muscle pain.
- Chills caused by low body temperature (hypothermia).
- Difficulty urinating.
- Fatigue.
- Headache.
- Itching.
- Nausea and vomiting.
- Pain, tenderness, redness or bruising at the injection site.
- Sore throat (pharyngitis).

What are the potential risks or complications of anesthesia?

Potential complications include:

- **Anesthetic awareness:** For unknown reasons, about 1 out of every 1,000 people who receive general anesthesia experience awareness during a procedure. You may be aware of your surroundings but unable to move or communicate.
- **Collapsed lung (atelectasis):** Surgery that uses general anesthesia or a breathing tube can cause a collapsed lung. This rare condition occurs when air sacs in the lung deflate or fill with fluid.
- **Malignant hyperthermia:** People who have malignant hyperthermia (MH) experience a dangerous reaction to anesthesia. This rare inherited syndrome causes fever and muscle contractions during surgery. It's important to relate a personal or family history of MH to your physician anesthesiologist before your anesthetic to avoid drugs that trigger this reaction.
- **Nerve damage:** Although rare, some people experience nerve damage that causes temporary or permanent neuropathic pain, numbness or weakness.
- **Postoperative delirium:** Older people are more prone to postoperative delirium. This condition causes confusion that comes and goes for about a week. Some people experience long-term memory and learning issues.

Who's at risk for anesthesia complications?

Certain factors make it riskier to receive anesthesia, including:

- Advanced age.
- Diabetes or kidney disease.
- A family history of malignant hyperthermia (anesthesia allergy).
- Heart disease, high blood pressure (hypertension) or strokes.
- Lung disease, like asthma or chronic obstructive pulmonary disease (COPD).
- Obesity (body mass index, or BMI, of 30 or more).
- Seizures or neurological disorders.
- Sleep apnea.
- Smoking.

Quizzes:

Can anesthesia cause stomach problems?

Sometimes anesthesia can make you **feel sick**. It's a **common side effect** and often **doesn't last long**. **Pain** also can make you feel sick or **vomit**. After the anesthesia wears off, you may feel pain from the incision (cut).

How does anesthesia affect the GI tract?

Anesthesia also **alters gastrointestinal motility**, **secretion**, and **absorption**; **postoperative opiate analgesia** in particular contributes to **delay** gastric **emptying**.

Does anesthesia affect gut bacteria?

Fasting, **stress**, **antibiotics**, **hypothermia**, **anesthesia**, and **surgery** can **all potentially lead to gut dysbiosis** خلل التنسج المعوي. The interactions between the **gut microbiota** and the **central nervous system** are **mediated** through **neural**, **immune**, **endocrine**, and **metabolic pathways**, collectively defined as the "microbiota-gut-brain axis".