PEPTIC ULCER

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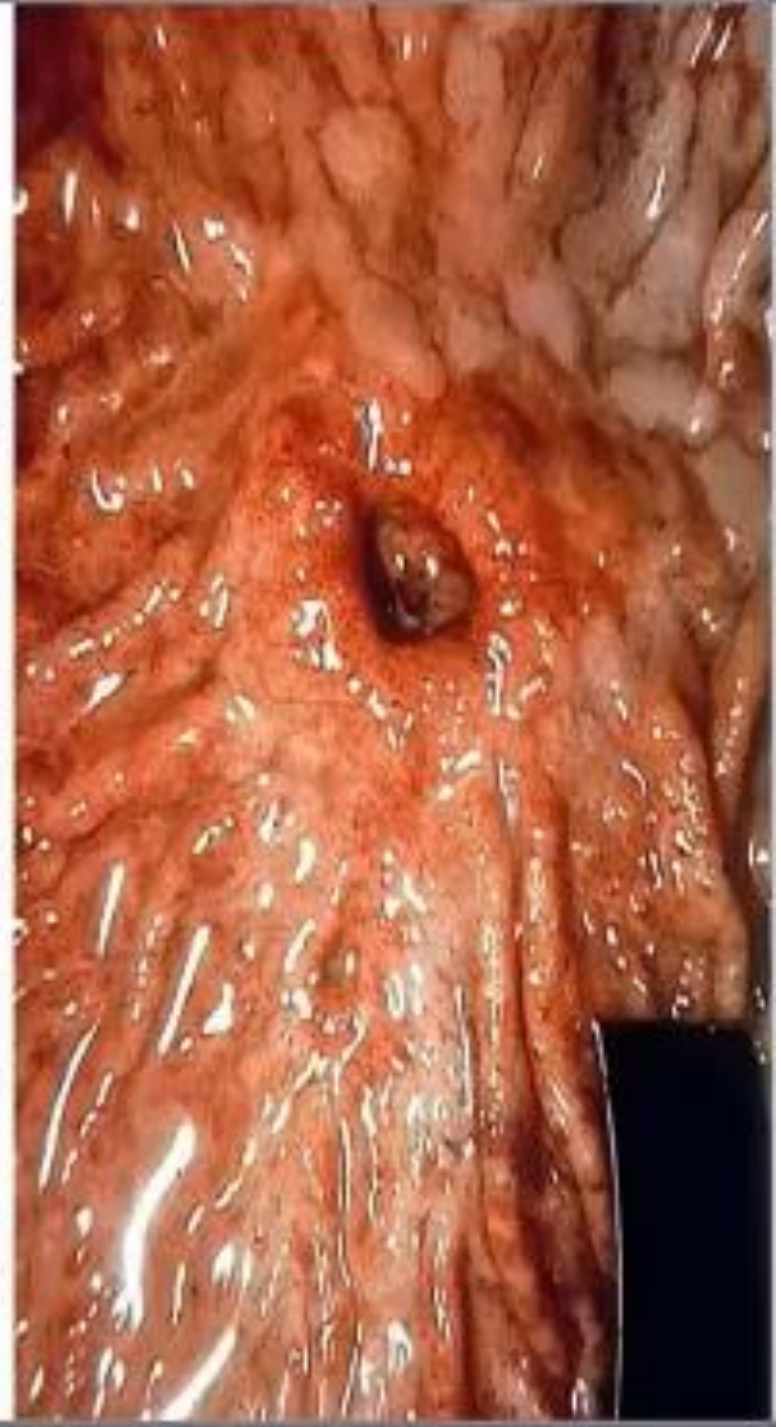
Lecture ILOs

- By end of this lecture the student will be able to:
  - Describe the etiology and clinical manifestations of peptic ulcer in order to plan the effective management (Knowledge).
  - Analyze the clinical features of peptic ulcer in order to reach an appropriate diagnosis.
  - Correlate the clinical features with diagnostic tests results to reach definite diagnosis and plan management of the patients(Cognitive).

Peptic ulceration and related disorders

Peptic ulcer disease encompasses disorders of the oesophagus, stomach and duodenum. The conditions share the symptom of epigastric pain and all have the common aetiology of mucosal inflammation associated, to a greater or lesser extent, with gastric acid-pepsin secretions. Recent work has demonstrated that perhaps the most important aetiological factor in gastric and duodenal ulcer disease is chronic mucosal infection with the bacterium *Helicobacter pylori*. Peptic disorders, together with gallstone disease, are the most common causes of organic upper abdominal pain.



PEPTIC ULCER_Role of surgery

- Over the last 2 decades, despite the advances in medical therapy to inhibit acid secretion and to eradicate H. pylori surgery is important in managing these patients. There is an increase in emergency operations for complicated peptic ulcer while the number of elective operations has been decreased markedly.

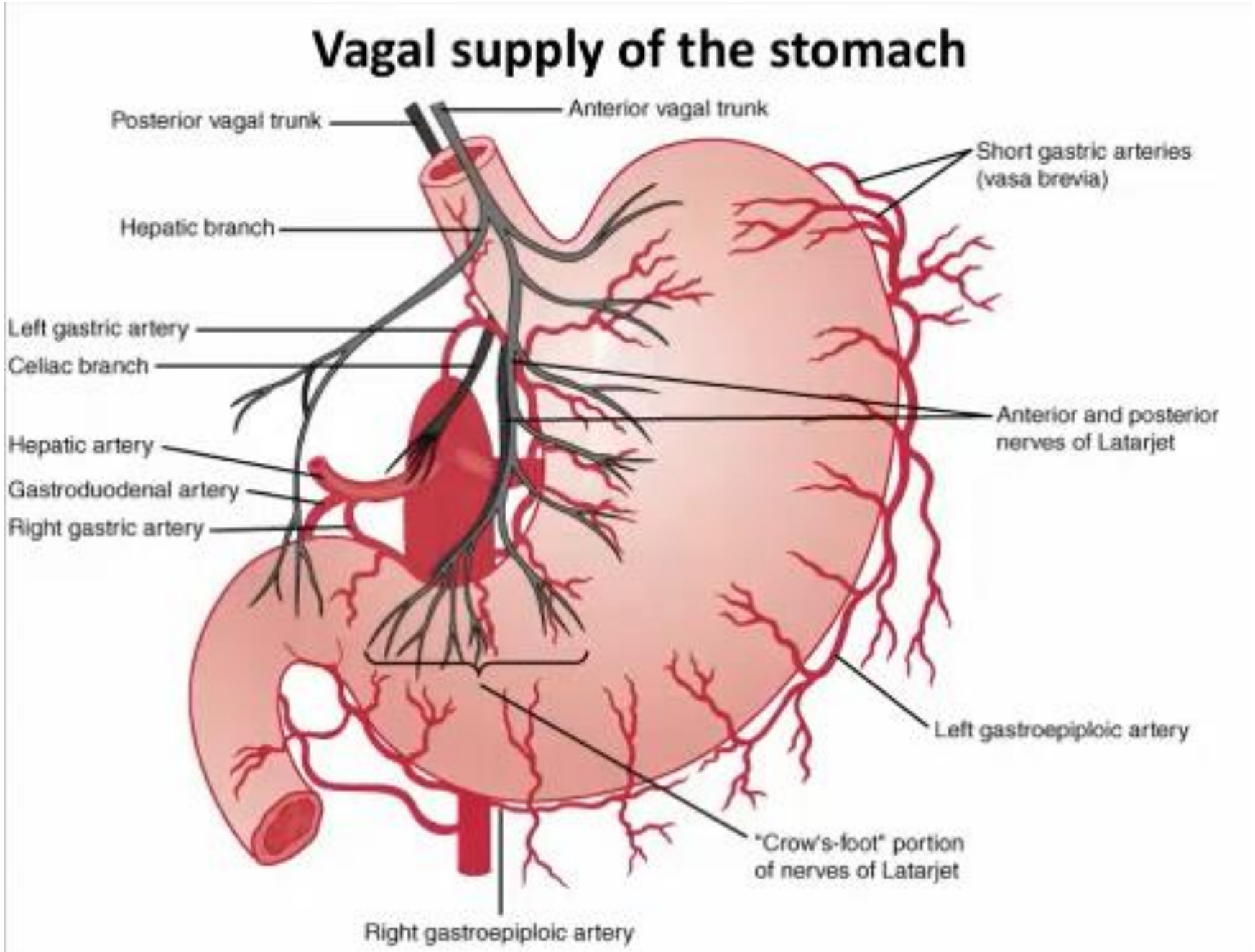
ULCER - EROSION

An ulcer extends through the muscularis mucosa in contrast to an erosion, which is superficial to muscularis mucosa.

Blood supply of the stomach

On the lesser curve, **left gastric artery (**of coeliac axis) anastomoses with **right gastric artery** (of common hepatic artery).**Gastroduodenal artery (**of common hepatic artery) passes behind the first part of the duodenum(highly relevant to bleeding duodenal ulcer), Here it divides into **superior pancreatico-duodenal artery** and **right gastroepiploic artery**. **Superior pancreatico-duodenal artery** supplies the duodenum and pancreatic head and anastomoses with **Inferior pancreatico-duodenal artery,(**of superior mesenteric). **Right gastroepiploic artery** runs along the greater curvature of the stomach, anastomoses with **Left gastroepiploic artery (**of splenic artery).**Short gastric arteries (**of splenic artery) supply the fundus.

Vagal supply of the stomach



Investigations of gastric disorders

- **Flexible endoscopy** is the most sensitive one .
- CT scan, useful in staging gastric cancer.
- PET scan: useful in diagnosis, staging gastric cancer
- Endoscopic ultrasound is the most sensitive in T staging of gastric and duodenal tumors
- Laparoscopy for peritoneal, lymph nodes and liver spread.

Etiology of Duodenal ulcer

- Increased secretion of acid and pepsin(mucolytic) in conjunction with H. pylori infection(duodenal ulcers 90% and gastric ulcers 75%) or ingestion of NSAIDs.
- Genetic factors, social stress(blood group O is the most common blood type in patients with duodenal ulcer/ patients who have bled from a duodenal ulcer. May be due to increased parietal cell mass???).
- Cigarette smoking predisposes to peptic ulcer, causes relapse after treatment.

Pathology

Duodenal ulcer

- Due to increased gastric acid secretion.
- A chronic ulcer penetrates the mucosa into the muscle coat, leading to fibrosis.
- Fibrosis causes deformities as pyloric stenosis.
- When an ulcer heals, a scar can be observed.
- Presence of Posterior and anterior duodenal ulcers is referred to as ‘kissing ulcers’.
- Anterior duodenal ulcers tend to perforate.
- posterior duodenal ulcers tend to bleed by eroding gastroduodenal artery.

Types and etiology of gastric ulcers

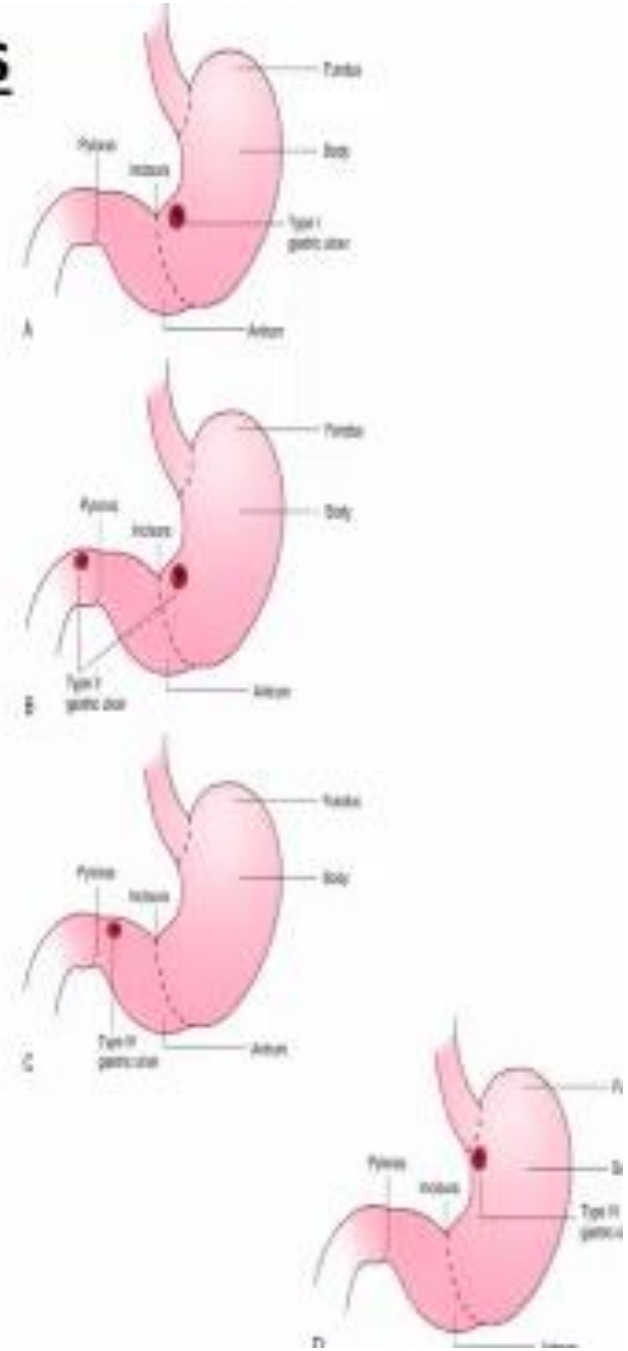
Type I (on the lesser curve- body)

Types II (GU+DU).

Type III (pyloric antrum or canal).

Type IV (paracardiac)

- **gastric ulcers 1,2 are defects in mucosal protection. type 3(due to acid hypersecretion and behave like DU).**
- **Gastric ulcer? turn malignant (DU not).**



Gastric ulcers

- Due to decreased gastric protection (Cytoprotection) (bicarbonate and mucus layer)(HCL is normal or low).
- Fibrosis, may result in rarely seen **hourglass stomach**.
- Large chronic ulcers may penetrate posteriorly through the pancreas or major vessel as the splenic artery.
- Less commonly, they may erode transverse colon.
- Chronic gastric ulcers are much more common on the lesser curve(especially the incisura angularis) than the greater curve.
- It is fundamental that any gastric ulcer may be malignant, so multiple biopsies should be taken

Other Gastric Ulcers

Carcinoma

Lymphoma

Lieomyoma

Sarcoma

Crohns

TB

Behjete

Clinical Manifestations

- Young and middle age patients
- Pain or one of the complications:

- ❑ **Perforation.**
- ❑ **Bleeding.**
- ❑ **Obstruction (**pyloric obstruction, hour glass stomach).
- ❑ **Penetration.**
- ❑ **Malignant transformation** in gastric ulcer

Abdominal pain

The most common symptom is epigastric pain: well localized /tolerable/frequently relieved by food(DU) but in GU food brings more pain. pain may be episodic, seasonal in spring, autumn (periodicity),may relapse due to stresses as work, worry, weather.

For these reasons and because it is relieved, many patients seek medical advice late.

Constant pain, referred to the back= deeper penetration of the ulcer (penetration into the pancreas).

perforation….peritonitis.

1. Perforation

- Acute/chronic(penetration)
- **Pathology 3 Stages:**

A. **Chemical peritonitis** (peritonism as HCL sterilizes gastroduodenal contents) **THEN**

B. **Delusion (masked peritonitis)(**6-12 hours due to neutralizing peritoneal exudate) **THEN**

C. **Septic (bacterial)peritonitis**

- NSAIDs is a possible cause.
- 80% of pts have peptic dyspepsia. 20% perforation of silent ulcer

- **Clinical features:** patient with a history of peptic ulcer, develops sudden sever abdominal pain/nausea/vomiting.
- The Patient is feverish/tachycardic/dehydrated with ileus.
- Abdominal examination reveals peritonitis(R/T/RT).
- A hallmark: free air under the diaphragm on upright chest radiograph (70-75%).
- **Treatment**: emergency surgery after resuscitation(fluid and electrolyte repletion+ anitbiotics).Choice of surgery is guided by comorbid diseases, and hemodynamic stability during the operation.

  DU= Omental patch (Graham patch)(will not prevent recurrence) followed by medical treatment? (may be vagotomy+ pyloroplasty in hemodynamically stable patients with less contamination).

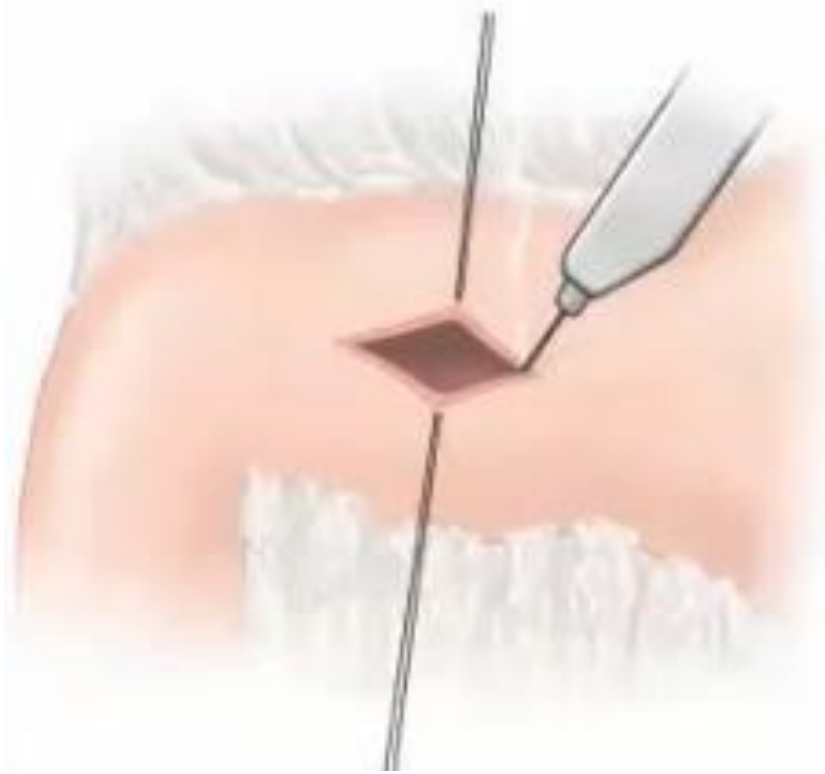
GU= Omental patch + biopsy+ H pylori test and treatment if positive. (may be Billroth I gastrectomy in hemodynamically stable patients with less contamination).

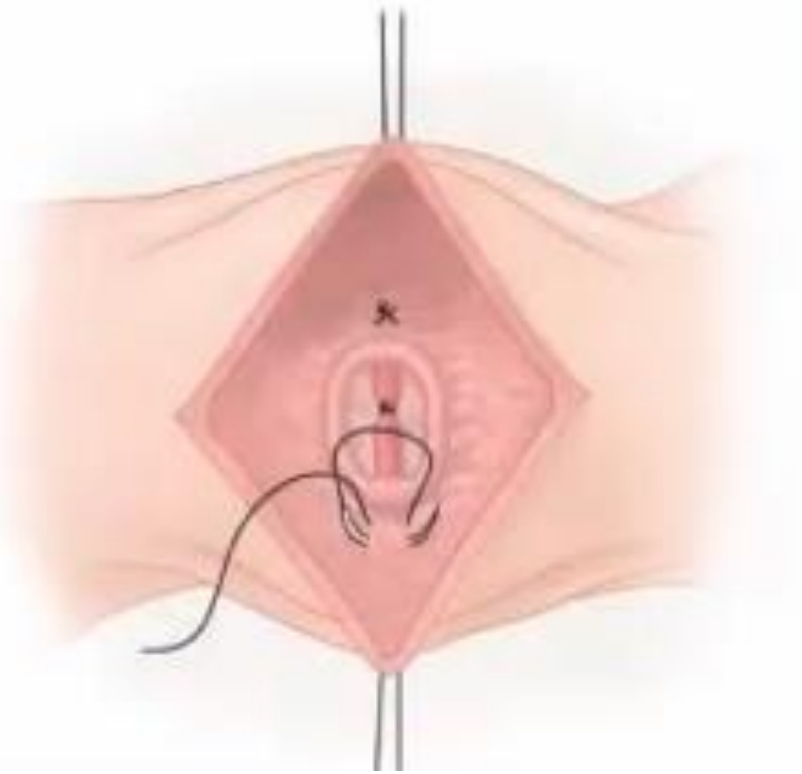
2. Bleeding

- 20% of patients with PUD develop bleeding.
- The most common cause of death in patients with peptic ulcer is bleeding in patients with major medical problems or older than 65 years.
- Peptic ulcer disease is the most common cause of UGI bleeding. 80% of bleeding ulcers will stop with conservative measures (self-limited). Duodenum has abundant blood supply and gastroduodenal artery lies directly behind the first part duodenum.
- Hematemesis/melena.
- **The Initial step in the management is resuscitation followed by endoscopy=** tool of choice for UGI bleeding (diagnostic /therapeutic).

- **Mortality** increases with age/severity of initial bleeding/ presence of shock/higher transfusion requirement/bright red blood in NG tube or in the stool/Recurrent bleeding /concomitant disease/Visible vessel seen at the base of the ulcer during endoscopy even intact(55% risk of re-bleeding) /spurter.
- When bleeding is controlled, long-term medical therapy includes PPIs + H. pylori test and treatment if positive.
- If bleeding continues or recurs= surgery(over sewing the ulcer +/- truncal vagotomy+ pyloroplasty)(parietal cell vagotomy is time consuming).
- **For bleeding gastric ulcers, Billroth I gastrectomy is usually performed.**

In general, surgery for peptic ulcer bleeding is indicated earlier in older patients because vessels are atherosclerotic (less likely to stop bleeding spontaneously)+ diminished perfusion of the heart, brain and kidneys is less well tolerated
At surgery, the gastroduodenal artery is oversewn+ vagotomy and drainage procedure.





3. Obstruction

- Gastric outlet obstruction is manifested by delayed gastric emptying, anorexia, nausea and vomiting.
- Patients are dehydrated+ hypochloremic, hypokalemic metabolic alkalosis. Fluid resuscitation(replacement of chloride and potassium deficiencies)+ nasogastric suction to relieve obstructed stomach.
- Gastric outlet obstruction may be **functional** (acute inflammatory edema) or **organic** (chronic inflammation+ fibrosis and scarring with narrowing of the lumen).
- Stomach becomes massively dilated, rapidly loses its muscular tone. Marked weight loss, malnutrition are also common.

Vomitus: foul/frothy/not bile stained/at the end of the day /not relieving the pain/contain undigested food).

Treatment:

- categorize the patient as acutely or chronically obstructed.
- **Acute obstruction=** treat non-operatively with NGT /IVF /nutritional support/acid suppressive therapy/test and treat H. pylori.
- **Chronic obstruction=** surgery.

Preoperative: NGT/Correct fluid, electrolyte imbalance/ Anti-secretory therapy/Endoscopy, biopsy.

Operative: Gastrectomy(**Billroth 1 OR 2?**can be done if easy, Alternatively: truncal vagotomy+ gastrojejunostomy

Gastric emptying

- **Delayed:** truncal vagotomy/diabetes/myxedema/ mechanical gastric outlet obstruction/hypokalemia/ anti-cholinergic or opiate drugs.
- **Rapid:** ZES/retained gastric antrum syndrome/ steatorrhea /massive small bowel resection where there is impaired ability to reduce HCL secretion. Failure of switch-off mechanism to inhibit acid secretion also results in rapid gastric emptying.
- **Acute gastric dilatation** may result in a vasovagal response characterized by marked gastric and abdominal distension. These are clearly demonstrable in an awake patient, But may occur in anaesthetic patient and thus go unrecognized.
- Vomiting, aspiration, hypoxia, or bleeding from erosive stress gastritis may occur.

Zollinger-Ellison syndrome(gastrinoma)

- A triad of **(Basal gastric acid hypersecretion + severe peptic ulcer (duodenal) disease+ non B islet cell tumor of pancreas or duodenum).**
- Basal acid secretion is increased above 15 mEq/h
- These tumors secrete gastrin and known as gastrinomas.
- It accounts for 0.1–1% of peptic ulcers

Diagnosis:

- History and physical examination+ Upper GIT endoscopy
- Fasting serum gastrin level(normal 60 pg/mL; in ZES >150 and can be over 1000 pg/mL or 100.000) in patients with **ulcers (refractory/recurrent / multiple /unusual sites).**

Gastrin level in ZES

- Gastrin is produced in the antrum(not in the fundus), duodenum, and small intestine. When distal stomach is removed gastrin levels decrease significantly.
- Gastrin stimulates parietal cells to secrete acid and stimulates chief cells to secrete pepsinogen.
- Most patients with gastrinoma have serum gastrin levels> 500 pg/mL.
- If mildly elevated (<200–500 pg/mL) a provocation or stimulation test with intravenous cacium or secretin is performed to confirm the diagnosis, Arise of 200 pg/mL after 15 minutes, or a doubling of the fasting level is diagnostic.

- ZES can occur sporadically or as part of multiple endocrine neoplasia (MEN I)
- 25% of ZES patients have MEN 1.
- ZES is due to a true pancreatic tumor in adults, but may be secondary to hyperplasia in children. Growth of the tumor is usually slow and survival is often prolonged.
- If an isolated tumor is found on CAT scan= surgical resection is indicated.
- About 2/3 of these tumors are malignant.

Helicobacter pylori testing

Urea breath test: based upon the ability of H. pylori to convert urea to ammonia and **CO_2**.(96% sensitive – 94% specific).

Patients swallow urea labelled with an isotope(either radio-active carbon 14 or non-radioactive carbon 13). In the subsequent 10–30 minutes, the detection of isotope-labelled **CO_2** in the exhaled breath indicates that the urea was split by urease enzyme secreted by H. pylori present in the stomach.

Mucosal biopsy: (histopathology examinations)(biopsies).

Blood test: serology detect IgG antibody(remain positive for 1 year post treatment)

Stool test (Fecal Ag test): detect protein Ag.

Breath and stool test are more accurate than blood test in detecting active H pylori infection(differentiate active/past infection).

PPIs-Bismuth-Antibiotics can interfere with test accuracy so better to be stopped 2 weeks pre-test)

Treatment :

- In 2005, **Barry Marshall** and **J. Robbin Warren** won the Nobel Prize in medicine for their work on H. pylori and its role in gastritis and peptic ulcer disease.
- 2 weeks treatment by (PPI+ 2 antibiotics)

  = Omeprazole + Clarithromycin + amoxicillin.

Treatment

Medical treatment (heals in 4-6 weeks).

- **6 weeks then endoscopy.**
- **Rest: physical-mental.**
- **Bland diet…small frequent meals/avoid heavy meals/avoid irritant food(hot-spicy)/stop smoking, Alcohol, NSAIDs.**
- **Antacids like aluminium hydroxide.**
- **Anticholinergic xx??.**
- **H2 blockers:CIMETIDINE-Ranitidine- Famotidine.**
- **PPIs: omeprazole.**
- **Sucralfate (gastrofate)**

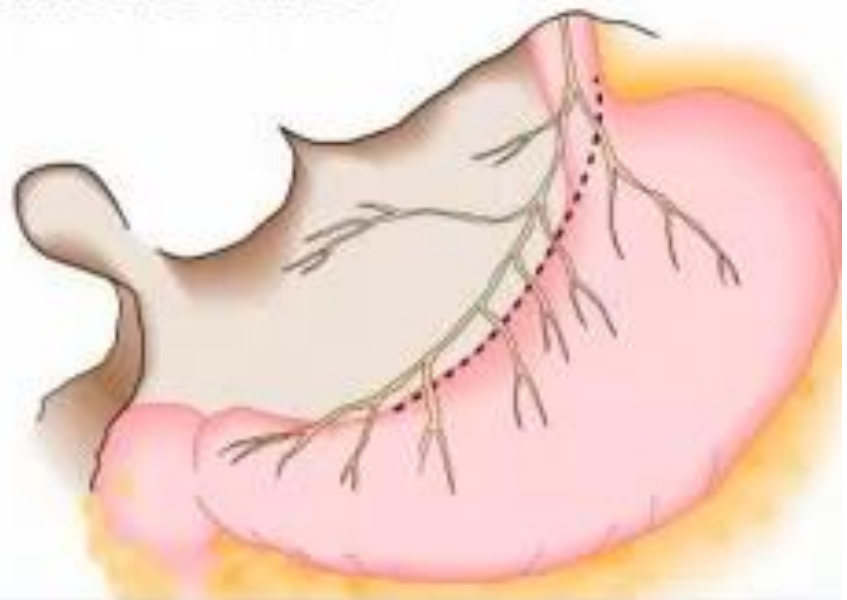
Surgical Procedures for Peptic Ulcer Disease

Vagotomy

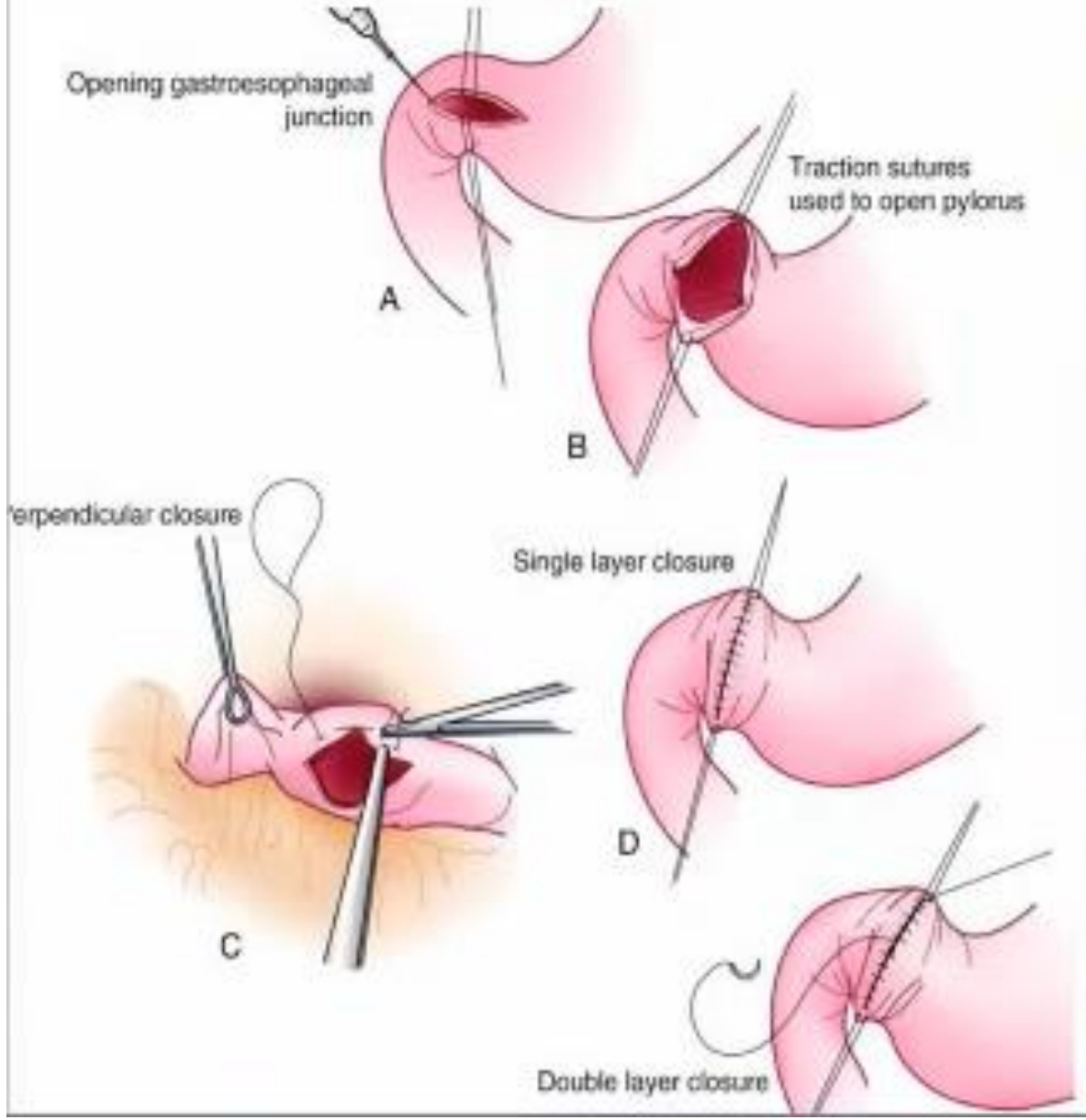
1. **Truncal Vagotomy with Drainage Procedures (Dragstedt 1948) (pyloroplasty Vs gastrojejunostomy)(40% of cases have gastric stasis).**
2. **Selective vagotomy with drainage procedures.**
3. **Highly Selective Vagotomy (parietal cell vagotomy).lower complications but higher recurrence rate.**

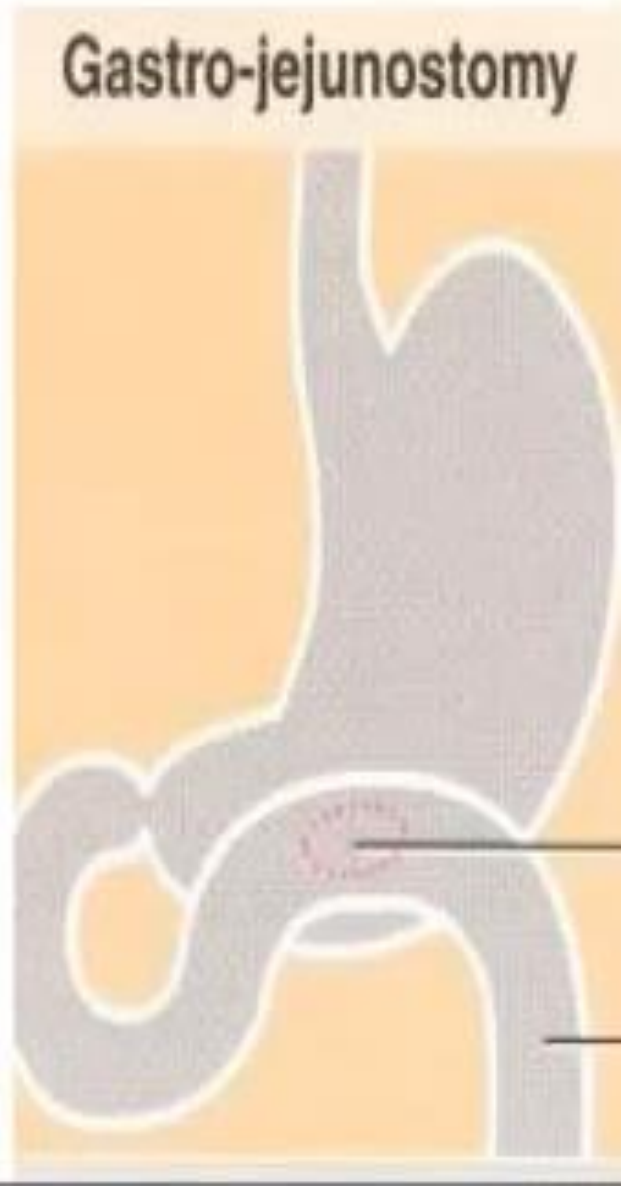
Taylor procedure:

posterior truncal vagotomy+ anterior seromyotomy **to avoid proximal(criminal) nerve of grassi.**



Heineke-Mikulicz pyloroplasty Vs gastro-jejunostomy





Gastrectomy

Billroth1:

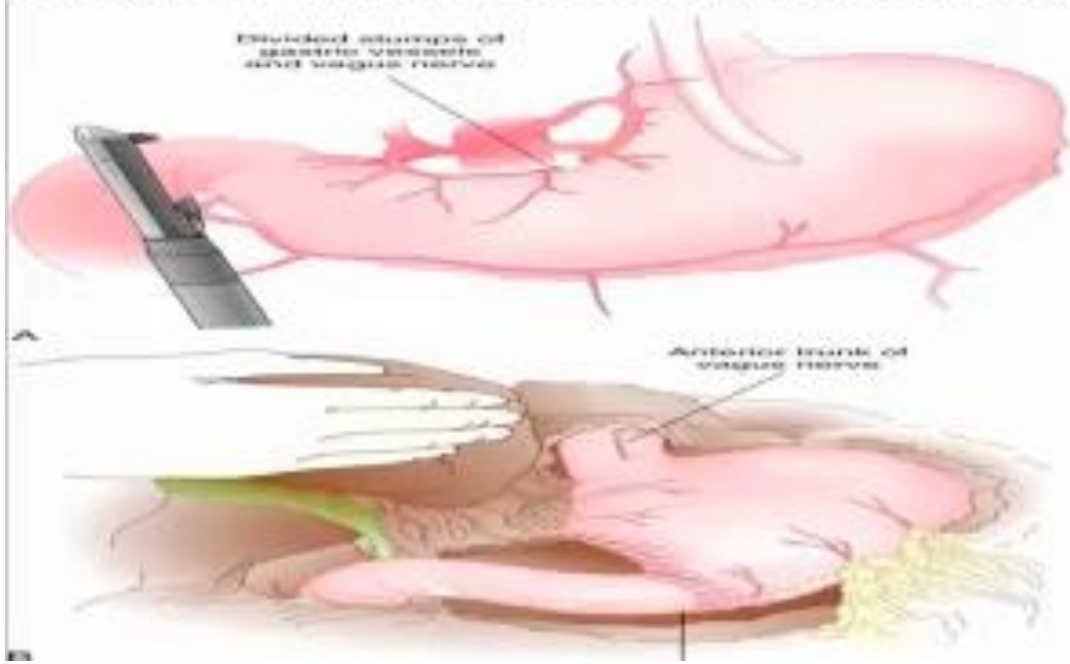
Hemigastrectomy + gastroduodenal anastomosis. for GU??

Billroth II:

Hemigastrectomy+ side to side gatrojejunostomy.

if end to side anastomosis = polya gastrectomy.

Polya with hofmeister valve to avoid reflux alkaline gastritis.



Postoperative Complications of Peptic Ulcer surgery

- Early complications :afferent loop obstruction /efferent loop obstruction /bleeding/stomal obstruction/duodenal stump blow-out
- Post-Vagotomy complications. + Gallstones
- Malignant transformation
- Post-gastrectomy Syndromes: Dumping syndrome/ metabolic

Dumping Syndrome

- Symptoms follow ingestion of a meal when portion of the stomach has been removed or normal pyloric sphincter mechanism has been disrupted.
- Dumping syndrome exists in either late or early forms, early form is more frequent.

❑ **Early dumping(hypovolemia):**

- Within 30 minutes following ingestion of a meal .
- Gastrointestinal symptoms: nausea/vomiting/epigastric fullness/Eructation/cramping abdominal pain/explosive diarrhea due to afferent loop distention by bile-pancreatic juices
- Cardiovascular symptoms :palpitations/tachycardia/ diaphoresis/fainting/dizziness/ flushing/blurred vision.

- This is because gastrectomy or interruption of pyloric sphincteric mechanism . The stomach can not prepare its contents or deliver them to the proximal bowel as small isotonic particles.
- Resultant hypertonic bolus passes to small intestine, induces rapid shift of intravascular fluid into the intestinal lumen to achieve isotonicity.
- Following this shift of extracellular fluid, luminal distention occurs and induces autonomic responses listed earlier.
- Dietary measures are usually sufficient to manage these patients.

Late Dumping: pancreatico-cibal asynchronism (HYPOGLYCEMIA)

Starts 2 hours (1-3) after meal.

- **Due to rapid gastric emptying. carbohydrates are delivered to the small intestine, quickly and absorbed, resulting in hyperglycemia . That triggers the release of huge amounts of insulin. This results later in profound hypoglycemia.**
- **This activates adrenal gland to release catecholamines, which results in diaphoresis, tachycardia, confusion.**
- **These patients are advised to ingest frequent dry small meals with low carbohydrate.**

Metabolic disturbances

Anemia: **Iron deficiency anemia:** due to

- ❑ **Decreased intake.**
- ❑ **Impaired absorption(site of absorption=duodenum) is bypassed(Billroth 1or 2??)/no binding to gastroferrin protein/An acid environment is necessary to release ferric ion from food and make it available for absorption in the small intestine.**
- ❑ **Chronic subclinical blood loss due to hyperemic, friable gastric mucosa connected to small intestine(Stoma).**

Treatment: Iron supplements.

Megaloblastic anemia: Vitamin B12 deficiency due to poor absorption /lack of intrinsic factor secretion.

treatment: IM injection of cyanocobalamin .

Metabolic disturbances

.Osteoporosis,osteomalacia: ca deficiency (duodenal absorption bypassed)

- Impaired absorption of fat, steatorrhea post Billroth II gastrectomy as a result of inadequate mixing of bile salts and pancreatic lipase with ingested fat because of duodenal bypass.

Treatment: pancreatic replacement enzymes.

Efferent loop obstruction

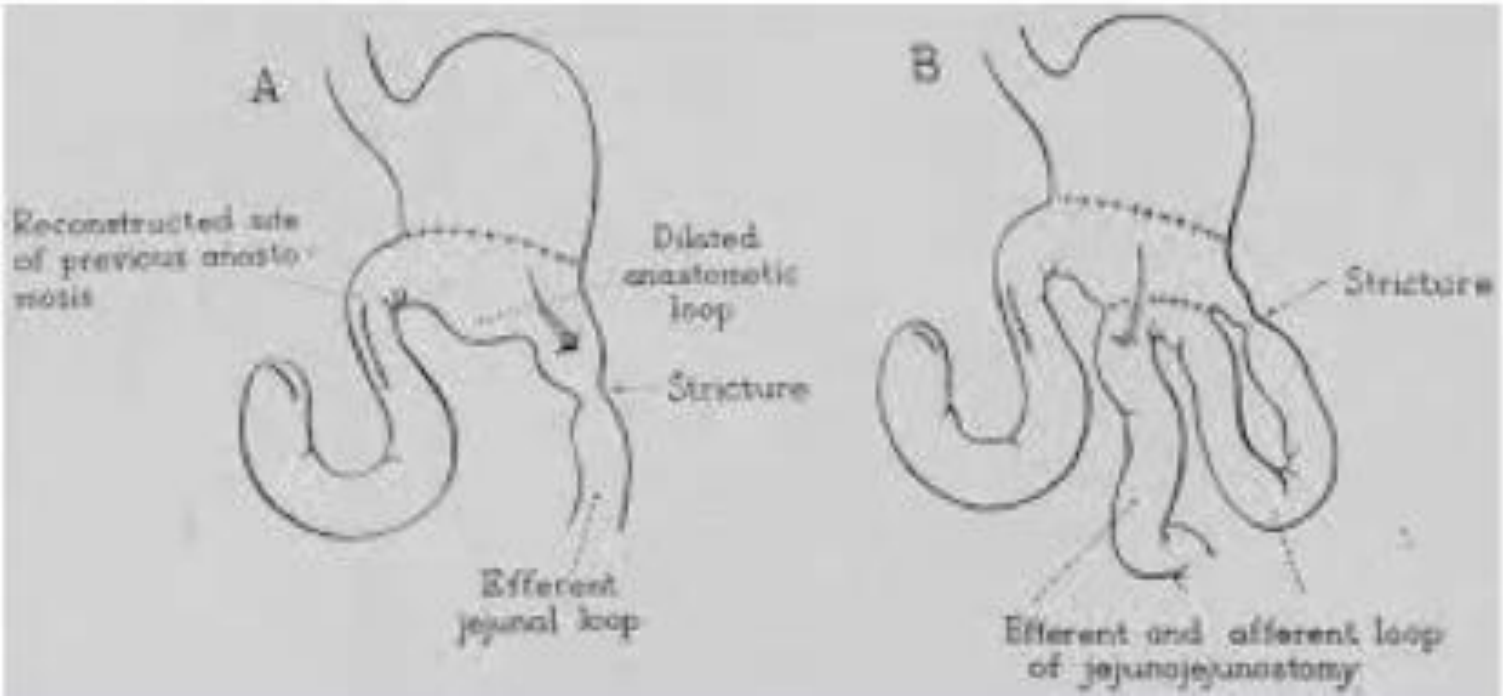
Commonest causes of efferent loop obstruction:

***Herniation of the limb behind the anastomosis.**

***Stomal edema.**

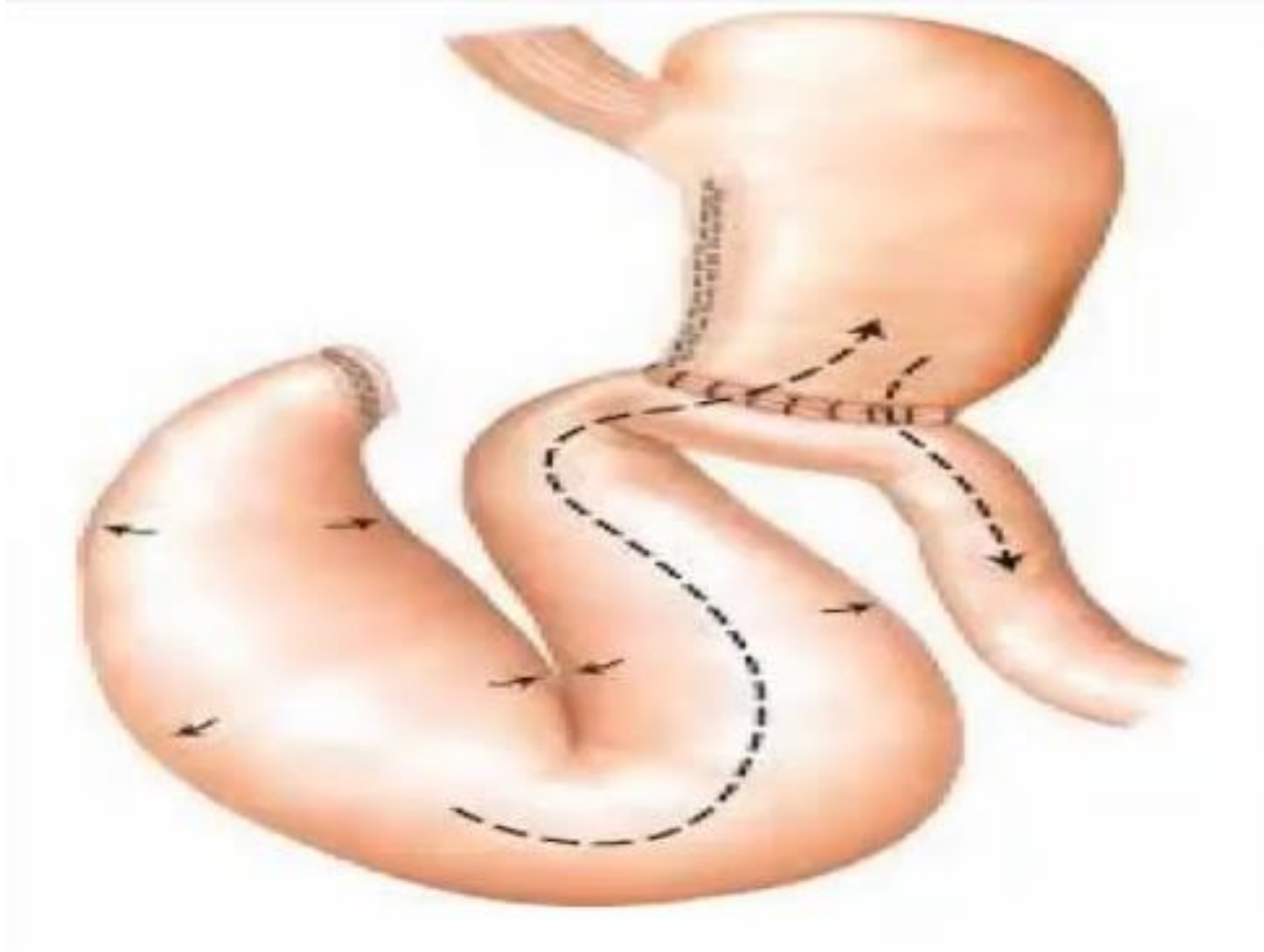
***Jejuno-gastric intussusception.**

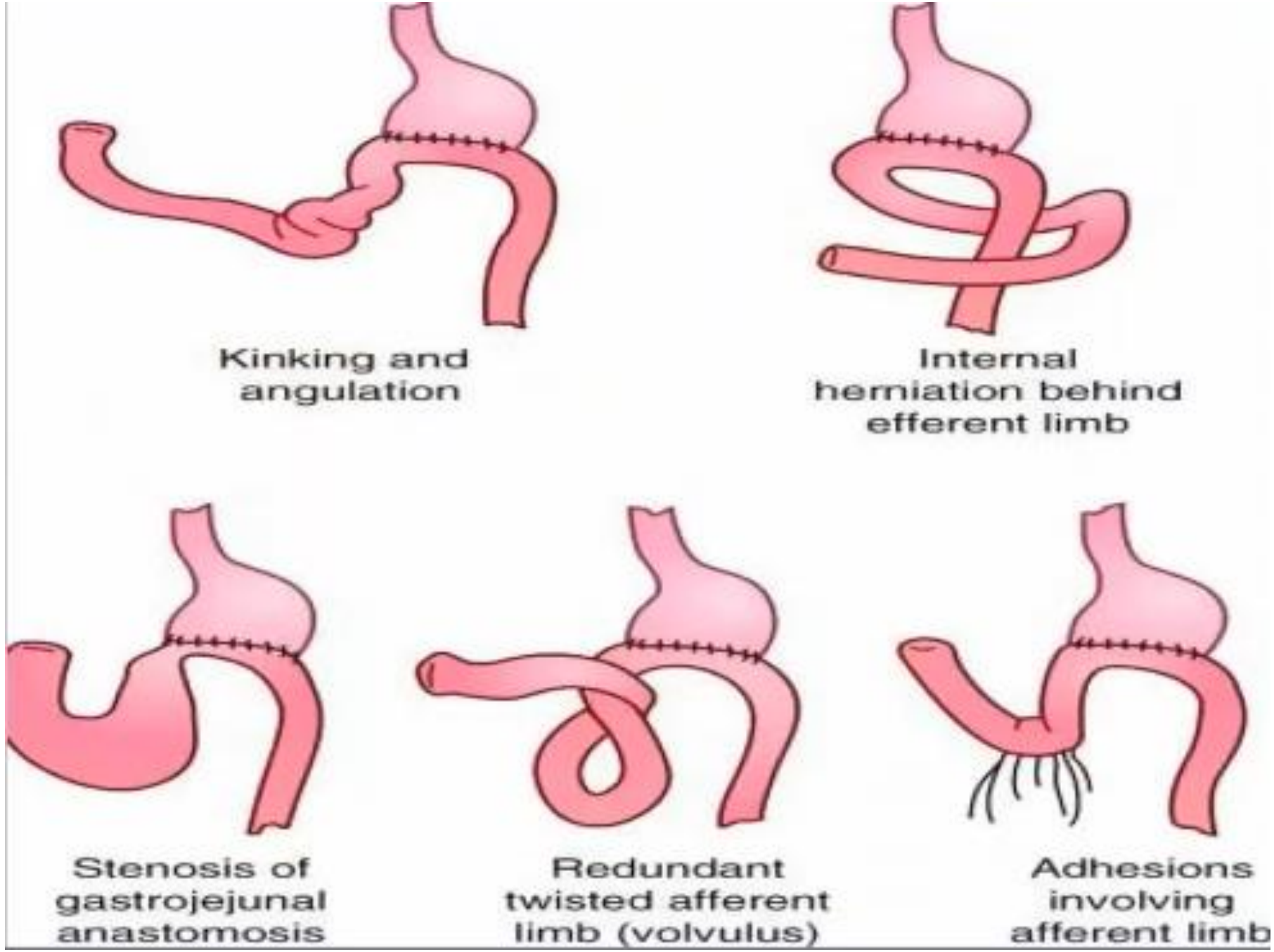
Surgery is necessary: reduce retro-anastomotic hernia and close retro-anastomotic window to prevent recurrence



Afferent loop syndrome

- Longer afferent limb more than 30 cm.
- Following obstruction of the afferent limb, accumulation of pancreatic-biliary secretions(in response to food ingestion) within the limb, resulting in its distention causing epigastric discomfort and cramps.
- In partial obstruction, intraluminal pressure increases to forcefully empty its contents into the stomach, results in a projectile bilious vomiting that offers immediate relief of symptoms. But NO food contents within the vomitus.
- In complete persistent obstruction: blind loop syndrome/ duodenal stump blow out/pancreatitis.
- Surgery is indicated(mechanical problem/not functional): enteroentrostomy.




Kinking and angulation
Internal herniation behind efferent limb
Stenosis of gastrojejunal anastomosis
Redundant twisted afferent limb (volvulus)
Adhesions involving afferent limb

Post-vagotomy complications

1.Diarrhea

- Approximately 30% of patients suffer from diarrhea after gastric surgery. it is mild(rarely fulminant) and disappears within 4 months.
- May be due to dumping syndrome/vagotomy due to drainage procedure so more with truncal than selective or highly selective Vagotomy.
- Post-vagotomy diarrhea usually resolve over time. If not, cholestyramine (chelates bile salts and renders them unabsorbable) decreases the severity of diarrhea.

2. Gall stones due to denervation.

Malignant transformation

- Gastrectomy or vagotomy with drainage are independent risk factors of gastric cancer(four folds risk)(10 years).
- Reflux alkaline gastritis(Bile) and gastric cancer(intestinal metaplasia)are linked.
- Highly selective vagotomy does not have increased risk.

Cushing's ulcer: PUD/gastritis with neurologic trauma or tumor.

Curling's ulcer: PUD/gastritis with major burn injury.

Marginal ulcer: ulcer at the margin of GI anastomosis (stoma).

Ménétrier's disease-hypertrophic gastritis

Rare, characterized by gross hypertrophy of gastric mucosal folds+ mucus production+ hypochlorhydria + GI protein loss

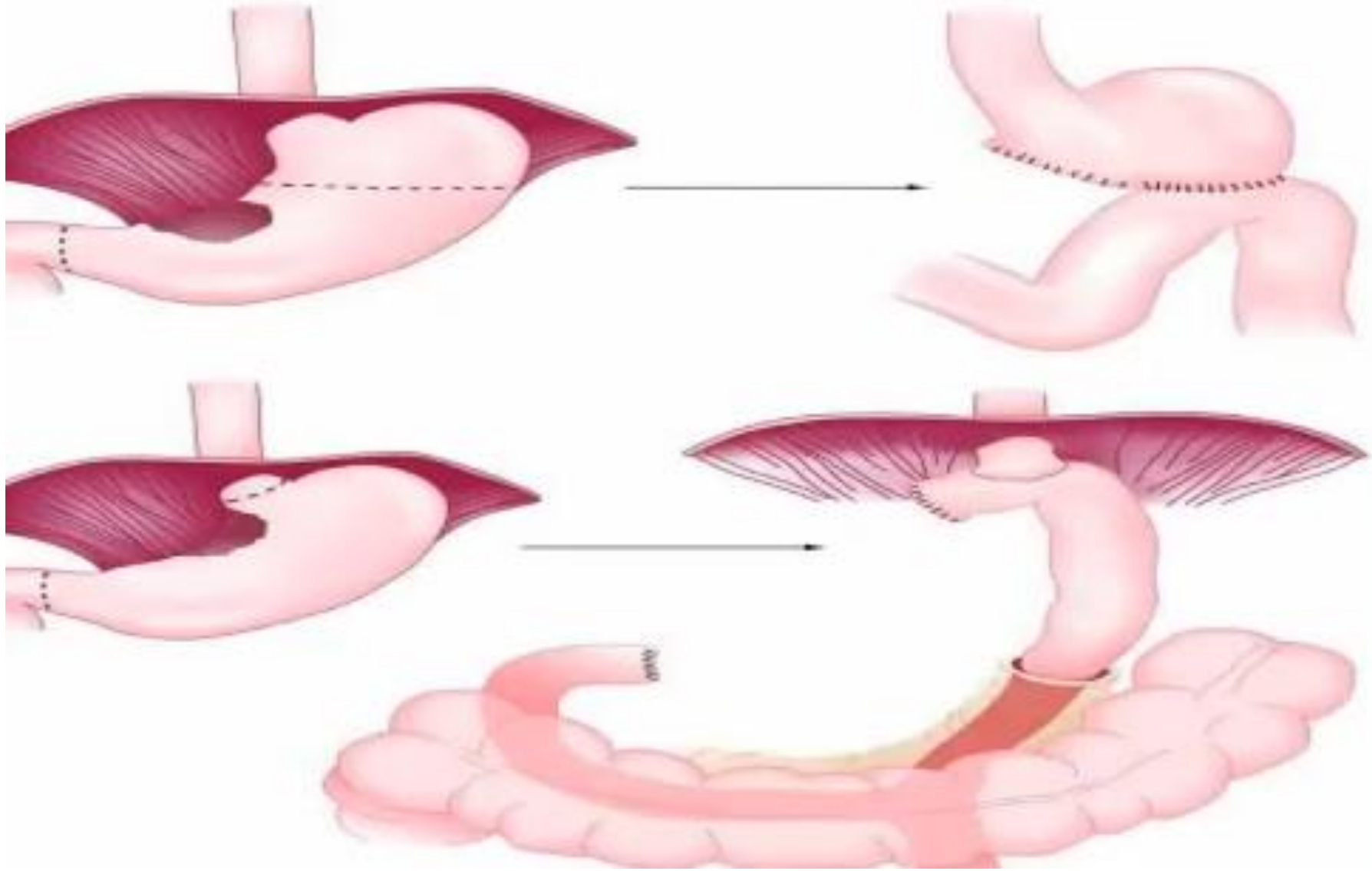
Pre-malignant?.

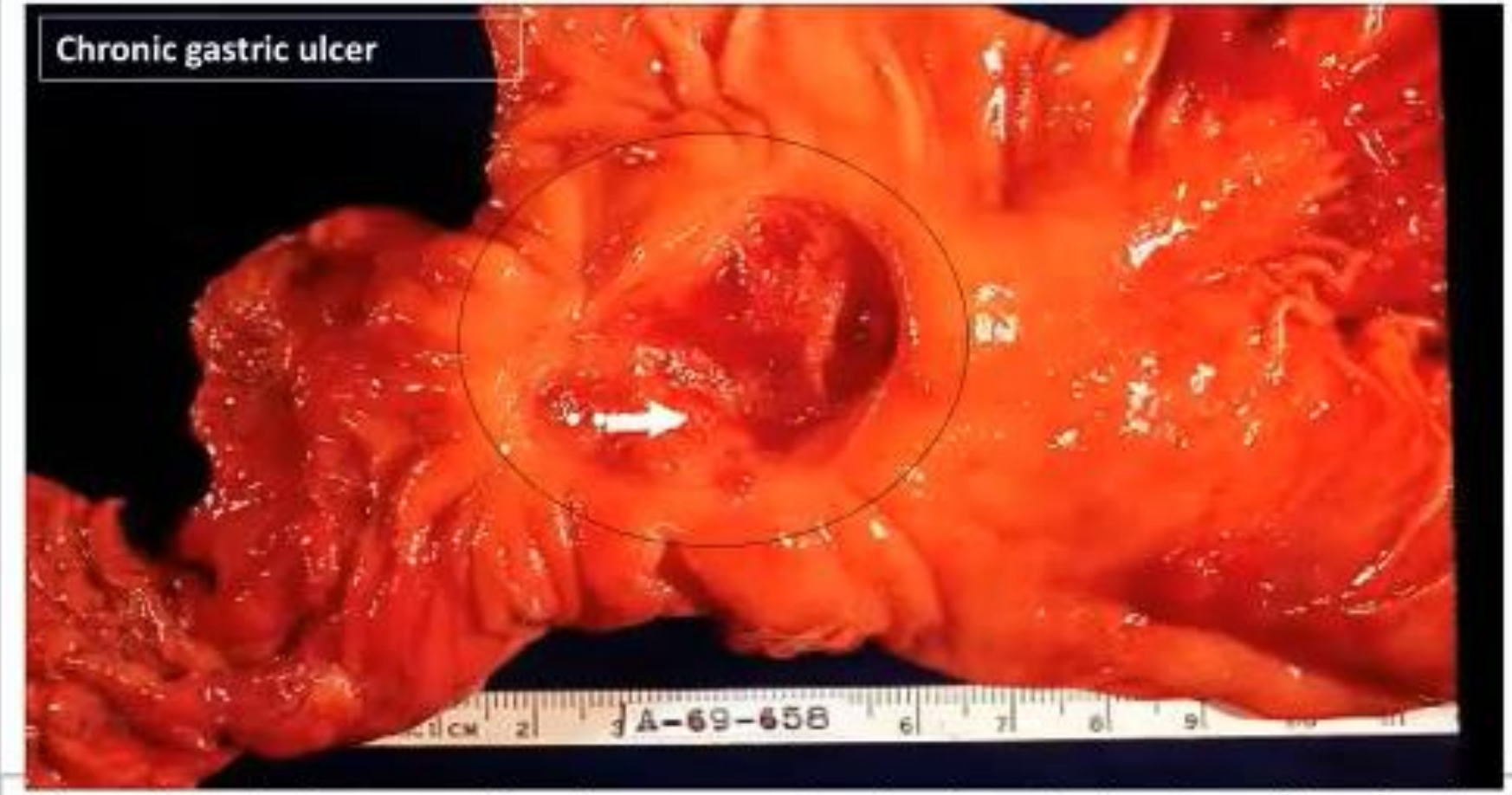
Presentation: hypoproteinemia and anemia.

Treatment i: maintain adequate nutrition+ gastrectomy to prevent cancer developing.

Caused by over expression of transforming growth factoralpha (TGFα).

Gastrectomy

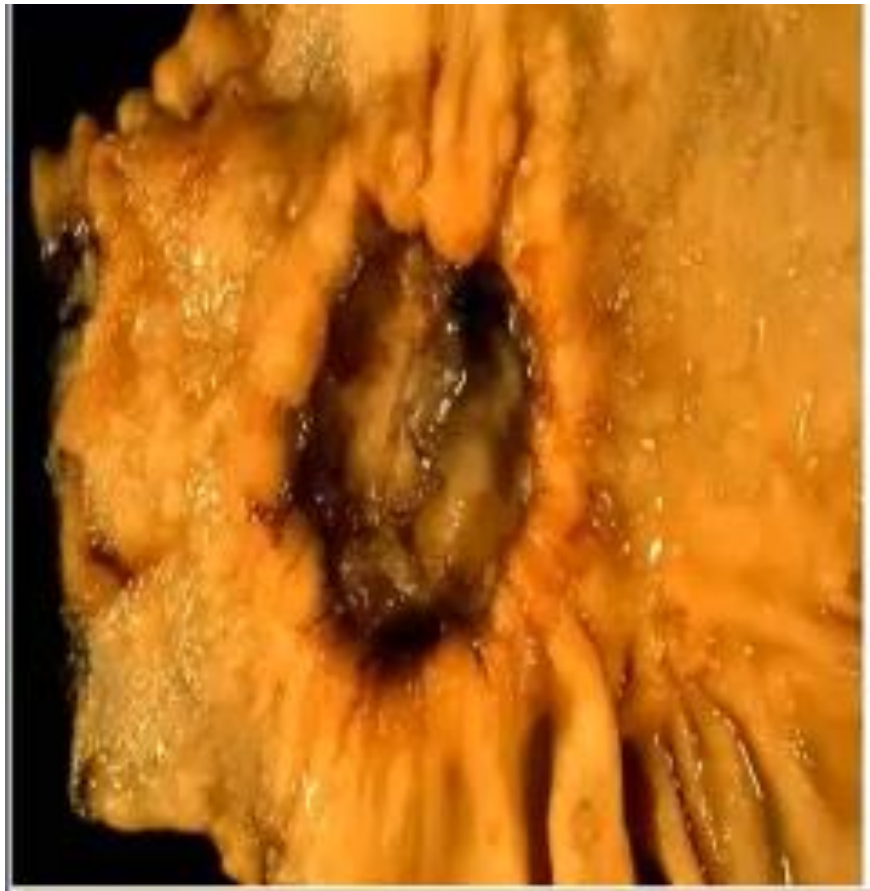


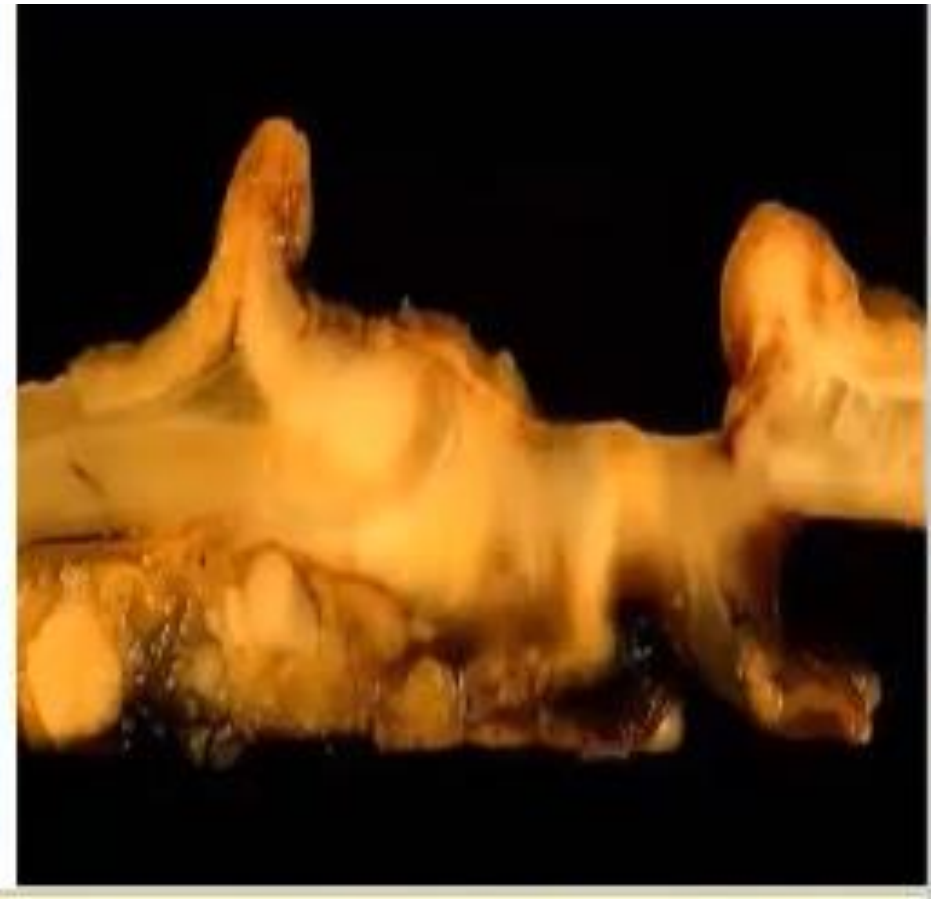


The ulcer is deep, with sharp proximal edge & sloping distal edge

The arrow points to eroded gastric artery which has caused fatal hemorrhage

What are the complications of chronic gastric ulcer?





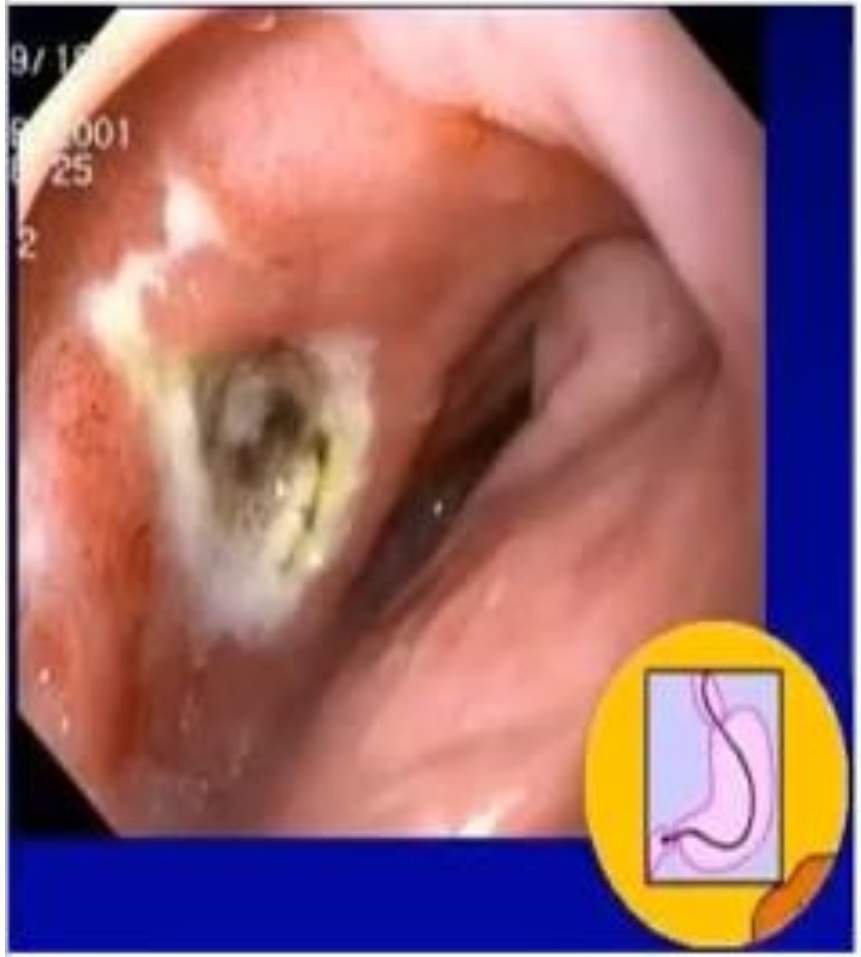
Malignant gastric ulcer (Adenocarcinoma)

The left view: ulcer is suspicious.

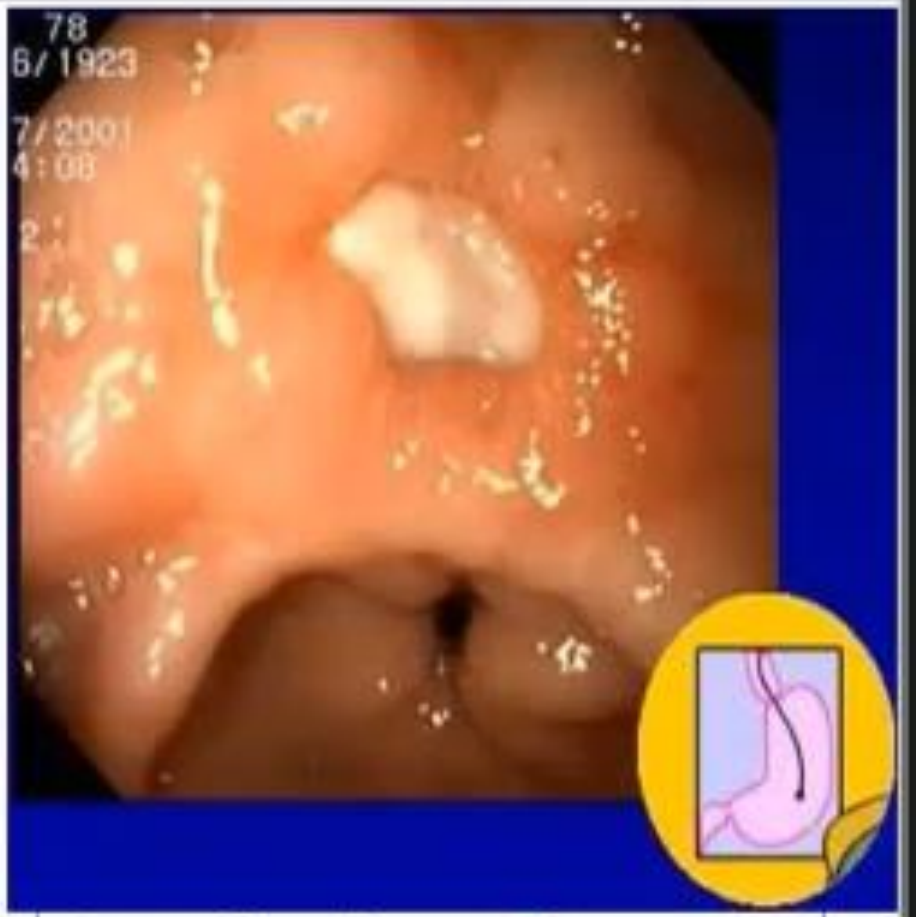
Longitudinal section: pylorus is to the left. Edges are everted
Several metastatic nodes within the lesser omentum.

Investigations for suspected peptic ulcer

Gastro-duodenoscopy is the most sensitive investigation



Duodenal ulcer



Gastric ulcer, Biopsy

Barium meal –2 duodenal kissing ulcers

Two well-defined filling excesses facing each other on the opposite contour of the duodenal bulb (arrows)