

# STUDY OF ENDOSCOPY IN GASTRIC LESIONS



Dissertation submitted in partial fulfillment of regulation for the award of  
M.S. Degree in General Surgery  
(Branch I)



The Tamilnadu  
Dr. M.G.R. Medical University  
Chennai  
March 2009

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Coimbatore Medical College  
Coimbatore - 641 014

## **CERTIFICATE**

Certified that this is the bonafide dissertation done by **Dr.A. KAVITHA PRIYA** and submitted in partial fulfillment of the requirements for the **Degree** of M.S., General Surgery, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai.

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## **DECLARATION**

I solemnly declare that the dissertation titled  
**“THE STUDY OF ENDOSCOPY IN GASTRIC LESIONS”**  
was done by me from 2006 onwards under the guidance and supervision of  
**Professor Dr. R. PERUMAL RAJAN M.S.**

This dissertation is submitted to the Tamilnadu Dr. MGR  
Medical University towards the partial fulfillment of the requirement for the  
award of MS Degree in General Surgery (Branch I).

Place: Coimbatore

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IN GASTRIC LESIONS

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Date : 8.10.2007

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## **AIM OF THE STUDY**

Inspection of upper gastrointestinal tract has been carried out for many years using rigid endoscopes and multiple lenses, such procedures were difficult and complete visualization of the stomach was not possible.

The present study envisages to fully exploit the advantages of flexible fibre optic endoscope in the detection of gastric lesions.

## **REVIEW OF LITERATURE**

### **HISTORY OF ENDOSCOPE**

In 1868, first practical rigid upper GI endoscope was demonstrated by Kussmaul -when he performed gastroscopy with the help of a cooperative Sword swallower<sup>8</sup>.

In 1881 Karl Stoerk, an Austrian Laryngologist, examined under dim light the entire length of the oesophagus with a rigid tube<sup>8</sup>.

In 1881, at the same time, Johann Von Mikulicz constructed a gastroscope that was angulated to 30\* at the distal third of a hollow tube<sup>8</sup>.

During the next 50 years Chevalier Jackson introduced rigid broncoesophagoscopy and several German physicians built

gastroscope, all of which proved to be both impractical and dangerous<sup>13</sup>.

A new era for endoscopy began in 1932, when the gastroscope designed by Rudolf Schindler and manufactured by George Wolf in Berlin was introduced. This semi flexible gastroscope was constructed on the principle that a series of convex lenses transmit light without distortion through a flexible tube if the distal portion is not bent beyond a certain angle<sup>15</sup>.

In the early 1950s, an intragastric camera was manufactured in Japan. This consisted of a flexible tube with a miniature camera incorporated in its tip<sup>15</sup>.

In 1954, Hopkins and Kapany produced a clad glass fiber that allowed light to travel from one end of the glass fiber to the other, no matter how the fiber was twisted or coiled<sup>17</sup>.

In 1957, Hirschowitz et al employed these fibers to construct a flexible fibre optic gastroscope<sup>18</sup>.

In 1969 charged couple device (CCD) was introduced in endoscopic instruments<sup>19</sup>.

In 1970, the length of the fibroesophagoscope was gradually increased to 105 cm so that nearly complete inspection of the oesophagus, stomach and duodenum was possible<sup>8</sup>.

In 1980s Welch Allyn introduced electronic endoscope for clinical trial<sup>19</sup>.

1980-1990 - Advent of video endoscopy and endoscopic ultrasonography.

1990-2000 - Explosion of laparoscopic surgery and small bowel endoscopy.

2000 - Video capsule endoscopy was introduced.

2002- Infrared Fibre optic endoscope - Angle research center,  
Hampton, Virginia.

## **PRINCIPLES OF ENDOSCOPY**

The advent of fibre optic endoscopy is undoubtedly one of the most exciting recent advances in gastroenterology and has markedly increased the diagnostic accuracy for certain diseases. Unfortunately this improvement has not always been accompanied by a similar benefit in the management of the patient and the accepted indications for endoscopy have frequently not been subjected to controlled clinical trials.

The mortality of the endoscopy varies quite widely in different centers, depending upon the expertise of the endoscopist. The major problems are respiratory (hypoxia due to sedation and aspiration pneumonia), perforation of the esophagus. Thus all patients presenting with dysphagia should have a barium swallow as an initial investigation to assess the level of stricture and look for unsuspected lesions such as pharyngeal pouch. Hepatic coma precipitated by sedation in patients with advanced liver disease may also occur.

## **INSTRUMENTS**

A wide range of endoscopes are now available and only those long enough to examine the whole of the stomach and upper part of the duodenum should be used (10 cm working length). The most important part of the endoscope is the fibro optic bundle, which consists of more than 2, 00,000 glass coated 10 micro meter fibers, bound firmly together in the same spatial relationship, but free in between (a coherent bundle). Some fibers transmit the light; others transmit the image by a series of total internal reflections along the length of the bundle. Each fiber transmits a minute proportion of the image and hence, the importance of the constant spatial relationship of the fibers in producing an accurate composite picture. The overall diameter of the shaft is limited to 10- 12 mm for easy passage through the esophagus. The distal endoscope is flexible and the tip can be angulated acutely in two planes often more than 180 degrees<sup>8</sup>.

The image is focused into the fiber bundle by a distal lens which may forward viewing (80 to 105 degrees wide angle lens) or

side viewing when it is set at the right angles to the direction of the fiber bundle. Attempts have also been made to combine the advantages of the end viewer and side viewer by setting the lens obliquely at the tip of the instrument (field of view 90 degrees diagonally, 60 degrees vertically and horizontally) in all circumstances other than endoscopic cannulation of the ampulla, the procedure should use the forward or forward oblique endoscope which allows examination of the esophagus. There are potential areas of the stomach, the proximal portion of the duodenal bulb and the upper portion of the second part of the duodenum that may be difficult to see with this instrument fig-1, 2, 3.

These blind spots have been reduced by increasing the angle of view of the modern end viewing endoscopes, and the smallest diameter (P scope) can often be inverted with the bulb and the second part of the duodenum to provide a good view of the whole mucosa in this region. Nevertheless, all trained endoscopists are also familiar with the side viewing endoscope, which provide a good view of the stomach and duodenal bulb. Ulcers surrounded by edematous fold



are often more easily seen 'en face' with side viewing endoscopes. Inverting the endoscope to view the Fundus (J maneuver) is now a routine part of the examination of the stomach and is made simple by the modern with increased tip flexibility.

The most modern instrument has a common channel to insufflate air or water to cleanse the lens and a separate suction and biopsy tunnel. Immediate cleansing of both channels after endoscopy is an essential ritual to prevent Clogging with mucus or blood, this result in need for extensive repair by the manufacturers. The most endoscopy units have now trained nursing personnel familiar with the techniques involved. However, these delicate and highly expensive instruments are also used in emergencies in out of duty hours and it is imperative that anyone using the endoscope be familiar with the cleaning routine<sup>8,11</sup>.

## **BIOPSY FORCEPS AND CYTOLOGY BRUSHES**

Biopsy forceps consists of sharpened cusps controlled by a flexible cable. Often there is a central spike between the cusps that enables larger specimens to be taken and is also helpful when lesions are biopsied tangentially. Cytology brushes have covering sleeve that protects the sample during withdrawal<sup>8,11</sup> fig-4.

## **ANATOMY OF STOMACH**

### **Embryology**

The stomach arises as a spindle shaped dilatation of the foregut during the 4<sup>th</sup> week of gestation. The fully developed stomach is the largest dilatation of the gut and lies between the esophagus and duodenum.

The stomach is divided into

- 1) Fundus
- 2) Body
- 3) Antrum

The stomach is situated in the upper part of the abdomen. It is roughly “J” shaped and has two openings. The cardiac and the pyloric orifices, two curvatures known as the “Greater curvature” and the “Lesser curvature” and two surfaces an anterior and a posterior surface fig-5,6.

The Fundus is a dome shaped projection upward and to the left of the cardiac orifice. The body extends from the level of the cardiac

orifice to the level of the incisura angularis a constant notch in the lower part of the lesser curvature. The pyloric Antrum extends from the incisura to the proximal level of the pylorus. The pylorus is the most tubular part of the stomach. The cavity of the pylorus is called the pyloric canal. Its thick muscular wall forms the pyloric sphincter.

Accordingly to the functions of the stomach it may be divided into

- a) Fundus -parietal cell or oxyntic gland area.
- b) Antrum -Pyloric gland area.

The Fundus secretes acid-peptic juice and the Antrum secretes a thick viscid, relatively alkaline mucous and the hormones gastrin and Somatostatin.

### **Arterial supply**

1. The left gastric artery: This arises from coeliac axis, and divides into an ascending branch and descending branch. The descending

branch, lying between the layers of the lesser omentum is closely opposed to the lesser curvature and sends branches to the stomach.

2. The right gastric artery: arising from the common hepatic artery also divides into a number of branches to supply.
3. Stomach along the lesser curvature and anastomosis with left gastric artery.
4. The right gastro epiploic artery, branch of the gastro duodenal artery which runs from right to left along the greater curvature to join the left gastro epiploic artery.
5. The left gastroepiploic artery: A branch of splenic artery contributes to the arcade along the greater curvature.
6. 5 to 7 small branches from the splenic artery supply the fundus.  
(Short gastric arteries).

7. The left inferior phrenic artery supplies a small area around the Fundus.
  
8. Oesophagus has a rich intramural arterial anastomosis in its submucosal layer, which is continuous with a network in submucosa of stomach. This explains how, following a near total gastrectomy an upper gastric remnant retains its blood supply fig-7.

Blood supply to the greater omentum comes from the right and left gastroepiploic arteries which form an arcade along the greater curvature of the stomach. The right epiploic artery (from right gastroepiploic artery) and left epiploic artery (from left gastroepiploic artery) form an anastomotic arcade in the lower part of the omentum which is joined by accessory epiploic arteries (from the two gastroepiploic arteries) and provide a rich blood supply to the omentum. When mobilizing greater omentum from the stomach, it is therefore not necessary to preserve the gastroepiploic arcade provided epiploic arcade is preserved<sup>3, 6, 7</sup>.

## **Venous Drainage**

These commence as straight vessels between the mucosal glands and drain into the sub mucosal veins. Larger veins accompany main arteries and drain into splenic and superior mesenteric vein and portal vein fig-8.

## **Lymphatic drainage**

These smaller vessels are said to resemble the veins in distribution. Lymph vessels of the stomach are situated along the following sites.

1. Sub mucosal plexus
2. Intra muscular plexus
3. Sub Serosal Plexus.

They anastomose freely with each other. This network drains into the large lymphatic vessels; accompany the four main vessels to the greater and lesser curvature. The sub mucosal lymphatics of the stomach and lower oesophagus communicate freely, facilitating the

spread of carcinoma from one organ to another. The lymphatics of the antrum drain into the right gastric node superiorly, and right gastro epiploic and sub pyloric node inferiorly fig-9.

The lymphatics of the pylorus drain into right supra pyloric node superiorly, and sub pyloric nodes situated along the gastro duodenal artery inferiorly. The efferent lymphatics from the supra pyloric lymph nodes converge on the Para aortic nodes around the coeliac axis, while the efferent lymphatics from the sub pyloric lymph nodes pass up to main superior mesenteric nodes situated around the origin of the superior mesenteric artery.

The Japanese research society for gastric cancer has assigned a number to each lymph node station to aid the pathological staging. They are grouped in to 3 tiers. These tiers are known as N1, N2, and N3 each of which is numbered.



<b>N1</b>	<b>N2</b>	<b>N3</b>
1. Right cardiac	7. Left gastric	12. Nodes in lesser omentum
2. Left cardiac	8. Common hepatic	13. Retro pancreatic nodes
3. Lesser curvature	9. Coeliac	14. Nodes in bowel mesentry
4. Greater curvature	10. Nodes along the splenic hilum	15. Nodes along middle Colic artery
5. Sub pyloric	11. Nodes along the splenic artery	16. Para aortic nodes
6. Supra pyloric		

The differentiation between a D1 and a D2 operation depends upon the tiers of nodes removed. Different tiers need to be removed depending upon the positions of primary tumour. In general, a D1 resection involves the removal of perigastric nodes and D2 resection

involves the clearance of the major arterial trunks. There remains some controversy about the extent of the lymphadenectomy required for the optimal treatment of curable gastric cancer.

In Japan, at least a D2 gastrectomy is performed for all operable gastric cancer; whether or not there is histological evidence of regional lymph node involvement affects the prognosis of operable cases of carcinoma of the stomach. Retrograde Spread may occur if the upper lymphatics are blocked. In Japan the lymph node Dissection is highly advanced.

### **Innervation**

The motor and sensory innervation of the stomach is by the vagus. The entire gastrointestinal tract has an extensive intrinsic innervations grouped in two principle plexus fig-10.

- i) Myentric plexus of Auer Bach.
- ii) The sub mucosal plexus of Meissner's<sup>3,6,7</sup>.

## **HISTOLOGY**

The wall of the stomach is composing of four layers fig-11.

- i) Mucosa
- ii) Sub mucosa
- iii) Muscle
- iv) Serosa<sup>3</sup>.

## **TYPES OF CELLS AND FUNCTION**

- i) Parietal cells - Hydrochloric acid and gastric intrinsic factor
- ii) Chief cells - Pepsinogen
- iii) Goblet cells - mucus
- iv) Epithelial cells - Extra cellular fluid (non parietal secretion)
- v) Gastrin cells/ G cells – Gastrin
- vi) Delta cells – Somatostatin
- vii) Mast cells – Heparins & histamine
- viii) Fundic argentaffin cells – Somatostatin<sup>3</sup>.

## **PHYSIOLOGY**

### **Functions of the stomach**

**1. Storage:** the function of the stomach is to act as a reservoir for ingested food. The main function of the stomach is to mix and churn the food so that it is delivered slowly to the duodenum. Swallowed food enters the stomach where it is mixed with gastric juice and changed to a more liquid form. The storage function of the stomach is mainly performed by receptive relaxation. The upper portion of the stomach relaxes as the intake of food is anticipated. Food particles are reduced in size by the grinding action of the Antrum Pylorus constantly returns ingested material to the proximal stomach to be churned repeatedly until and unless it is ready for delivery to the duodenum.

**2. Digestion:** Small amount of digestion takes place in the stomach, mostly by proteolysis. Pepsin, the proteolytic enzyme of the stomach is active in acid environment (pH below 5). It also secretes mucus,

which prevents auto digestion of the stomach. The liquefied, churned food which has undergone slightly proteolysis is then delivered slowly into the duodenum. The mixing and slow emptying of the mixed meal from the stomach is performed by 'antral pump'. Contraction of the body of the stomach propels contents into the gastric antrum. As the antrum fills the pylorus opens to allow the escape of some chyme. When contraction wave reaches the pylorus it closes. The antral contents are now pushed back into the body of the stomach. Thus each contraction wave produces both the escape of a small quantity of chyme and mixing of the remainder. The amount of chyme passed through the pylorus with each contraction will depend upon the viscosity and amount of solid in the gastric contents. Fluids are emptied more rapidly. Starches undergo enzymatic breakdown by low pH, which is favourable for the activity of salivary alpha-amylase. Peptic digestion is mainly aimed at to start proteolysis and initiate dispersion of fats, proteins and carbohydrates by breaking down cell walls. Gastric mucosa also secretes a lipase which assists in the early stage of fat digestion.

**3. Haematopoiesis:** It produces intrinsic factor (by the parietal cells) which is essential for absorption of vitamin B12 and thus helps haematopoiesis.

**4. Sterility:** of the foregut is mainly maintained by gastric acid. Majority of the bacteria die due to the low gastric PH. Only a few unusual fusiform bacilli can withstand the gastric acid.

**5. Defence mechanism:** Gastric mucosa has got the capacity to protect its surface from harmful ingestants. Rapid mucus release is the first line of defense. If potentially dangerous material permeates through the mucosa, the lamina propria stands in the way with the army of mast cells, macrophages and lymphocytes.

**6. Heat exchange:** The stomach, due to its abundant mucosal microcirculation, can act as heat exchanger. Due to this mechanism a stable thermal environment is maintained against too cool or too warm ingestant. This offers protection to the adjacent viscera against thermal damage.

Stimulation of gastric secretion occurs through three phases.

**1. Cephalic phase** in which the secretion is stimulated by thinking of food.

**2. Gastric phase** in which the secretion is stimulated by presence of food in the stomach.

**3. In the intestinal phase**, the presence of chyme in the duodenum and small bowel inhibits gastric emptying and the acidification of duodenum leads to production of secretin that also inhibits gastric acid secretion, along numerous other peptides originating from the gut<sup>2,3,5</sup>.

## **DISEASES OF THE STOMACH**

### **PEPTIC ULCER**

It occurs due to the presence of increased acid and pepsin secretion.

#### **Types of Peptic Ulcers**

1. Gastric - Ulcer > 3 cm is called – giant gastric ulcer
2. Duodenal - > 2cm is called - giant duodenal ulcer
3. Gastrojejunostomy Stoma
4. Lower end of esophagus
5. Diverticula's with ectopic gastric mucosa.

#### **Acute Peptic Ulcers**

##### **Etiology**

These are due to disruption of the gastric mucosal barrier, they appear as multiple erosions, classically they present with hemorrhage.



## **Pathology**

These are frequently multiple. In  $\frac{3}{4}$  of the patients three or more lesions are present. In the stomach they occur anywhere, but usually confined to the first part in the duodenum. The ulcers are oval or circular ranging from 1 mm to 1 cm. They are usually shallow punched out and seldom invade the muscular coats. When healing occurs they are unlikely to leave scars.

## **Clinical Features**

1. Pain
2. Transient Dyspepsia
3. Haematemesis & melaena
4. Perforation <sup>2,3,5</sup>.

## **Zollinger – Ellison Syndrome**

It is associated with increased gastrin secretion and peptic ulceration; patients have high serum gastrin levels.

It is suspected in the following circumstances:

1. Recurrent peptic ulcers
2. Duodenal ulcers with massive hyper secretion of acid
3. Duodenal ulcer and diarrhoea
4. Post bulbar or jejunal ulcerations
5. Duodenal ulcer in patients under 20 yrs of age<sup>2,3,5</sup>.

## **CHRONIC GASTRIC ULCER**

### **Aetiology**

Chronic gastric ulcer is associated with either normal acidity or hyper secretion and atrophic gastritis; one constant factor is smoking.

### **Incidence**

Sex ratio is equal.

### **Pathology**

A chronic gastric ulcer is usually larger than the duodenal ulcer; the floor of the ulcer is situated in the muscular coats.

As with duodenal ulcer, gastric ulcers tend to occur in the non acid secreting mucosa of the boundary with the body of the stomach.

### **Giant gastric ulcer**

Ulcer of more than 3 cm size is called giant gastric ulcer. The chance for bleeding is high.

### **Clinical features of chronic gastric ulcer and chronic duodenal ulcer**

#### **CHRONIC GASTRIC ULCER**

##### **(1) Periodicity**

Attacks for several weeks followed by intervals of freedom.

##### **(2) Pain**

In the epigastrium upto 2 hrs after food, practically never occurs at night.

##### **(3) Vomiting.**

It relieves pain may be self induced.

##### **(4) Haematemesis and Malena.**

(5) **Appetite** usually good, but afraid to eat.

(6) **Diet** avoids spicy food.

(7) **Weight** loss of weight.

## **CHRONIC DUODENAL ULCER**

### **(1) Periodicity**

Usually well marked, classically the attack comes in the spring and autumn precipitated by work, worry and whether.

(2) **Pain** it is severe; occurs 1- 2 and half hours after food; and is relieved by taking food often awakens the patient round about 2 am.

(3) **Vomiting** it is rare unless stenosis has occurred.

(4) **Haematemesis and Malena.**

(5) **Appetite** is good.

(6) **Diet** patients mostly eat any kind of food but some patients avoid fried foods.

(7) **Weight** usually there is no weight loss<sup>2, 3, 5</sup>.

## **JOHNSON'S CLASSIFICATION OF PEPTIC ULCER**

### **TYPE I**

Gastric ulcer.

60-70%.

Location-lesser curvature at or proximal to incisura, near the junction of oxyntic and antral mucosa.

Associated with diffuse antral gastritis or multifocal atrophic gastritis.

### **TYPE II**

Gastric ulcer associated with active or chronic duodenal ulcer.

Location same site as type I.

### **TYPE III**

Pyloric channel ulcer.

Location within 2 cm of pylorus.

#### **TYPE IV**

Gastric ulcer.

Located in proximal stomach or in the gastric cardia.

Type 2 and 3 gastric ulcers appear to behave more like duodenal ulcers and associated with excess acid.

Type 1 and 4 are not associated with excess acid and NSAID induced ulcers. They are typically multiple and located anywhere in stomach where protective mucosal barrier was damaged.

#### **PROGNOSTIC FINDINGS AT ENDOSCOPY FOR PEPTIC ULCER**

Actual appearance of ulcer at endoscopy is the most important predictor of bleeding.

#### **APPEARANCES OF ULCERS**

A clean ulcer base.

A flat ulcer.

Pigmented spot (purple/brown/black).

Adherent clot.

A visible vessel which may be smooth surfaced or tubular protuberance on ulcer surface or active bleeding with sprouting blood, continuous oozing or oozing around adherent clot fig-12.

Later four appearances are considered stigmata of haemorrhage.

### **PROBABILITY OF REBLEEDING**

Clean ulcer base – rarely bleeds.

Flat pigmented spot – 10%.

Adherent non bleeding clot – 20%.

Visible vessel – 40-80%.

Ulcer size > 1 cm.

### **MALLORY WEISS SYNDROME**

This condition has specific features. The patient is usually a male over 50 years of age has prolonged vomiting bout after taking alcohol, suddenly starts to vomit blood profusely and continuously.

As a result of which longitudinal tear of the mucosa just below the cardia occurs.

Clinical features

1. Faintness
2. Sweating
3. Pallor
4. Haematemesis
5. Malena<sup>2,3,5</sup>.

## **HIATUS HERNIA**

**Types**

1. Sliding - 85%.
2. Para oesophageal or rolling- 5%.
3. Mixed – 10%.



## **Sliding hiatus hernia**

Mechanism of herniation:

The cardiac orifice and a portion of the stomach immediately adjacent pass into the posterior mediastinum carrying with them a small peritoneal sac applied to the left side of the stomach.

Endoscopy reveals varying degrees of inflammation of the lower end of the oesophagus. Most valid sign is reflux of gastric juice through cardia, best seen during oesophagoscopy under surface anaesthesia. Furthermore the cardia will open on inspiration, whereas it normally closes and descends fig-14.

## **Para esophageal (rolling) hernia**

This is a true hernia into which the greater curvature of the stomach or very rarely, the whole stomach itself, ascends into a preformed sac lying in the mediastinum.

### **Mixed type**

It is a combination of the above mentioned hernias<sup>2, 3, 5</sup>.

### **MUCOSAL EROSIONS**

Erosion is break in the mucosa that does not penetrate the muscularis mucosa and submucosa. Often occur in stomach, may involve lower oesophagus and duodenum.

At endoscopy the lesions are generally multiple with white bases, commonly encircled by a halo of erythema. When erosions have recently bled their bases may be black fig-15, 16.

Haemorrhage refers to the appearance of discrete petichiae or bright red streaks and patches not associated with breaks in mucosa. Also called submucosal or subepithelial haemorrhage.

Major cause of erosions in stomach is due to disturbances of gastric mucosal barrier. NSAIDs and alcohols are common causes.

Pathogenesis related to a combination of both gastric acid and activated pepsin injuring gastric mucosa, exacerbated by mucosal ischemia secondary to hypo perfusion<sup>2,3,5</sup>.

## **GASTRO OESOPHAGEAL REFLUX DISEASE**

It is a common disease that accounts for 75% of the oesophageal pathology.

### **ETIOLOGY AND PATHOGENESIS**

1. Mechanically defective lower oesophageal sphincter.
2. Inefficient oesophageal clearance of refluxed gastric juice.
3. Abnormalities of the gastric reservoir that augment physiologic reflux such as
  - a. Increased gastric pressure.
  - b. Excessive gastric dilatation.
  - c. Increased gastric emptying.
  - d. Increased gastric acid secretion.

## **COMPLICATIONS**

1. Chronic blood loss.
2. Deep ulceration with periesophagitis.
3. Formation of strictures and webs.
4. Columnar cell change (Barrett's oesophagus)<sup>2,3,5</sup>.

## **ESOPHAGOGASTRIC VARICES**

It is one of the major manifestations of portal hypertension.

The sub mucosa of the esophagus is richly supplied with veins situated both above and below the muscularis intera, fine anastomosis between the caval and portal circulation exists at the lower end of the esophagus, as the veins engorge, the vessels in the sub mucosal plexus increase in size and become dilated, extend in the fundal and subfundal region of the stomach also become varicose, gastric varices occur predominantly in the cardiac end of the stomach.

### **Causes of bleeding.**

1. Increased pressure within the varices.
2. Ulceration secondary to oesophagitis<sup>2,3,5</sup>.

### **UPPER GI BLEEDING**

Upper gastro intestinal bleeding is defined as bleeding from a source proximal to the ligament of Treitz.

Haematemesis and melaena are most frequent clinical findings. However, massive bleeding from an upper source may also cause haematochezia.

## CAUSES OF UPPER GI BLEEDING

Condition	Percentage
ULCERS	60%
Oesophageal	6%
Duodenal	21%
EROSION	26%
Oesophageal	13%
Gastric	9%
Duodenal	4%
OESOPHAGEAL VARICES	4%
MALLORY WEISS TEAR	4%
TUMOURS	0.5%
VASCULAR LESIONS	0.5%
OTHERS	5%

Upper GI endoscopy is the single most important investigation. The dramatic improvement in diagnosis management will be achieved only by flexible endoscopy. The diagnostic accuracy of this is great if performed within 12 hours of bleeding. Thus examination should be accomplished as soon as possible after initial resuscitation and stabilization of the patient. 95% of diagnosis will be arrived by single endoscopic examination<sup>2,3,5</sup>.

## **TUMOURS OF THE STOMACH**

1. Benign lesions
2. Malignant lesions

## **BENIGN LESIONS**

### **Classification of benign tumours of the stomach**

#### **Polyps**

Hyperplastic polyp (type 1 & 2 in Japanese literature)

Neoplastic or adenomatous polyps (type 3& 4 in Japanese literature)

Mixed polyps (hyperplastic and neoplastic)

Fundic gland polyp.

Familial polyposis and other polyposis syndromes.

Peutz-jeghers (hamartomatous) polyp.

Inflammatory fibroid polyp.

Retention (juvenile polyp).

### **BENIGN HYPERPLASTIC GASTROPATHY**

1. Menetrier's disease

2. Pseudo lymphoma.

### **Intramural tumours**

1. Leiomyoma

2. other mesenchymal tumours

3. Heterotopic pancreas

4. Brunner's gland adenoma

### **Inflammatory conditions**

1. Eosinophilic gastritis

2. Granulomatous lesion (sarcoid, crohn's disease)



3. Syphilis

4. Tuberculosis

### **Cysts**

1. Intramucosal cyst (mucocele)

2. Sub mucosal cyst (gastritis cystica profunda)

3. Duplication cyst.

### **Miscellaneous conditions**

1. Gastric varices

2. Aneurysms of gastric vessels (Dieulafoy's disease)

3. Antral vascular ectasia (watermelon stomach)<sup>2,3,5</sup>.

## **CARCINOMA OF STOMACH**

### **LAUREN'S HISTOLOGICAL CLASSIFICATION OF CA STOMACH**

1. Diffuse gastric cancer
2. Intestinal gastric cancer
3. Others with mixed morphology.

### **EARLY GASTRIC CARCINOMA**

This is defined as cancer limited to mucosa and sub mucosa with or without lymph node involvement

.

Early gastric cancer has been classified by Japanese into three types

- (a) Protruding
- (b) Superficial – elevated, flat and depressed.
- (c) Excavated

### **ADVANCED GASTRIC CARCINOMA**

This is defined as a tumour which has involved the muscularis propria of the stomach.

**BORRMANN'S CLASSIFICATION OF ADVANCED  
GASTRIC CANCER (MACROSCOPIC APPEARANCE)**

Type 1 - Proliferate.

Type 2 - Ulcerative

Type 3 - Excavated

Type 4 -Diffuse infiltrative

Type 5 –Unclassifiable

**Malignant epithelial tumours**

(1)Adenosquamous fig-18.

(2)Squamous

(3)Anaplastic tumours

**Malignant non epithelial tumours**

(1)Leiomyosarcoma

(2)Fibro sarcoma

(3)Angiosarcoma

## **Other tumours**

### **Lymphoma**

- (1) Primary gastric lymphoma.
- (2) Secondaries due to systemic lymphoma<sup>2,3</sup>.

### **Endoscopy:**

This has revolutionized the diagnosis of gastric cancer. In early stage when the patients only complain of dyspepsia, gastroscopy is justified particularly if the patient is above 40 years of age. This will diagnose early case of gastric cancer. Whenever a lesion is detected by gastroscopy biopsy should be taken. Endoscopic appearance of gastric cancer ranges from small plaque like lesion to polypoid growth or small ulcers. Differentiating feature of benign from malignant ulcer is that the mucosal folds converge radially towards the crater in benign ulcer, and disruption of folds and blurring of the ulcer edge will be evident in malignant ulcer. At least 6 biopsies should be taken from the edge of the ulcer from all the four quadrants avoiding the necrotic areas of the ulcer bed. The sensitivity of biopsy alone varies from 75-85% in most series fig-17.

**Exfoliative cytology:**

Examination of cells detected by washing the stomach may diagnose a case of carcinoma. 75% accuracy has been claimed in this technique.

Chymotrypsin lavage may soften the mucous lining and may extrude more carcinomatous cells in the lavage for detection. There are four ways for obtaining cellular material is available now.

They are

1. Lavage techniques.
2. Abrasive techniques
3. Fluorescence techniques
4. Fibrogastrosopic techniques<sup>8,9,11</sup>.

**CARCINOID TUMOUR OF THE STOMACH**

This is a very difficult disease to diagnose early, not only because of the diversity of its presentation but also because of the time lag between the commencement of symptoms.

### **Clinical features**

1. Gastric distention- inability to take a normal meal& vomiting.
2. Anorexia leading to loss of weight.
3. Anemia, tiredness, weakness & pallor.
4. Persistent pain; no response to treatment- no periodicity.

### **Clinical groups**

- a. dyspepsia
- b. insidious onset
- c. obstructive types
- d. lump
- e. Silent <sup>2,3,5</sup>.

## **Materials and methods**

This is a prospective study of 150 patients from 2006 to 2008 done in the department of General Surgery Coimbatore medical college and hospital Coimbatore.

### **Inclusion Criteria**

The patients to be studied are above 15 years of age with the complaints of haematemesis, nausea, vomiting, regurgitation, heart burns ,dysphagia, flatulence, abdominal pain, loss of appetite, weight loss and recent changes in bowel habits have been taken up for the study. History of alcohol ingestion was enquired.

### **Exclusion criteria**

Children and pregnant women.

### **Method of collection of data**

The patients presented with the above complaints will be evaluated by detailed clinical examination followed by endoscopy after getting informed written consent from the patient.

Complete haemogram, chest x ray and ultrasound abdomen were done before embarking on endoscopy.

### **Methodology and techniques**

1. Endoscopy is performed with the video endoscopic instruments.
2. The patients were kept on empty stomach from previous day 10pm onwards.
3. Ryle's tube aspiration is done in the night and in the morning half an hour before the procedure.
4. 0.012 mg /kg body weight of atropine, 1cc will be given to reduce the secretions. Patient's throat swabbed with 4% xylocaine jelly or 10% xylocaine spray for anesthesia. Injection Diazepam will be given to alley apprehension of the patients.
5. Patient is put on left lateral position and the endoscopy is done under vision past the epiglottis into the oesophagus. The patient was then encouraged to swallow the tube along with the manual guidance and the mucosa was constantly visualized. The instrument should not be passed blindly or undue force should



not be used to pass the instrument. Whenever there is “Red out” pull out the instrument and reintroduce the instrument.

### **A.EXAMINATION OF THE OESOPHAGUS**

The mucosa is fully examined and the level of the diaphragm is observed as a slight indentation. The oesophagogastric junction is easy to identify as a change from the slight opaque grey squamous esophageal mucosa to the red glistening gastric folds.

### **B. EXAMINATION OF THE STOMACH**

Air is insufflated into the stomach when the endoscope is at the level of the oesophagogastric junction. The tip is maneuvered slightly downward and to the left initially to obtain a view of the stomach and then upward until the pyloric ring comes into view. The stomach is usually examined completely or withdrawal of the instrument, with particular attention paid to the area just below the angulus on the lesser curve, which is relatively difficult to see but is a common site

for ulceration as mentioned previously, complete examination of the fundus of the stomach requires inversion of the endoscope.

### **C.EXAMINATION OF THE DUODENUM**

The endoscopist must be convinced that the entire mucosa of the duodenal cap has been visualized. This may be difficult, particularly for the area just distal to the pyloric ring. The mucosa of the cap has a velvety appearance and close examination often reveals small polyps which are usually areas of heterotopic gastric mucosa, Brunner glands or pancreatic rests. This diagnosis can be confirmed by biopsy.

The examination is not complete without attempting to enter the second part of the duodenum which is easily recognized because of the circular muscle folds. Occasionally it may be difficult to exit from the cap. The superior duodenal fold is identified, and the tip is advanced over it, slightly to the right and then strongly downward. At this point withdrawal of the endoscope often results in advancement as the loop in the stomach is straightened<sup>8</sup>,

## **CLEANING AND DISINFECTION**

Instrument is cleaned with soap and water.

Biopsy channels are brushed. Endoscope is left in cleaning solution for 20 min.

2% glutaraldehyde is the most widely used disinfectant<sup>8,9</sup>.

## **INDICATIONS-DIAGNOSTIC**

1. Dyspepsia
2. Heart burns
3. Dysphagia
4. Vomiting
5. Gastrointestinal bleeding.

## **CONTRAINDICATIONS**

1. Anterior osteophytic proliferation of the cervical spine
2. Zenker's diverticulum
3. Serious systemic disease
4. Recent myocardial infarction
5. Postoperative status.

## **COMPLICATIONS**

1. Perforation
2. Bleeding
3. Cardiopulmonary accidents
4. Reactions to medication
5. Infection<sup>8,9,11</sup>.

## RESULTS

### SURVEY AND ANALYSIS OF ESOPHAGEAL DISEASES IN 150 CONSECUTIVE PATIENTS

S.No.	Disease	Male	Female	Total	Percentage
1.	Reflux Esophagitis	7	2	9	6%
2.	Hiatus Hernia	1	0	1	0.6%
3.	Esophageal Varices	9	2	11	7.3%
4.	Esophageal Moniliasis	1	0	1	0.6%
5.	Esophageal Stricture	3	0	3	2%
6.	OG Junction Growth	0	1	1	0.6%
7.	Esophageal Growth				
	Upper 1/3	0	0	0	13.3%
	Middle 1/3	2	1	3	
	Lower 1/3	9	8	17	
		32	14	46	30.6%

**SURVEY AND ANALYSIS OF GASTRIC DISEASES IN 150  
CONSECUTIVE PATIENTS**

S.No.	Disease	Male	Female	Total	Percentage
1.	Gastritis	14	6	20	13.3%
2.	Gastric Erosion	5	2	7	4.6 %
3.	Gastric Ulcer	7	2	9	6 %
4.	Pre-pyloric ulcer	2	0	2	1.3%
5.	Pyloric ulcer with GOO	3	1	4	2.6%
6.	Antral Growth with GOO	9	5	14	9.3%
7.	FB Stomach	0	1	1	0.6%
8.	Posterior wall bulge	2	0	2	1.3%
6.	Gastric Growth				
	Fundus + cardia	1	1		
	Body	2	3	18	12%
	Pyloric Antrum	8	3		
		53	24	77	51.3%

**SURVEY AND ANALYSIS OF DUODENAL DISEASES IN  
150 CONSECUTIVE PATIENTS**

S.No.	Disease	Male	Female	Total	Percentage
1.	Duodenal Ulcer	1	0	1	0.6%
2.	Duodenal ulcer with obstruction	1	0	1	0.6 %
3.	Periampullary Growth	1	0	1	0.6%
		3	0	3	2%

**SURVEY AND ANALYSIS OF ESOPHAGO - GASTRIC  
DISEASES IN 150 CONSECUTIVE PATIENTS**

S.No.	Disease	Male	Female	Total	Percentage
1.	Gastric Erosion + Esophagitis	1	0	1	0.6 %
2.	Oesophageal varices + Gastritis	1	0	1	0.6 %
3.	Oesophageal varices+congestive gastropathy	1	0	1	0.6%
		3	0	3	2%



**OVER ALL ANALYSIS OF UPPER GI DISEASE – IN 150  
CONSECUTIVE PATIENTS**

S.No.	Disease	Male	Female	Total	Percentage
1.	Oesophagus	32	14	46	30.6%
2.	Stomach	53	24	77	51.3%
3.	Oesophagus +Stomach	3	0	3	2%
4.	Oesophagus+ duodenum	1	0	1	0.6%
5.	Duodenum	3	0	3	2%
6.	OG Junction	0	1	1	0.6%
7.	Normal	15	4	19	12.6%
		107	43	150	100%

**INCIDENCE OF GASTRIC LESIONS IN MALE/FEMALE:  
BENIGN / MALIGNANCY**

BENIGN		MALIGNANT	
MALE	FEMALE	MALE	FEMALE
22%	8%	13.3%	8%
Total 30%		Total 21.3%	

### AGE INCIDENCE OF GASTRIC MALIGNANCY

YEARS	MALE	FEMALE	PERCENTAGE
20-30 years	0	1	0.6%
31-40 years	3	1	2.6%
41-50 years	5	3	5.3%
51-60 years	7	2	6%
61-70 years	2	3	3.3%
71-80 years	3	2	3.3%
	20	12	21.3%

### SEX INCIDENCE OF GASTRIC MALIGNANCY

SEX	TOTAL	PERCENTAGE
Male	20	13.3%
Female	12	8%

## **DISCUSSION**

Out of the 150 cases done, in this series, 12.6% of the patients were found to be endoscopically normal.

In this study, 13.3% of males and 8% females had gastric malignancy.

In spite of normal GI endoscopy, these patients could still have lower abdominal problems, which need special investigations like ultrasound and CT Abdomen plain/contrast to exclude disease in them.

Most of the patients with gastric malignancy, presented with malena, vomiting and dyspepsia.

In this series, 51.3% of the patients presented with gastric diseases, 30% presented with oesophageal diseases.

The gastric malignancy was more in the age group between 50-60 years.

One patient, who was totally asymptomatic, was found to have growth in the stomach while doing endoscopy.

In this series, all the biopsies which were taken from gastric ulcers were negative for malignant cells.

Even though there is changing scenario of increasing incidence of gastric malignancy in the Fundus and cardia, in this series most of the patients -7.3% had antral growth.

During the survey most of the gastric malignancies were found to be in the advanced stage. So only palliative treatment was given to those patients.

So the need for early endoscopy in symptomatic patients is stressed to diagnose the lesions much earlier.

## **CONCLUSION**

Endoscopy is less invasive, less complicated, affordable and easily available and it should be performed in every medical center in the patients who are presenting with upper GI symptoms.

By doing so, the chances of detecting the lesion at an earlier stage will progressively increase, thereby improving the response to treatment and life span.

## **PROFORMA – I**

### **Study of upper GI endoscopy in gastric lesions**

NAME                      AGE                      SEX                      SERIAL NO.

OP/IP NO.              ADDRESS              OCCUPATION

DOA                      DOP                      DOD

#### **1) PRESENTING COMPLAINTS                      DURATION**

- a. Heart burns
- b. Regurgitation- postural, non postural
- c. Flatulence
- d. Dyspepsia- recent, chronic
- e. Pain abdomen- upper abdomen, anterior chest, back
- f. Dysphagia- intermittent, progressive
- g. Haematemesis- quality , quantity
- h. Malena-present or absent
- i. Jaundice
- j. Loss of weight- sudden, slow
- k. Loss of appétit

l. Abdominal lump

m. Nausea

n. Vomiting

## **2) PAST HISTORY**

1. DM/HT

2. H/O acid peptic disease

3. H/O intake of NSAIDS

4. Any H/O surgery

## **3) PERSONAL HISTORY**

Smoking- duration

Consumption of alcohol- duration

Diet- veg/non veg

Betel nut chewing

## **4) FAMILY HISTORY**

Any H/O Ca stomach in the family

## **5) SOCIAL STATUS**

## **6) MARITAL STATUS**



## **GENERAL EXAMINATION**

Built/ nourishment

Pallor / icterus/ pedal edema/generalized

lymphadenopathy/left supraclavicular node

PULSE RATE BLOOD PRESSURE

ABDOMEN

Tenderness

VGP/ VIP

Dilated veins /scars

Mass/ lump

Hepatomegaly

Ascitis /free fluid

## **PER RECTAL EXAMINATION**

## **CARDIOVASCULAR SYSTEM**

## **RESPIRATORY SYSTEM**

## **CENTRAL NERVOUS SYSTEM**

## **SPINE /CRANIUM**

## **INVESTIGATIONS**

1). Blood

Hb, Tc, Dc, LFT, Urea, Sugar and Sr. Creatinine.

2) Stool for occult blood

3) ECG, X-ray chest, X-ray abdomen

4) Barium meal

5) USG Abdomen

6) UGI scopy

7) Biopsy

8) CT scan

## **DIAGNOSIS**

## **MANAGEMENT AND FOLLOW UP**

## PROFORMA II      ENDOSCOPIC STUDY

NAME            AGE            SEX            GE NO.    DATE            TIME

SCOPE USED MODEL      REFERRED FROM: SURGICAL/MEDICAL

PROVISIONAL DIAGNOSIS      PREMEDICATION/ANESTHESIA

ENDOSCOPIC DIAGNOSIS

PROCEDURE                      ENDOSCOPIST

EMERGENCY                      ASSISTANT

ROUTINE                          STAFF

FOLLOW UP

THERAPUETIC                      POST OP

FINDINGS

OESOPHAGUS

OESOPHAGO GASTRIC JN

STOMACH

DUODENUM

ENDOSCOPIC PICTURES TAKEN: YES/ NO

SPECIMEN TAKEN: YES/ NO

BRUSH CYTOLOGY

FORCEPS BIOPSY

SITE OF BIOPSY

PATHOLOGY

STUDY

- i. COMPLETE / INCOMPLETE
- ii. NORMAL/ ABNORMAL
- iii. NEXT FOLLOW UP NEEDED / NOT NEEDED

FINAL DIAGNOSIS

SITE OF LESION

ADVICE - MEDICAL

SURGICAL

COMBINATION

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# FIBER OPTIC ENDOSCOPE



Fig - 1



Fig - 2

## MONITOR



Fig – 3

## BIOPSY FORCEPS & CYTOLOGY BRUSH



Fig - 4

# ANATOMY STOMACH

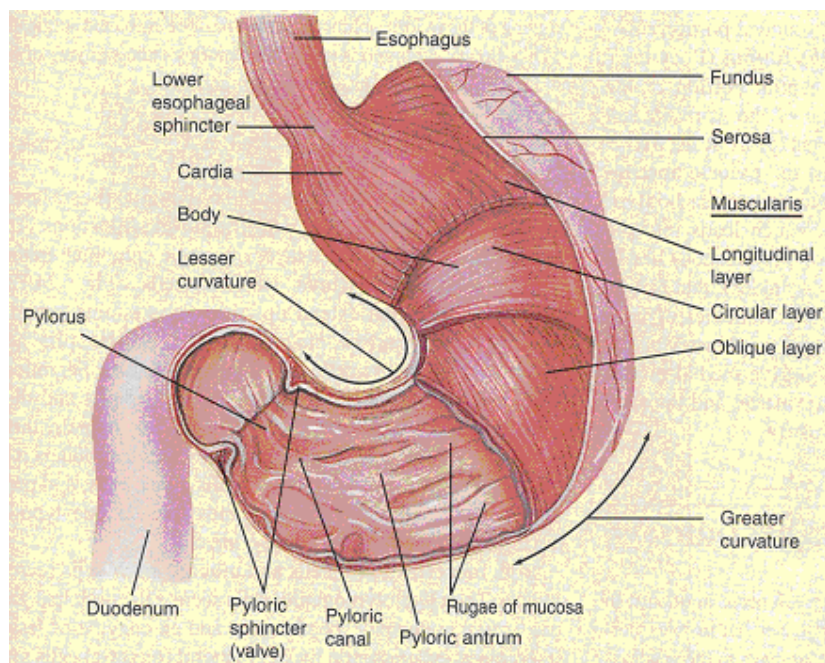


Fig -5

# ANATOMY STOMACH

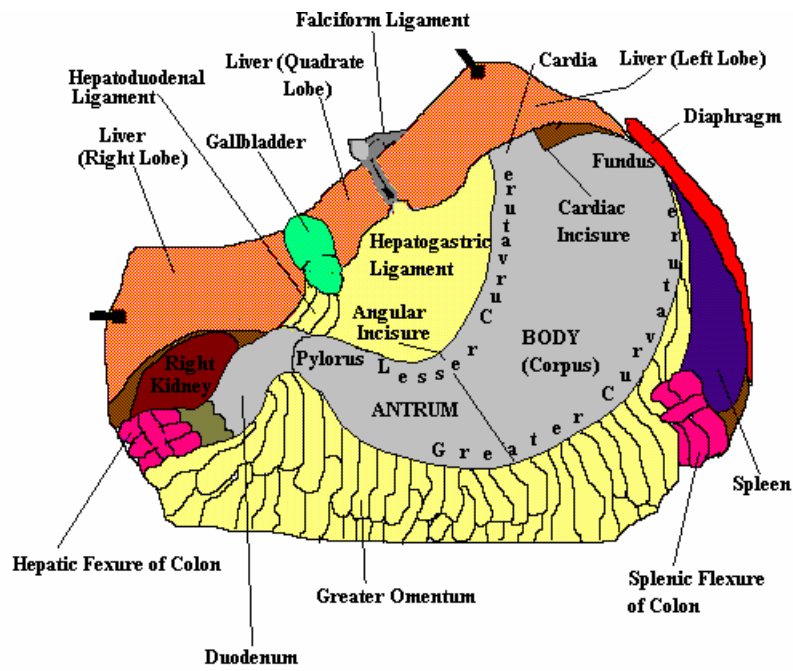


Fig - 6

## ARTERIAL SUPPLY- STOMACH

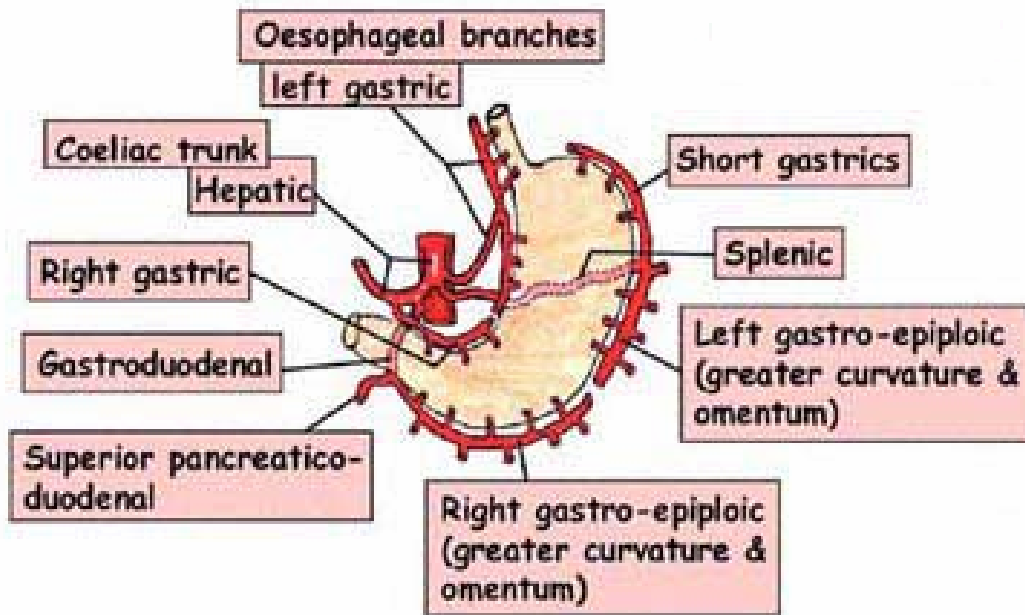


Fig - 7

## VENOUS DRAINAGE - STOMACH

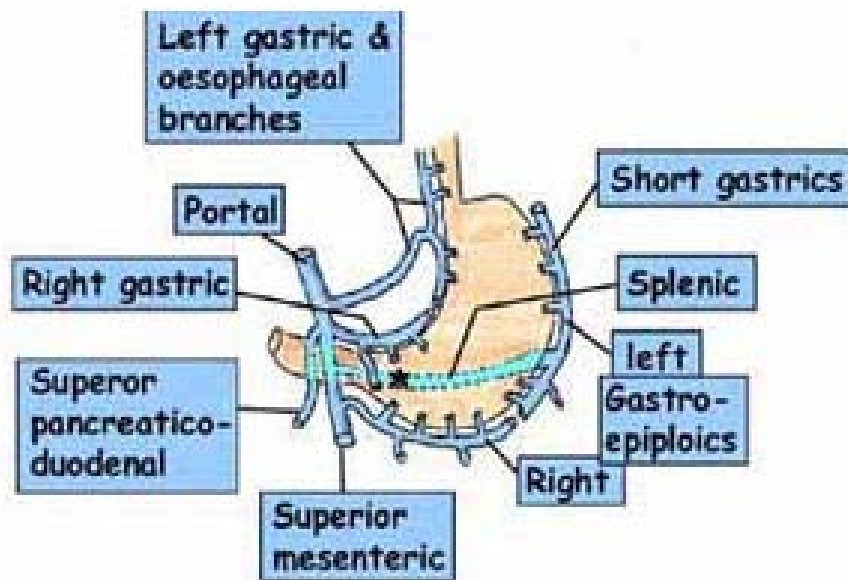


Fig- 8

## LYMPHATIC DRAINAGE -STOMACH

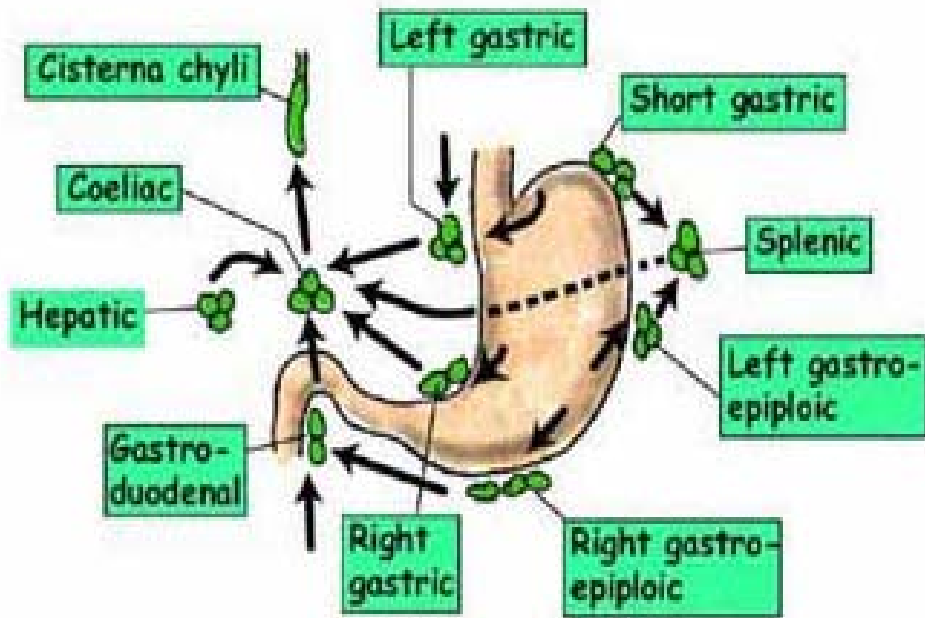


Fig- 9

## NERVE SUPPLY - STOMACH

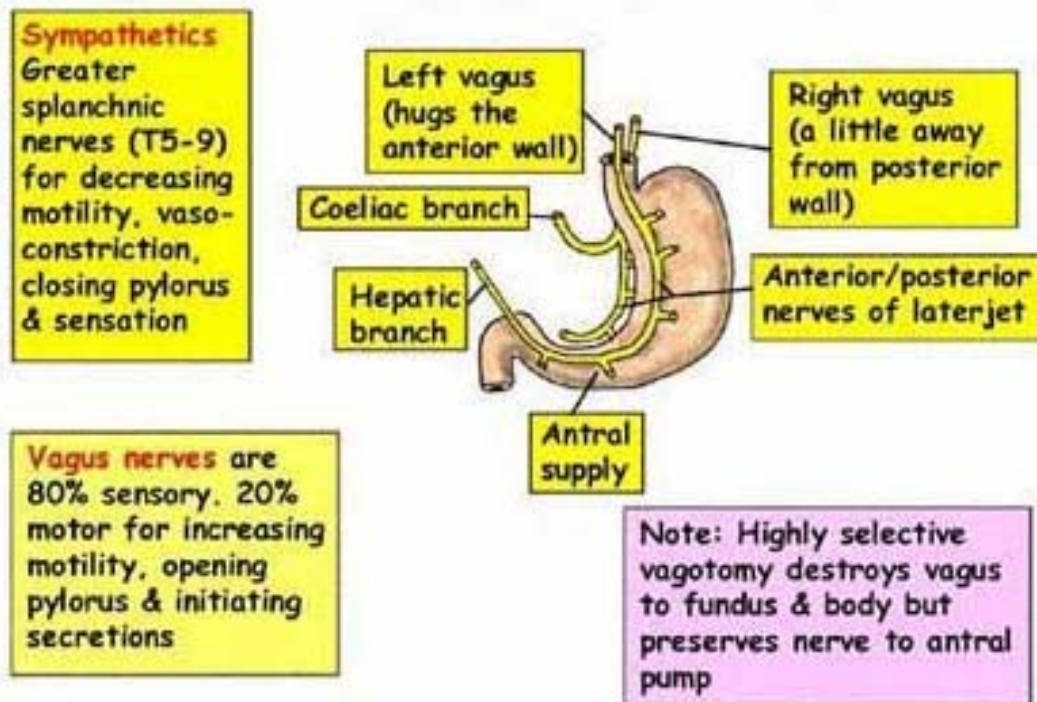


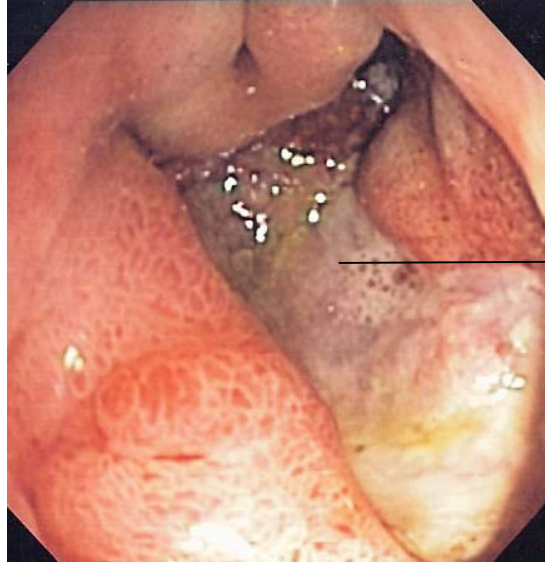
Fig – 10



## STOMACH NORMAL HISTOLOGY

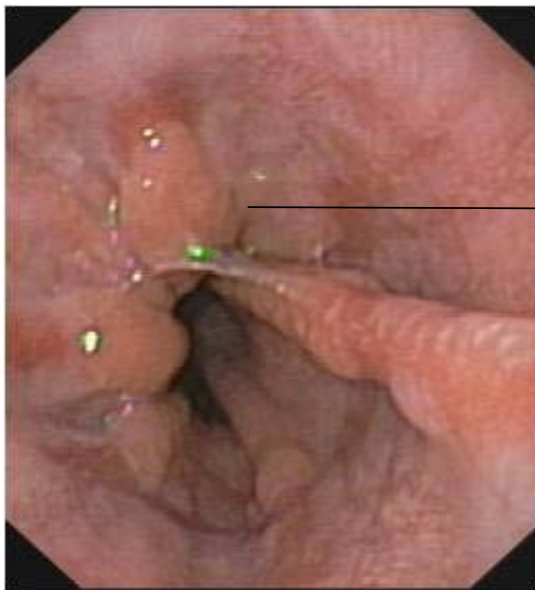


Fig - 11



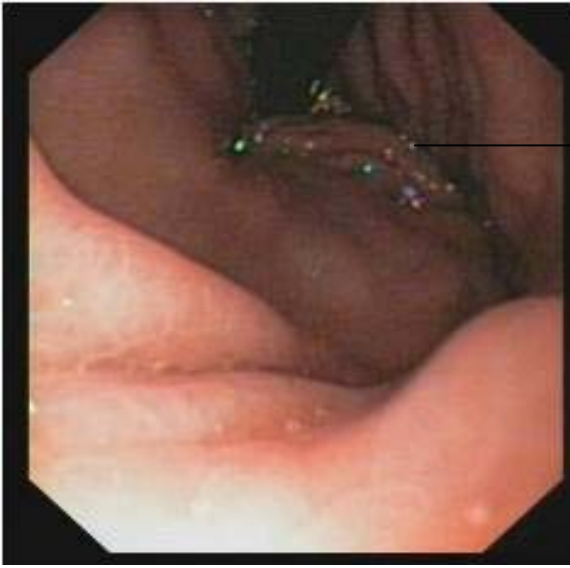
**DEEP GASTRIC  
ULCER**

Fig - 12



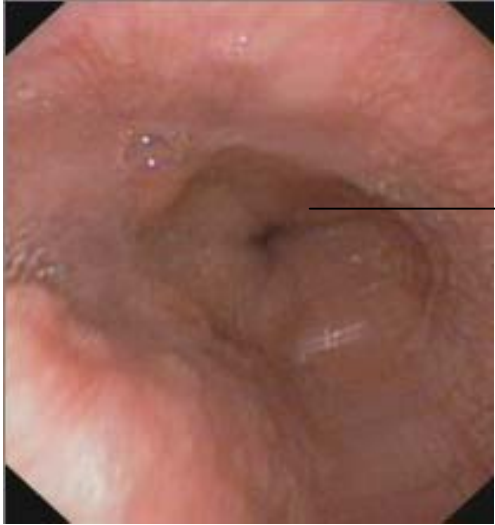
**GASTRIC OUTLET  
OBSTRUCTION**

Fig-13



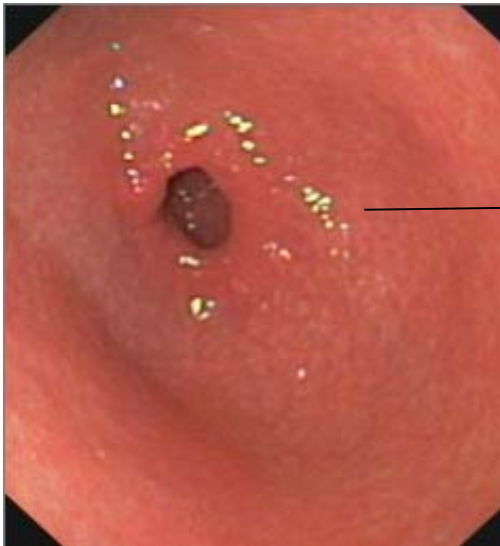
→ **HIATUS HERNIA**

Fig - 14



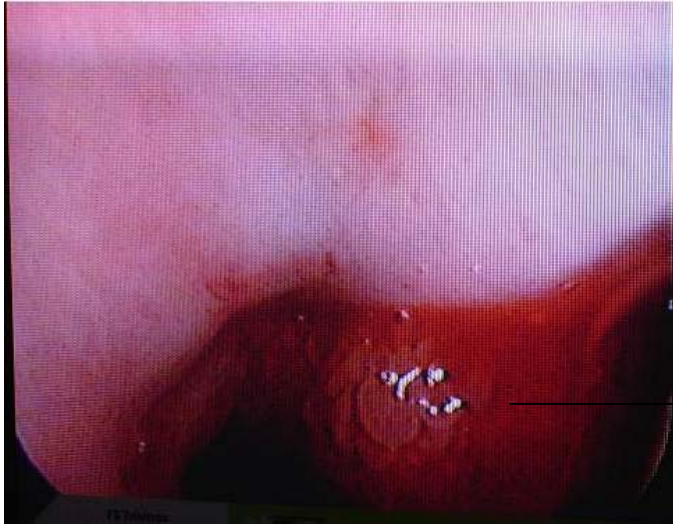
**ANTRAL  
GASTRITIS**

Fig -15



**EROSIVE  
GASTRITIS**

Fig - 16



**CARCINOMA  
STOMACH**

Fig - 17

**ADENOCARCINOMA STOMACH-  
MODERATELY DIFFERENTIATED**

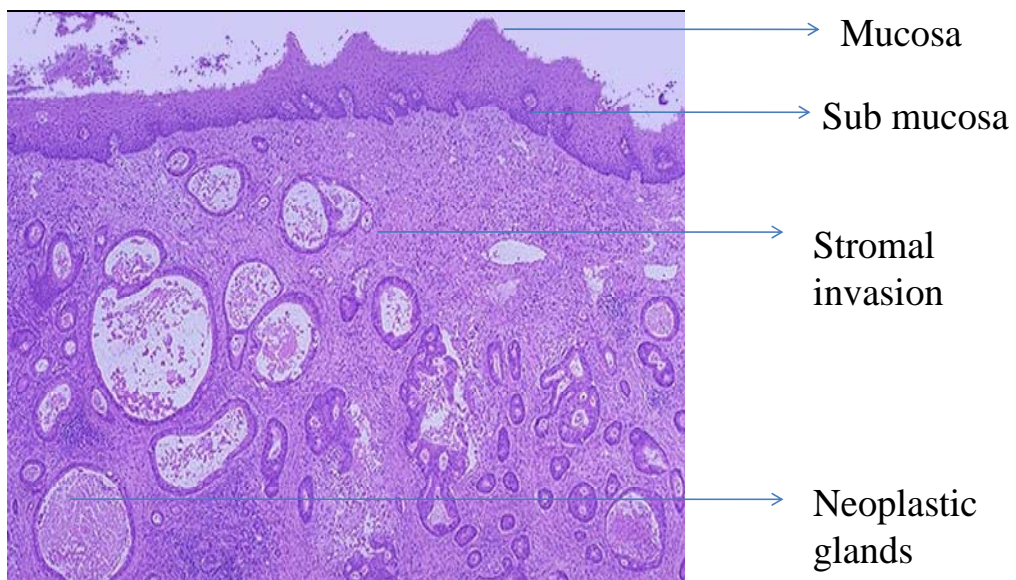
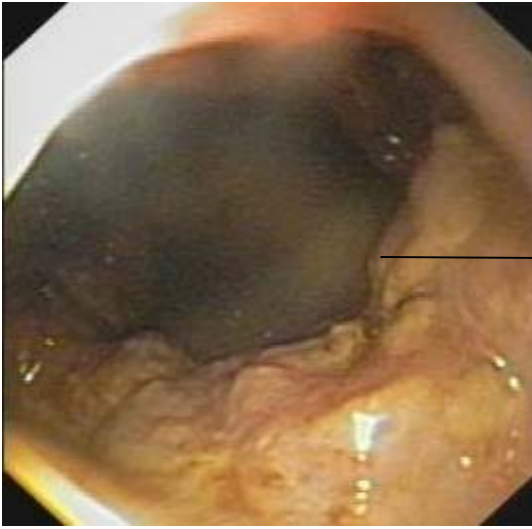


Fig – 18. Showing increased mitosis, nuclear cytoplasmic ratio and hyperchromatism.



→ **CARCINOMA  
OESOPHAGUS**

Fig - 19



→ **CARCINOMA  
OG JUNCTION**

Fig - 20