SURGERY OF STOMACH AND DUODENUM

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General and Laparoscopic Surgeon

FICMS General Surgery

CABS General Surgery

Surgical Anatomy

- Food reservoir & mechanical digestion.
- Arterial blood supply

1- Left gastric artery: from CA

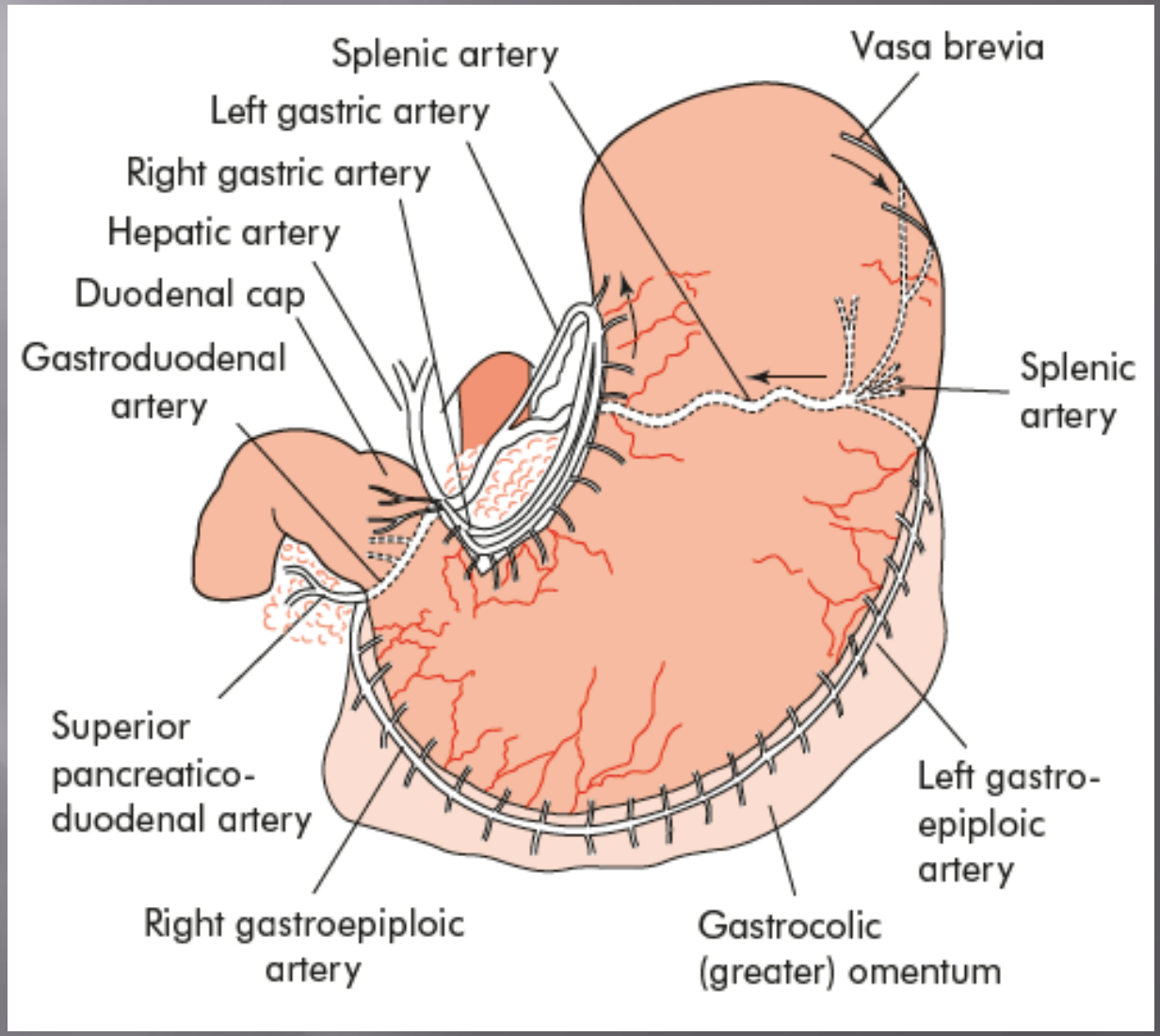
2- Right gastric artery: from CHA

3- Gastroduodenal artery (from hepatic artery) divide into a –superior pancreaticoduodenal A., b- right gastroepiploic artery.

4-Inferior pancreaticoduodenal artery a branch of superior mesenteric artery

5- Left gastroepiploic artery a branch of splenic artery..

6- Short gastric arteries from splenic artery


Splenic artery
Vasa brevia
Left gastric artery
Right gastric artery
Hepatic artery
Duodenal cap
Gastroduodenal artery
Splenic artery
Superior pancreatico-duodenal artery
Left gastro-epiploic artery
Right gastroepiploic artery
Gastrocolic (greater) omentum

- **Venous drainage**

1- Lesser curve: right & left gastric (coronary) veins drain into portal vein

2- Greater curve: left gastro epiploic vein & vasa brevia join splenic vein

3- G curve: right gastro epiploic vein join superior mesenteric vein

4- vein of mayo

- **Nerve supply**

1-Intrinsic:

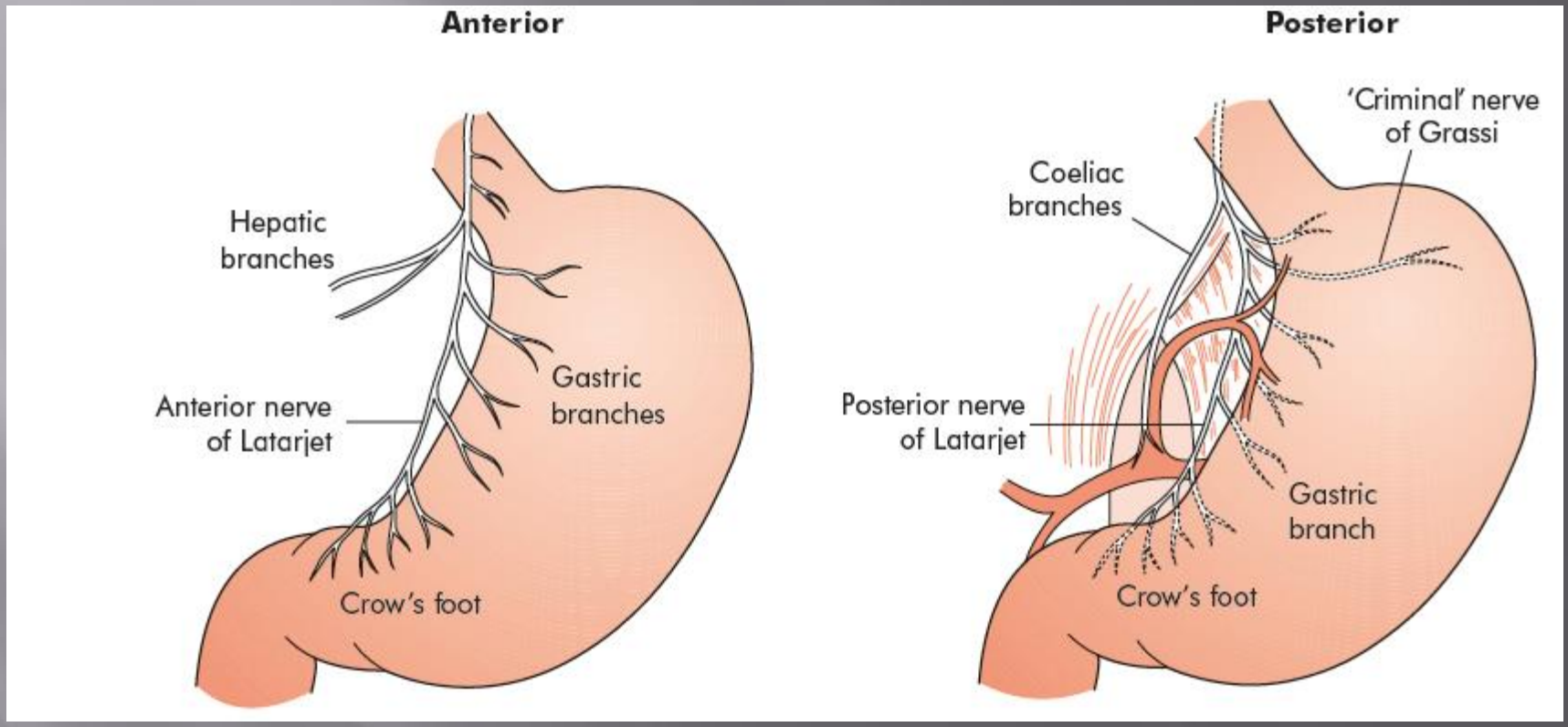
a- myenteric plexus of Auerbach,

b- submucosal plexus of Meissner

2-Extrensic:

- Ant. Vagus & post vagus (afferent sensory; efferent secretomotor).

- Sympathetic from celiac ganglion.


Anterior
Hepatic branches
Gastric branches
Anterior nerve of Latarjet
Crow's foot
Posterior
'Criminal' nerve of Grassi
Coeliac branches
Posterior nerve of Latarjet
Gastric branch
Crow's foot

Physiology

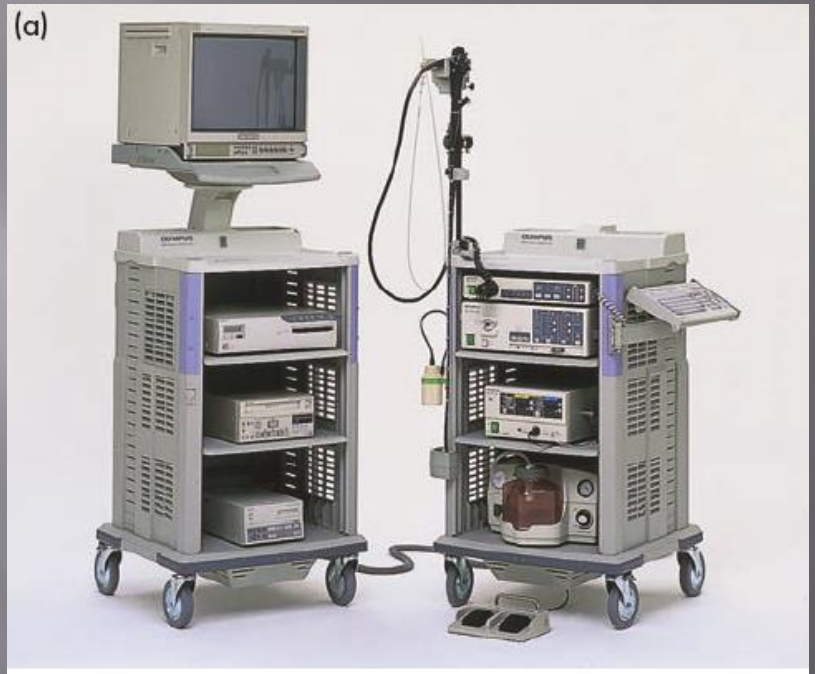
1-Parietal cells secrete HCL

2-Chief cells secrete pepsin

3- Endocrine cells secrete gastrin (G cell), somatostatin(D cell)..

Investigations of stomach & duodenum

- OGD flexible endoscopy
- Endoscopic ultrasound: more sensitive than CT for tumor staging.
- Barium meal
- Ultrasound
- Computerised tomography scanning (CT Scan) and Magnetic resonant imaging.
- Laparoscopy: detect peritoneal metastasis, + U/S to check liver & LN.
- PET Scan.
- Angiography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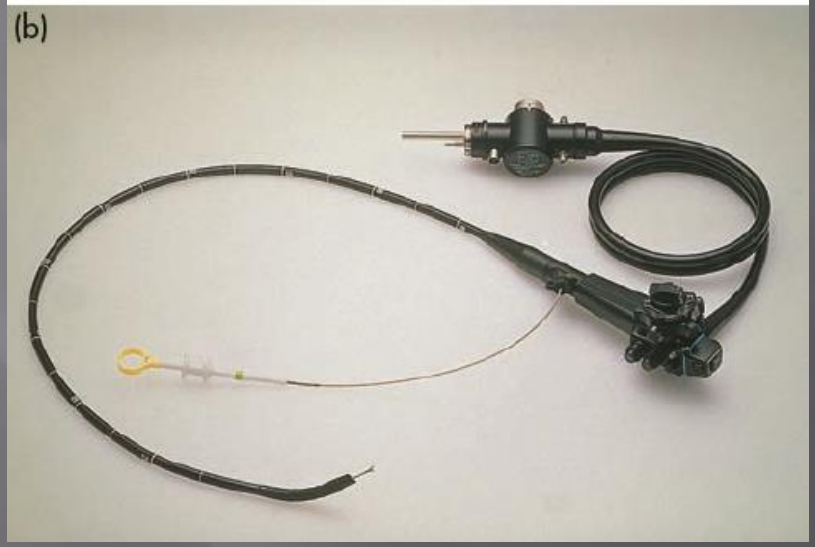
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SURGICAL PATHOLOGY

- 1-Congenital hypertrophic pyloric stenosis

2-Duodenal atresia

- **HELICOBACTER PYLORI**

- Spiral bacteria

- It is important in the aetiology of gastroduodenal diseases such as

1- Chronic gastritis

2- Peptic ulceration

3- Gastric cancer

Types of Gastritis

- Type A gastritis
- An autoimmune condition; there is circulating antibody against parietal cell mass resulting in hypochlorhydria then to achlorhydria ; a premalignant condition
- Deficient secretion of intrinsic factor by parietal cell lead to malabsorbtion of vitamin B12 leading to pernicious anemia which is again premalignant condition.
- **Type B Gastritis**

- Associated with Helicobacter pylori, affect the antrum
- Can develop peptic ulcer, pangastritis with risk of gastric cancer.
- Intestinal metaplasia is associated with chronic pangastritis with atrophy.
- Intestinal metaplasia with dysplasia is pre malignant condition that needs regular OGD follow up.

- **Reflux Gastritis**

Occur after gastric surgery ,treatment medical very rare surgical..

- **Erosive Gastritis**

Caused by NSAID & alcohol.

Stress Gstritis

- Serious illness or injury, cardiopulmonary bypass
- Reduction to blood supply of the superficial mucosa
- Can lead to stress ulcer +/- bleeding
- Prevention by H2 antagonist +/- barrier agent e.g. sucralfate.

Menetriers Disease

- Gross hypertrophy gastric mucosa, mucus production, & hypocholhydria.
- Premalignant condition ,only treatment is gastrectomy.

Other forms of Gastritis

- Lymphocytic gastritis : T cell infiltration , + celiac disease
- Esinophilic Gastritis
- Granulomatous Gastritis in Crohn' dis.
- AIDS Gastritis
- Phlegmonous (Bacterial) gastritis

- **PEPTIC ULCER**

Common sites: 1-first part duodenum. 2-lesser curve stomach. 3- oesophagus. 4- stoma following gastric surgery. 5- Meckel's diverticulum.

1-Acute peptic

- (gastric & duodenal) ulcer & acute stress ulcer.
- Causes: major illness, uremia, food poisoning, bacteraemia, burn, aspirin, steroid & NSAID.

- Pathology

Single or multiple as diagnosed by OGD.it involve the mucous membrane & does not penetrate the muscles, acute ulcer may bleed, or perforate..

- **Treatment**.

Treat the cause plus anti ulcer treatment.

2-Chronic peptic ulcer.

Deeper penetrating ulcer.

A-Gastric ulcer having relatively normal level or below normal gastric acid secretion.

B-Duodenal ulcer occur in presence of very high acid level; also occur in Zollinger Ellison syndrome, genetic factors may be involved, H pylori, social stress.. emotional, smoking, NSAID

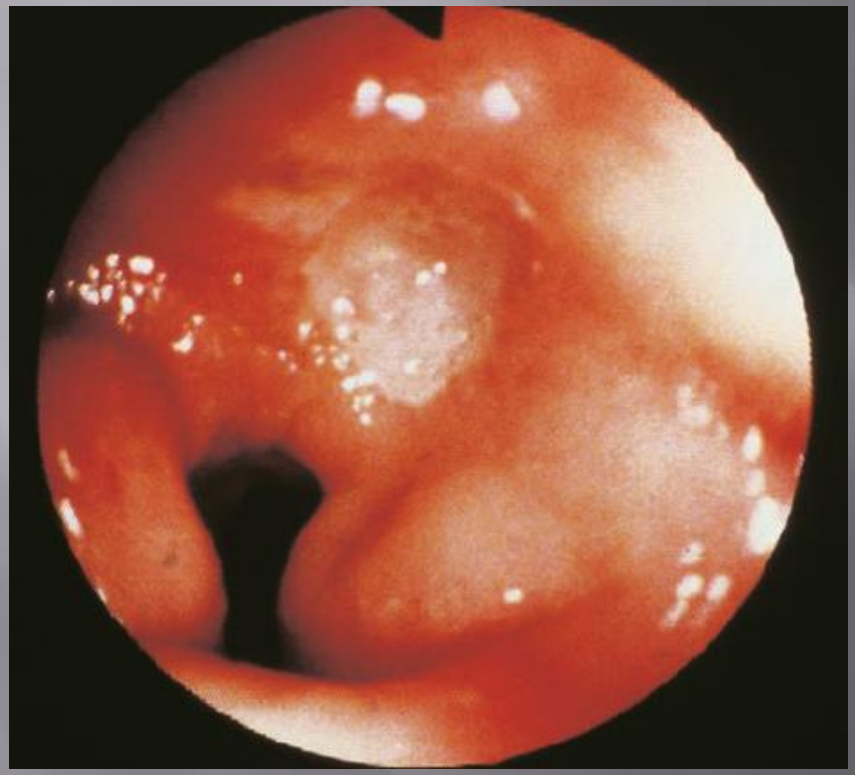
DUODENAL ULCER

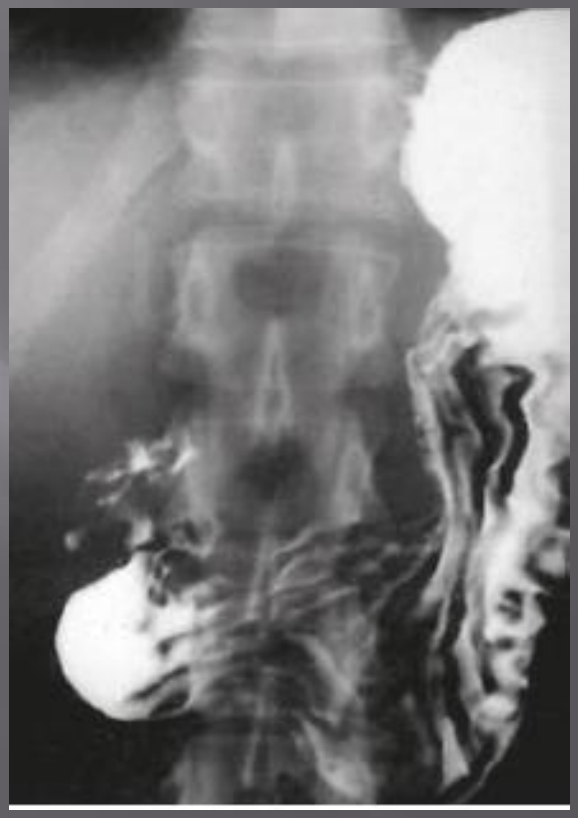
Incidence

- The incidence of duodenal ulcer & the frequency of elective surgery are falling therapy except in cases of complications:
- Use of OGD
- Wide spread use of anti ulcer drugs & eradication of H.pyl.
- More common in male than female, older age, & developing countries.

Pathology

- Occur in first part duodenum it involve the mucosa, muscle coat, leading to fibrosis and pyloric stenosis, it may penetrate post. to pancreas & invade gastro duodenal artery,
- Sometimes multiple ulcers,, or ANT,& POST. ulcer called kissing ulcers,,
- Ant. ulcer tend to perforate while Post. ulcer tend to bleed.
- Big ulcer called GIANT ulcer.(more than 2 cm.)
- Chronic duodenal ulcer never become malignant.
- Chronic gastric & chronic duodenal ulcer may co exist.





GASTRIC ULCER

- Incidence

Same etiological factors

Gastric ulcer is less common than D.U

Sex are equal male to female,

Population is older than d.u.

More prevalent in low socioeconomic patients,,.

- **Pathology**

Usually on the lesser curve of stomach, but it is larger than DU.

Invade mucosa & muscle coat & fibrosis may cause stomach deformity: hour glass stomach or tea pot deformity, may penetrate to pancreas or blood vessel or invade transverse colon ..

- **Malignancy in gastric ulcer**

1-On the long run ch. GU might become malignant ; take multiple biopsies during OGD

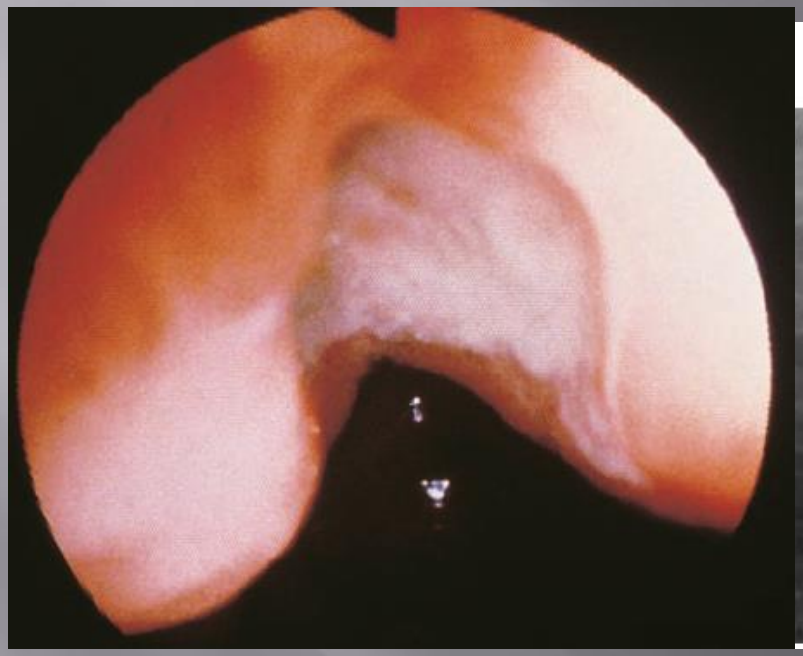
2-other type of GU is malignant from the start.

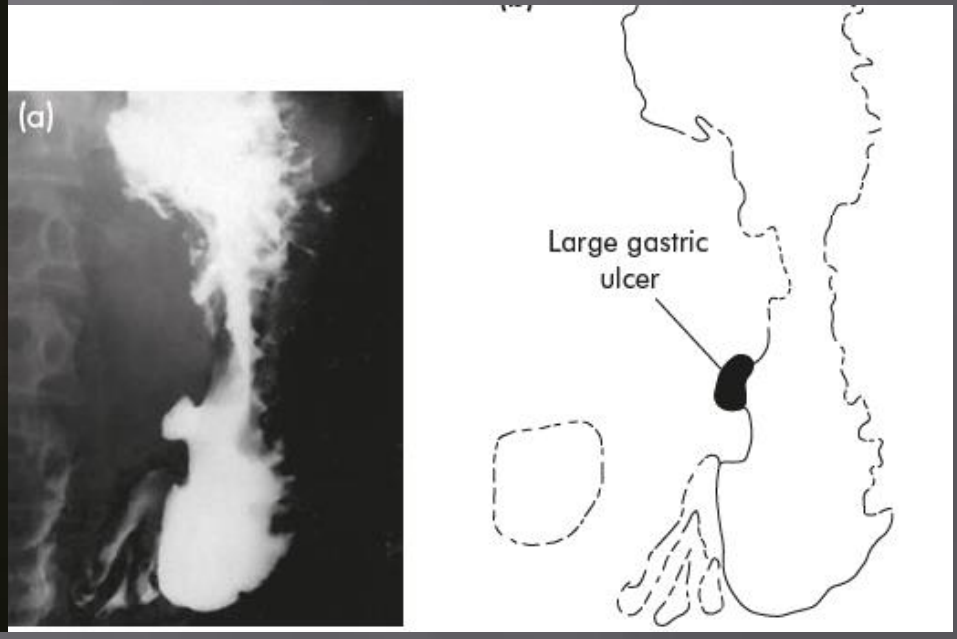
- **Other types of peptic ulcer**

1-stomal ulcer at jujunal site of gastrojejunostomy.

2-Billroth II (polya) gastrectomy.

3- Prepyloric gastric: risk of cancer so it need biopsy.




(a)
Large gastric
ulcer

- **Clinical features of gastric & duodenal ulcers**

- PAIN in the epigastric region, may radiate to the back, it is intermittent, may last weeks or months with interval of pain free, eating aggravates pain in GU & relieve pain of DU.

- PERIODICITY; attack of pain last from 2—6 weeks, attack is more in spring & autumn.

- VOMITING: absent unless there is pyloric stenosis .

- Alteration in weight.

- BLEEDING: either chronic loss presented as anemia; or fresh as hematemesis & melena.

- **CLINICAL EXAMINATION**

Tenderness at epigastric region, stomach splash in case of pyloric stenosis.

- **INVESTIGATIONS OF PEPTIC ULCER**

1--OGD with or without multiple biopsy (CHECK ESOPHAGUS ,,, STOMACH,,DUODENUM),,& STOMA OF GASTROJEJUNOSTOMY to exclude stomal ulcer.

2—BARIUM MEAL

3—C.B.P.

TREATMENT OF CHRONIC PEPTIC ULCER

- The vast majority of uncomplicated peptic ulcer are treated medically ,,surgical treatment of uncomplicated peptic ulcer has decreased markedly since 1990.due to the use of H2 recepter antagonist or proton pump inhibitor & eradication therapy.
- The aim of surgical treatment is to reduce gastric acid secretion,(now it is reserved for the complicated P.U.)

MEDICAL TREATMENT

1 - Cessation of cigarette smoking ,,NSAIDs & Cortisone.

2- H2 receptor antagonist a-cemetidine (tagamet)- b-ranetidine (zantac).

3- Mucosal protective agent: sucralfate (ulcar).

4- Proton pump inhibiter (omeprazole)

5- Eradication therapy: flagyl x3, plus amoxil 500mg x3 ; or flagyl plus Clarithromycin 500mg twice a day for 2 weeks.

SURGICAL TREATMENT OF UNCOMPLICATED PEPTIC ULCER

- Although it is rare now, if medical Rx fails.
- Operations for D. U. aim is diversion of the acid away from duodenum,
- HISTORICAL SURGICAL PROCEDURES

1-BILLROTH I & BILLROTH II (polya) GASTRECTOMY, by Billroth in 1881.

2-GASTRO JEJUNOSTOMY (by Wolfler in 1881) will end with high rate of stomal ulcer.

At present time the operation of choice in cases of DU. are:

1—Truncal vagotomy (Dragstedt 1943) plus drainage procedure (due to gastric stasis):

A—PYLOROPLSTY.

B—GASTROJEJUNOSTOMY: posteriorly at antrum.

2- Selective vagotomy plus drainage procedure.

3-Highly selective vagotomy only (with preservation to the nerve of Latarjet that supply the pylorus).

4- Truncal vagotomy and antrectomy (Billroth I): very low recurrence but more side effects & mortality.

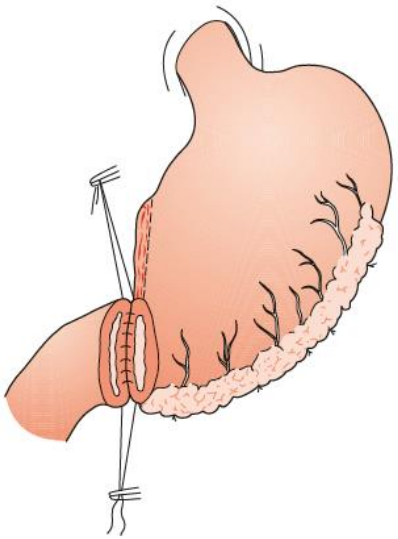


Figure 63.16 Billroth I gastrectomy. The lower half of the stomach is removed and the cut stomach anastomosed to the first part of the duodenum.

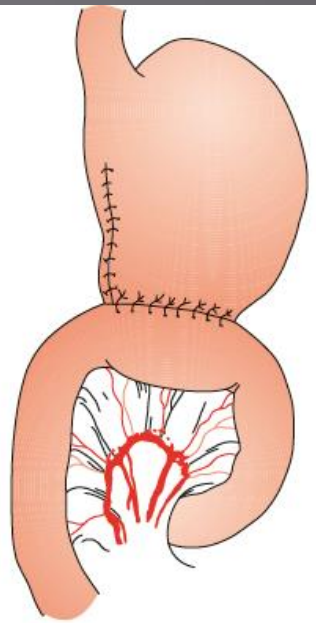


Figure 63.17 Billroth II. Two-thirds of the stomach removed, the duodenal stump is closed and the stomach anastomosed to the jejunum.

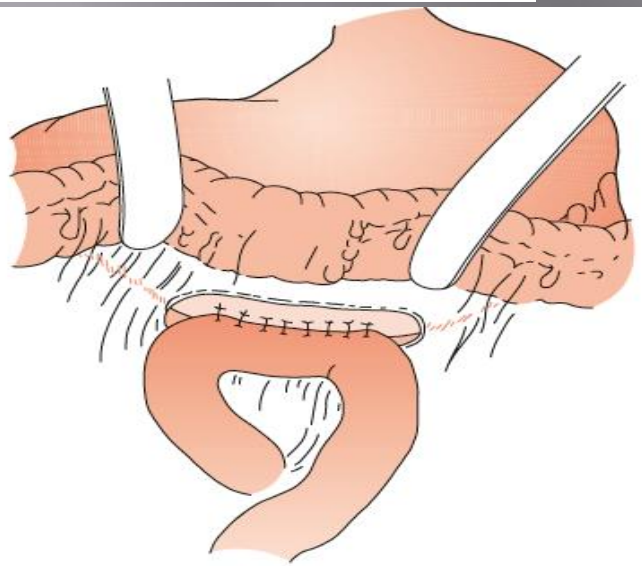


Figure 63.18 Gastroenterostomy. The jejunum is anastomosed to the posterior, dependent, wall of the stomach.

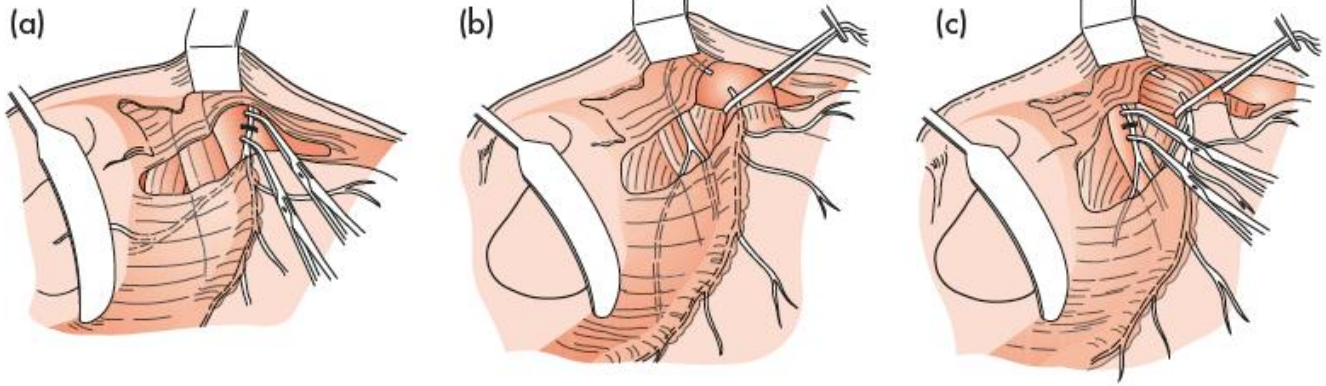


Figure 63.19 Truncal vagotomy. (a) Division of the anterior vagus. (b) Mobilisation of the oesophagus. (c) Division of the posterior vagus.

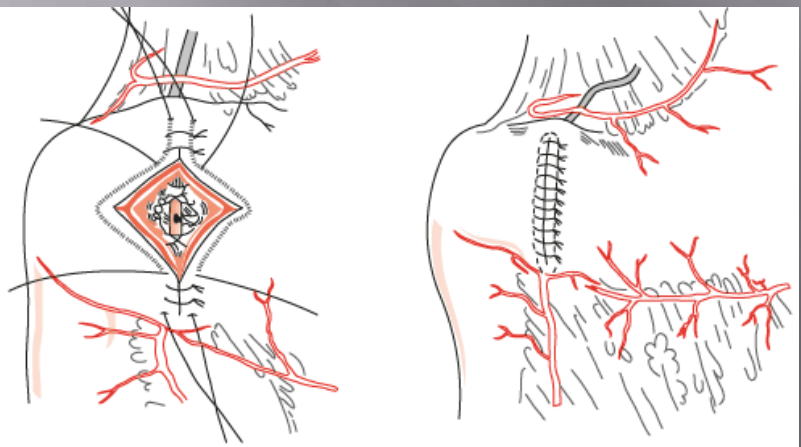


Figure 63.20 Pyroplasty.

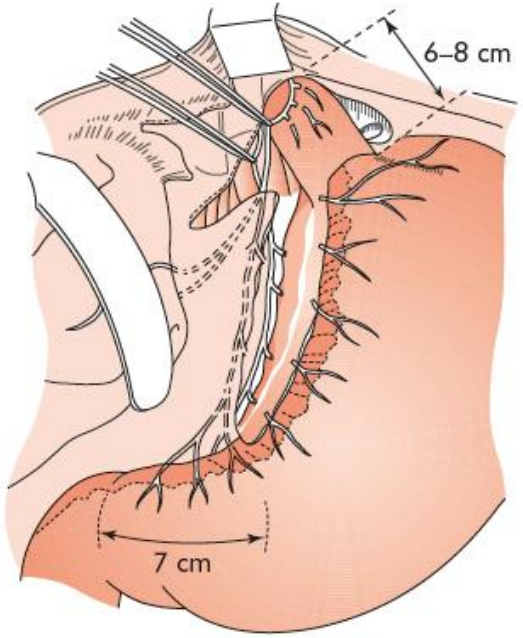


Figure 63.21 Highly selective vagotomy. The anterior and posterior vagus nerves are preserved but all branches to the fundus and body of the stomach are divided.

Operations for gastric ulcer

1—BILLROTH I OR BILLROTH II OPERATION PLUS EXCISION TO GASTRIC ULCER FOR HISTOPATHOLOGY TO EXCLUDE MALIGNANCY

2—Or Vagotomy plus drainage procedure plus excisional biopsy of the ulcer..

COMPLICATIONS OF PEPTIC ULCER SURGERY

1- Recurrent ulceration –a-either same ulcer, or b-stomal ulcer (complication may cause gastrojejunocolic fistula).

2- Bile vomiting: may respond to bile-chelating agents; or may require dismantling of gastrojej., or antrectomy + Roux en Y gastrojejunostomy.

3- Small stomach syndrome.

4-Dumping due to rapid gastric emptying:

a—early dumping: osmotic effect of food.

b—late dumping: reactive hypoglycemia.

5-Post vagotomy diarrhea .

6-Malignant changes: bile reflux gastritis then intestinal metaplasia then malignancy.

7-Gallstones formation.

8-Nutritional disorder: wt. loss, anemia, vit.B12 deficiency.

	Early	Late
Incidence	5–10%	5%
Relation to meals	Almost immediate	Second hour after meal
Durations of attack	30–40 minutes	30–40 minutes
Relief	Lying down	Food
Aggravated by	More food	Exercise
Precipitating factor	Food, especially carbohydrate-rich and wet	As early dumping
Major symptoms	Epigastric fullness, sweating, light-headedness, tachycardia, colic, sometimes diarrhoea	Tremor, faintness, prostration

COMPLICATIONS OF PEPTIC ULCER

1-PERFORATION PEPTIC ULCER:

1- Ant. 1st part duodenum most common, 2- Gastric ulcer,
3- prepyloric ulcer.

- **SYMPTOMS**

Some give history of D.U. or silent ulcer ,,there will be Sever sudden onset epigastric pain then generalized due to irritant effect of gastric acid & gastric contents of food on the peritoneum, first chemical peritonitis then bacterial peritonitis,

- **SIGNS**

At first temp. subnormal or normal then temp. increase (pyrexia by bacterial peritonitis),pulse increases,

Abdomen moves little or not move with respiration,

Severe abdominal tenderness, abdominal rigidity called board like rigidity,

On percussion dull, on auscultation first bowel sound +ve then become –ve.

P.R. exam elicit tenderness at rectovesical pouch.

In advanced peritonitis: septicemia & septic shock & mortality will be high.

INVESTIGATIONS

- Xray abdomen in erect ,,supine & lateral position,,, shows gas under diaphragm in 70% of chest x-ray,,
- Blood tests, serum amylase, abdomen ultrasound & C.T.Scan abdomen..

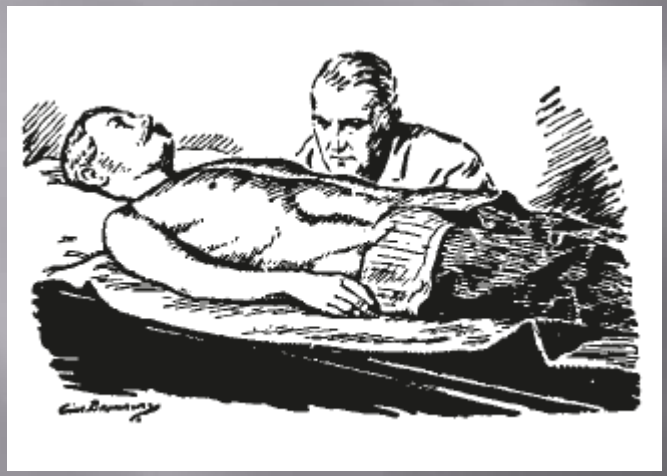
TREATMENT

- Resuscitation: antibiotics, tagamet injection, NG tube, Foley's catheter.
- Emergency laparotomy by upper mid line incision, localize the perforation site close the perforation by interrupted sutures with or without omental patch, then peritoneal toilet, then put drain.
- Rarely partial gastrectomy indicated:

1-perforation in carcinoma 2-sever bleeding 3-perforation is big & can not be closed.

- Postop. period & Follow up by anti ulcer treatment & O.G.D.
- Other types of treatment:

- Conservative (small perforation & localized peritonitis)
- Definitive surgical treatment of PU if patient is fit.



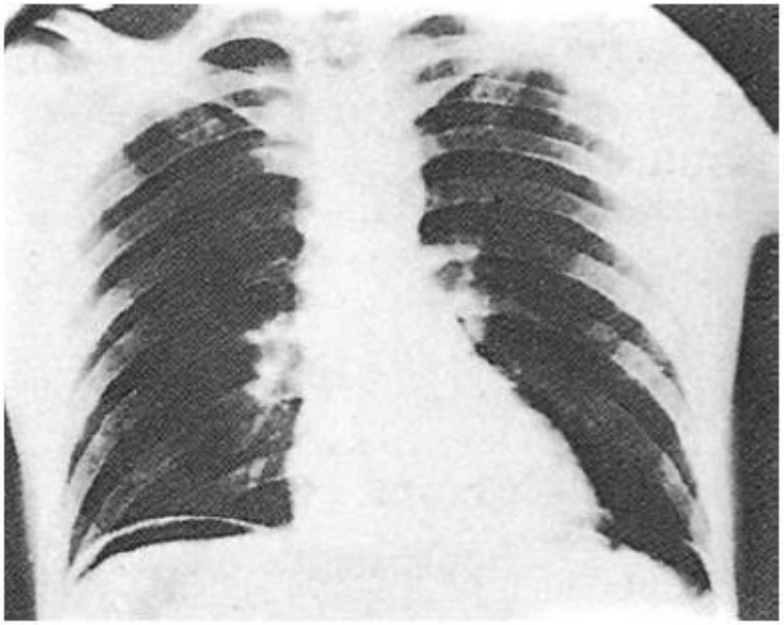


Figure 63.23 Plain abdominal radiograph of a perforated ulcer, sho
ing air under the diaphragm.

2—PYLORIC STENOSIS

- Due to fibrosis or cicatrisation of duodenum or sometimes due to oedema associated with prepyloric ulcer
- Also called GASTRIC OUTLET OBSTRUCTION DUODENAL STENOSIS.
- Other causes of gastric outlet obst:
- carcinoma pylorus, adult hypertrophic pyloric stenosis, pyloric mucosal diaphragm.

CLINICAL FEATURES

- There is a history of long standing peptic ulcer .
- SYMPTOMS

1-Pain, 2- Vomiting (foul) no bile, undigested food.

SIGNS

- 1-Wt loss, 2 - dehydration , pale 3-distended epigastric region, 4- visible peristalsis from left to right, 5- succussion splash positive (audible) on shaking abdomen of the patient.

METABOLIC EFFECT

- There will be HYPOKALEMIC METABOLIC ALKALOSIS
- PARODOXICAL ACIDURIA.
- DEHYDRATION PLUS ANAEMIA PLUS VITAMINE DIFICIENCY.

INVESTIGATIONS

1-O.G.D. + BIOPSY. 2- BARIUM MEAL
3-BLOOD+ELECTROLYTES TEST.

TREATMENT

- 1-N.G TUBE 2- I.V.FLUID +NACL + KCL. 3-WATCH URINE OUT PUT. 4- ANTI ULCER TREATMENT.
- MILD CASES DUE TO OEDEMA WILL RESPOND TO CONSERVATIVE.SEVER CASES WILL NEED SURGERY
- IF THE ULCER HEALED DO ONLY DRAINAGE BY GASTROJEJUNOSTOMY ONLY.
- 2-IF THERE IS ACTIVE ULCER DO TRUCAL VAGOTOMY PLUS GASTROJEJUNOSTOMY OR PYLOROPLSTY.
- ***FOLLOW-UP AS FOR PATIENT WITH PERFORATED PEPTIC ULCER.***

3—BLEEDING (HAEMATEMESIS & MELENA) OF PEPTIC ULCER

- Either 1- slight 2- major bleeding.
- Sometimes there is history of peptic ulcer, drug intake like NAIDs &ASPIRIN.
- THERE IS EROSION OF AN ARTERY IN THE FLOOR OF AN ULCER, LIKE GDA OR SPLENIC ARTERY,IF PATIENT ABOVE 40 YS THERE IS ATHEROSCLEROSIS SO THE ARTERY WILL NOT CONTRACT TO ARREST BLEEDING
- **SYMPTOMS & SIGNS**

FEATURES OF BLEEDING.

- **TREATMENT**

RESUSCITATION

- I.V.CANNULA, CVP LINE, BLOOD SAMPLE FOR BLOOD TEST, I.V. FLUID, BLOOD TRASFUSION, INJACTABLE ANTI ULCER TREATMENT, NG TUBE, FOLEY 'S CATHETER, PULSE & BP CHART, OXYGEN,

SPECIFIC INVESTIGATIONS FOR DIAGNOSIS SPEC. O.G.D.; ANGIOGRAPHY…

CONSERVATIVE TREATMENT: IF BLEEDING STOP CONTINUE MEDICAL TREATMENT +/- MINIMAL INTERVENTION.

- **MINIMAL INTERVENTION**

MANY O.G.D. DEVICES ARE AVAILABLE CAN ACHIEVE HAEMESTASIS : INJECTION DILUTED ADRENALINE ,,OR NACL INJECTION ,OR BY ANGIOGRAPHY TO FIND THE SITE OF THE BLEEDER AND EMBOLIZATION.

INDICATION OF SURGERY:

– IF BLEEDING STOPPED BUT RECCUR AGAIN AFTER ADMISSION TO HOSPITAL .

– IF BLEEDING NEVER STOP SINCE ADMISSION TO HOSPITAL & PATIENT RECEIVE MORE THAN 6 UNITS BLOOD.

SURGICAL TREATMENT

- AIM OF SURGERY IS TO STOP THE BLEEDING.
- For POST. D.U:

DO LAPARATOMY BY UPPER MIDLINE INCISION, MOBILIZE THE DUODENUM, DO LONGITUDINAL INCISION 3,5 CM STOMACH SITE 2,5 CM DUODENUM SITE, BY CUTTING PYLORIC SPHINCTOR

- MULTIPLE INTERRUPTED UNDERRUNING SILK SUTURES,
- THEN DO PYLOROPLASTY,
- EITHER DO TRUNCAL VAGOTOMY OR CONTINUE GIVING ANTI ULCER TREATMENT

- ON RARE OCCASION THERE WILL BE A NEED TO DO LIGATURE TO GASTRODUODENAL ARTERY OR TO DO PARTIAL GASTRECTOMY.

- IN BLEEDING GASTRIC ULCER PROCEDURE THE SAME

1-EITHER ARREST BLEEDING +BIOPSY,, OR, 2—EXCISION BIOPSY TO THE ULCER AND CLOSE STOMACH.

HAEMATEMESISS & MELAENA (UPPER GASTROINTESTINAL BLEEDING)

- **CAUSES**

1- CHRONIC PEPTIC ULCER 60%

2- ACUTE PEPTIC ULCER 26% (EROSION)

3- OESOPHAGEAL VARICES 4%

4- MALLORY –WEISS TEAR (SYNDROME) 4%: Longitudinal tear below the GEJ due to repetitive strenuous vomiting.

5- TUMOUR O.5%

6- VASCULAR e.g.DIEULAFAYS DISEASE O.5%

7- OTHERS 5% e.g.PURPURA,,HAEMOPHILIA

- **MANAGEMENT**

- SAME RESUSCITATION PLUS INVESTIGATIONS

- SPECIFIC TREATMENT ACCORDING TO THE CAUSE

Comparing Duodenal and Gastric Ulcers

- **DUODENAL ULCER**
- Age 30–60
- Male: female 2–3:1
- 80% of peptic ulcers are duodenal

GASTRIC ULCER

- Usually 50 and over
- Male: female 1:1
- 15% of peptic ulcers are gastric

Signs, Symptoms, and Clinical Findings

- **DUODENAL ULCER**
- Hypersecretion of stomach acid (HCl)
- May have weight gain
- Pain occurs 2–3 hours after a meal; often awakened between 1–2 AM;
- ingestion of food relieves pain
- Vomiting uncommon

- **GASTRIC ULCER**
- Normal—hyposecretion of stomach acid (HCl)
- Weight loss may occur
- Pain occurs 1/2 to 1 hour after a meal; rarely occurs at night; may be relieved by vomiting;
- ingestion of food does not help, sometimes increases pain
- Vomiting common

Comparing Duodenal and Gastric Ulcers

- **DUODENAL ULCER**
- **Hemorrhage less** likely than with gastric ulcer, but if Present, **melena** more common than Hematemesis
- More likely to perforate than gastric ulcers

- **GASTRIC ULCER**
- **Hemorrhage more** likely to occur than with duodenal ulcer; **hematemesis** more common than melena

Comparing Duodenal and Gastric Ulcers

- **GASTRIC ULCER**
- **Hemorrhage more** likely to occur than with duodenal ulcer; **hematemesis** more common than melena

- **GASTRIC ULCER**
- Occasionally
- *H. pylori*, gastritis, alcohol, smoking, use of NSAIDs, stress

GASTRIC NEOPLASMS

BENIGN TUMOURS.

- LEIOMYOMA
- NEUROFIBROMA
- POLYP(S): Metaplastic (H. pylori), Inflammatory, Fundic gland polyp(+ use of PPI), Adenoma (10%, premalignant), Carcinoid.

GASTRIC CANCER

- GASTRIC CANCER IS ONE OF THE MOST COMMON CAUSES OF DEATH IN THE WORLD.
- THE OUTCOME IS GENERALY POOR, OWING TO THE ADVANCED STAGE AT TIME OF PRESENTATION.
- EARLY PRESENTATION OF GASTRIC CANCER IS ASSOCIATED WITH VERY HIGH CURE RATE.

INCIDENCE

- Variable worldwide, high incidence in Japan.
- MEN AFFECTED MORE THAN WOMEN.
- INCIDENCE INCREASE WITH AGE.
- IN THE WEST GASTRIC CANCER IS CONTINUING TO FALL BY 1%PER YEAR.THIS REDUCTION AFFECT CANCER ARISING FROM BODY & PYLORUS, WHILE THERE IS INCREASE IN CANCER IN THE PROXIMAL STOMACH.
- CANCER OF BODY, PYLORUS ARE MORE COMMON IN LOW SOCIOECONOMIC GROUP.
- PROXIMAL GASTRIC CANCER AFFECT HIGH SOCIOECONOMIC GROUP..
- PROXIMAL GASTRIC CANCER NOT ASSOCIATED WITH H,PYLORI
- BODY & PYLORUS CANCER IS ASSOCIATED WITH H. PYLORI..

AETIOLOGY

1- H.PYLORI ASSOCIATED WITH BODY & PYLORIC TUMOR.

H.PYLORI CAUSE GASTRITIS , THEN GASTRIC ATROPHY, & INTESTINAL METAPLASIA

2- PERNICIOUS ANAEMIA & GASTRIC ATROPHY ARE RISK FACTOR.

4-GASTRIC POLYP.

5-AFTER GASTRIC SURGERY eg. BILLROTH II, PYLOROPLASTY, GASTROJEJUNOSTOMY,WILL INCREASE THE RISK 4 TIMES, DUE TO REFLUX BILE GASTRITIS.

7—CIGARETTE SMOKING, DUST INGESTION,,

8—INGESTION OF SPIRIT CAUSE GASTRITIS ON LONG RUN CHANGE TO CANCER.

9—GENETIC FACTOR,

CLINICAL FEATURES

- **SYMPTOMS**
  - DYSPEPSIA,
  - POOR APPITITE, WEIGHT LOSS.
  - DISTENSION,
  - BLEEDING,
  - ANAEMIA,
  - DYSPHAGIA,VOMITING,
  - LUMP AT **EPIGASTRIC REGION**
- **SIGNS**
  - EPIGASTRIC TENDERNESS,
  - ?MASS,
  - STOMACH SPLASH,
  - DEHYDRATION,,FEATURES OF PYLORIC OBSTRUCTION,
  - THROMBOPHLEBITIS OF SUPERFICIAL Vs. OF THE LEG (Trousseau' s sign),
  - ENLARGED LEFT SUPRACLAVICULAR LYMPH NODES(Troisier'sign),
  - LIVER METASTASIS CAUSE JAUNDICE,
  - ASCITES.

PATHOLOGY

1 – INTESTINAL GASTRIC CANCER (Arise in intestinal metaplasia)

A – POLYPOIDAL.

B – ULCERATIVE

2 – DIFFUSE GASTRIC CANCER INFILTRATES DEEPLY IN STOMACH WITHOUT OBVIOUS MASS (LINITIS PLASTICA) OR CALLED (LEATHER BOTTLE STOMACH). BAD PROGNOSIS.

- MICROSCOPICALLY IT IS COLUMNAR CELL
- MOST COMMON SITE IS AT PROXIMAL STOMACH , WHILE IN JAPAN IT IS MORE DISTAL.

STAGING CARCINOMA STOMACH (TNM)

- Early cancer: limited to mucosa & submucosa i.e T1, any N.
- Advanced cancer involves the musculais.

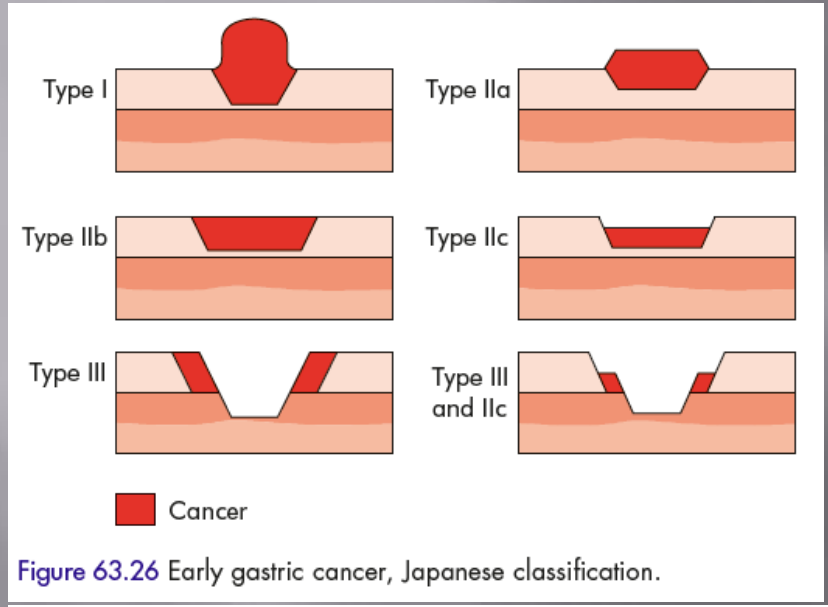


Figure 63.26 Early gastric cancer, Japanese classification.

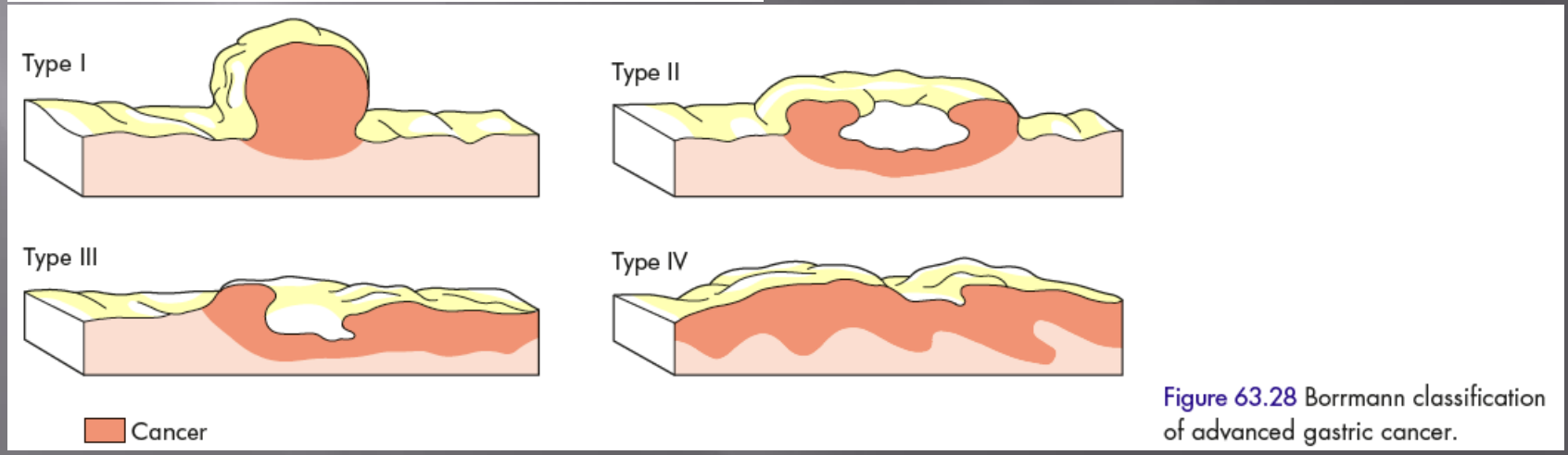


Figure 63.28 Borrmann classification of advanced gastric cancer.

Table 63.5 International Union Against Cancer (UICC) staging of gastric cancer.

T1	Tumour involves lamina propria, submucosa		
	T1a lamina propria		
	T1b submucosa		
T2	Tumour invades muscularis propria		
T3	Tumour involves subserosa		
T4a	Tumour perforates serosa		
T4b	Tumour invades adjacent organs		
N0	No lymph nodes		
N1	Metastasis in 1–2 regional nodes		
N2	Metastasis in 3–6 regional nodes		
N3a	Metastasis in 7–15 regional nodes		
N3b	Metastasis in more than 15 regional nodes		
M0	No distant metastasis		
M1	Distant metastasis (this includes peritoneum and distant lymph nodes)		
Staging			
IA	T1	N0	M0
IB	T1	N1	M0
	T2	N0	M0
IIA	T1	N2	M0
	T2	N1	M0
	T3	N0	M0
IIB	T1	N3	M0
	T2	N2	M0
	T3	N1	M0
	T4a	N0	M0
IIIA	T2	N3	M0
	T3	N2	M0
	T4a	N1	M0
IIIB	T3	N3	M0
	T4a	N2	M0
	T4b	N0–1	M0
IIIC	T4a	N3	M0
	T4b	N2–3	M0
IV	Any T	Any N	M1

SPREAD OF GASTRIC CANCER

1-DIRECT INVADE THE WALL THEN, LIVER, PANCREAS, COLON, OMENTUM.

2-LYMPHATIC SPREAD BY PERMIATION & EMBOLIZATION.

3-BLOOD-BORNE METASTASIS.

4-TRANSPERITONEAL SPREAD.

LN number		Site of cancer			
		Antrum	Middle	Cardia	Cardia and oesophagus
1	Right cardia	N2	N1	N1	N1
2	Left cardial		N1	N1	N1
3	Lesser curve	N1	N1	N1	N1
4sa	Short gastric	N1	N1	N1	N1
4sb	Left gastroepiploic	N1	N1	N1	N1
4d	Right gastroepiploic	N1	N1	N2	N2
5	Suprapyloric	N1	N1	N2	N2
6	Infrapyloric	N1	N1	N2	N2
7	Left gastric artery	N2	N2	N2	N2
8a	Anterior hepatic artery	N2	N2	N2	N2
9	Coeliac artery	N2	N2	N2	N2
10	Splenic hilum		N2	N2	N2
11	Splenic artery		N2	N2	N2
19	Infradiaphragmatic				N2
20	Oesophageal hiatus			N2	N1
110	Lower oesophagus				N2
111	Supradiaphragmatic				N2

The nodes in stations 12–18 are not routinely removed in a D1 or D2 gastrectomy.

INVESTIGATIONS

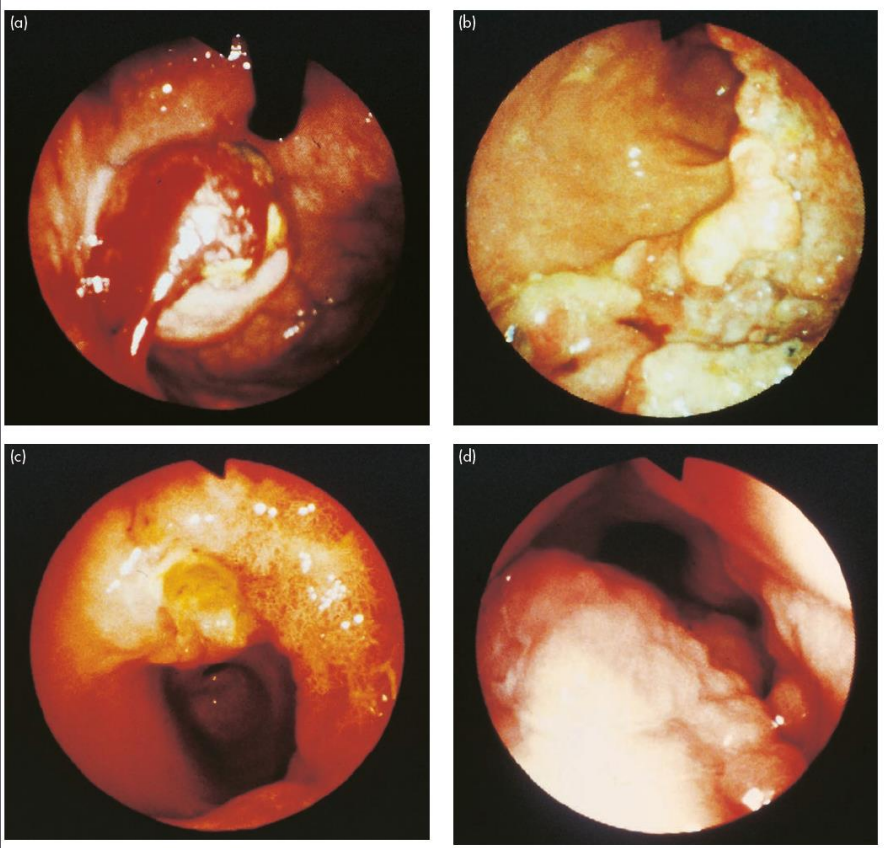


Figure 63.29 Advanced gastric cancer. (a) Type I; (b) type II; (c) type III; (d) type IV (linitus plastica) (courtesy of Dr GNJ Tytgat, Amsterdam, The Netherlands).

TREATMENT OF GASTRIC CANCER

1-PALLIATIVE SURGERY

- GASTROJEJUNOSTOMY
- ENDOSCOPIC STENTING.

2-RADICAL RESECTION

- TOTAL GASTRECTOMY + ROUX EN Y ESOPHAGOJEJUNOSTOMY.
- SUBTOTAL (BILLROTH II) GASTRECTOMY for distal cancer.
- Gastric cancer is chemosensitive: given as neoadjuvant therapy (Cis-platinum, Epirubicin, 5-FU).

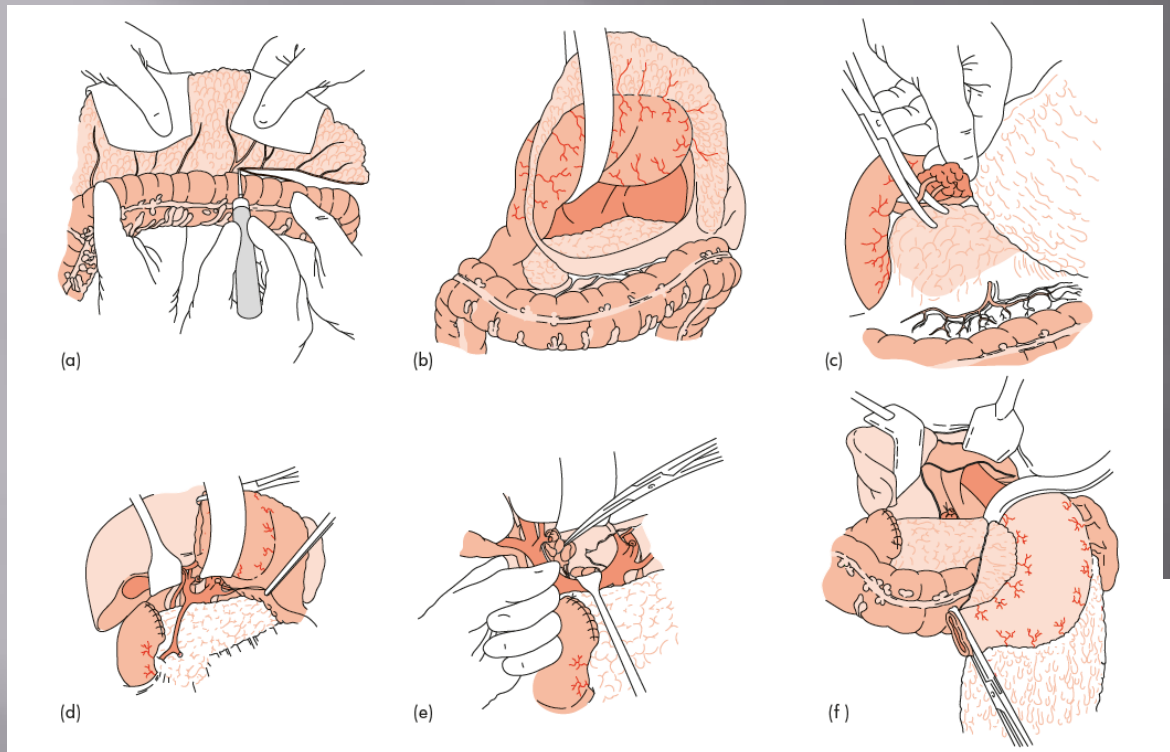


Figure 63.31 Radical total gastrectomy. (a) Dissection of omentum off the transverse colon. (b) Exposure of the lesser sac. (c) Splenectomy Division and oversewing of the duodenum. (e) Dissection of the left gastric artery nodes (group 17). (f) Mobilisation of the oesophagus.

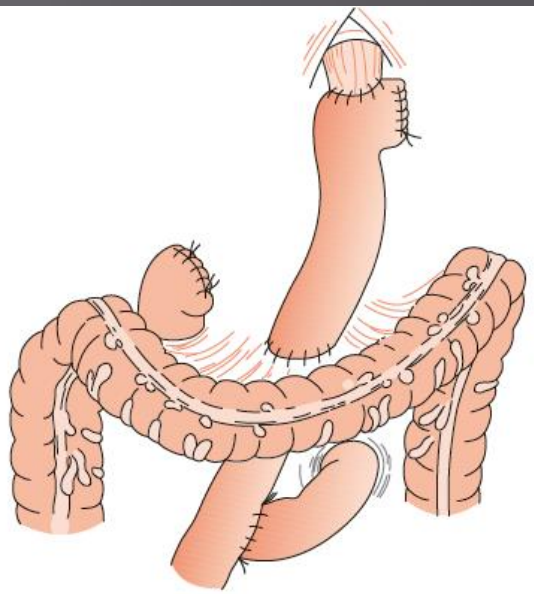
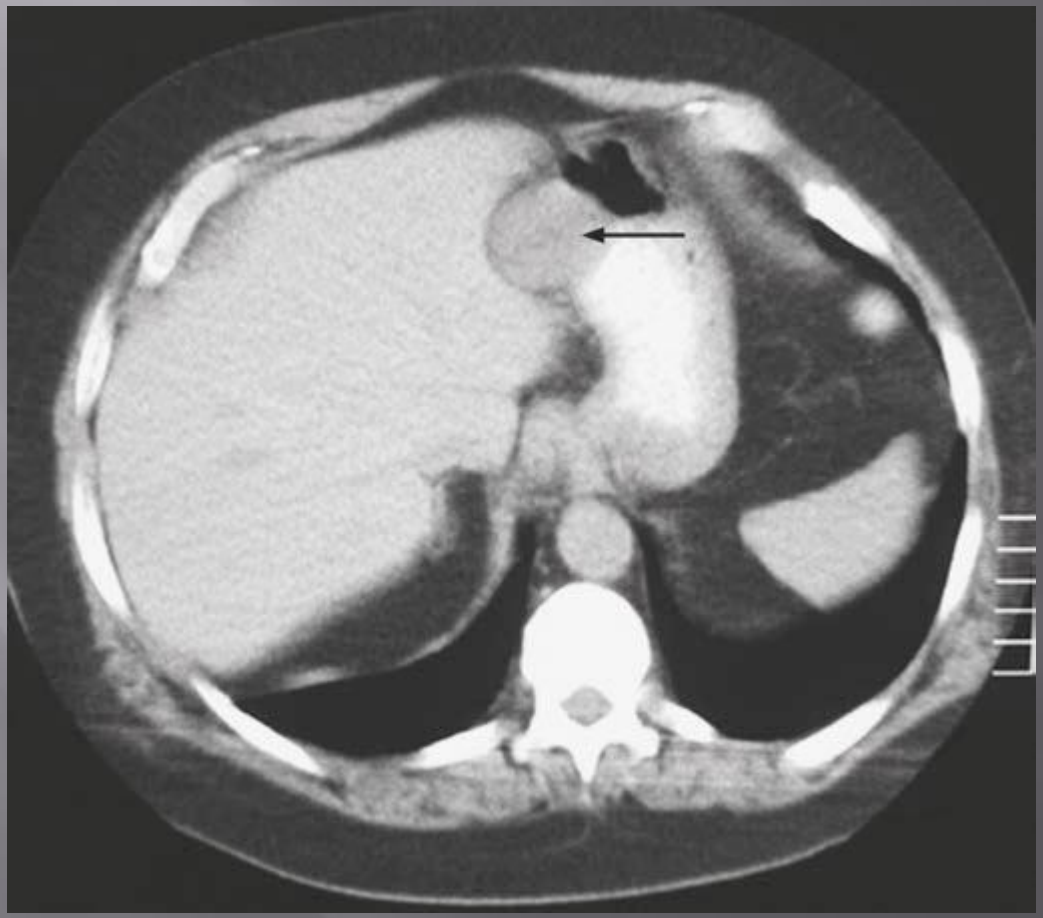


Figure 63.32 Oesophagojejunostomy Roux-en-Y.

GASTROINTESTINAL STROMAL TUMOURS

- GIST comprise 1–3 per cent of all gastrointestinal neoplasia, 50 % affect the stomach.
- equally in males and females.
- associated with a mutation in the tyrosine kinase *c-kit* oncogene.
- mucosa overlying the tumour ulcerates, leading to bleeding, or that they are noticed incidentally at endoscopy.
- Because the mucosa overlying the tumour is normal, endoscopic biopsy can be uninformative; Targeted biopsy by endoscopic US is more helpful.
- Tumours over 5 cm in diameter should be considered to have metastatic potential.

- treated by wedge excision.
- These tumours are sensitive to the tyrosine kinase antagonist imatinib.



GASTRIC LYMPHOMA

1- Primary gastric lymphoma: ~5 % of gastric neoplasms; B cell derived, arising from the mucosa-associated lymphoid tissue (MALT)

2- Stom. involvement in generalized lymphoma.

- most common in the sixth decade.
- Symptoms: pain, weight loss and bleeding. Acute presentations of gastric lymphoma, such as haematemesis, perforation or obstruction, are not common.
- form a diffuse mucosal thickening, which may ulcerate
- Diagnosis made by endoscopic biopsy.
- Surgery for localized disease, & chemotherapy for systemic disease.
- Early lymphoma may regress by H.pylori eradication.

Duodenal tumours

- Duodenal villous adenomas are commonly found around the ampulla of Vater and are premalignant
- Duodenal carcinoma is uncommon, but the most common site for adenocarcinoma is in the small intestine
- Both adenoma and carcinoma occur commonly in patients with familial polyposis and screening these patients is advised
- Pancreatic cancer is the most common cause of duodenal obstruction

DUODENAL OBSTRUCTION

Duodenal obstruction in the adult is usually due to malignancy, and cancer of the pancreas is the most common cause. About one-fifth of patients with pancreatic cancer treated with endoscopic stenting will develop obstruction. Treatment is usually by gastroenterostomy but duodenal stenting is increasingly being used.

Other causes ofduodenal obstruction,
including metastases from colorectal and gastric cancer.
Primary duodenal cancer is much less common as a cause of obstruction than these other malignancies.

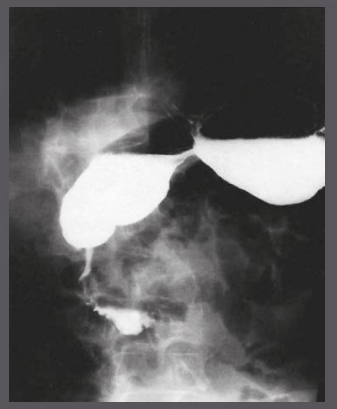
Acute gastric dilatation

This condition usually occurs in association with pyloroduodenal disorders or postsurgery without nasogastric suction. The stomach, which may also be atonic, dilates enormously. Often the patient is also dehydrated and has electrolyte disturbances.

Failure to treat this condition can result in a sudden massive vomit with aspiration into the lungs. The treatment is nasogastric suction, with a large-bore tube, fluid replacement and treatment of the underlying condition.

Volvulus of the stomach

Rotation of the stomach usually occurs around the axis and between its two fixed points, i.e. the cardia and the pylorus. In theory, rotation can occur in the horizontal (organoaxial) or vertical (mesenteroaxial) direction but, commonly, it is the former which occurs. This condition is usually associated with a large diaphragmatic defect around the oesophagus (paraoesophageal herniation) (Figure 63.35). What commonly happens is that the transverse colon moves upwards to lie under the left diaphragm, thus taking the stomach with it, and the stomach and colon may both enter the chest through the eventrationof the diaphragm. The condition is commonly chronic,the patient presenting with difficulty in eating. An acute presentation with ischaemia may occur. Endoscopically, it can be



Treatment

A-open surgery
B-Laparoscopic treatment

Include :
1-close defect in the diaphragm with mesh
2-separate the stomach from the transverse colon
3- anterior gastropexy to fix the stomach to the anterior abdominal

THANK YOU