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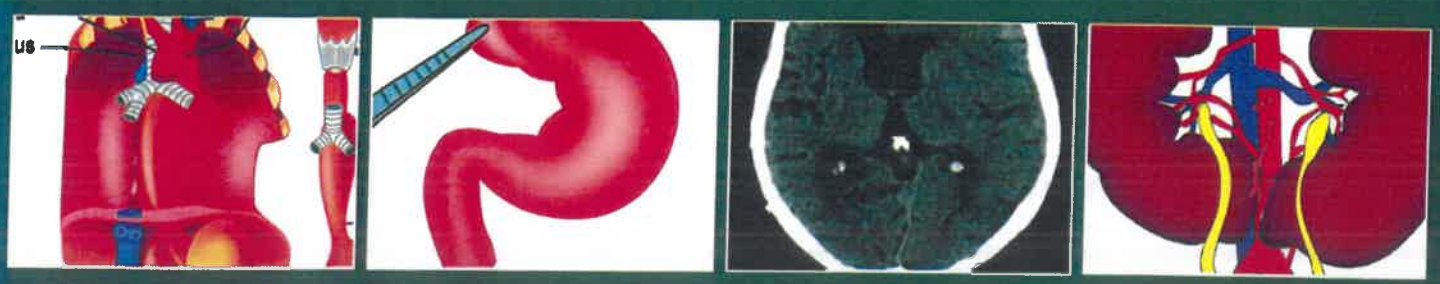

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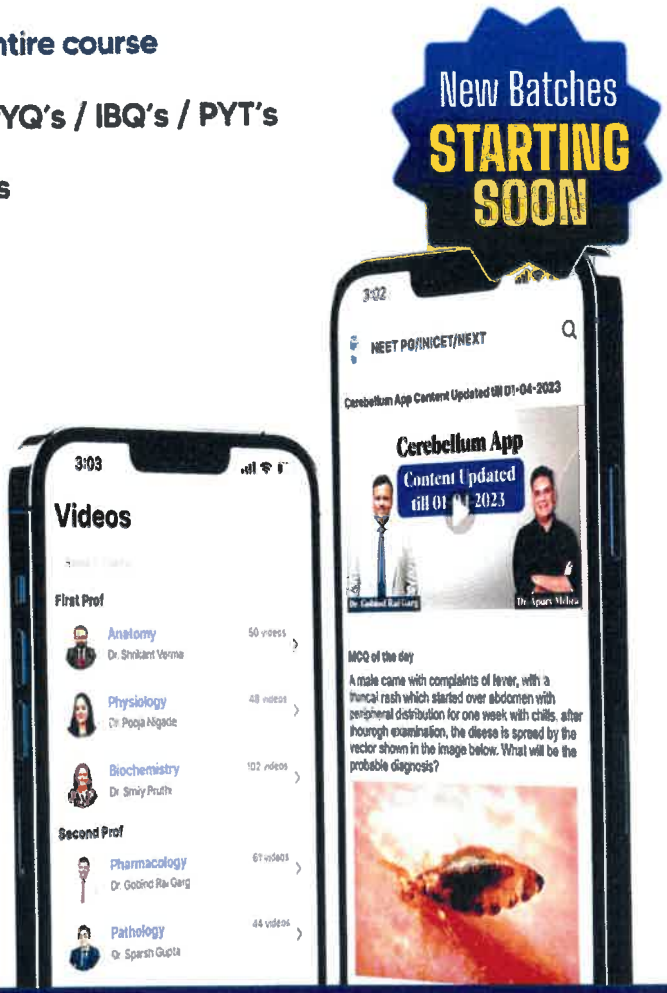

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Table of Content

List of Topics OPTIONAL for FMGE-Aspirants (Less Important):

1. Billiary tree
2. Transplant surgery
3. Bariatric surgery
4. Elective Neurosurgery and Cardiothoracic surgery
5. Miscellaneous in GI and HPB

Section 1

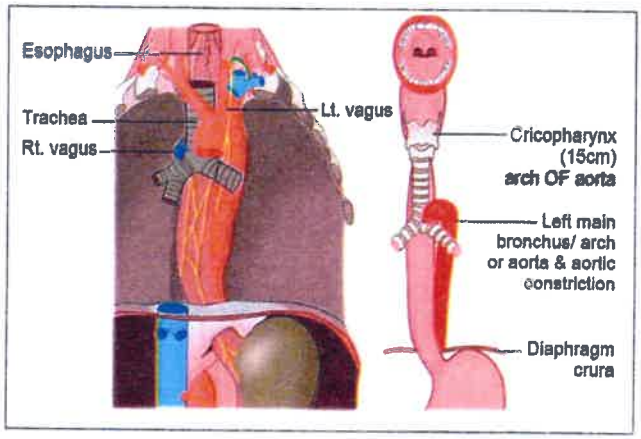
Gastrointestinal Tract

1 Chapter

ESOPHAGUS PART - 1

ANATOMY OF ESOPHAGUS

- **Located at 3 regions -**
  - i. Upper part in neck (cervical esophagus)
  - ii. Thorax (middle part, longest part)
  - iii. Abdominal esophagus
- **Upper esophagus:** cricopharyngeal sphincter present (muscular sphincter)
- **Lower esophagus:** lower esophagus sphincter (LES) present - physiological sphincter
- Trachea lies in front of esophagus in neck and thorax.
- 3 parts where esophagus has physiological narrowing which are measured from upper incisors:
  - i. 15 cm from upper incisors cricopharynx (narrowest part of esophagus)
  - ii. 25 cm indentation of Arch of aorta/left main bronchus
  - iii. 40 cm diaphragmatic crura



Blood Supply And Lymphatic Drainage

- Upper part of esophagus - receives blood supply from inferior thyroid artery
- Middle part of esophagus - receives blood supply from descending thoracic aorta (Direct branches) and bronchial artery branches
- Lower part - receive blood supply from celiac trunk (left gastric artery) and from lateral branches of abdominal aorta (inferior phrenic artery)
- GI organs metastasize to lymph nodes across 3 stations:

1st station - on surface of the esophagus (peri-esophagus group)

2nd station - along blood vessels - inf. thyroid artery/ branches of thoracic aorta/left gastric artery

3rd station - at the root (origin) of the vessels

- Level 5,6 neck nodes
- Para-aortic nodes in thorax
- In abdomen - pre-aortic (LGA)/para-aortic (inferior phrenic artery)

- During lymphadenectomy, we will remove level 1, 2, 3 lymph node stations
- Esophagus has rich submucosal lymphatic plexus, so that's why esophagus metastasis can move across all 3 stations.
- Lower esophageal cancer can have LN mets in thorax/neck.

Applied Anatomy of Esophagus - Prevention of Reflux

1. LES is a physiological sphincter - has a resting pressure of 10 to 25 mmHg - keeps the esophagus closed
2. Pinch cork effect of diaphragmatic crura - where the esophagus pierces the diaphragm, the diaphragm has pinching effect on lower esophagus

3. Intrabdominal length of esophagus – 1 to 3 cm (also prevents reflex)
4. Rosette shape muscular fibers at GE junction
5. Angle of His (angle between lower esophagus and fundus of stomach)
6. Phrenico-esophageal ligament hitches the esophagus to the diaphragm

*Overall length of LES is 4 to 5 cm from which intra-abdominal length is 1 to 2 cm

Development Of Gut Tube

- From endoderm
- Endoderm lines the tube, with fold at head end, and fold at the tail end.
- Head end forms the foregut, central part forms midgut (longest part), lower part forms hindgut.
- Foregut vascularity comes from artery of celiac trunk
- Artery of mid gut – superior mesenteric artery, and hindgut – inferior mesenteric artery

CONGENITAL TRACHEO-ESOPHAGEAL FISTULA

- Fistula is an abnormal communication between two tubular structures like between trachea and esophagus

Associated Disease

V. Vertebral anomalies

A. Anorectal

C. Cardiac (septal defects)

T. E. TE fistula

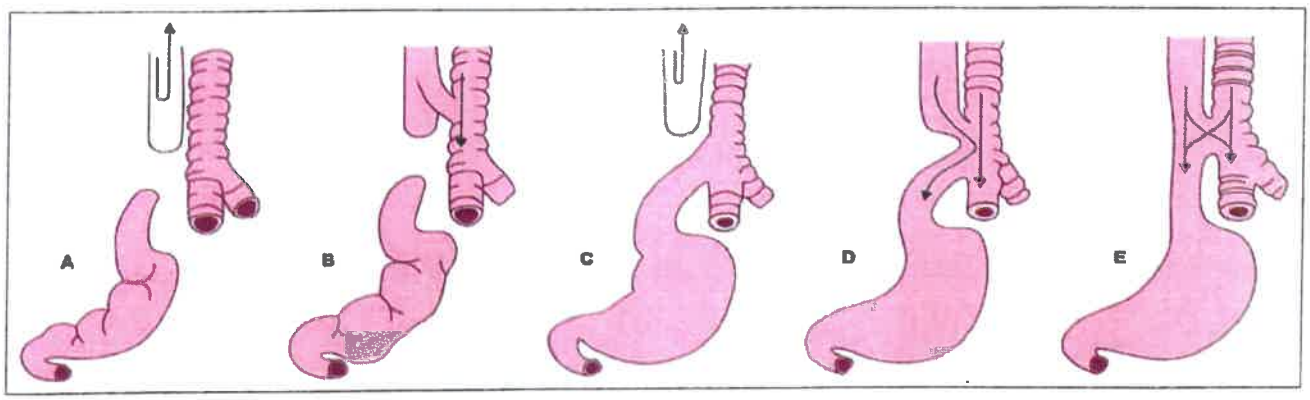
R. Renal agenesis

L. Limb abnormalities (absent radius)

- CHARGE Syndrome also associated with tracheo-esophageal fistula (coloboma, heart defects, choanal atresia, growth retardation, genital anomalies and ear anomalies)

Types And Presentation

5 types:



- Type A- no fistula, upper blind esophagus, blind stomach, no communication
- Type B- upper esophagus connect with trachea and lower esophagus is blind
  - Both type A, B have a long gap between proximal and distal segments of esophagus
- Type C- upper esophagus atresia, lower esophagus communication with trachea
- Type D- Both upper and lower esophagus are communicating with trachea separately
- Type E- H-type of configuration between upper and lower esophagus with communication
  - Type C, D, E are easier to reconstruct
  - Type A, B required some esophagus conduit to fill gap
  - Most common type is type C (85%)

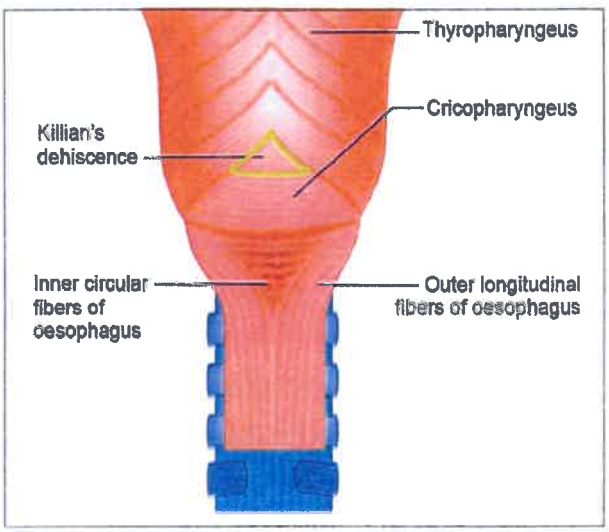
- Age of presentation – at birth (when mother tries to feed child will start choking, frothing, coughing)
- Can die due to aspiration pneumonitis

Investigation And Rx

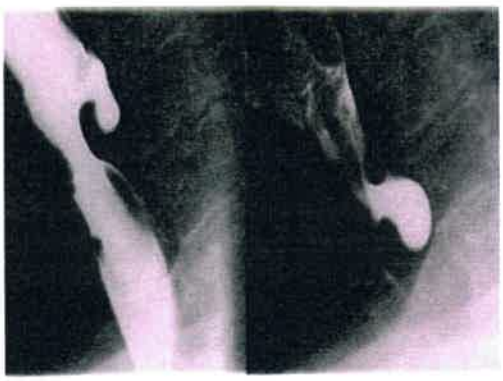
- Infant feeding tube will not pass in stomach (coiling of tube) and can be seen on X-ray, stuck in esophagus
- Air in the stomach will only be seen in type C, D, E fistula
- IOC – CECT Thorax with neck
- Rx – Reconstruction soon after birth: disconnect the fistula, reconstruct the esophagus for type C, D, E, and esophagus conduit for type A, B as gap is big

ZENKER'S DIVERTICULUM/ PHARYNGEAL POUCH

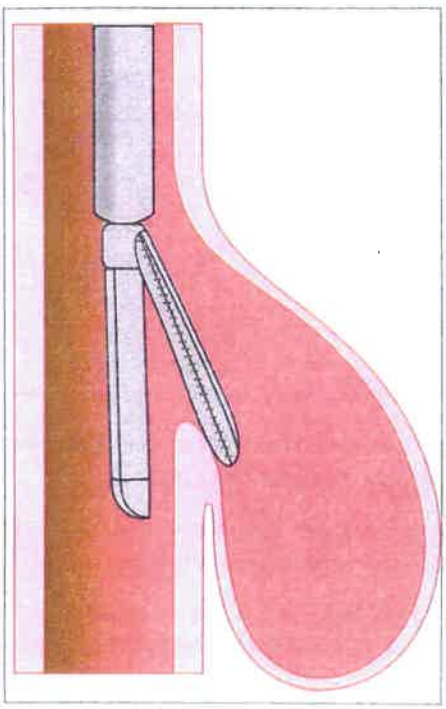
- It is an acquired, false, pulsion diverticulum
- Inferior constrictor has two fibres- thyropharyngeus and cricopharyngus
- Out pouching b/w 2 layers of inferior constrictor – mucosa of pharynx out pouches (false or pulsion diverticulum)
- Pathogenesis: abnormal motility disorder such that inferior constrictor contracts but failure of synchronised relaxation of cricopharyngeal sphincter → mucosal outpouching through Killian's dehiscence → Zenker's diverticulum (Posterolateral)



- C/F: 40-60 yrs
- On going to bed, patient complains of regurgitation of food (Earliest)
- Also c/o halitosis
- 1st investigation: upper GI endoscopy - may show mouth of diverticulum
- Diagnosis: CT with oral contrast/Ba swallow



- Rx: Surgery – Diverticulectomy + cricopharyngeal myotomy
- Nowadays, endoscopic diverticulotomy (Dohlman's procedure) or stapled diverticulopexy are minimally invasive treatments



Stapled Diverticulopexy

MOTILITY DISORDER OF ESOPHAGUS

- MC motility disorder – achalasia cardia
- MC hyper motility disorder of esophagus – nut cracker/jackhammer esophagus

ACHALASIA

- Failure to relax of LES, ineffective peristalsis in body of esophagus
- Risk: 30-60 years of age, Chaga's disease, Allgrove syndrome (Achalasia, Alacrymia, Adrenal insufficiency)
- Pathogenesis:

due to absent parasympathetic ganglia in LES

↓

LES fails to relax.

Due to reduced ganglia in body

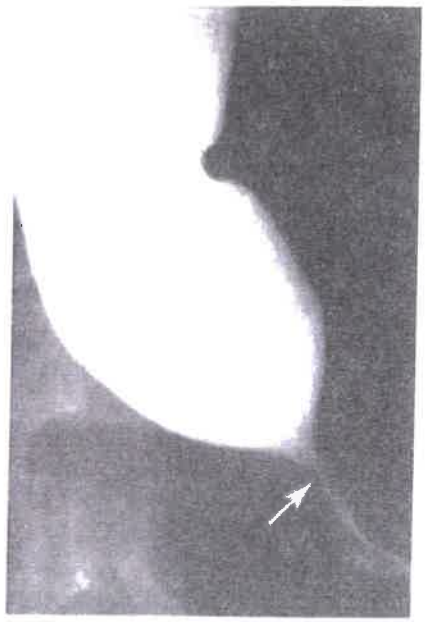
↓

peristalsis in weak/ineffective

- Sequelae: Aspiration pneumonitis, pre-malignant condition – Squamous Cell Carcinoma
- Pseudoachalasia: features of achalasia like regurgitation seen in patients of CA esophagus
- C/F: Middle aged female with clinical triad:
  1. Dysphagia – Liquids > solids (initial stage)
  2. Regurgitation
  3. Weight loss
- Clinical score for severity: Eckhardt score – includes dysphagia, regurgitation, retrosternal pain and weight loss

Investigation

1. 1st step: upper GI endoscopy
2. Esophageal manometry – investigation of choice
   - Chicago classification: manometric classification of Achalasia
   - All types will show elevated IRP (Integrated relaxation pressure) at LES:
     - Type 1: 100% failed peristalsis
     - Type 2: 100% failed peristalsis + "Pan-Esophageal pressurization" in >20% swallows
     - Type 3: 100% failed peristalsis + "Premature contractions/spasms" in >20% swallows
3. BA Swallow:



a) Bird beak/rat tail appearance

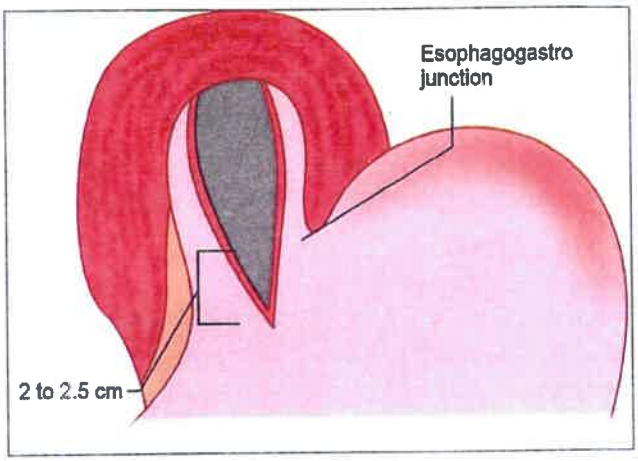
b) Lower esophageal narrowing + smooth tapering = achalasia

* Narrowing in middle + narrowing is abrupt = Ca esophagus

* Corkscrew esophagus – Diffuse esophageal spasm

- RX:
  - Medical management: calcium channel blocker – early
  - Minimal invasive surgery: inj. Botox, endoscopic dilatation of LES
  - Surgery: Heller's cardio myotomy, Per Oral Endoscopic Myotomy (POEM).

* For type 3 Achalasia, POEM with long myotomy is preferred



- Length of Myotomy in Hellers: 6 to 7cm on lower esophagus, 2 to 2.5cm on Cardia
- This will lead to Postoperative GERD, hence a partial fundoplication (180 degrees Dor or 270 degrees Toupet) is done at the same time

Extra Edge

1. Constriction at 25cm from upper incisors is due to left main bronchus (not right)
2. Middle aged female with Dysphagia - more likely to be Achalasia > Cancer
3. POEM is preferred for type 3 Achalasia

2 Chapter

ESOPHAGUS PART - 2

GERD =ACIDITY = ESOPHAGITIS

- Reflux of gastric acid into esophagus (Montreal definition)
- Acid reflux esophagitis & its symptoms
- Risk factors:
  - Anatomical defense mechanism hindered - Hiatus Hernia
  - Smoking, spirits, spicy food (Triggers)
  - Obesity (central) - ↑ intra-abdominal pressure
- Pathogenesis:

Failure of anatomical mechanisms which prevents reflux like

1. Intra-Abdominal length of esophagus (normally > 2cm)
2. Angle of his
3. Diaphragmatic Pinch
4. LES
5. Phreno-Esophageal Ligament

↓

Triggers leading to increased T.L.O.S.R

↓

Reflux of acid into esophagus

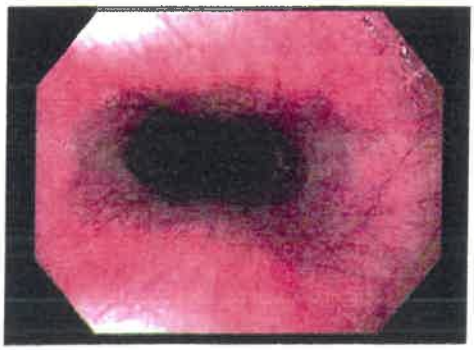
↓

Esophagitis

(TLOSR: Transient lower oesophageal sphincter relaxation)

- Sequelae:
  1. Barret esophagus - adeno carcinoma
  2. Hiatus hernia (sliding)
  3. Esophageal Stricture
  4. Schatzski Ring
- C/F: heart burn, epigastric pain, regurgitation/ volume reflex (late finding)
- Investigation:
  1. Initial - upper GI endoscopy - diagnostic

Erosions in lower Esophagus

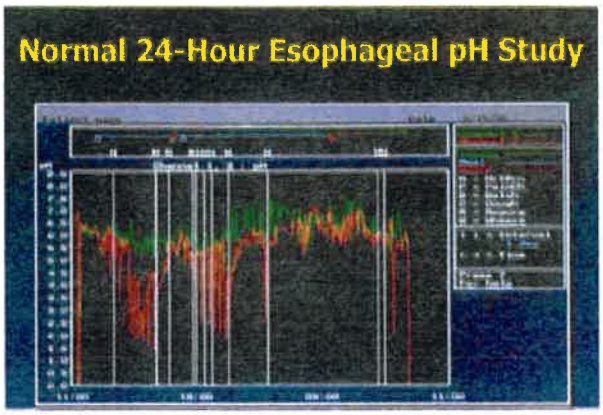


Normal Esophagus

- Grades - Los Angeles types - A to D

2. Gold standard - 24 hrs pH study done when:
   - Endoscopy is normal or
   - Patient has Atypical symptoms (cough/ asthma) or

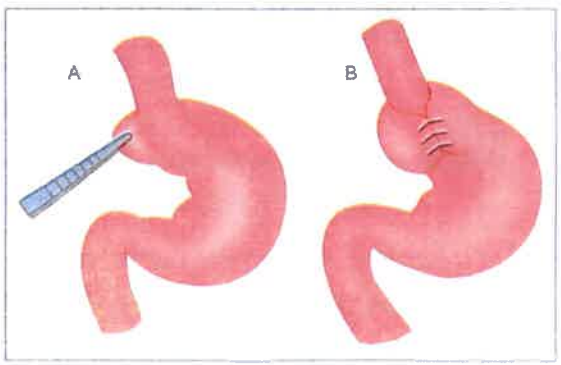
- Surgery is planned



- De-meester > 14.7 = symptom match GERD
- 48-96 hr pH study - Capsule (Bravo)
- Note: stop PPIs at least a week before performing 24 hr pH study

3. Esophageal manometry may aid diagnosis but is not required unless Planning surgery.

- Treatment:
  - Medical: PPI - long term 2-8 weeks - ulcer heals
  - Lifestyle changes-Avoid trigger - TLOSR
  - Surgery is done when
    1. Severe Symptoms: medically not controlled/ severe symptom /volume reflex / regurgitation
    2. Complication - Barrett's esophagus, Hiatus hernia
- Sx:
  1. Floppy Nissen fundoplication: 360-degree wrap



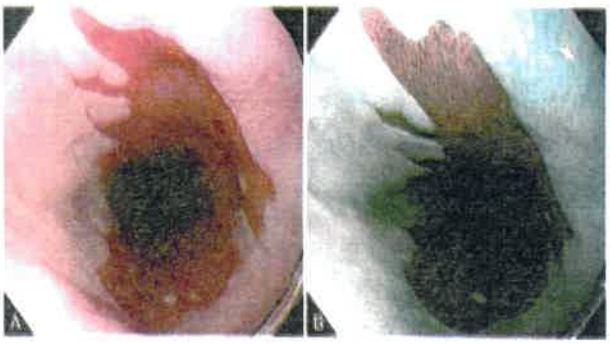
- MC Complication: Short term Dysphagia (< 3 months)
- Other Complications: Gas bloat syndrome, wrap migration

2. Toupet: 270-degree posterior wrap
3. DOR: 180-degree anterior wrap (usually done along with Heller's myotomy for antireflux effect)

- Newer treatment: Magnetic sphincter augmentation ring - encircle lower esophageal zone (Linx procedure)

BARRETT'S ESOPHAGUS

- Columnar/intestinal metaplasia of lower esophagus
- Length of metaplasia > 1 cm
- Risk factors: GERD, Obesity
- Pathology: Mucous secreting goblet cell
- Prague C & M: c - circumference, m - maximum higher the Prague score, more risk of cancer
- C/F: feature of GERD
- Diagnosis: endoscopy + Biopsy - Salmon Coloured Tongue- like projections at GE Junction
- Additional aid:
  - NBI light (image) consists of only two wavelength: 415-nm blue light and 540-nm green light. The differential absorption and refection of these spectra facilitates detection of mucosal abnormalities.



- Risk of malignancy:
  - Non-dysplastic Barrett's: 0.2-0.5% per year to around 0.7% per year for low-grade dysplasia.
  - For high-grade dysplasia, the risk of cancer progression can be as high as 7%
- Diagnostic protocol:
  - Seattle protocol: 4 quadrant biopsy at

- Risk factors

SCC	Adeno carcinoma
Smoking, spirit, hot beverage, dietary (N-nitroso compound)	Risk factor: GERD/ Barrett esophagus, obesity
Pre malignant: Caustic injury, achalasia, Kelly Paterson (upper esophageal CA)	

- Pathology:
  - Exophytic - more bleeding
  - Endophytic - more pain
  - Infiltrative - Obstruction → dysphagia
  - Microscopic: Middle 3rd - squamous cell carcinoma (MC type of CA overall);
  - Lower Third: Adenocarcinoma (MC in Western world)
- Spread:

a. Direct spread: Neck - RLN; Thorax - Trachea, Aorta; Abdomen - Upper Stomach

b. Lymph node mets:
  - Station 1 in lymph node = peri esophageal
  - Station 2 = along the blood vessels
  - Station 3 = (Neck) level 6/para aortic/pre aortic

a. Hematogenous mets:
  - 2L - lung/liver
  - 2B - brain/bone

- C/F:
  - Elderly smoker male with Progressive dysphagia initially more to solids + regurgitation (pseudo achalasia)
  - Retrosternal Pain, hematemesis
  - Weight Loss
- TNM staging:

T: tumour depth

T1: restricted to mucosa/submucosa

T2: muscularis

T3: adventitia

T4a: adjacent organ - resectable (pleura/diaphragm)

T4b: adjacent organ - not resectable (trachea/ aorta)

- Nodal stage:

N1: 1 to 2 regional nodes

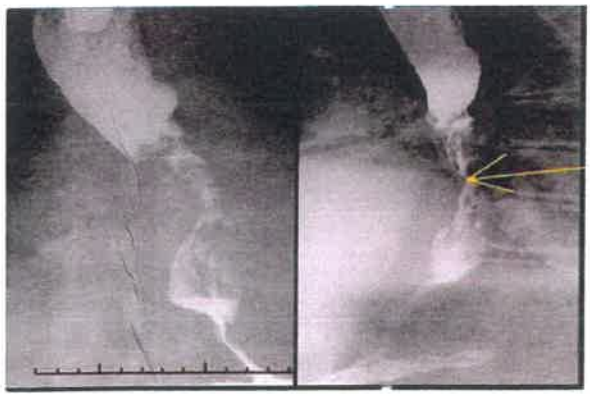
N2: 3 to 6

N3: >7

- Metastasis

M1: distant haematogenous metastasis

- Investigation:
  - 1st investigation upper GI endoscopy + biopsy of lesion
  - Chromoendoscopy (Lugol's iodine) is used to highlight suspicious areas (esp. in sq. cell carcinoma) if gross appearance is inconclusive
  - TNM stage: T- depth - all GI cancer - endoscopy ultrasound (EUS)
  - N, M: CECT/PET scan
  - BA swallow
    - long segment narrowing depending on location of growth
    - Apple core
    - Shouldering/abrupt narrowing



- RX:
  - Depend on staging:
    - Surgery for operable: T1/T2, N0
    - For T3, N1/N2: Multimodal treatment
    - Inoperable Palliation: SEMS (Self-

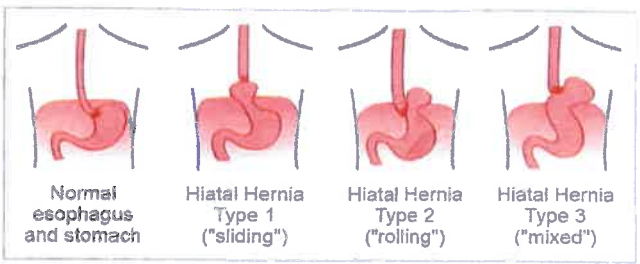
intervals of 2cm along the length at time of diagnosis and in future surveillance

- Dysplasia should be confirmed by 2 independent pathologists

- RX:
  - GERD + Barrett esophagus (no dysplasia): GERD Rx + endoscopic surveillance (3-5 years)
  - Barrett esophagus + low grade dysplasia: Radio frequency ablation + endoscopic mucosal resection + GERD Rx + 6 monthly surveillance
  - Barrett esophagus + high grade dysplasia: Assess risk, multidisciplinary discussion→ local ablation/esophagectomy (Treat like cancer)

HIATUS HERNIA

- Migration of intra-abdominal organs through esophageal hiatus into thorax
- MC Organ to herniate is stomach
- Pathogenesis: GERD
  ↓
  Ulcerations
  ↓
  Fibrosis of esophagus
  ↓
  Shortening of GE junction as it gets pulled up due to healing and fibrosis
  ↓
  stomach migrates into thorax = sliding (type I) type of hiatus hernia (80-85%)
  - The above pathogenesis leads to increase in GERD symptoms thereby increasing the hiatal hernia, subsequently causing a vicious cycle (GERD begets GERD)



Type I: Sliding type (MC type), GE junction migrates into thorax, associated with GERD

Type II: Rolling type, fundus of stomach herniates, without migration of GE Junction

Type III: Mixed type

Type IV: Other organs like colon herniate (Rare)

- C/f:
  - Sliding hernia: GERD, heart burn
  - Rolling hernia: risk of gastric volvulus - episodic abdominal pain, retching, inability to pass Ryles tube (Borchardt's triad)
  - Mixed hernia: both
- Investigation:
  - 1st Investigation - upper GI endoscopy - Lax diaphragmatic crura - migration of Z-line upwards
  - IOC: CT with oral contrast
- RX:
  - Surgery → Laparotomy/Laparoscopy → reduce the hernia → remove the sac → close the defect/ mesh
  - Add an anti-reflux procedure to prevent GERD.

KEY POINTS

- GERD = esophagitis due to acid reflux
- Anatomical aberration + TLOSR
- 24 hr pH role: symptoms with drop pH
- Anti-reflux surgeries: medically uncontrolled/ HH
- BE: risk of malignancy: 0.2 to 0.5% per year if non-dysplastic
- Hiatus hernia IOC: CT with oral contrast

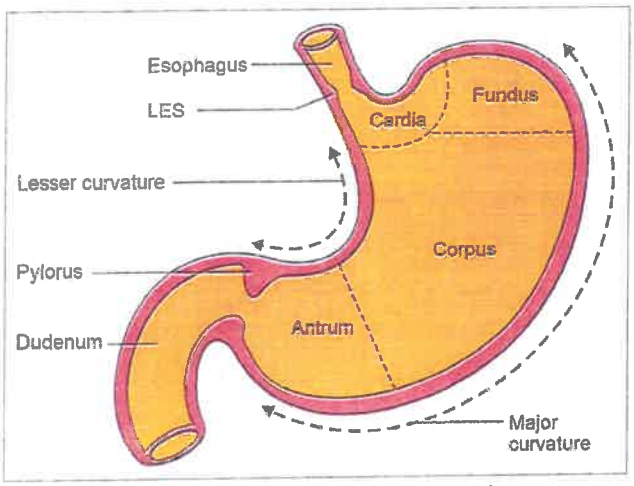
CARCINOMA ESOPHAGUS

- 5-year survival: <10%
- With multimodal rx, it can increase to 40-50%

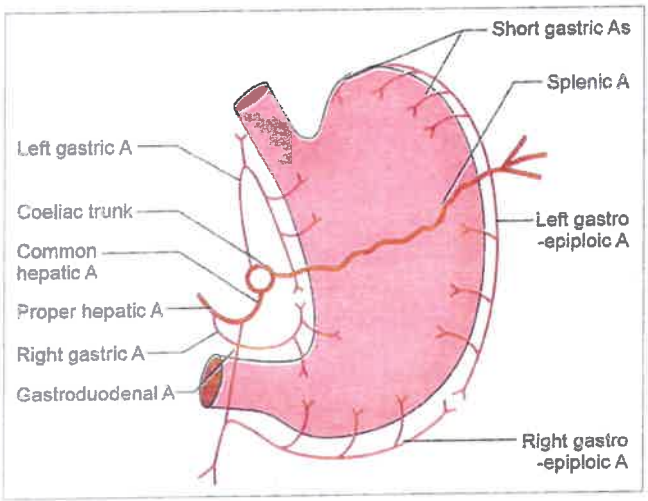
3
Chapter

STOMACH PART-1

ANATOMY OF STOMACH



Incisura: common site of gastric ulcer



Blood supply- from the 3 branches of celiac trunk :

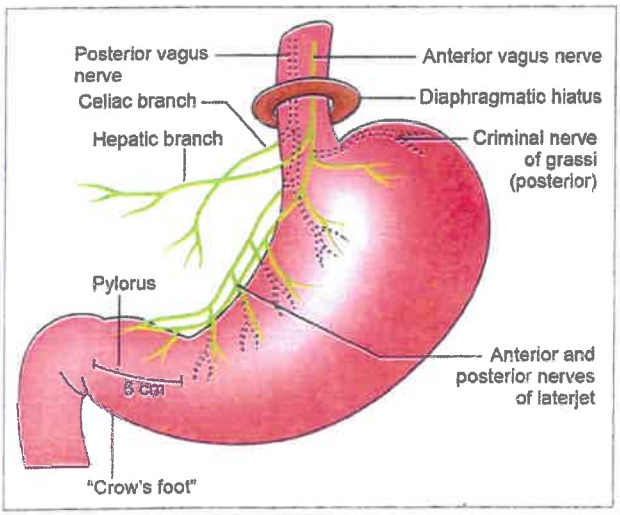
i. Left gastric artery

ii. Splenic artery

iii. Common hepatic artery

- Lesser curvature - left gastric artery/ right gastric artery
- Greater curvature - LGE / RGE

Nerve Supply of Stomach



1. Left /Anterior vagus nerve runs anterior to GE junction, giving a hepatic branch
2. Right/ posterior vagus nerve runs posterior to GE junction giving celiac branch as well as the "criminal nerve of Grassi"

Both Vagus nerve divide on surface of stomach into multiple nerves of Laterjet (sero-muscular branches of stomach)-which are responsible for acid production and gastric motility

Branches supplying the pylorus (crow's foot) - spared in highly selective vagotomy to maintain gastric emptying

On the other hand, during "Truncal vagotomy", both ant. & post. Vagus are cut at GE junction (trunk)

↓

Loss of peristaltic effect and pyloric relaxation

↓

Always add drainage procedure to stomach

Expanding Metal Stent)

1. If Operable, then approaches:
   - Lower esophageal tumor – single – left thoraco-abdominal incision; double – abdomen + right thorax (Ivor-Lewis)
   - Middle esophageal tumor – double approach/Ivor Lewis; triple/Mckeown
2. Extent of resection: >10cm proximal and > 5 cm to 10 cm distal margin (depending on location)
   - For middle esophageal tumors – total esophagectomy with lymph node clearance
   - For lower esophageal – esophagectomy with proximal gastrectomy + LN
3. Reconstruction:
   - Most preferred Conduit → Gastric pull up (based on RGE vessels) → esophago-gastric anastomosis
   - Alternate conduits – colon (transverse colon/ left colon) – jejunum
   - Post Op: adjuvant chemo therapy – platinum based
   - FLOT regimen: 5 FU + Leucovorin + Oxaliplatin + Taxane

KEY POINTS

- MC type of Ca esophagus: SCC
- Cause of death: TE fistula/Aortic erosion
- 1st investigation: upper GI endoscopy
- RX: Depends on stage:
  1. Early stage/Operable – Surgery
  2. Advanced – Multimodal treatment.

Extra Edge

1. Mc Bening solid tumor of esophagus: Leiomyoma - Removed by Submucosal Tunneling Endoscopic Resection (STER)
2. Dysphagia Lusoria - Retro-Esophageal right Subclavian Artery Causing Compression leading to dysphagia

Lymphatic Drainage

Goes along the blood vessels of stomach

- 1st station: Peri-gastric nodes (numbered 1 to 6)/> 16mm
- 2nd station: along left gastric artery, along common hepatic artery, & splenic artery
- 3rd station: At Celiac trunk
- Based on this principle, lymphadenectomies are described for CA Stomach:
- D1 Lymphadenectomy: Peri-gastric group removed (1 to 6)
- D2 Lymphadenectomy: D1 + 2nd and 3rd stations (LGA, CHA, celiac, splenic), numbered 7 to 11 also removed
- D2 Lymphadenectomy: lymph node stations 1 to 6 and 7 to 11 removed

Development

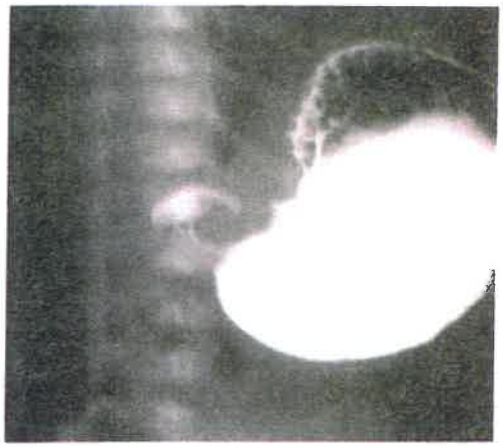
Entire stomach arises from the foregut, from distal part hence blood supply - celiac trunk

INFANTILE PYLORIC STENOSIS

- What is IHPS? Hypertrophy of pyloric sphincter (circular fibres) at birth leading to gastric outlet obstruction.
- Clinical presentation: 4 to 8 weeks, typically in male child
  - Mother claims that child has non-bilious vomiting of curdled milk (feeds), after which child is hungry again.
  - On examination:
    - During feed: left to right peristaltic wave seen on abdomen.
    - When baby is relaxed (Feeding): olive shaped mass (pylorus) palpable in Epigastrium/ right hypochondrium.
    - Dehydration may be the presentation due to repeated vomiting

Investigation

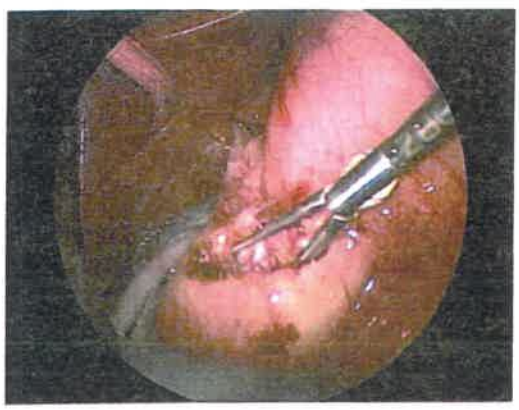
- Blood electrolytes: ↓H+, Cl-↓, K+↓ = hypokalemic hypochloremic Metabolic alkalosis
- + Paraoxidical aciduria (due to RAAS activation causing Na retention and K^+, H^+ losses in urine)
- IOC: USG abdomen - pylorus thicker >4mm, longer > 16mm distended stomach.
- Ba meal will show mushroom sign.



Mushroom sign on barium meal

Treatment

- 1st step: Correct the electrolyte abnormalities with DNS + 0.15% KCl
- Endoscopic pyloric dilatation if low grade of hypertrophy
- TOC: Ramstead's pyloromyotomy -pyloric serosa and muscle cut along its length till duodenal fornix (only mucosa kept intact) to release the pyloric obstruction



KEY POINTS

- IHPS presents at 4 to 8 weeks- non bilious vomiting
- Metabolic abnormality: Hypokalemic hypochloremic metabolic alkalosis with paradoxical aciduria.

PEPTIC ULCER DISEASE

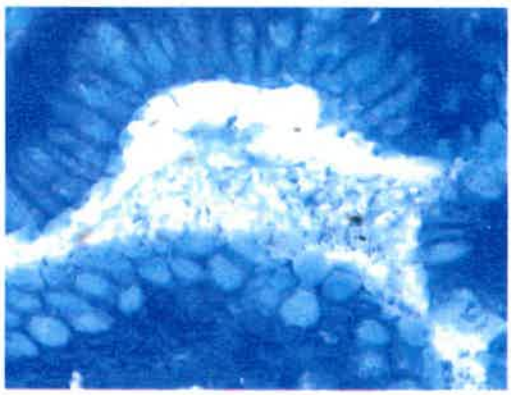
- Ulcer means breach of mucosa

Peptic ulcer: Ulceration in either gastric mucosa or duodenal mucosa due to acid induced damage.

- Risk factors:

Gastric ulcer	Duodenal ulcer
Mucous barrier lost	Due to increased acid secretion
Caused by Smoking, NSAID,H pylori	H Pylori infection

H Pylori:



Gets lodged usually in antral mucosa, produces urease → leading to NH3 release → reflex hypergastrinemia (negative feedback)

1. It causes inflammation of gastric mucosa- chronic gastritis (Type B)
2. Causes ulceration of both gastric, duodenal mucosa
3. Causes gastric cancer (not duodenal) - Adenocarcinoma/MALtoma

Direct toxic damage due to:

- Cag-A
- Vac-A

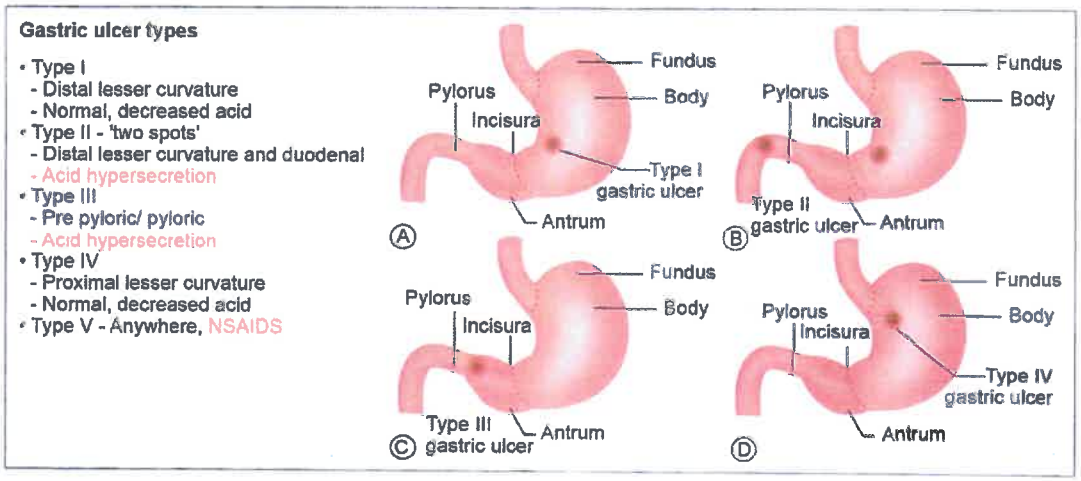
Both are cytotoxic

Histopathology: Warthin starry staining / modified GIEMSA stain. (Seen in the image)

Pathology/ Types Of Peptic Ulcer

Location: Johnson's classification

I. Lesser curvature
II. Gastric+duodenal ulcer
III. Pre pyloric region
IV. Proximal stomach
V. NSAID induced



Type II and Type III: associated with increased acid production

Sequelae

- Bleed (Duodenal > Gastric ulcer)- MC complication of peptic ulcer
- Perforation → peritonitis (duodenal > gastric)
- Stricture → gastric outlet obstruction
- Malignancy → only in gastric ulcers

Presentation

- Pain in epigastrium

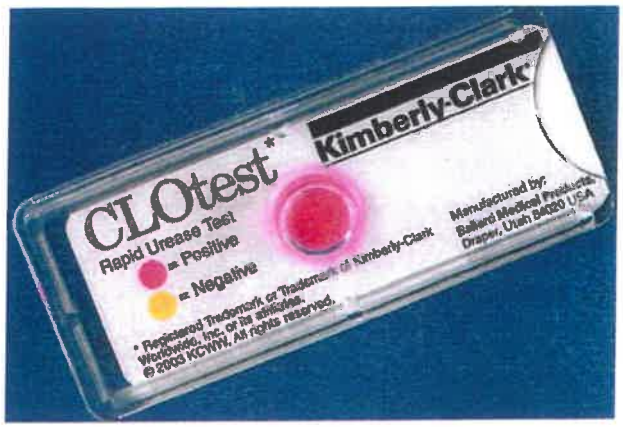
- May aggrevated or improve after meals
- Periodicity of pain
- Vomiting
- Weight loss

Examination

- No finding

Investigation

- Upper GI endoscopy : gastric/ duodenal/ both



CLO Test / Rapid Urease Test

Antral mucosal biopsy → slide → change in color tells urease activity → Pink: positive → Yellow: negative

- Antral mucosal biopsy : diagnosis of h.Pylori + Rapid urease test (Slide in image)
- Ulcer wedge biospy (for gastric ulcers only): rule out malignancy

Treatment

MEDICAL MANAGEMENT: T/T OF CHOICE

i) Ppi/ h2 blocker: given long term : 4-8 weeks

ii) Stop smoking/nsaids

iii) H. Pylori eradication :

Triple therapy: two antibiotics → amoxicillin + doxycycline ± metronidazole and one ppi 10-14 → days

iv) If ulcer recurrs/ more ulcers/ulcers in unusual locations → suspect zollinger ellison syndrome (gastrinoma)

SURGICAL MANAGEMENT

Indications

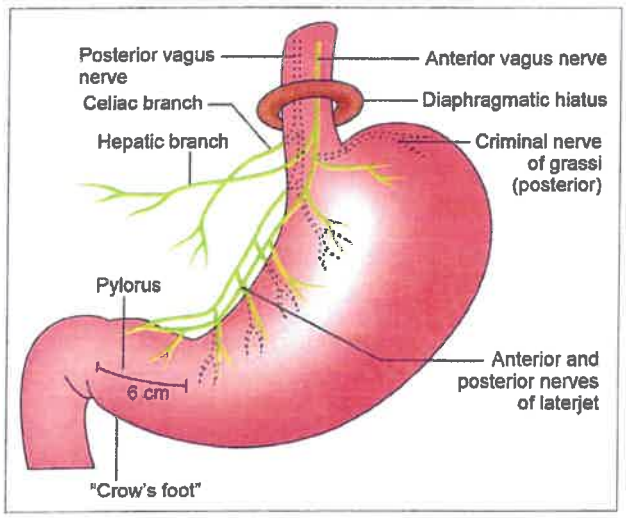
- Recurrent ulcer despite optimal treatment
- Complications of peptic ulcer disease

Principles of surgery

- Gastric ulcer: mucus barrier is lost → excise the ulcer → since have a risk of a malignant transformation
- Duodenal ulcers are due to increased acid production → acid lowering surgery done

1. **Vagotomies (for duodenal ulcer only)**

   A. **Truncal vagotomy** - both anterior and posterior vagus are cut at trunk (GE junction)



- **Acid production reduces drastically** to below 50% - low recurrence
- However, vagotomy also reduces peristalsis and pyloric spincter doesn't relax
- Hence add a drainage procedure
  - i. Pyloroplasty or
  - ii. Gastro - jejunostomy

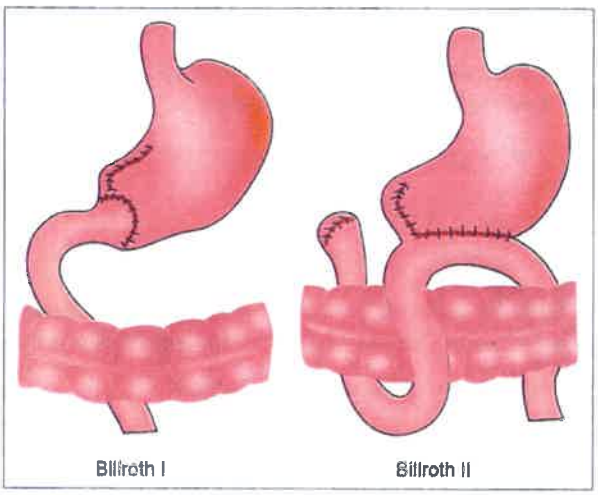
Both of these increase morbidity of surgery and time of surgery → so an alternative is "highly selective vagotomy"

B. **Highly selective vagotomy-**

- Only denervate individual branches of the anterior and posterior vagus

- Preserve the crow's feet (last 6 cm over pylorus)
- Hence preserving the innervation of pylorus (last 6 cm) → pylorus will relax → hence no need for a drainage procedure
- Best tolerated acid lowering procedure → FAIRLY good ulcer healing procedure
- Very low complication rate (<5%)

2. **Antrectomy/distal gastrectomy procedures (will reduce gastrin)**
   - Distal gastrectomy (antrectomy) → decrease gastrin production
   - Reconstruction by billroth
     A. **Billroth I: distal gastrectomy with gastro-duodenostomy** → after distal gastrectomy, join duodenum to stomach, suture the rest of the stomach
     B. **Billroth II: distal gastrectomy with gastro-jejuonostomy** → after distal gastrectomy, close the duodenal stump + pull the loop of jenunum and join it to stomach
   - For gastric ulcers, no need of acid lowering procedure
   - If ulcer on lesser curvature, just excise that part with reconstruction by doing Billroth
   - So, Billroth I and II also done in distally located gastric ulcers
   - If ulcer in proximal part, just resect that part (Like Csendes, Paucet procedure)



Recurrence rates after surgery for peptic ulcer:

A. Truncal vagotomy with pyloroplasty- 2 to 7%
B. Selective vagotomy with pyloroplasty- 5 to 10%
C. Highly selective Vagotomy- 2 to 10%
D. Truncal vagotomy with antrectomy- 1%

Though truncal vagotomy with antrectomy has the least recurrence rate, its side effect profile is poor (10 to 20%) compared to HSV <5%.

COMPLICATIONS

1. **Anatomical changes**
   A. Small stomach syndrome: peristalsis is weak
   B. Post vagotomy diarrhea
   C. Gall stones
   D. Iron deficiency anemia (MC Nutritional problem)
   E. Megaloblastic anemia (Decreased intrinsic factor)
   F. In loop gastro jejunostomy: billiary juices can go in stomach → bile reflux gastritis, ulcers at anastomosis → marginal ulcers → can be avoided by Roux-En-Y Gastro jejunostomy

2. **Physiological changes**
   A. Dumping : food gets rapidly dumped in jejunum → this happens in a drainage procedure → not seen in highly selectively vagotomy
      I. **Early dumping:**
         - Food which is highly osmolar → food pulls the liquid with it from the intravascular compartment → jejunum swells up → intra vascular fluid reduces → hypotension
         - Occurs 15-30 mins after meal → light headed, dizzy, bloated, sometimes diarrhea
         - Diagnosed by rise in hematocrit after meals and hypotension

      II. **Late dumping :**
         - Food rich in simple sugars, refined flours: absorbed rapidly → transient hyperglycemia → insulin surge → rebound hypolycemia