Hematemesis 1997

Causes, Varices, Bleeding Peptic Ulcers

- Gastroesophageal reflux
- Esophageal varices
- Mallory Weis syndrome
- Carcinoma of the esophagus, leiomyoma

Gastric
- Gastric ulcer
- Varices
- Acute erosive gastritis
- Gastric cancer
- Gastric cyst, lymphoma
- AV malformation, polyps
- Gastric intravascular ectasia

Duodenum: Ulcers, Carcinoma, AV malformation, polyps

Rare causes: Purpura, Hemophilia, Hemobilia, Acute pancreatitis.
GI Haemorrhage

NG tube aspiration

Nonbiliious nonbloody fluid

Possible UGI source

Blood fluid

UGI source

Bloody fluid

Bilious nonbloody fluid

Possible cause of source

Endoscopy

Identified

Treat

Massive haemorrhage

not identified

No source found

Abdominal angiography

Selective visceral angiography

Bleeding peptic ulcer

Variceal bleed

Ca esophagus

Ca Gastroic
CIF8 - H/o PUB, Hematemesis or melena features of shock

- Perforation produce abdominal signs
- Shock produce systemic signs

- Abdominal pain
- Distension

- BP ↓
- Elevated PT
- Acute mental status
- Dysfunction
- Myocardial
- Renal
- Comorbidity

Immediate - Endoscopy = Both dx and mxt

Forrest Clas of Endoscopic appearance of bleeding ulcers

Ia - Spurting
Ib - Non-spurting

IIa - Visible vessels
IIb - Non-bleeding ulcer
IIc - Ulcer w/ hematin clot
III - Clean ulcer base
Management

- Resuscitation is first priority than UGI endoscopy
  T/I

Conservative
- Resuscitation
- Ryle's tube
- Cold Saline
- Cold Antacid
- Ranitidine

Non-surgical

Surgical

1. Laser coagulation - Nd: YAG
2. Botox therapy
   - Epinephrine
   - 2% ethanolamine
3. Hemoclip application
4. Bipolar electrocoagulation

Indications of Sx:
- Failures of non-Sx
- Rebleeding in hospital
- >2lit. Blood needed
- Elderly
- Shock
- Recurrent

Surgical

Gastric
- Laparotomy gastrostomy
- Under cutting
- Partial gastrectomy
- Ulcer excision + Vagotomy + Pyloroplasty

Ant. Gastroduodenotomy
- Under cutting of ulcer base - but if chance of recurrence than
- Throm vagotomy

Degluttion +
95. Ans. a. Gastric ulcer (Ref: Sabiston 19/e p1164)

96. Ans. a. Esophageal varices

- Variceal bleeding often occurs without obvious precipitating factors and usually presents with painless but massive hematemesis and associated with tachycardia and shock.

97. Ans. b. Angiography can image bleeding at a rate of 0.05-0.1 ml/min or less (Ref: Sabiston 19/e p1174)

Selective angiography, using either the superior or inferior mesenteric arteries, can detect hemorrhage in the range of 0.5 to 1.0 ml/min.

- Selective angiography, using either the superior or inferior mesenteric arteries, can detect hemorrhage in the range of 0.5 to 1.0 ml/min.
- Only employed in the diagnosis of ongoing hemorrhage.

- Particularly useful in identifying the vascular patterns of angiodysplasias.
- It may also be used for localizing actively bleeding diverticula.
Notes of Dr. Ravindra Goswami (IAS-2015, AIR-153)

Eckovation App
Group Code: 873541

Section 3: Gastrointestinal Surgery

376

- Catheter-directed vasopressin infusion can provide temporary control of bleeding, permitting hemodynamic stabilization, although as many as 50% of patients experience rebleeding when the medication is discontinued.
- It can also be employed for embolization.
- Complications: Hematomas, arterial thrombosis, contrast reactions, and acute renal failure.

- Radionuclide scanning with technetium-99m (99m-Tc)-labeled RBCs is the most sensitive but least accurate method for localization of GI bleeding.
  - With this technique, the patient's own RBCs are labeled and reinjected.
  - The labeled blood extravasates into the GI tract lumen, creating a focus that can be detected scintigraphically.
  - Initially, images are collected frequently and then at 4-hour intervals for up to 24 hours.
  - The tagged RBC scan can detect bleeding as slow as 0.1 ml/min and is reported to be more than 90% sensitive.
- Unfortunately, the spatial resolution is lacking, and blood may move retrograde in the colon or distally in the small bowel.
- Reported accuracy of localization is 40-60%, and it is particularly inaccurate in distinguishing right from left-sided colonic bleeding.

98. Ans. d. CA stomach
99. Ans. c. Endoscopy

100. Ans. c. Endoscopy can best diagnose it; d. Peptic ulcer is the MC cause; e. Lab BUN (Ref: Sabiston 19/e p1161-1167; Schwartz 9/e p917; Bailey 26/e p1042-1043, 25/e p1063-1064; Shackelford 7/e p710-712)

- Actively bleeding ulcers and ulcers with non-bleeding visible vessels (protruding discoloration) warrant endoscopic therapy.
  - The management of ulcers with adherent clot consists of aggressive irrigation followed by endoscopic therapy (injection of epinephrine combined with thermal treatment).
  - Most frequent endoscopic characteristic is clean base (type III).
  - NG aspirate may be negative in the presence of significant duodenal bleeding and a competent pylorus preventing duodenogastric reflux. When emergency surgery for life-threatening lower GI hemorrhage is being contemplated, preoperative or intraoperative EGD is appropriate, particularly relevant if blind subtotal colectomy for massive hemorrhage is being considered.
  - Early EGD (within 24 hours) results in reductions in blood transfusion requirement, a decrease in the need for surgery, and a shorter length of hospital stay.

- Around 20-35% of patients undergoing EGD will require a therapeutic endoscopic intervention, and 5-10% will eventually require surgery. In 1-2% of patients with upper GI hemorrhage, the source cannot be identified because of excessive blood impairing visualization of the mucosal surface. Lavage of the stomach with room temperature normal saline solution can be helpful.
- When surgery for upper GI hemorrhage is undertaken, such operations are typically confined to the eldest and often the sickest patients.

**Medical Management:**
- PPI have been shown to reduce the risk for rebleeding and need for surgical intervention. Only 60-70% of patients with a bleeding ulcer test positive for H. pylori; after H. pylori eradication there is no need for long-term acid suppression.

**Endoscopic management:**
- Epinephrine injection (1:10,000) to all four quadrants in large volume (>13 mL); electocautery for bleeding ulcers and argon plasma coagulation for superficial lesion; a second attempt at endoscopic control before surgical intervention is encouraged.

**Ulcers >2 cm, posterior duodenal ulcers and gastric ulcers have a significantly higher risk for rebleeding.**

101. Ans. b. Duodenal ulcer (Ref: Sabiston 19/e p1164, 18/e p1524, 1529)

- Peptic ulcer is the most common cause of upper GI bleeding, present in one-half to two-thirds of patients with upper GI bleeding. Bleeding may be the initial presenting symptom in up to 10% of patients with peptic ulcer. Duodenal ulcer bleeding is more common than gastric ulcer bleeding.
- Only 10-15% of 'Heavy' drinkers develop alcoholic cirrhosis (leading to esophageal varices).
Acute erosive gastritis, chronic peptic ulcer and oesophageal varices constitute almost 90% of the cases.

Initial assessment (Table 23.6)

Intrusive endoscopy should be done on an emergency.

Massive hemorrhage preventing endoscopy

- IV H2 blocker/PPI Foley

- Endoscopy
  - Positive
  - Negative
    - Evaluate for LGIB

Varices

- Duodenal ulcer
- Gastric ulcer

AGML

- Manage accordingly
  - Endoscopic control by electrocoagulation or injection
  - Medical management
    - Endoscopic therapy

Succeeds

- Observe
- OR

Fails, rebleeds

Succeeds

- OR

Fails
### Table 23.1: Initial Assessment

<table>
<thead>
<tr>
<th>History</th>
<th>Probable causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abuse of drugs and alcohol</td>
<td>Acute erosive gastritis, Chronic peptic ulcer</td>
</tr>
<tr>
<td>Previous abdominal pain</td>
<td>Chronic peptic ulcer, Atherosclerosis</td>
</tr>
<tr>
<td>Toxemia, liver cell failure</td>
<td></td>
</tr>
<tr>
<td>Anaemia, loss of weight, loss of appetite</td>
<td>Acute erosive gastritis, Chronic peptic ulcer</td>
</tr>
<tr>
<td>Violent vomiting—haematemesis</td>
<td>Cirrhosis of the liver,</td>
</tr>
<tr>
<td>General physical examination</td>
<td>Carcinoma stomach,</td>
</tr>
<tr>
<td>Peculiar atrophy, gynaecomastia</td>
<td>Acute erosive gastritis (use of NSAID),</td>
</tr>
<tr>
<td>Palpable left supraclavicular node</td>
<td>Bleeding tendencies,</td>
</tr>
<tr>
<td>Swellings—multiple joint involvement</td>
<td>Portal hypertension,</td>
</tr>
<tr>
<td>Purpuric spots (Fig. 23.45)</td>
<td>Carcinoma stomach,</td>
</tr>
</tbody>
</table>

### Abdominal examination

- Palpable spleen, ascites
- Palpable stomach mass
- Tenderness in the epigastrium

### Assessment of haemorrhage

- Massive haemorrhage (more than 1,000 ml of blood)
- Moderate haemorrhage (500-1,000 ml of blood)
- Mild haemorrhage (less than 500 ml of blood)

---

### Table 23.2: Surgery for haematemesis

<table>
<thead>
<tr>
<th>Causes</th>
<th>Conservative</th>
<th>Failure</th>
<th>Surgical method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute erosive gastritis</td>
<td>Yes</td>
<td>Rarely—gastrectomy</td>
<td></td>
</tr>
<tr>
<td>Chronic duodenal ulcer</td>
<td>Yes</td>
<td>Vagotomy, under-running, pyloroplasty</td>
<td></td>
</tr>
<tr>
<td>Chronic gastric ulcer</td>
<td>Yes</td>
<td>Partial gastrectomy</td>
<td></td>
</tr>
<tr>
<td>Mallory-Weiss syndrome</td>
<td>Yes</td>
<td>Suturing of the tear</td>
<td></td>
</tr>
<tr>
<td>Cancer of the stomach</td>
<td>Yes</td>
<td>Gastroscopy</td>
<td></td>
</tr>
<tr>
<td>Duodenal polyp</td>
<td>Yes</td>
<td>Surgery if endoscopic facility not available</td>
<td></td>
</tr>
<tr>
<td>Haemobilia</td>
<td>Yes</td>
<td>Ligation of feeding vessel</td>
<td></td>
</tr>
<tr>
<td>Variceal bleeding</td>
<td>Yes</td>
<td>Devascularisation</td>
<td></td>
</tr>
<tr>
<td>Pseudocyst of pancreatitis</td>
<td>Yes</td>
<td>Ligation of pseudoneurysm</td>
<td></td>
</tr>
<tr>
<td>Duodenal lesion</td>
<td>Yes</td>
<td>Wide excision</td>
<td></td>
</tr>
</tbody>
</table>

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Stomach and Duodenum

**Notes of Dr. Ravindra Goswami (IAS-2015, AIR-153)**

- The surgical treatment depends on the ulcer size, distance from the GE junction and the degree of surrounding inflammation.
- Whenever possible, the ulcer should be excised.
- The most aggressive approach is to perform a gastrectomy that includes a small portion of the oesophageal wall and the ulcer followed by a Roux-en-Y oesophago-gastro-jejunostomy to restore intestinal continuity.
- For type 4 gastric ulcers that are located 2 to 5 cm from the gastro-oesophageal junction, a distal gastrectomy with a vertical extension of the resection to include the lesser curvature with the ulcer can be performed (i.e., Pauchet procedure). After resection, bowel continuity is restored with an end-to-end gastroduodenostomy.
- The Csendes procedure may be useful in stable patients or the Kelling-Madlener operation in unstable ones (Fig. 23.29).
- Some have even advocated leaving the ulcer in place or locally excising it in conjunction with truncal vagotomy and pyloroplasty.

**Summary of the management of peptic ulcer**

(Fig. 23.30)

**Complications of peptic ulcer**

A. **Acute**
- Perforation
- Haematemesis and/or melena

B. **Subacute**
- Residual abscess

C. **Chronic**
- Gastric outlet obstruction (pyloric stenosis)
- Peptic deformity
- Hour glass contracture of the stomach
- Penetration into the pancreas
- Carcinoma of stomach

**Type 4 gastric ulcers (see Fig. 23.10)**

- The type 4 gastric ulcer presents a difficult management problem.
ACUTE COMPLICATIONS OF PEPTIC ULCER

PERFORATED PEPTIC ULCER

Introduction

More common in males. The ratio is 8-10 men to 1 woman in India.

Anterior duodenal ulcer perforates and posterior duodenal ulcer bleeds. An ulcer on the posterior wall of the stomach can perforate into the lesser sac.

Usually, patients with a long history of peptic ulcer, suddenly complain of feeling something that has given way in their abdomen. It may be precipitated by excessive smoking alcohol, drugs, etc. Rarely a ‘silent’ ulcer can also perforate (especially those patients treated with cortisone).

Patients taking NSAIDs (often elderly) can present less dramatically.

Two factors are associated with most perforated peptic ulcers: Chronic use of NSAIDs and Helicobacter pylori infection.

Patients with H. pylori infection and perforated peptic ulcers tend to be younger, with a male preponderance and more prolonged period of dyspepsia. However, NSAIDs category of patients are elderly with equal proportion of both sexes being affected.

- Perforated peptic ulcers have a mortality rate of 5-10%.
- Perforated gastric ulcers in the elderly have 20-30% mortality rate.
- Golden time to operate is within 6 hours.

Stages of duodenal ulcer perforation

1. Stage of chemical peritonitis
   - Immediately after the perforation, gastric and duodenal contents leak into the peritoneal cavity and produce severe agonising pain in the right hypochondrium. It is mainly H. pylori which produces pain.
   - There may be an episode of coffee-ground vomitus, followed by melaena later.
   - The pulse rate increases. The patient is pale and anxious.
   - Blood pressure may be normal in the initial few hours.
   - Per abdomen, there is guarding and rigidity of the abdominal wall.
   - Rebound tenderness is present all over the abdomen. This sign is called Blumberg’s sign.
   - On percussion, liver dullness is obliterated because of collection of free air (gas) under the right dome of diaphragm. This stage is seen for about 2-4 hours from the time of perforation.
   - Bowel sounds are usually absent.
Injection gentamicin 60-80 mg IV, 8th hourly against gram-negative organisms.

Injection metronidazole 500 mg IV, 8th hourly to treat anaerobic organisms.

Cephalosporins can also be used depending upon the severity of the shock.

Exploratory laparotomy is done through a midline incision. The perforation is identified and closed with interrupted nonabsorbable silk sutures, which is strengthened by placement of omentum (Fig. 23.34). Peritoneal toilet/wash is given to avoid residual abscess. Abdomen is closed with a drain which is removed after 3–5 days. If it is a large gastric ulcer, it is better to do a gastrectomy, if condition of the patient permits.

It is the posterior duodenal ulcer which commonly bleeds, because it erodes into the gastro-duodenal artery which runs posterior to the duodenum. A gastric ulcer on the lesser curvature erodes into one of the branches of left or right gastric artery.

Precipitating factors for haemorrhage

Chronicity, results in destruction of the layers of the stomach, exposing the vessel.

Sudden, severe acid peptic digestion brought about by irritants such as alcohol, drugs, etc.

Atherosclerosis: Sclerotic artery does not contract, resulting in massive haemorrhage.

Characteristics of individuals at an increased risk of developing acute GI bleeding

- Increased age
- Male sex
- Cardiovascular disease
- Diabetes mellitus and renal disease
- Increased number of medications
- Oral anticoagulant use

Clinical features of bleeding peptic ulcer

1. History of abdominal pain of peptic ulcer disease.

2. History of haematemesis or melaena (black tarry stools), one or more attacks.

3. There may be features of haemorrhagic shock such as febrile, thready pulse, hypotension, syncope.

4. Oliguria, due to inadequate renal perfusion.

5. Brainstem hypoxia results in change in rate and depth of respiration.

6. There may not be any abdominal signs. However, due to accumulation of blood in the intestines and stomach, child distension may be present. Perforation produces abdominal signs and haemorrhage produces systemic signs.

BLEAD risk classification

The following are some of the factors associated with increased morbidity and mortality (Key Box 23.11).

**Key Box 23.11**

**BLEED RISK CLASSIFICATION**

- Blood pressure low
- Elevated prothrombin time
- Altered mental status
- Dysfunction—myocardial, renal, comorbid disease.
2. Stages of reaction

The peritoneum reacts to the chemical irritants by secreting peritoneal fluid. As a result of this, TCI and bile are diluted by the peritoneal secretions (reaction of peritoneum to the insult) resulting in an improvement of symptoms. Hence, it is also called stage of delusion or stage of illusion. This stage lasts for 3-6 hours. However, the signs are worse.

- Pulse is feeble, more than 120/min.
- Hypotension persists
- Evidence of dehydration due to loss of fluid into peritoneal cavity.
- Shifting dullness is present
- Abdominal distension is due to fluid and paralytic ileus.
- Bowel sounds are absent
- Guarding and rigidity are worsened.

3. Stage of bacterial peritonitis

- The peritoneal contents get contaminated with gram-negative organisms resulting in bacterial peritonitis (the organisms are from the intestine itself and not from the peritoneum).
- The patient becomes severely ill, dehydrated, toxic with drawn in cheeks. The tongue is dry and coated but with bright eyes (Hippocratic facies, Fig. 23.31).
- Features of hypovolaemic and septicemic shock such as feeble thready pulse, cold peripheries, shallow respiratory, high grade fever and persistent hypotension are present. Gross abdominal distension, guarding, rigidity, abdominal tenderness all over suggest generalised peritonitis.

![Fig. 23.31: Peritonitis—Hippocratic facies—sunken eyes, mould due to sepsis, nasogastric tube showing altered blood](image)

Investigation of perforated duodenal ulcer

- Complete blood picture and electrolyte study.
- Plain X-ray chest or abdomen in erect position shows collection of free gas under the right dome of diaphragm in majority of cases. If patient is unable to stand, left lateral decubitus films are taken (Figs 23.32 and 23.33).
- When in doubt, request a CT scan with contrast which can demonstrate pneumoperitonium, fluid in the abdomen, site of perforation and some surprises also.

![Fig. 23.32: Plain X-ray abdomen erect showing collection of free gas under the right dome of the diaphragm](image)

![Fig. 23.33: Plain X-ray abdomen lateral decubitus showing collection of free gas under the abdominal wall](image)

Treatment (ABCDEF)

A. Aspiration of stomach contents with Ryle’s tube to reduce further contamination and to decrease biliary and pancreatic juice.
B. Blood grouping and cross-matching may be necessary for surgery.
C. Charts: Temperature, pulse, BP, respiration, urinary output (urinary bladder is catheterised using a Foley’s catheter).
D. Drugs:

- Injection ampicillin 500 mg IV, stat and 6th hourly against gram-positive organisms.

It resembles a strike by employees paralysing the work of a factory in response to an insult. Students should remember that ABCDEF are the basic principles of treatment of any acute abdomen. In majority of cases of acute abdomen, these principles can be applied with minor modifications.
Varices (Portal HTN) = Portal Pressure > 10 mmHg

<table>
<thead>
<tr>
<th>Causes</th>
<th>Pre-sinusoidal</th>
<th>Sinusoidal</th>
<th>Post-sinusoidal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extrahepatic or Sinistral:</td>
<td>Schistosomiasis 🔥</td>
<td>Congenital hepatic fibrosis</td>
<td>Budd-Chiari syndrome 🔥</td>
</tr>
<tr>
<td></td>
<td>Splenic vein thrombosis 🔥</td>
<td>Nodular regenerative hyperplasia</td>
<td>Congestive heart failure 🔥</td>
</tr>
<tr>
<td></td>
<td>Splenomegaly 🔥</td>
<td>Idiopathic portal fibrosis</td>
<td>IVC web 🔥</td>
</tr>
<tr>
<td></td>
<td>Splenic arteriovenous fistula 🔥</td>
<td>Myeloproliferative disorder</td>
<td>Constrictive pericarditis 🔥</td>
</tr>
<tr>
<td></td>
<td>Veno-occlusive disease 🔥</td>
<td>Sarcoid and GVHD</td>
<td></td>
</tr>
</tbody>
</table>

MC cause of intrahepatic presinusoidal portal hypertension: Schistosomiasis 🔥
MC cause of sinusoidal portal hypertension: Cirrhosis 🔥

- Hepatic
  - > 40 yrs
  - Alcohol History 🔥
  - M > F
  - Mild to moderate

- Splenomegaly moderate
- Ascites 🔥
- Liver function N
- Encephalopathy 🔥

- Inv. Blood: Anemia, BT, CT, Albumin
  - M.F.
  - Albumin, BT, CT, OT/Pt

- Biopsy
  - HBs Ag
3. Endoscopy - For both AX and Mx
4. Superselective angiography:
   - To know anatomy of portal tract
   - To locate the obstruction
   - Assess the diameter of portal vein
     - if < 1 cm it is an indication of "bino renal shunt"
   - To measure the pyle pressure
5. USG

Management
X ABCDEF
1. Primary prophylaxis (Before bleed)
   - Pharama - β blockers
   - Endotherapy - endoscopic band ligation
2. Secondary prophylaxis:
   - Modified Child's criteria:
     - Enceph PT Ab Bic
     - EPABE to Asci
3. Tilt of massive bleed
   (i) General measures, ICU, Resuscitate
   (ii) Measure to prevent encephalopathy:
      - Bowel wash
      - Neomycin, Oral lactulose

Note by Dr. Ravindra Goswami (IAS-2015, AIR-153)
(iii) Pharmacotherapy
- IV - vasopressin - 20 unit - 250 ml in 250 ml
or - IV somatostatin
or - IV Octreotide
+ IV metoclopramide to constrict GE sphincter

Bleeding controlled

(iv) Endotherapy - Sclerotherapy - 2 x Ethanolamine
S/E = Higher complication - Perforation, ulcers
Higher rebleeding rate

Bleeding

Stopped than

Definitive secondary
Prephylaxis & definitive T/E

If not stopped than

Re endotherapy
If not than

Balloon tamponade
Through Sengstaken tube
or Minnesota tube

If bleeding continues

TIPS
CTP (class B or C)

Surgery
Tips

Vascular placement of stent across tract between Rt. hepatic vein and portal system

- Stent (CTP CLASS A)
  - Both as rescue and definitive
  - In cirrhosis & good liver function
    - End to side
      - To urgent bleed control
      - S/E = Encephalopathy
    - Side to side
      - To urgent bleed control
  - S/L = Spleenectomy

- Proximal spleno renal stent
  - Spleenectomy
  - Spleenic vein to renal vein
  - Done in children

- Distal spleno renal stent
  - In portal vein thrombosis & Portal vein > 1 mm
  - In cirrhosis
  - Distal end of splenic vein to renal vein
  - Proximal end cut and ligated
**Prevention of Variceal Bleeding**

- Current measures aimed at preventing variceal bleeding include:
  - Improvement of liver function (abstention from alcohol)\(^\text{a}\)
  - Avoidance of aspirin and NSAIDs\(^\text{a}\)
  - Administration of propranolol or nadolol (nonselective beta blockers)\(^\text{a}\)

- Beta-blockers reduce the index variceal bleed by 45% and reduce bleeding mortality by 50%\(^\text{a}\).
- 20% of patients do not respond to beta blockers and 20% cannot tolerate beta blockers due to medication side effects\(^\text{a}\).

Prophylactic endoscopic variceal ligation (EVL) is associated with a lower incidence of first variceal bleed\(^\text{a}\).
- EVL is recommended for medium to large varices, performed every 1 to 2 weeks\(^\text{a}\) until obliteration, followed by endoscopy 1 to 3 months later and surveillance endoscopy every 6 months to monitor for recurrence of varices.

### Injection Sclerotherapy

- Tetradecyl sodium (1-3%), sodium morrhuate (5%), ethanolamine olate (5%) and 3% phenol are most common sclerosing agents\(^\text{a}\).
- A combined para and Intravariceal technique is used for the management of acute variceal bleeding and a predominantly Intravariceal technique for long term management\(^\text{a}\).
- Variceal eradication is considered the end point of EST\(^\text{a}\).
- Variceal obliteration can be achieved in 80-95% patients with a mean of 4-6 sessions\(^\text{a}\).
- Esophageal stricture\(^\text{a}\) is a common complication.

### Endoscopic Variceal Ligation

- For control of acute variceal bleeding EVL has been found to be as effective as EST\(^\text{a}\).
- Technically more difficult than EST\(^\text{a}\) in presence of massive bleeding due to reduction in field of view.
- Variceal recurrence rate is higher in EVL since gastroesophageal varices\(^\text{a}\) (perforating veins) are not obliterated.
- Not suitable for small varices (grade I, II)\(^\text{a}\).
- EVL is associated with lesser complications and reblooding\(^\text{a}\).
- Variceal eradication in fewer sessions but higher recurrence of varices\(^\text{a}\).

### Sengstaken-Blakemore Tube

- If the rate of blood loss prohibits endoscopic evaluation, a Sengstaken-Blakemore tube may be inserted to provide temporary hemostasis\(^\text{a}\).
- Once inserted, the gastric balloon is inflated with 300 ml of air\(^\text{a}\) and retracted to the gastric fundus, where the varices at the esophagogastric junction are tamponaded by the subsequent inflation of the esophageal balloon to a pressure of 40 mmHg\(^\text{a}\).
- The two remaining channels allow gastric and esophageal aspiration\(^\text{a}\).
- A radiograph is used to confirm the position of the tube\(^\text{a}\).
- The balloons should be temporarily deflated after 12 hours to prevent pressure necrosis of the esophagus\(^\text{a}\).

31. Ans. a. 40 mm

32. Ans. b. Banding (Ref: Sabiston 19/e p1434; Schwartz 9/e p1113; Bailey 26/e p1075, 25/e p1089; Blumgart 5/e p1135-1138; Shackelford 7/e p1600-1601; Harrison 18/e p2598)
TIPSS

39. Ans. a. HPS (Ref: Sabiston 19/e p1437-1440; Schwartz 9/e p1114; Bailey 26/e p1075-1076, 25/e p1089-1090; Bhargav 5/e p1180-1188; Shackelford 7/e p1602-1603; Harrison 18/e p2599)

**Fig. TIPS (transjugular intrahepatic portosystemic shunt)**

**TRANSJUGULAR INTRAHEPATIC PORTAL-SYSTEMIC SHUNT (TIPSS)**

- TIPSS is a non-selective shunt, created between portal and hepatic vein.
- TIPSS is portohepatic or intrahepatic shunt.
- TIPSS in the acute situations should be avoided in patients requiring ventilation and with evidence of sepsis and renal failure.

**Technique of Placement**

- Initial venous access is through the right internal jugular vein because this is the shortest and most direct path to catheterize the hepatic vein.
- The right hepatic vein is MC used because it is the largest hepatic vein and usually has the most favourable orientation.
- Portal vein is cannulated by Bovas needle.
- Portal vein is localized by carbon dioxide wedge hepatic venography.
- Portal venogram before dilating parenchymal tract is crucial, provides confirmation that portal vein has been accessed.

**Stent**

- BIVIATORR is a stent-graft specifically designed for TIPSS.
- The device has a 2 cm long bare stent segment that sits in the portal vein; the covered portion consists of three PTFE layers, one of which is an impermeable film to prevent bile leak into the shunt.

### Indications of TIPSS

| 1. Prevention of rebleeding from varices (MC) |
| 2. Acute variceal bleeding |
| 3. Refractory ascites |
| 4. Hepatorenal syndrome |
| 5. Refractory hepatic hydrothorax |
| 6. Budd-Chiari syndrome |
| 7. Hepatic veno-occlusive disease |
| 8. Portal hypertensive gastropathy |

### Contraindications of TIPSS

<table>
<thead>
<tr>
<th>Absolute</th>
<th>Relative</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Portal vein thrombosis</td>
<td></td>
</tr>
<tr>
<td>2. Hypervascular liver tumors</td>
<td></td>
</tr>
<tr>
<td>3. Encephalopathy</td>
<td></td>
</tr>
</tbody>
</table>

### Complications

- Encephalopathy (10-20%)
  - Usually occurs within 1 month of the procedure
  - Relatively easy to manage with protein restriction and lactulose
  - Declines after the first 3 months as the stent develops spontaneous closure
- Stenosis or thrombosis (5-15%)
  - Half of the stenoses occur in the hepatic vein, and half are due to intimal hyperplasia in the parenchymal segment.
  - Shunt stenosis is usually secondary to neointimal hyperplasia and is more common than thrombosis.
  - Shunt thrombosis occur in <30 days, whereas stenosis after 30 days.
  - Embolization of the stent to the pulmonary artery
  - Inadvertent puncture of the gallbladder or laceration of the liver capsule
  - Hemobilia, bacteremia with septic shock, intravascular hemolysis
  - Contrast induced oliguric renal failure, worsening hepatic function, right heart failure.

### Surveillance

- Doppler duplex ultrasonography at 24 hours, 1 month, 3 months, and 6 months after the initial TIPS procedure.
### Types of Portosystemic Shunts

<table>
<thead>
<tr>
<th>Non selective</th>
<th>Selective</th>
<th>Partial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Divert all portal flow away from the liver</td>
<td>Decompresses the gastroesophageal and splenic segments</td>
<td>Only diverts part of portal venous flow</td>
</tr>
<tr>
<td>End-to-side or side-to-side anastomosis &gt;11 mm</td>
<td>Disto splenorenal shunt (Warren shunt)</td>
<td>Side-to-side anastomosis &lt;10 mm in diameter</td>
</tr>
<tr>
<td>Side-to-side shunts</td>
<td>Inokuchi shunt consists of interposition of a vein graft between the left gastric or coronary vein and IVC</td>
<td></td>
</tr>
</tbody>
</table>

- **MC causes of death in Portal Hypertension**
  - Medically treated patients: Rebleeding
  - Shunted patients: Accelerated hepatic failure
  - Rex shunt is an internal jugular vein graft (mesenteric-left portal vein bypass) used in EHPVO
  - Eck's Fistula is an end-to-side portacaval shunt

#### Notes
- **Transplant is the ultimate therapy for cirrhosis**
- **TIPS = Acute EHPVO**
- **Shunt = Rex Shunt**
- **EVL**
- **Defn:** Endoscopic
- **Acute portal venous obstruction**
- **Anticoagulation**
- **Acute Portal Venous Obstruction**
- **Bye-pass**

Adynamic Intestinal obstruction & MnX
- Pathogenesis & Tilt of Cervical Lymphadenitis