# COMPARATIVE STUDY BETWEEN LAPAROSCOPIC NISSEN FUNDOPLICATION AND LAPAROSCOPIC NISSEN ROSSETTI FUNDOPLICATION FOR TREATMENT OF GASTROESOPHAGEAL REFLUX DISEASE

## Thesis

*Submitted in Partial Fulfillment of the M.D. Degree In **General Surgery***

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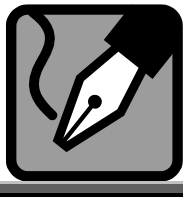
| | |
|---|---|
| **CD** | Crural Diaphragm |
| **EGF** | Epidermal growth factor |
| **EGS** | Esophagogastric segment |
| **GEJ** | Gastroesophageal junction |
| **GERD** | Gastroesophageal reflux disease |
| **HPZ** | High pressure zone |
| **LES** | Lower esophageal sphincter |
| **LESP** | Lower esophageal sphinter pressure |
| **PD** | Persistent dysphagia |
| **PEL** | Phrenoesophageal ligament |
| **SCJ** | Squamocolumnar junction |
| **TLESRs** | Transient lower esophageal sphincter relaxations |



# INTRODUCTION

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Gastroesophageal Reflux Disease [GERD] is a chronic disorder affecting a significant proportion of the population ***(Anvari, 1995)***.

Gastroesophageal reflux disease is a common condition in which the gastric content gains access to the esophageal lumen or respiratory tract. Symptoms such as heartburn, regurgitation, dysphagia and respiratory difficulty may occur. The lower esophageal sphincter is often mechanically incompetent in severe gastroesophageal reflux disease. Damage to the esophageal mucosa may results in severe esophagitis, ulceration, stricture or Barrett's esophagus ***(Hinder, 1994)***.

The relationship between Barrett's esophagus and gastroesophageal reflux disease is being established ***(Morson et al., 1992)***. In all studies dealing with adenocarcinoma of the esophagus the high prevalence of Barrett's metaplasia has been stressed ***(Blot et al., 1991)***.

Gastroesophageal reflux disease can be treated either medical or surgical. Medical therapy is the firstline of management for GERD. Esophagitis will heal in approximately 90% of cases with intensive medical therapy. However, the symptoms recur in more than 80% of cases within 1 year of drug withdrawal. In addition, while medical therapy may effectively treat the acid induced symptoms of GERD,

esophageal mucosal injury may continue due to ongoing alkaline reflux ***(Vaezi and Richter, 1995).***

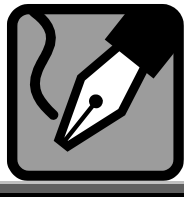
Surgery may be considered in patients with failure of medical therapy or whom develop complication of GERD. Failure of medical therapy is due to non compliance of the patients, inability to afford medications, relapse of symptoms soon after medication stopped or relapse of symptoms despit continuous use of medications ***(Hinder et al, 1997)***.

There are many operations for GERD. Nissen fundoplication is the most commonly performed antireflux procedure in the world, because it is one of the earliest antireflux operations described, is conceptually simple in design, and has passed the test of the time as being a reliable mean of preventing reflux ***(Peters et al, 1995)***.

Nissen fundoplication can be done either open or laparoscopic. The benefits of a laparoscopic approach include a shorter and more comfortable recovery with an earlier return to normal activities. Several reports document the feasibility, safety and favorable results of laparoscopic antireflux procedure ***(Hunter, 1996)***.

Nissen Rossetti fundoplication is a modified Nissen fundoplication in which short gastric vessels are not divided. The main benefit is to decrease the time of the operation ***(Peters et al., 1995)***.

Peters, DeMeestre and associates noted that incomplete fundic mobilization places lateral tension on the sphincter area and impair the sphincter relaxation with increase possibility of postoperative dysphagia ***(Peters et al, 1995)***.



# AIM OF THE WORK

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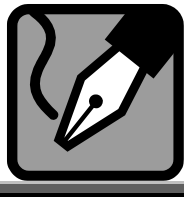
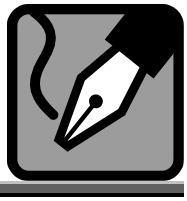
The aim of this work is to compare between laparoscopic Nissen fundoplication and laparoscopic Nissen Rossetti fundoplication as different surgical techniques for treatment of gastroesophageal reflux disease.



# REVIEW OF LITERATURE



# ANATOMY OF THE ESOPHAGUS

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## Configuration:

The esophagus is the narrowest tube of the intestinal tract. It ends by widening into the most voluminous part, the stomach. At rest, the esophagus is collapsed and forms a soft muscular tube that is flat in its upper and middle parts with a presenting diameter of 2.5 x 1.6 cm. the lower esophagus is rounded, and its diameter is 2.5 x2.4 cm. ***(Postlethwait, 1997).***

Compression by adjacent organs, vessels or muscles causes narrowing which can be visualized by means of fluoroscopy and endoscopy. The cricopharyngeal narrowing is identified at a site of 15 cm from the incisors. The aortic compression, which is left sided and anterolateral, is caused by crossing of the aortic arch and the left main bronchus at a location 22cm from the incisors. The third narrowing is not constant and is located at or about 44 cm from the incisors. It may be caused by the functional effect of lower esophageal sphincter rather than by mechanical imprint of the diaphragm ***(DeMeester and Levin, 1996).***

There are two functional constrictions: the upper and lower esophageal sphincters. They can be defined manometrically at the esophageal opening, at 14-16 cm from the incisors, and at the entrance into the stomach, at 40-45 cm from the incisors ***(Georg and Orringer, 1996).***

## Length of the esophagus:

The length of the esophagus is defined anatomically as the distance between the cricoid cartilage and gastric orifice. In adult, it ranges from 22 to 28 cm, of which 2 to 6 cm are located in the abdomen ***(Enterline, and Thompson, 1994)*.**

The identification and marking of the cricoid cartilage is rather difficult for practical reasons. Clinicians measure the distance between both ends of the esophagus by using the incisors as a direct macroscopic landmark during endoscopic procedures ***(Postlethwait, 1997).***

# COMPARTMENTS

## The bed of the esophagus:

Unlike the general structure of the digestive tube, the esophagus has no mesentery and no serosal coating. Its position within the mediastinum and a complete envelope of a loose connective tissue allows the esophagus extensive transversal and longitudinal mobility. Respiration may induce movement over a few millimeters, and swallows may result in displacement over as much as the height of one vertebral body ***(Dodd, 1998)***.

There are surgical implications to this observation. It is because it is surrounded by loose areolar connective tissue that the esophagus may be subjected to a blunt stripping from the mediastinum where there are no periesophageal contra-

indications to use this technique, such as fixation or invasion by malignant tumor ***(Orringer and Orringer, 1993).***

## Supporting and anchoring structures:

The esophagus, both proximally and distally, is stabilized by bony, cartilaginous or membranous structures. At the cranial end, the esophageal musculature is firmly inserted on the posterior margin of the cricoid cartilage with the help of the cricoesophageal tendon. There are minute membranes 170 um thick and 1.5 mm cranio-caudal extension (mean values) that anchor the esophageal wall to the trachea, pleura, prevertebral fascia and surrounding tissue of the posterior mediastinum ***(Enterline and Thompson, 1994)***.

The distal esophagus traverses the diaphragm through the esophageal hiatus, which is bounded by the two diaphragmatic crura, their insertion on the anterolateral surface of the first two or three lumbar vertebrae and the organization of their fibers may give a varying shape to the hiatus. This shape is influenced by respiration, swallowing and altered thoraco-abdominal pressure ***(Postlethwait, 1997).***

The Phreno-esophageal membrane, also known as Laimer`s ligament or Allison`s membrane, is an important anatomical structure. Macroscopically, the Phreno-esophageal membrane can be recognized by its well defined lower edge and its slightly yellow color, even in severe periesophagitis. The membrane is composed of equal proportion of elastic and collagenous fibrous element which guarantees sufficient

pliability. The Phreno-esophageal membrane splits into two sheets. One sheet extends upward 2-4 cm through the hiatus, where elastic and collagenous fibers traverse the esophageal musculature to insert on the submucosa. The other sheet passes across the cardia down to the level of the gastric funds to blend into gastric serosa, the gastro-hepatic ligament and the dorsal gastric mesentery ***(Eckhard, et al., 1998).***

This structural arrangement allows the terminal esophagus and the junction to move in relation to the diaphragm and to slip through the hiatus like in a tendon sheath. With advancing age, the elastic fibers are replaced by inelastic collagenous tissue and the adhesion of the Phreno-esophageal membrane to the lower esophagus become looser. This leads to a loss of pliability. Disruption of the anchoring structure of the cardia and proximal stomach in conjunction with a wide hiatus may result in protrusion of the gastro-esophageal junction and the cardia or even part of the stomach into the thoracic cavity ***(Postlethwait, 1997).***

## REGIONAL ANATOMY

The esophagus is a midline structure lying on the anterior surface of the spine. It descends through three compartments: the neck, the chest and the abdomen.

Three minor deviations are present along its trajectory. The first one is toward the left at the base of the neck. The second is observed at the level of the seventh thoracic vertebra, where the esophagus runs slightly to the right of the spine. The

third and most prominent angulation occurs just above the esophagogastric junction, where the esophagus shifts toward the left ***(Akiyama, 1998).***

## Cervical esophagus (Fig. 1):

Cervical esophagus starts as a continuation of the pharynx, at the lower margin of the cricoid cartilage opposite the six cervical vertebrae. It passes into the chest at the level of the sternal notch ***(Akiyama, 1998).***

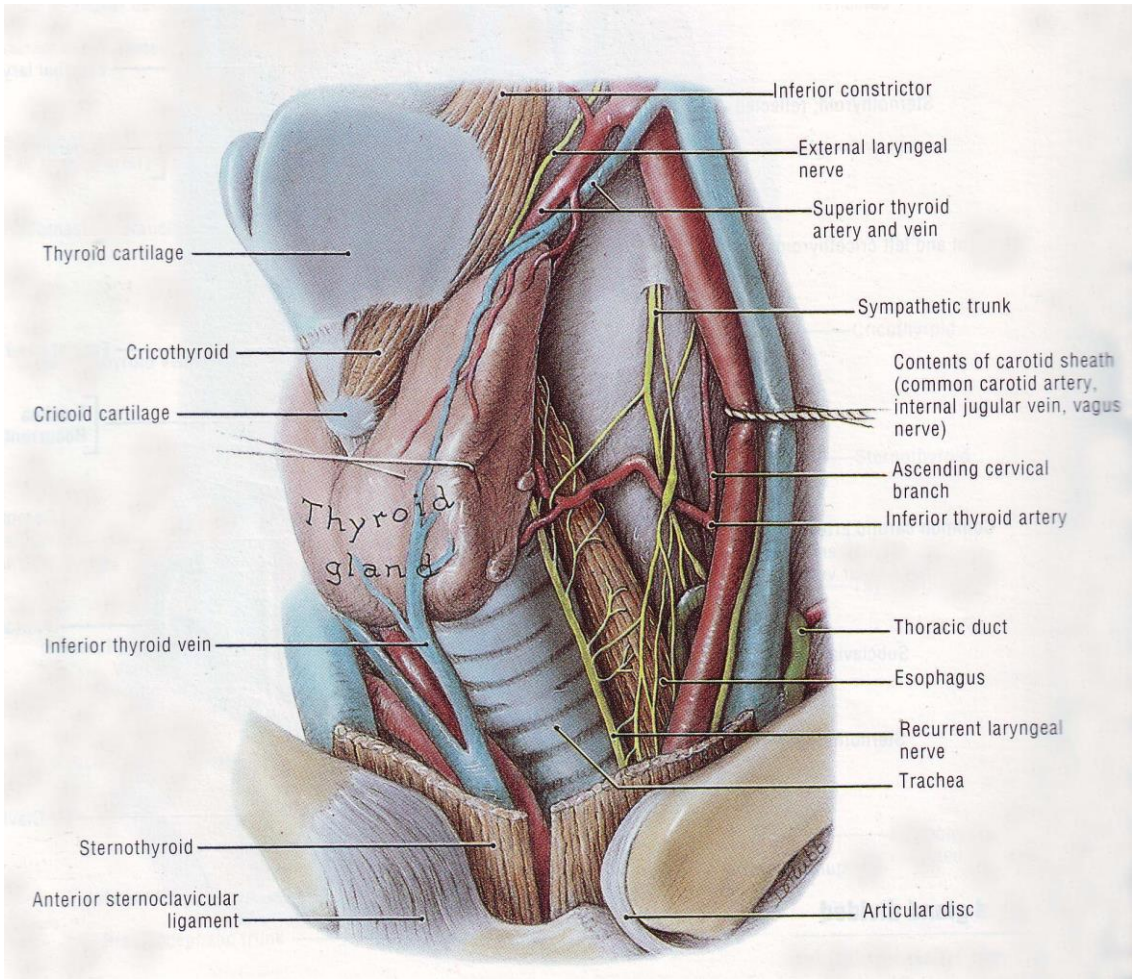



**Fig. (1):** Cervical esophegous and its relations ***(Anne and Ming, 1992)***

Trachea lies ventral to the cervical esophagus. The cervical vertebrae and both longus colli muscles are in close contact with its posterior wall. The posteromedial faces of both thyroid lobes cover both side with the paired common carotid arteries, the jugular veins and in the left lower neck the thoracic duct which ends at the confluence of the subclavian and jugular veins. In the respective grooves between the esophagus and the trachea the left and right recurrent laryngeal nerves courses cranially toward the larynx. The paired superior and inferior thyroid arteries are found 1 cm lateral to the esophagus, and their position can vary ***(Georg and Orringer, 1996).***

## Thoracic esophagus (Fig 2, 3):

In the upper part of the thoracic esophagus, between the thoracic inlet and tracheal bifurcation (at the level of fifth thoracic vertebra), the esophagus retains its relationship to the trachea ventrally and to the prevertebral fascia posteriorly. On he right, in close contact with the esophagus is the mediastinal pleura, the lung hilus, vessels originating from the aortic arch and the right vagus nerve. Lateral to the esophagus the azygos vein, which arches over the right main bronchus to end in the superior vena cava ***(Georg and Orringer, 1996).***

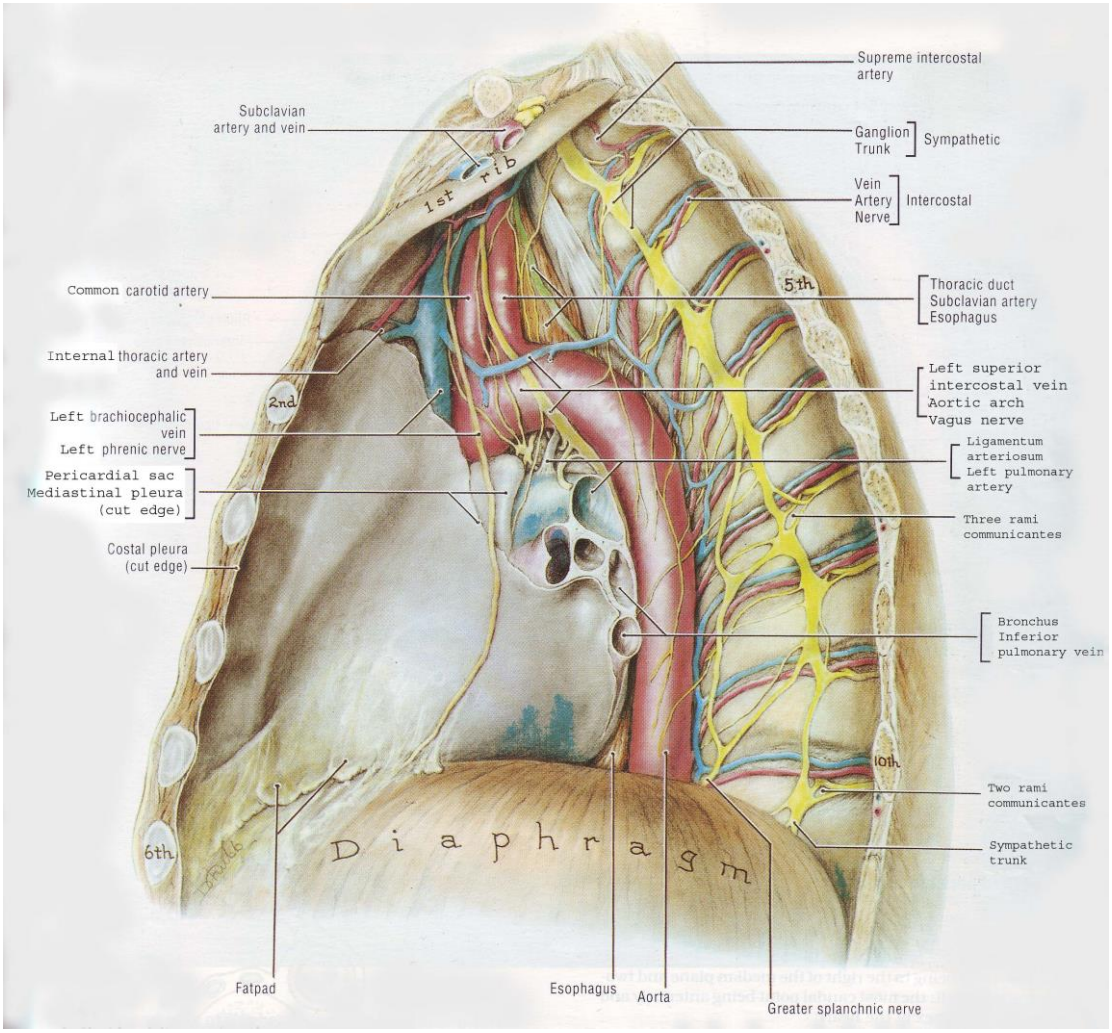



**Fig. (2):** Thoracic esophagous and its relations (left side)
***(Anne and Ming, 1992)***

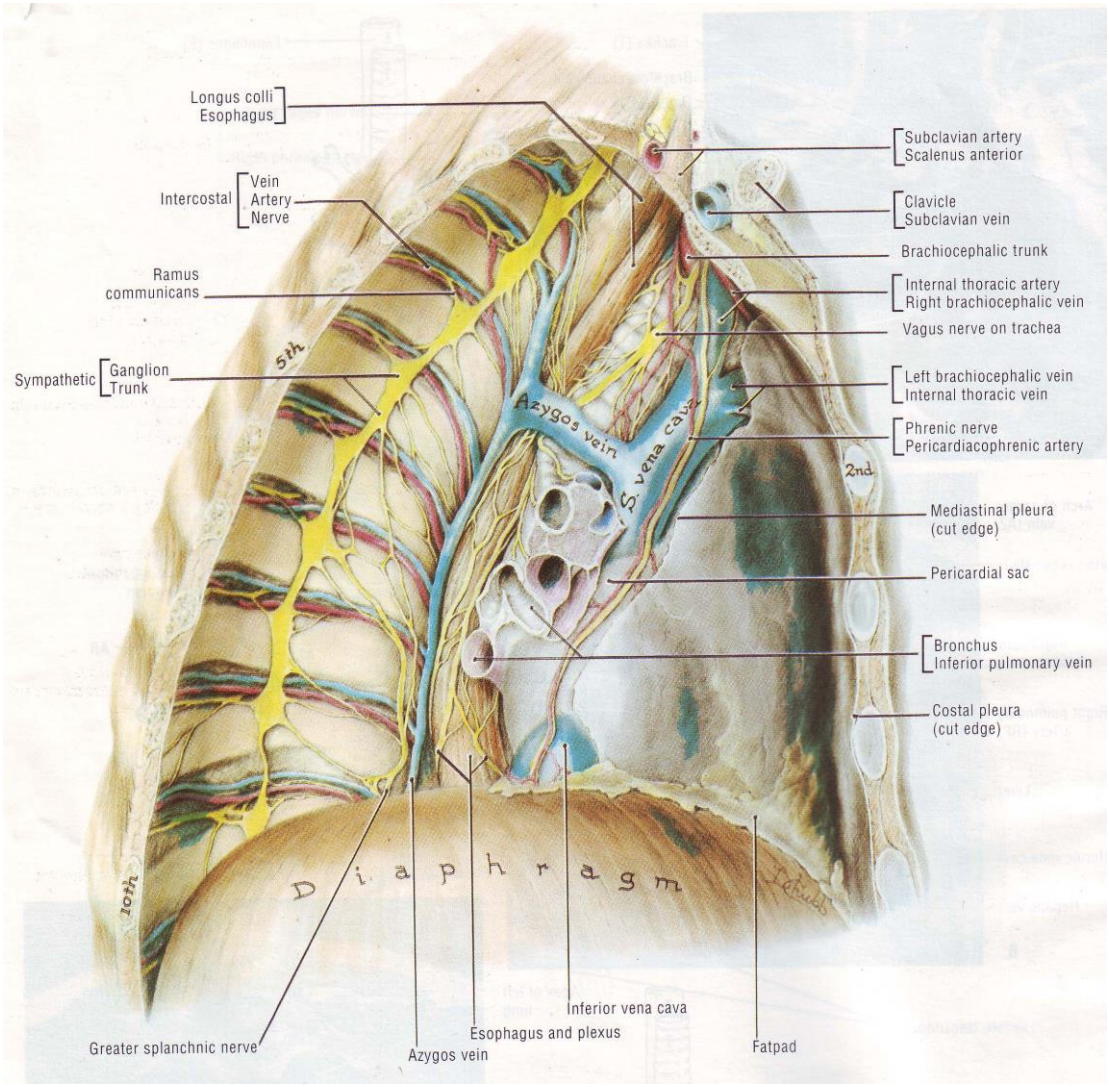



**Fig. (3):** Thoracic esophagous and its relations (right side)
***(Anne and Ming, 1992)***

Left of the esophagus lies the subclavian artery and posteriorly, the left pleura and thoracic duct. At a slightly lower level the aortic arc and the left main bronchus cross over the anterolateral side of the esophagus. The left recurrent laryngeal nerve is found in tracheobronchial groove of that side after it

emerges from under the aortic arch. Below the tracheal bifurcation, the pericardium together with the underlying left atrium and the left vagus trunk form the close anterolateral limits of the esophageal wall. Dorsally and toward the right, the azygos vein, the thoracic duct and the right vagus nerve lie parallel to the esophagus. The thoracic duct crosses from the right to the left just above the arch of the azygos at the T4-T5 level. The descending aorta and hemiazygos veins are dorsal and to the left. The pleura on the left side of the mediastinum may occasionally extend behind the esophagus. Both vagi accompany the esophagus while it passes through the hiatus at the level of the tenth thoracic vertebrae ***(Georg and Orringer, 1996).***

## Abdominal esophagus:

Part of the left lobe of the liver lies ventral to the esophagus. Both diaphragmatic crura are lateral and posterior. The inferior vena cava lies laterally to the right crus, whereas the aorta is found posterior to the left crus. The cranial pole of the spleen is close relationship to the terminal esophagus ***(Georg and Orringer, 1996).***

## TISSUE ORGANIZATION OF THE ESOPHAGUS

The overall tissue structure of the esophagus parallels the basic tissue organization of the digestive tract. Which comprises an external fibrous layer (tunica adventitia), a muscle layer (tunica muscularis), a submucosal layer (tela submucosa),

and internal mucous layer (tunica mucosa) ***(Georg and Orringer, 1996).***

## Tunica adventitia:

The tunica adventitia is composed of loose connective tissue. It envelops the esophagus, connect it with adjacent structures, and contain small vessels, lymphatic channels and nerve fibers ***(Georg and Orringer, 1996).***

## Tunica muscularis:

The tunica muscularis layer consists of two complete muscle coats that are separated by a thin connective tissue sheet. The outer layer is the longitudinal layer and the inner layer is the circular layer.

The longitudinal layer originates from the fascia at the dorsal plane of the cricoid cartilage. Some muscle fibers continue into the inferior laryngeal constrictor muscle. The long bundles of this muscular layer course straight down the esophagus to cross the gastric inlet, where some of the fibers change their arrangement ***(Liebermann-Meffert, et al.: 1994)***.

The circular layer begins at the level of the cricoid cartilage, possibly as an independent sheet. In their descent, the fibers of the circular muscle layer forms imperfect circles with overlapping ends. Approximately 3 cm above the junction with the stomach, the number of the muscle fibers increases causing a stepwise thickening in the muscle. Those on the lesser

curvature side retain their main orientation as short muscle clasps, whereas those on the greater curvature side change to become the oblique gastric sling fibers ***(Netter, 1991).***

## *Upper esophageal sphincter:*

Manometrically, this is a zone of elevated pressure 2 to 4 cm in length just at the entrance of the esophagus. This high pressure zone results from the effect of cricopharyngeus muscle, which loops around the hypo pharynx. It is inserted on both cricoid processes. Although it is not a true sphincter, this muscle behaves like one. During its sling like contraction, the muscle closes the esophageal opening by exerting its effects ventrally against the plane of the cricoid cartilage. This is the reason why the upper sphincter owns an asymmetrical pressure profile in manometric measurement ***(Winans, 1992).***

## *Lower esophageal sphincter:*

Manometrically, this is a zone of elevated pressure 3 to 5 cm in length, immediately above the junction of the esophagus with the stomach. Using small markers in a simultaneous radiomorphololgic study, it has been shown that this high pressure zone correlates with a thickened muscular structure at this site. The specific arrangement of the musculature accounts for the sphincteric asymmetry ***(Liebermann-Meffert, et al., 1995).***

Asymmetry of the high pressure zone at this position has been demonstrated manometrically. The manometric pressure

image of the lower esophageal high pressure zone obtained by the recently developed technique of the three dimensional computerized vector diagram matches the muscular asymmetry at the human cardia ***(Stein, et al.:1991)***

Some confusion remains about the exact distribution of the striated and smooth muscle in the esophagus. Liebermann-Meffert found that in the two most proximal centimeters of the esophageal musculature, the muscle fibers of both layers were exclusively striated. In the following 8 cm, the muscularis showed a structure composed of progressively more smooth muscle. The transition is neither abrupt nor restrictive to individual muscle bundles; both types of muscle are without any distinct anatomic separation. In all subjects, the transition zone between the two musculatures was seen at the same general level. Below the tracheal bifurcation, there were never any striated muscle cells present. When the muscularis is compared to the muscle of the muscularis mucosa, it is seen that the latter is composed uniquely of the smooth muscle fibers over the entire esophagus ***(Liebermann-Meffert, et al., 1995).***

## Tela submucosa:

The tela submucosa connects the muscular coat and the mucosa. It contains elastic and collagenous fibers, a meshwork of blood vessels, abundant lymph vessels, nerves and mucous glands. These deep esophageal glands are small branching glands of a mixed type, and their ducts pierce the muscularis mucosa ***(Georg and Orringer, 1996).***

## Tunica mucosa (Fig. 4):

This inner layer is made up of the muscularis mucosa, the tunica propria, and a stratified squamous epithelium. The muscularis mucosa, when contracted, creates the fold of the mucosa. These long folds run in the longitudinal axis of the esophagus, and also show small transversal rippled folds mostly in the distal esophagus. The fibrous tunica propria projects into the epithelium, thus forming the papillae. It contains lymph channels, occasional lymphocytes, and in the distal esophagus, superficial glands that resemble cardiac glands ***(Eckhardt, et al., 1998).***

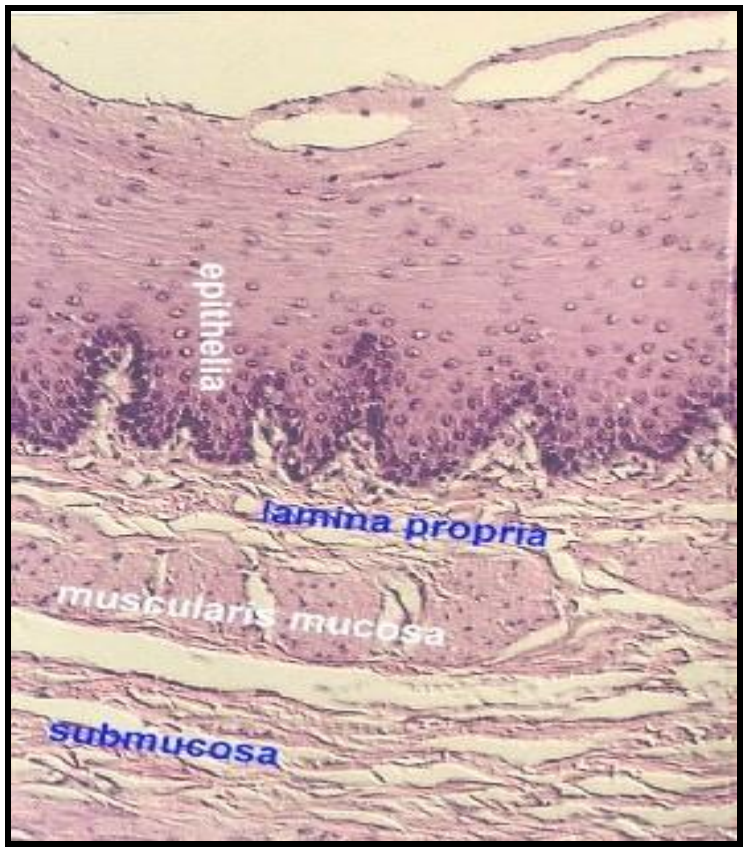



**Fig (4):** Tissue structure of the esophageal wall ***(Bremner, 1994)***

Clinically, the surface of the esophageal mucosa is reddish in color in its cranial portion and becomes paler toward the lower third of the esophagus. The smooth esophageal mucosa can be easily distinguished from the dark mamillated gastric mucosa. The mucosal transition at the squamo-columnar junction is an objectively recognizable reference point for endoscopist. On fresh anatomic specimens it is characterized by an abrupt demarcation line or Z line. This serrated line is located near the gastric orifice or just a few centimeters above it. Any proximal extension of a gastric like or intestinal-type columnar epithelium is pathologic and is attributable to long lasting gastro-esophageal reflux causing chronic severe esophageal mucosa and submucosal damage ***(Bremner, 1994).***

## Arterial supply:

There are three principal sources of the arterial blood supply of the esophagus. One in the neck, one at the aortic arch level and one at the cardia. In the neck, the superior and inferior thyroid arteries send small arteries to the cervical esophagus. Immediate branching reduces the diameter of these vessels that are already minute. At the level of the aortic arch, a group of three to five tracheobronchial arteries arising from the concavity of the arch and a single tracheobronchial artery arising more caudally from the anterior surface of the aorta give rise to the several esophageal tributaries (Fig. 5). Occasionally, one or two esophageal arteries proper arise from anterior thoracic aorta. Again, all esophageal vessels are reduced to a

small diameter by branching when they approach the esophageal wall. At the esophagogastric junction, the left gastric artery gives off two to sex branches that will supply mostly the anterior and right faces of the lower esophagus. Vessels from the splenic artery regularly supply the posterior esophageal wall and part of the greater curvature of the stomach ***(Liebemann-Meffert et al., 1997)***.

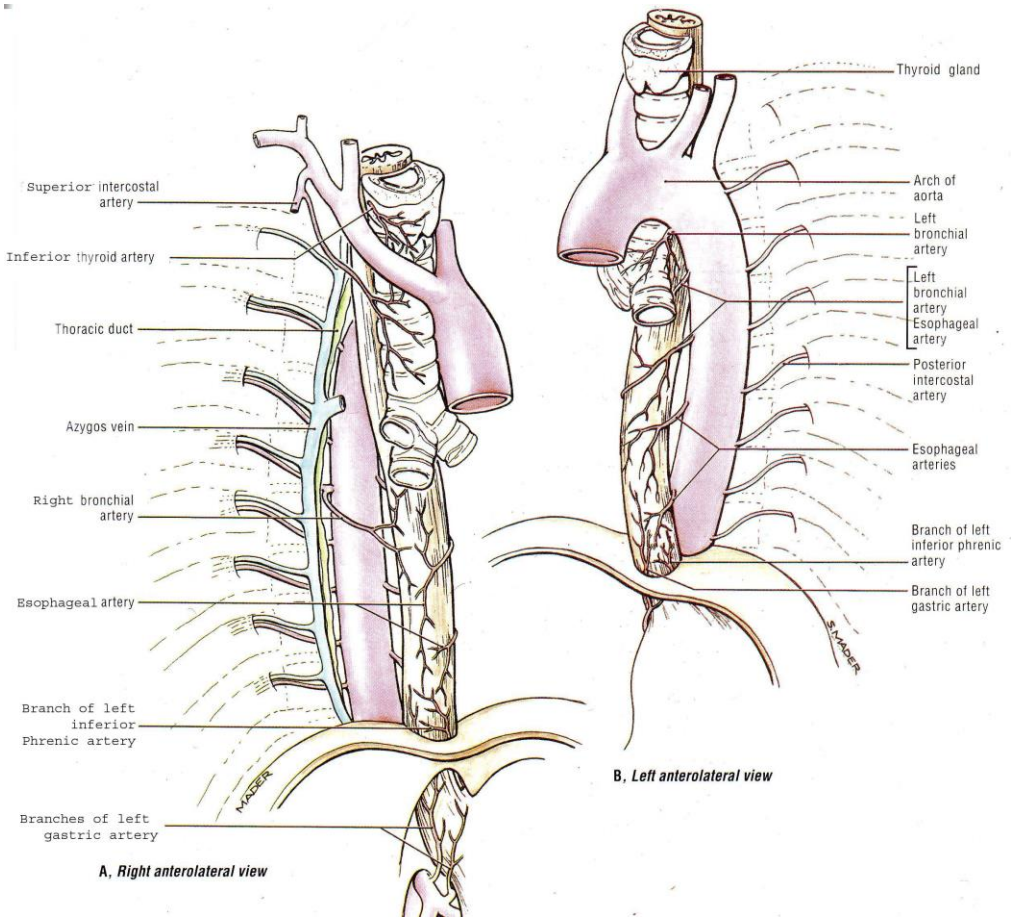



**Fig. (5):** Arterial supply of the thoracic esophagous
***(Anne and Ming, 1992)***

The extra-esophageal branches enter the esophageal wall, pass through the tunica muscularis and give off branches to the muscle and form a vascular plexus to the submucosa and mucosa. The evident continuity of the vessels and the rich anastmosing intramural vascularity explain why the mobilized esophagus retains blood supply over a long distance ***(Liebemann-Meffert et al: 1997).***

## Venous drainage:

The most comprehensive description of esophageal venous drainage is presented by Butler. He classified the esophageal veins into intrinsic and extrinsic veins, referring to intraesophageal and extraesophageal wall veins. The intraesophageal veins include a sub-epithelial plexus in the lamina propria close to the epithelium. This plexus forms a meshwork. In respect to esophageal axis, the vessels are arranged longitudinally and extend over the whole length of the esophagus. This plexus receives its blood supply from the capillaries located within the lamina propria. They drain into the submucous plexus, composed of vessels that unite to form small communicating veins, which in turn are arranged mainly in the longitudinal axis. In the submucosa and lamina propria of the lower end of the esophagus, anastmosis between the systemic and the portal system are possibly present. The thin walled superficial veins may enlarge in portal venous obstruction to form varices. Perforating veins pierce the muscular wall of the esophagus. They receive tributaries from

the muscle coat and form the extraesophageal veins at the surface of the esophagus. These extraesophageal vessels drain into locally corresponding large veins: the inferior thyroid veins, which empty into the brachiocephalic veins, the azygos and hemiazygos, the left gastric or coronary vein, the splenic vein via short gastric veins and the left gastroepiploic vein ***(Georg and Orringer, 1996).***

## Lymphatic drainage:

Sakata, Rouviere and Idanov emphasized the existence of rich lymphatic network in the mucosa and submucosa of the esophagus. The lymph vessels are abundantly interconnected and may surpass the blood capillaries number ***(Bruna, 1994).***

Most important is Sakta and Lehnert`s observation that the submucosal lymphatics form long channels that run parallel to the esophageal axis. Sakata claimed that the lymph flow through these channels may go, for example, from the midesophagus to the cervical or lower esophagus rather than through the few channels that pierce the muscular coat and only finally lead into the mediastinal lymph nodes ***(Lehnert, et al.: 1995).***

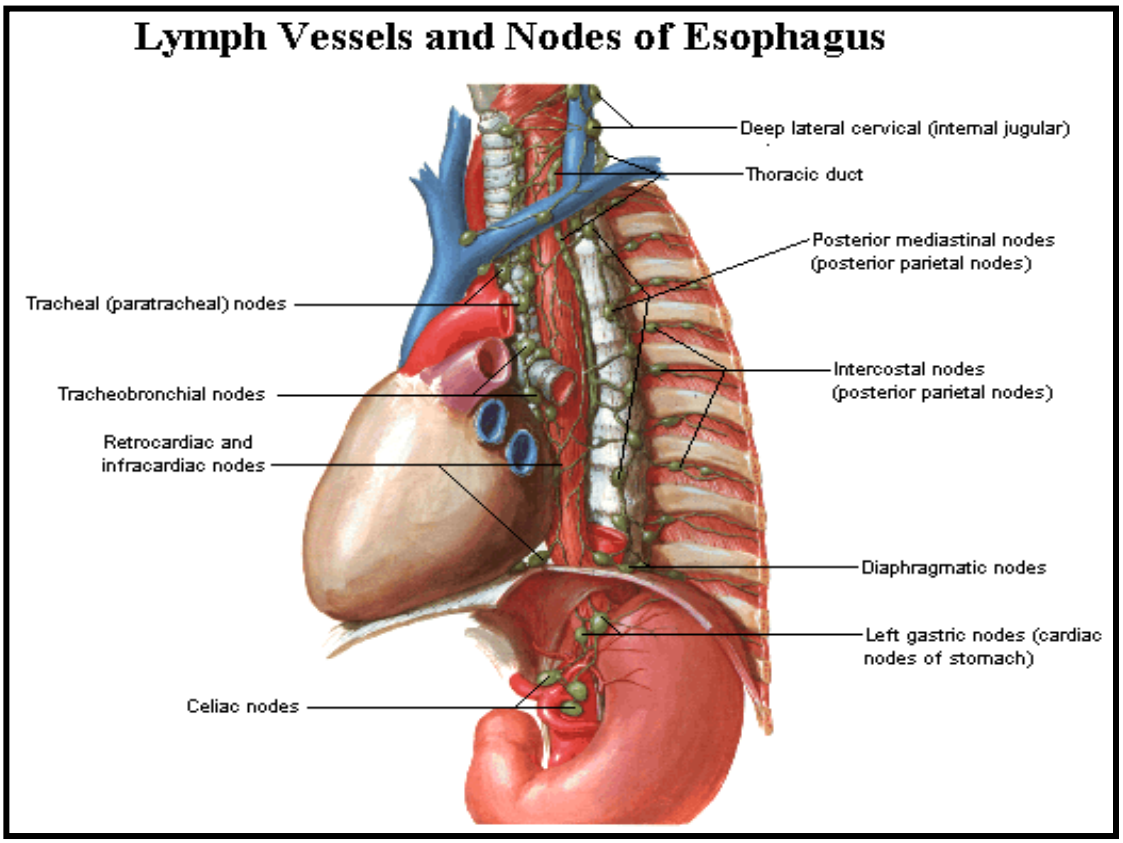


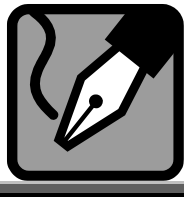

**Fig (6):** Lymph vessels and Nodes of esophagus ***(Bruna, 1994).***

Consequently, a primary tumor of the mucosa may extend over a considerable length within the esophageal mucosa and submucosa, and free tumor cells may follow the lymphatic channels over a considerable distance before passing through the muscular coat into the regional lymph nodes ***(Lehnert, et al.: 1995).***

Regional lymph nodes, according to Szabo and Shdanow, receive lymph outflow as follows: for the cervical esophagus, the lymph flow to the paratracheal cervical, the internal jugular, and the pre-clavicular lymph nodes. The thoracic esophagus

drains into the paratracheal, the tracheo-bronchial, the bifurcations, the juxta-esophageal, inter aortico-esophageal lymph nodes. For the abdominal esophagus, lymph drains toward the following node group: the gastric superiors, and the inferior diaphragmatic lymph nodes ***(Szabo, et al., 1991).***

From clinical observation of Akiyama, Haagensen, and Lam and associates, a clinically important suggestion can be deducted: that the lymph draining from areas above the tracheal bifurcation will drain mostly cranially toward the thoracic duct, whereas draining lymph from below the carina may flow mainly toward the cisterna chyli through the lower mediastinal, left gastric, and celiac lymph nodes. Flow in the area of the tracheal bifurcation seems to be bidirectional. It is uncertain whether this flow direction is the same under pathologic conditions. The lymph channels may be obstructed, for example, by tumor spread leading to reverse of the flow. Therefore, there may be a considerable variation in the eventual pathways of drainage (Fig. 6) ***(Bruna, 1994).***



# PHYSIOLOGY OF THE ESOPHAGUS

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The primary function of the esophagus is the aboard transport of an ingested food bolus by a neuromuscular process called primary peristalsis. Both primary and secondary peristalses clear refluxed gastric fluid from the esophagus into the stomach. Saliva is transported from the oral cavity by primary peristalsis to neutralize trace acid in the distal esophagus. The main distinction between primary and secondary peristalsis is in their initiation and neuromuscular control. Primary peristalsis is initiated by a pharyngeal swallow and impulses are autonomically controlled by the swallowing center of the brain within the central nervous system. Secondary peristalsis is an autonomic response to distention of the esophageal lumen within the enteric nervous system at the smooth muscle muscularis along the esophageal lumen ***(Meyer et al., 1999***).

The esophagus passes through the thoracic and mediastinal cavities, and passes into the abdominal cavity though opening within the diaphragm (Hiatus). Crural muscle fibers wrap sling like around the esophageal body and attach to the vertebral body. These external striated muscle fibers form an extrinsic sphincteric component called the crural diaphragm (CD). The intrinsic lower esophageal sphincter (LES) is located within the esophago-gastric segment (EGS) at roughly the same level as the crural diaphragm in the mid breath state ***(Meyer et al., 1999).***

Esophageal muscle is fully striated in the proximal 2-6% and fully smooth in the distal 50-60% with a broad transition region of mixed muscle fibers in between. The speed of peristaltic propagation varies along the esophagus depending on the muscle type with higher speed of the propagation in the upper esophagus ***(Clouse and Hallett, 1995)***.

The esophagus and the gut in general consist of concentric tissue layers. The innermost layer (mucosa) of the esophagus covered with stratified squamous epithelial cells with conversion to columnar cells of the stomach at the squamo-columnar junction (SCJ) in the esophago-gastric segment. The mucosa consists of a thin layer called lamina propria surrounded by the muscularis mucosae, containing a layer of nerve endings called the Meissner's plexus. the mucosa folds on itself in its resting state and occludes the lumen. The mucosae are surrounded by circular and longitudinal muscle layers, between which are inter-connective tissue and the Auerbach's plexus of nerve endings. Contraction of circular muscle layer generates circumferential tension and peristalsis wave ***(Brasseur; 1993).*** The longitudinal muscle layer shortens the esophagus locally during peristalsis ***(Pouderoux et al., 1997).***

Auerbach's plexus form the nerve supply for the muscle layers. Peristalsis in the proximal striated muscle section is directly innervated by the central nervous system through the vagus nerve, while peristalsis in the smooth muscle section is

controlled by the interaction between the central and the enteric nervous system ***(Edmundowicz and Clouse, 1991).***

## Anatomy and physiology of the esophago-gastric segment (Fig. 7):

The esophago-gastric segment (EGS) is defined as the esophageal segment between the lower esophagus and the stomach. Anatomically the EGS includes the intrinsic or lower esophageal sphincter (LES), the crural sphincter and the phreno- esophageal ligament (PEL) ***(Kwok et al., 1999).***

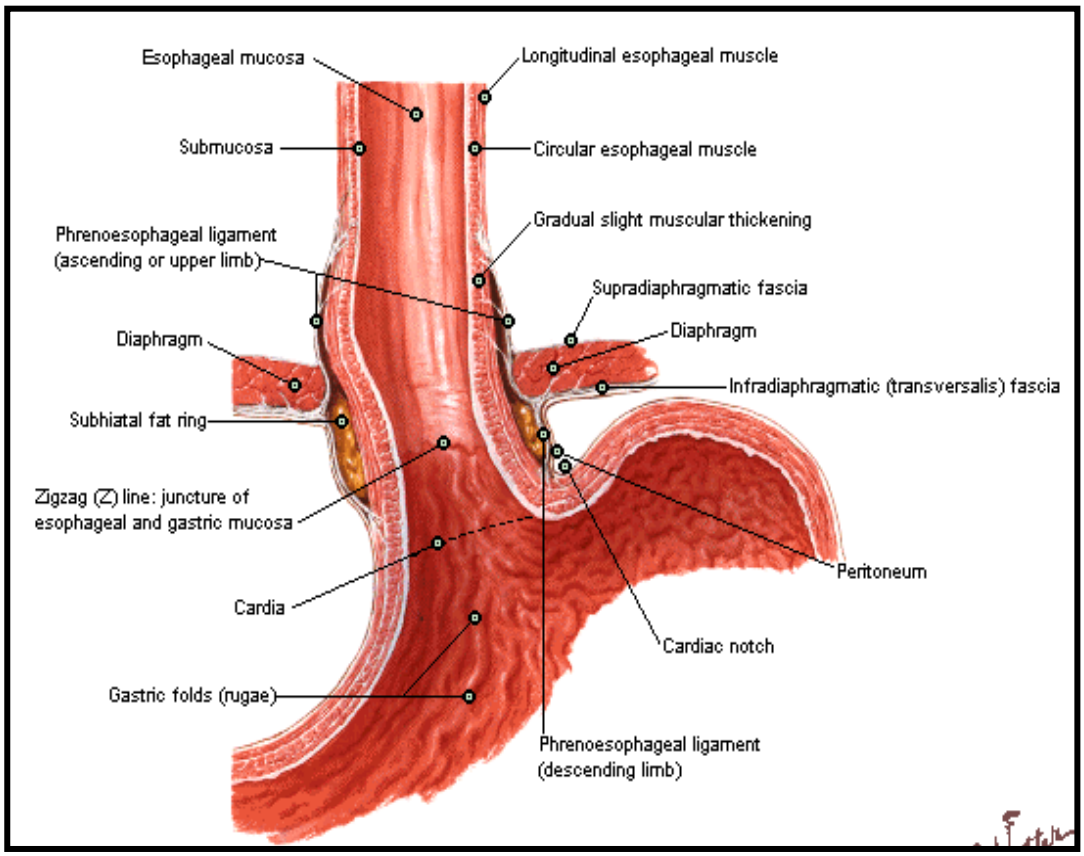



**Fig. (7):** Gastro-esophageal junction ***(Massey, 2001)***

Physiologically, EGS contains a high pressure zone (HPZ) measured manometrically in the resting state, reflecting lower esophageal sphincter and crural diaphragm tone that prevent gastro-esophageal reflux. During breathing, the costal diaphragm contracts in a phasic manner resulting in a pressure difference between abdominal and thoracic cavities between 30-70 mmHg. This provides a favorable pressure gradient for gastro esophageal reflux and a HPZ acts a barrier to reflux by keeping EGS closed. By contrast, with its protective function, the EGS must also allow unimpeded transport of a swallowed bolus from the esophagus to the stomach, the regular venting of the swallowed air from the stomach into the esophagus and the occasional regurgitation of gastric contents ***(Mittal et al., 1998).***

The high pressure antireflux barrier of the EGS arises from the intrinsic lower esophageal sphincter and the extrinsic crural diaphragm. The phreno-esophageal ligament extends from the costal diaphragm and merges circumferentially with the esophageal wall. The competence of the phreno-esophageal ligament is an important factor to preserve the abdominal esophagus and therefore may affects the gastro-esophageal reflux ***(Kwok et al., 1999).***

The lower esophageal sphincter is a 3-4 cm long segment of sphincteric muscle. Unlike the esophageal muscle, the lower esophageal sphincter muscle is contracted in the resting state and maintains a pressure of about 10-30 mmHg above the intra-gastric pressure ***(Massey, 2001).***

The crural muscles that constitute the external sphincter are attached to the vertebral column. The crural diaphragm has an opening through which the esophagus enters the abdomen. This canal is formed primarily by the right crus muscle of the diaphragm, and is observed as a constriction called the hiatus in radiographic studies. Contraction of the crural diaphragm exerts a pinchcock- like action on the lower esophageal sphincter, thereby extrinsic sphincter mechanism. It is also to be noted that the skeletal muscles of the crura contracts slightly before those of the costal part of the diaphragm, possibly acting as a barrier to the enhanced possibility of the reflux due to a higher abdominal to mediastinal pressure gradient during inspiration ***(Massey, 2001).***

Recent research suggests a third potential component to the protective function of the EGS. Oblique sling fibers extend in a C- like shape with closed ends located along the greater curvature of the stomach ***(Netter; 2000).*** This confirmation of muscle fibers may augment the high pressure barrier of the EGS as an additional component to the reflux barrier ***(Muinuddin et al., 2004).***

## Transient lower esophageal sphincter relaxation

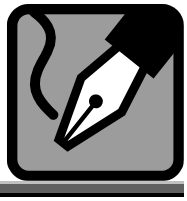
Transient lower esophageal sphincter relaxations (TLESRs) are abrupt decreasing in resting EGS pressure to the level of the intra-gastric pressure resulting from the ablation of the lower esophageal sphincter and crural diaphragm tone ***(Mittal and Fisher, 1990).***

TLESRs typically last 10-45 sec, longer than swallowing induced LES relaxation. The normal function of the TLESR is to enable the venting of gases from the stomach through the normally closed EGS. However, TLESRs are also implicated in most normal gastro-esophageal reflux events, aided by a positive intra-gastric to intra-esophageal pressure gradient. Reflux has been reported to occur at a frequency about 2-6 per hour in normal subject ***(Mittal et al., 1995).***

The role of TLESRs in reflux disease is an issue of some controversy. Whereas, it has been reported that TLESRs are not the primary mechanism underlying the reflux events in patients with hiatus hernia ***(Van Herwaarden et al., 2000)***.

Acid reflux has been reportedly observed in 35-65% of all TLESRs events in chronic reflux patients, compared to about 20-35 of all TLESRs in normal subjects ***(Grossi et al., 2001).***

During TLESRs, the EGS muscle tone ablates to within 2 mmHg of the intra-gastric pressure, practically implying the absence of any high pressure barrier. The possibility of mucosal damage resulting from the acid reflux during TLESRs is further increased in patients suffering from hiatal hernia due to accumulation of acid in the herniated gastric funds ***(Holloway et al., 1995).***



# PATHOGENESIS OF GERD

# PATHOGENESIS OF GERD

Gastro-esophageal acid reflux results in a myriad of symptomatic conditions such as heart burn and regurgitation, collectively referred to as gastro-esophageal reflux disease (GERD). A common consequences of GERD is reflux esophagitis, in which acid reflux causes erosions and modulation of the mucosal wall of the distal esophagus and manifests as a sensation of heart burn ***(Kahrials et al., 1996).***

The gastro-esophageal reflux disease (GERD) is resulting from the interaction of multiple patho-physiologic mechanisms. Beside incompetence of lower esophageal sphincter (LES) as indicated by transient LES relaxations and reduced basal LES pressure, other mechanisms are thought to play a role in GERD pathogenesis like disturbances in esophageal motility results in impaired esophageal clearance, anatomical abnormalities like hiatal hernia, gastric acid hypersecretion, duodeno-gastro-esophageal reflux of bile acids, delayed gastric emptying, disturbances in salivation and impaired mucosal resistance of the esophagus. A possible role of Helicobacter pylori is being evaluated ***(Janssens and Vantrappen, 1999).***

## Lower esophageal sphincter dysfunction:

The first cause in increased esophageal exposure to gastric juice in patients with gastro-esophageal reflux disease is mechanically defective lower esophageal sphincter, this cause

accounts for about 60% of cases of gastro-esophageal reflux ***(Zaninotto et al., 1999).***

LES dysfunction is the most common cause of gastro-esophageal reflux. It has been shown that LES competence is dependant on the resting sphincter pressure, total sphincter length and intra-abdominal esophageal length. The number of defective component of the LES has been observed to correlate with the severity of the esophagitis ***(Rakic et al., 1997).***

Some patients with GERD have a defective resting LES pressure or reduced sphincter pressure during periods of increased intra-abdominal pressure but much more frequently the underlying abnormality is an increased frequency of transient relaxation of the LES. Gastric distention significantly increases the rate of transient LES relaxations contributing to post-prandial reflux seen in patients with GERD. Evidence suggests that transient LES relaxations account for approximately 40-70% of reflux episodes in GERD patients ***(Holloway et al., 1995).***

An adequate abdominal length of the lower esophageal sphincter is important in preventing reflux caused by increases in intra-abdominal pressure and an adequate overall abdominal esophageal length is an important factor increasing the resistance to reflux caused by increase in intra-gastric pressure independent of intra-abdominal pressure. Therefore, patients with low sphincter pressure or those with a normal pressure but

a short abdominal length are unable to protect themselves against reflux caused by fluctuations of intra-abdominal pressure that occur with daily activities or changes in position and unable to protect themselves against reflux related to independent increase in gastric pressure caused by outlet obstruction, aerophagia or gluttony.

Patient with gastro-esophageal reflux disease and a deficiency of one or all components of a mechanically competent lower esophageal sphincter have a patho-physiologic defect of the anti-reflux barrier. Identification of a mechanically defective lower esophageal sphincter is important because the anti-reflux surgery is designed to correct this defective LES ***(Clement and Heibert, 1991).***

**Intra-abdominal esophagus:**

In patients with GERD, it has been shown that the level of acid exposure is inversely proportional to the length of the intra-abdominal esophagus. The probability of reflux being 19% when the length is greater than 2 cm, but rising to 90% when the length is 1 cm or less in the presence of normal esophageal sphincter pressure ***(Johnsson et al., 1997).***

In the normal situation, the effect of positive intra-abdominal pressure on the intra-abdominal segment of the esophagus maintains apposition of its walls, as shown by the flattened cross sectional appearance of the esophagus CT scan ***(Clark, 1996).***

## Esophageal body dysfunction:

In normal circumstances, esophageal mucosa contact time during episodes of esophageal reflux is minimized by both primary and secondary esophageal body peristalsis. Primary peristalsis is that which occurs in response to a swallow, and frequently follows reflux episodes. Furthermore, distension of the distal esophagus by a bolus of refluxate elicits peristaltic activity, known as secondary peristalsis .Impairment of peristaltic function is present in around 25% of patients with mild GERD and around 50% of those with severe disease. Such impairment may manifest either as simultaneous non-propagated peristalsis, or low amplitude contractions, both of which abnormalities impair both primary and secondary peristalsis ***(Kahrilas et al., 1996).***

There is a significant association between impaired peristaltic function and the presence of hiatal hernia, which is present in 82% of patients with severe peristaltic impairment, and only 14% of reflux patients with normal manometric body function ***(Jenkinson et al., 1991).***

Continual acid reflux eventually leads to esophageal injury and can result in permanent esophageal body dysfunction ***(Stein et al., 1990).***

The failure of peristaltic pump, either from reflux or from other causes, such as scleroderma, results in frequent and prolonged episodes of esophageal acid exposure. There are

several reports of improvement of reflux related esophageal dysfunction after fundoplication surgery ***(Hunter et al., 1996)***.

Long lasting acid reflux episode were taken to indicate a problem with acid clearance, and nocturnal acid reflux was felt to be especially important in the development of esophagitis because of infrequency of peristaltic contractions at night ***(Barham et al., 1995).***

**Gastric dysfunction:**

Abnormal gastric function may results in esophageal exposure to gastric juice. Conditions like increased intra-gastric pressure, excessive gastric distension, increased gastric acid secretion or delayed gastric emptying may result in increased esophageal acid exposure ***(De Meester and Jetal, 1990).***

Delayed gastric emptying can be an important factor in the development of GERD. The effect of impaired gastric emptying, irrespective of the cause, include increased pressure against the anti-reflux barrier, an increased rate of transient lower esophageal sphincter relaxation, shortening and consequent impairment of function of the lower esophageal sphincter, likened to the shortening of a neck of a balloon consequent on inflation, and an increased in the gastric content available for reflux. Interestingly, gastric emptying is increased following anti-reflux surgery, which may reflect the effect of fundoplication on the fundal baroreceptors ***(Maddern et al., 1995).***

**Bile reflux:**

Bile salt may be implicated in the etiology of esophagitis particularly in Barrett's esophagus which may be accompanied by significant increase in bile salt concentration in esophageal aspirates ***(Stein et al., 1994).***

The relationship between the toxic effect of bile salt and PH is more complex. In order to cause mucosal damage, bile salts must be both soluble and in a non-ionized form. Below PH 2, bile salts precipitates out and are, therefore, incapable of mucosal injury. At the normal duodenal PH of 7, bile salts are mostly in solution, but are completely ionized, and again are not cytotoxic. However, in the PH range of 2-7, bile salts exist as a mixture of ionized and non-ionized forms, and in the presence of acid and pepsin, are capable of producing the most severe form of cellular damage. An alkaline PH, bile salts are in solution and partly dissociated, and again are capable of producing severe cellular damage.

The question of whether reflux of duodenal contents is of theoretical or practical importance has long been debated, but has recently become clarified by the availability of ambulatory bilirubin monitoring by means of portable spectrophotometric system (Bilitic). Recent studies combining ambulatory PH and bilirubin monitoring have shown that pure acid and pepsin reflux occurs in 42% of patients and in 58% the reflux comprises of a mixture of gastric and duodenal juices with the

assumption that bilirubin is a marker for duodenal juice ***(Kauer, et al., 1995).***

**Esophageal mucosal resistance:**

Normally, the esophageal epithelium is relatively thick, composed of squamous cells with tight junctions and lipid rich intercellular space and is resistant to abrasions from food material. With prolonged exposure to regurgitated gastric contents erode the mucosa, promoting infiltration by neutrophils and eosinophils and ultimately epithelial cell necrosis ***(Rouvk et al., 1994).***

Mucosal injury which occurs as a consequence of gastro-esophageal reflux episode depends not only on contact time, PH and nature of refluxate, but also on the integrity of the mucosal defense mechanism. Mucosal damage, possibly mediated by tissue prostaglandins and back diffusion of hydrogen ions, occur causing cellular damage and increasing permeability, thus allowing vicious circle of further damage by acid and other constituents of the refluxate. The factors responsible for the integrity of the mucosal defense mechanisms are not completely understood. Although it is likely that bicarbonate in mucous plays a part, as gastro-duodenal mucosa, and those surface active phospholipids which are relevant in mucosal defense in many tissues such as lung and synovial membranes, may also be relevant ***(Kwun et al., 1992).***

Epithelial renewal after damage by mechanical or chemical injuries is an important defensive mechanism of the esophageal mucosa. The presence of epidermal growth factor (EGF) receptors in the esophageal epithelium and the high EGF concentration in human saliva suggest a role of EGF in epithelial renewal process in the esophagus. Salivary EGF is strongly enhanced by acid perfusion of the esophagus in healthy controls but in patients with esophagitis, this response is markedly impaired ***(Rouvk et al., 1994).***

While EGF receptors are increased in inflamed esophageal mucosa, EGF levels are significantly lower in esophagitis, suggesting that local production of EGF may be impaired in esophagitis. On the other hand, the reduction of EGF in esophagitis could results from depletion of mucosal EGF stores secondary to inflammation ***(Jankwski et al., 1992).***

**Salivation:**

Saliva has an important role in mitigation of the effect of the gastro-esophageal reflux. Its bicarbonate content is capable of neutralizing small acid volumes, and salivation induces swallowing, which in turn induces esophageal body peristalsis. It has been shown experimentally that acid clearance time is decreased after salivary stimulation, and, conversely acid clearance time is increased if saliva is removed by suction, even when a constant swallowing frequency is maintained.

Salvia production may be increased by the presence of acid in the lower esophagus, when this situation is present clinically, the patient experiences excessive mucus in the throat. Medically, this condition is referred to as "water brash" ***(Helm et al., 1998).***

**Acid factors:**

Although GERD is considered primarily a motor disorder its clinical severity is related to the concentration of acid in the refluxate, frequency and duration of esophageal acid exposure. In most case, this is not caused by gastric acid hyper-secretion.

Several studies in animal models have shown that esophageal mucosal injury is pH dependent. The lower the pH of the refluxate, the longer the time required for intra-esophageal pH to return to non injurious levels and the higher the risk for severe manifestation of the disease ***(Bell et al., 1992).***

It was reported that there is a correlation between acidity and esophageal pain in patients with GERD who were administered solutions with pH concentrations in the range of 1.0 - 4.0 ***(Bell et al., 1992).***

Factors that precipitate reflux include overeating; a diet rich in fatty foods, caffeine and chocolate; alcohol use; tobacco use; and stress ***(Smith et al., 1999)***.

Patients with mild GERD experience the majority of reflux episodes during day time post prandial periods; more nocturnal reflux is observed as the severity of GERD progresses ***(Bell et al., 1992).***

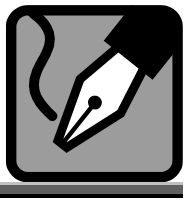
Nocturnal reflux may be more damaging to the esophageal mucosa due to reduced acid clearance and salivary neutralization during sleep ***(Smith et al., 1999)***.

## Helicobacter pylori and gastroesophageal reflux disease:

Epidemiological, patho-physiological and clinical studies have demonstrated that Helicobacter pylori infection, particularly the cagA-positive strain, is protective against the development of gastroesophageal reflux disease and its complications. This protective effect is dependant on the extent of Helicobacter induced corpus gastritis causing profound reduction of acid secretion. In addition, development of reflux esophagitis after H.pylori eradication therapy has been reported suggesting that the disappearance of H.pylori increase the risk of development of reflux esophagitis.

Although the mechanism of reflux esophagitis development after eradication therapy is unknown, the severity of corpus gastritis before eradication has been shown to be an important factor in predicting the occurrence of reflux esophagitis. The severity of corpus gastritis is known to be

negatively associated with gastric acid secretion. Several studies have shown that gastric acid secretion increase to normal or near normal levels in patients with hpochlorohydria after successful H.pylori eradication therapy. It is presumed that healing of gastritis (parietal cell dysfunction) and increased acid secretion after successful H.pylori eradication therapy contributes to the development of reflux esophagitis ***(Cuschieri, 2002).***



# DIAGNOSIS OF GERD

# DIAGNOSIS OF GERD

## Clinical features of gastro-esophageal reflux disease

Gastro-esophageal reflux disease can occur in all age groups, but it is most common in infancy and middle age. There is a female preponderance of approximately 1.5 to 1 ***(Watson, 1998)***.

For some patients, the symptomatology is so typically that a confident diagnosis of reflux disease can be done on symptoms alone. For other patients, investigatory tools may be necessary to establish abnormal esophageal acid exposure. Finally, a confident diagnosis of GERD can be made when the characteristic reflux associated lesions are observed in the esophagus endoscopically. Whether targeted biopsy of such lesions is useful in clinical practice or not remains controversial ***(Bokus and Berk, 1995).***

**Symptoms:**

a) Typical

1. Heart burn
2. Regurgitation
3. Dysphagia
4. Odynophagia
5. Bleeding

b) Atypical

1. Non cardiac chest pain
2. Pulmonary symptoms
3. Laryngeal symptoms
4. Palatal and dental erosions

***(Bokus and Berk, 1995)***

**Typical symptoms of GERD:**

**Heart burn:**

Heartburn is the classical symptom of gastro-esophageal reflux and is present in over 80% of patients. It consists of a retrosternal burning pain, which usually comes on after eating, but occasionally at night and after bending or stooping. It is frequently exacerbated by ingestion of acidic substances, such as citrus fruits, fruit juice and red wine, as well as by substances such as coffee, foods containing fat and nicotine, which reduce lower esophageal sphincter pressure. Heartburn is usually relieved by milk, antacids and agents, which suppress gastric acid secretion. Experimentally, heartburn can be induced not only by intra-esophageal infusion of acid, but also by a variety of other stimuli, including balloon distension. The efficacy of acid suppression agents therefore is related not only to alteration in pH, but also to reduction in the volume of refluxate ***(Watson, 1998).***

Heartburn is often intermittent, often occurring within half an hour after meals, an exercise or after bending or in lying down ***(Watson et al., 1995).***

There is an imperfect correlation between the presence and severity of heartburn and of gastro-esophageal reflux. Firstly, heartburn may be a symptom of biliary tract disease or an upper gastro-intestinal motility disorder and secondly, patients with mild symptoms may have severe disease and vice versa. Patients with stricture and Barrett's usually have the most severe patho-physiological abnormalities, but rarely complain of reflux symptoms, which has been shown to be associated with impairment of sensitivity to infused acid, compared to patients with erosive esophagitis. Furthermore, a group of patients exists in whom symptoms may be very severe, even in the absence of esophagitis and pathological acid exposure on 24 h pH monitoring. These patients are believed to have a sensitive esophagus whereby physiological reflux episodes of short duration may result in severe symptoms and demonstrating a close correlation between symptoms and reflux episodes on pH monitoring usually identifies these patients ***(Ball and Watson, 1998)***.

**Regurgitation**

Regurgitation may be present in addition to heartburn or it may be the only symptom. Food may regurgitate into the pharynx, particularly on bending or stooping, but, more

commonly, it is gastric juice that regurgitates and produces a bitter or acidic taste. Regurgitation is usually associated with very poor LES function, but it is important to recognize that many patients with regurgitation do not have esophagitis, and may not necessarily have pathological acid exposure on pH monitoring, if the pH of their refluxate is greater than 4. In these circumstances, symptomatic control may be difficult to achieve by acid suppression therapy. Surgical treatment is more likely to be necessary when regurgitation is a predominant symptom, particularly if there are demonstrable objective abnormalities, such as an anatomical hiatal hernia, lower esophageal sphincter failure and indeed esophagitis or pathological acid exposure when these are present ***(Watson, 1998)***.

In some patients with GERD, the predominant symptom is frequent effortless vomiting. Some patients may even complain of a burning sensation of the tongue, lips and buccal mucosa, and a chronic unpleasant taste in the mouth. The effortless return of gastric or esophageal contents into the pharynx is distinguished from the vomiting by the absence of nausea, retching and abdominal contractions ***(Watson et al., 1995).***

***Dysphagia***

Dysphagia may be a symptom of gastro-esophageal reflux disease in 40-50% of patients. Although only around

10% of patients with the more sever forms of gastro-esophageal reflux disease will develop a stricture with dysphagia, which is usually persistent and often progressive. Many patients have co-existing motility disorders resulting in ineffective peristalsis, which can give rise to occasional, brief of dysphagia, which may range from an awareness of slow transit of a solid bolus up to bolus impaction. Dysphagia may be experienced at any level retro-sternly, depending on the nature and site of the peristaltic impairment. Dysphagia may be felt at cricopharyngeal level (globus sensation), as gastro-esophageal reflux is known to induce cricopharyngeal spasm in some patients ***(Watson, 1998).***

In the majority of cases, dysphagia resolves with successful treatment of the gastro-esophageal reflux, although caution should be exercised in patients with a severe degree of peristaltic impairment, in whom the performance of a total fundoplication procedure may exacerbate the dysphagia. The complete reversibility of motility disturbances following successful anti-reflux surgery remains controversial, and many factors are involved. localized areas of high amplitude peristalsis and indeed low amplitude contractions in the presence of normal propagated peristalsis are much more likely to be improved than disordered peristalsis or low amplitude contractions in all segments, and current evidence suggests that improvement is more likely after partial rather than total fundoplication procedures, because of the less obstructing nature of the wrap ***(Watson, 1998).***

## Odynophagia

Odynophagia means painful swallowing and may occur on swallowing solids or liquids, particularly if very hot or very cold. This symptom can be a useful clinical guide to the presence of gastro-esophageal reflux and is usually associated with the presence of esophagitis. Pain on swallowing solids may occur as a result of organic obstruction from a stricture or a functional obstruction caused by a motility disorder. Such pain is often excruciatingly severe, being felt across the chest and radiating to the inter-scapular region and occasionally up to the neck and gums, mimicking the pain of ischemic heart disease ***(Bremner, 1998)***.

## Bleeding:

Bleeding is an uncommon symptom of gastro-esophageal reflux disease and only occurs in the presence of sever esophagitis or esophageal ulcer. Rarely, the bleeding is overt, resulting in hematemesis or melena, but more usually, bleeding is slow and results in iron deficiency anemia. Caution must be exercised in attributing occult bleeding to the presence of a hiatal hernia or gastro-esophageal reflux disease unless this is associated with severe esophagitis or frank ulceration ***(Bremner, 1998)***.

## Atypical symptoms of GERD:

### Non cardiac chest pain

Non cardiac chest pain is a common clinical situation in which patients complain of chest pain, which is characteristic of ischemic heart disease, even including its pattern of radiation, but cardiac investigations including coronary angiography fail to identify a cardiac cause. While some of these patients undoubtedly have micro-vascular angina, over 50% of such patients have, in fact, been identified as having an esophageal disorder, usually gastro-esophageal reflux disease, but, occasionally, a primary motility disorder. Difficulties can occur, as gastro-esophageal reflux and ischemic heart disease frequently co-exist. Reflux episodes and experimental instillation of acid into the esophagus can cause ECG abnormalities in such patients, and in some patients without demonstrable cardiac abnormalities, their reflux and chest pain is only induced by exertion. Clearly, careful assessment of these patients is necessary and as well as full cardiological assessment, both ambulatory manometry and pH monitoring is required, and occasionally needs to be combined with ECG stress testing in difficult causes. However, once correctly diagnosed, and particularly where gastro-esophageal reflux disease is the cause of the problem, the results of effective reflux control are quite dramatic, both in relieving a disabling symptom which often is the subject of several hospital

admissions, and the associated anxiety resulting from the fear of cardiac disease ***(Watson, 1998)***.

**Pulmonary symptoms**

Pulmonary symptoms associated with gastro-esophageal reflux are common. The commonest of these is asthma, which may commence unusually late in life and coexist with reflux symptoms. This is thought either to be associated with micro-aspiration during reflux, or a vagal mediated bronchospasm consequent with esophageal acidification in particularly sensitive subjects. Occasionally, patients may present with recurrent episodes of pneumonia which may occur in patients with gastro-esophageal reflux, with or without the presence of esophagitis, indicates the necessity for reflux control. The symptomatic response is usually quite dramatic ***(Fennerty, et al., 2000)***.

In patients with bronchial asthma, during the attack of asthma changes in the intra-thoracic and intra-abdominal pressure and in diaphragmatic performance may impair the anti-reflux barrier and cause gastro-esophageal reflux. It might be supposed that gastro-esophageal reflux provokes asthma and asthma causes gastro-esophageal reflux. A vicious circle could arise with exacerbation of asthma symptoms ***(Boyle et al., 1995).***

***Laryngeal symptoms***

Laryngeal symptoms may occur in association with gastro-esophageal reflux, and may manifest as a persistent dry cough, change in voice or chocking episodes. These symptoms may be associated with laryngeal erythema, edema and even ulceration. In such cases, esophagitis is often present in the proximal esophagus and, in addition to demonstrating pathological acid exposure by a pH probe situated at the usual site, 5 cm above the manometrically determined lower esophageal sphincter, excessive acid exposure is usually, demonstrated in the proximal esophagus using a second probe, 5 cm distal to the cricopharyngeal sphincter. Once more, effective reflux control usually results in dramatic relief of the symptoms and reversal of the pathological changes ***(Watson, 1998)***.

Chronic cough may be symptom of gastro-esophageal reflux. The number of the cough correlated significantly to the total number of reflux episodes, the number of reflux episodes longer than 5 minutes and the percentage of total time with pH of less than 4 ***(Irwin et al., 1999)***

***Palatal and dental erosions:***

Palatal and dental erosions have more recently been shown to be associated with gastro-esophageal reflux and proximal pathological acid exposure ***(Boyle et al., 1995).***

Dental surgeons are beginning to seek exclusion of gastro-esophageal reflux in atypical cases ***(Boyle et al., 1995)***

## Investigation of GERD:

Although a high percentage of the population suffer from symptoms of GERD, but only a small percentage present to the clinician. That is why cases which present with reflux symptoms should be investigated correctly and seriously on certain sequence of tests evaluating their symptoms. Some of the physiological tests require special expertise in their execution and interpretation and are, therefore only available in major or specialized centers ***(Cuschieri, 2002).***

Gastro-esophageal reflux disease GERD investigations can be divided into two main categories, radiological procedures, which are non invasive and provide anatomic and qualitative information, and physiological procedures, which are invasive tests and provide more functional and quantitative data and in some instances, tissue diagnosis ***(Stevens et al., 1999).***

### I. Non-Invasive

1. Chest radiology
2. Barium swallow
3. Radio-isotope studies

**II. Invasive**

1. Endoscopy
2. Manometry
3. Evaluation of esophageal motility (fluoroscopy)
4. PH monitoring
5. Acid clearance test ***(Stevens, et al., 1999).***

## I. Non Invasive Testing

### 1. Chest radiology

Radiological studies are usually the first tests obtained in patients who present with esophageal complaints. Patients are willing to undergo this examination because they are not invasive and can be performed in an ambulatory setting at a very modest cost ***(Stevens, et al., 1999).***

The presence of a gas filled portion of the stomach above the level of the diaphragm, best observed in lateral view. It is suggestive of the presence of hiatal hernia. In the antero-posterior view, the cardiac shadow may obliterate the hernia outline ***(Ott and Abernethy., 1990).***

This investigation is necessary in all patients who have esophageal symptoms to exclude aspiration pneumonia. Chest radiography (CXR) is an essential investigation which performed before contrast studies ***(Cuschieri, 2002).***

## 2. Barium swallow

The first diagnostic test in patients with suspected GERD should be a barium swallow and meal including a full assessment of the stomach and duodenum. Esophageal motility is assessed by observing several individual swallows of barium traversing the entire length of the organ, with the patient in the horizontal position ***(Jeffery and DeMeester, 1999).***

The standard examination should include three techniques: a full column view, a mucosal relief maneuver, and an air/contrast interface**.**

The patient is asked to quickly drink 250 ml of barium solution. This provides a full column view of the esophagus. After the contrast passes into the stomach and esophageal mucosal relief images are taken, the esophagus is now coated with a thin layer of contrast while the walls collapse to baseline configuration. Finally, to detect more changes in the esophageal mucosa, an air contrast interface appears when gas producing granules are added to the barium suspension and ingested ***(Stevens et al., 1999).***

However, radiological demonstration of reflux in this way is present in 20% of individuals without esophagitis and radiological reflux is absent in 40% of patients with moderate to severe esophagitis, so the use of Barium swallow in the detection of GERD is therefore suspected ***(Cuschieri, 2002).***

A carefully performed study can also detect moderate or severe reflux esophagitis, stricture and help in the assessment of motor function and volume clearance of the esophagus ***(Stevens et al., 1999).***

The clinician should be suspicious of reflux; even in the presence of a "no GERD" was seen in the radiology report. If the barium filled esophagus and stomach look like a trumpeting elephant, also if the delayed film shows barium in the esophagus when the small bowel is also well filled ***(Jeffery and DeMeester, 1999).***

Only the most severe cases of esophagitis produce changes in the wall of the esophagus that can be seen on X rays and are also useful to evaluate peptic strictures and the complications of reflux ***(Ott and Abernethy, 1990).***

**Radiological signs of GERD**

The important radiological finding directly indicating significant gastro-esophageal reflux is observation of barium suspension refluxing into the esophagus in an amount sufficient to distend the distal half of the esophagus. The impression is strengthened if a hiatus hernia or a patulous esophago-gastric junction is also observed. Most of these patients will provide a history of regurgitation of food or liquid into pharynx ***(Chen et al., 1985).***

***A) Mucosal erosion and ulceration***

The accuracy of the radiological diagnosis of reflux esophagitis has been the subject of several investigations. These rather uniformly suggest that mild reflux esophagitis is detectable radiological in about 50% of cases. However, moderate reflux esophagitis, characterized by erythema with erosions, is detectable in approximately 80% of cases, and severe reflux esophagitis with more extensive ulcerations or an esophageal stricture is detectable radiologically in virtually 100% of cases ***(Chen et al., 1985).***

The characteristic lesion of reflux esophagitis is the mucosal erosion, which may be seen as round dots or streaks of barium in the distal esophagus. They tend to have a linear configuration extending perpendicularly from the squamo-columnar junction. Discrete ulcer may have a linear configuration in the distal esophagus ***(Ott and Abernethy, 1990).***

***B) Scaring:***

Healing of these ulcers is associated with variable degree of scaring. In some patients, the radiological finding ranges from slight rigidity and asymmetry of the distal esophagus to a typical peptic stricture. Occasionally, prominent sacculations of diverticulae are seen because of scarring in the distal esophagus. These large collections of barium should not be mistaken for ulcers ***(Chen et al., 1985)***.

***C) Esophageal fold and polyp:***

This sign consists of a gastric fundal fold that crosses the gastro-esophageal junction and ends as polypoid protuberance in the distal esophagus ***(Stevens et al., 1999).***

The presence of an anatomical hiatal hernia can be demonstrated by barium radiology ***(Watson, 1998).***

Esophagogram should also be performed to evaluate surgical anatomy if operative intervention is being contemplated ***(Stevens et al., 1999).***

## 3. Radio-Isotope studies (Gastro-esophageal scintigraphy)

The scintigraphy method for detection of gastro-esophageal reflux disease (GERD) is non invasive, sensitive, specific and well tolerated by the patient ***(Hillemeier, 1997).***

The esophagus and stomach are scanned after the patient has ingested a solution of Tc99m sulfur colloid. After its administration, a gamma camera is employed to acquire a number of scintiscans of the esophago-gastric region over a 15 to 20 minutes period. The esophagus is compressed if no spontaneous reflux is seen. Reflux episodes are demonstrated as radioactivity in the region of the chest overlying the esophagus ***(Ogorek and Fisher, 1999).***

The test has good specificity but is considered less sensitive than prolonged pH monitoring in detecting reflux. In spite of this, the esophageal scintigraphy has an advantage over the traditional pH probes because it can detect postprandial reflux where an esophageal pH probe may not be able to detect reflux because the gastric content are buffered by ingested food, also scintigraphy can able to detect the volume of reflux ***(Jenkins, 1985).***

The gastro-esophageal scintigraphy allows the detection of rates of gastric emptying that may be helpful in the evaluation of children who have gastro-esophageal reflux disease (GERD). This may be valuable in those children who are undergoing surgical fundoplication and suggests whether or not they need to have a procedure to facilitate gastric emptying simultaneously with the fundoplication ***(Hillemeier, 1997).***

## II. Invasive Testing

### I. Endoscopy

Gastro-esophageal reflux disease (GERD) is difficult to define because of its extreme heterogeneity. Peptic lesions of the squamous epithelium have a characteristic endoscopic appearance but these lesions can occur transiently and may even be absent in severely symptomatic patients. Therefore, the endoscopic appearance has a high specificity but a low sensitivity for the diagnosis of GERD ***(Bokus and Berk, 1995).***

Endoscopy provides direct visualization of esophageal mucosa and thus high sensitivity and specificity for detection of mucosal injury. Endoscopy examination is particularly helpful in patient with typical symptoms and more advanced and complicated reflux disease. Nonetheless many patients with typical symptoms of GERD and abnormal esophageal acid exposure on pH monitoring have no endoscopic or histological esophagitis. Endoscopy remains relatively insensitive in patients with only low grade disease ***(Pace et al., 1991).***

Despite these limitations, it is quite clear that endoscopy remains the investigation of choice for making the diagnosis of reflux esophagitis and grading its severity. The spectrum of endoscopic findings in reflux disease ranging from minimal, equivocal mucosal changes and erosions, to deep ulcerations, strictures and columnar lined esophagus. Data indicates that, the clinical response to treatment and the subsequent prognosis are dependant upon the severity of the mucosal lesions observed endoscopically. Therefore, it is important to classify accurately the severity of the peptic lesions which affects the esophagus ***(Tytgat et al., 1992).***

## Classification of endoscopic grading of esophagitis

The most popular grading system is that advocated by Savary and Miller and modified subsequently.

**Savary and Miller grading of esophagitis**

**Grade I:** Single or isolated erosive lesion(s), oval or linear but affecting only one longitudinal fold.

**Grade II:** Multiple erosive lesions, non-circumferential, affecting more than one longitudinal fold, with or without confluence.

**Grade III:** Circumferential erosive lesions.

**Grade IV:** Chronic lesions: ulcer(s), stricture(s) and/ or short esophagus. Alone or associated with lesions of grade I-III.

**Grade V:** Columnar epithelium in continuity with the Z line, non-circular, star shaped or circumferential. Alone or associated with lesions of grade I-IV. ***(Cuschieri, 2002)***

The Savary and Miller grading system suffers from a significant inter-observer variability and has not been submitted to a validation program. An alternative system, which clinically and functionally correlates with the severity of the reflux and its treatment and which has been submitted to validation with success, is the "Los Angeles classification system".

**Los Angeles classification of esophagitis**

**Grade A:** One (or more) mucosal break no longer than 5 mm, that dose not extends between the tops of tow mucosal folds.

**Grade B:** One (or more) mucosal break more than 5 mm long, that dose not extends between the tops of tow mucosal folds.

**Grade C:** One (or more) mucosal break that is continuous between the tops of two or more mucosal folds but which involves less than 75% of the circumference.

**Grade D:** One (or more) mucosal break which involve at least 75% of the esophageal circumference.

***(Cuschieri, 2002)***

**Beauchamp grading of esophagitis**

***Grade I:*** Red mucosa

***Grade II:*** Two longitudinal ulcers > 3 cm, confluent ulceration

***Grade III:*** Cobble stoning aspect of LOS mucosa and disappearance of "Z" lines.

***Grade IV:*** Presence of an ulcer or stricture.

***(Phillipe et al., 1995)***

## Endoscopic finding

### *A) Reflux esophagitis*

Because of the specific histological change attributable to GERD, an endoscopic biopsy is the most sensitive test for esophagitis. Unfortunately, as many as 40% of patients with symptoms of esophagitis have no microscopic evidence of mucosal pathology. This lack of correlation appears to result from the inherent difficulty of distinguishing mild or grade I esophageal inflammation from normal mucosa endoscopically. Notably, the common finding of erythema in the distal esophagus is not diagnostic for esophagitis as it is also seen frequently in normal individuals. Granularity and friability in the distal esophagus is a more helpful endoscopic sign of early esophagitis ***(Kenneth et al., 1994).***

Advanced mucosal lesions include shallow erosions and exudates arising at gastro-esophageal junction, discrete ulcers, diffuse hemorrhagic mucosa and esophageal strictures. Even endoscopic biopsies are frequently not diagnostic, especially in mild to moderate reflux disease because the histological picture is difficult to interpret. Superficial specimens obtained by biopsy forceps demonstrate a significant inflammatory cell response in many patients with symptomatic reflux disease. Infiltration of the epithelium and lamina propria by neutrophils and eosinophils is in fact, relatively specific. Although, insensitive indicator of a reflux esophagitis, being found in 40

to 60% of patients with moderate to severe disease. Lymphocyte and plasma cell infiltrate do not appear to correlate with the diagnosis. The endoscope findings of reflux esophagitis are redness, erosion, hemorrhage, ulceration and nodulation ***(Phillipe et al., 1995).***

***B) Esophageal stricture***

In grade IV esophagitis stricture is present. Its severity can be assessed, by the ease with which a no. 36 French endoscope can be passed. Esophagoscopy should be performed in all patients with strictures whether the lesions have been demonstrated on contrast studies or are suspected on bases of progressive dysphagia. The principles of diagnostic objectives of endoscopy are to document the presence of stricture and to characterize the degree of esophageal involvement. When a stricture is observed, the severity of esophagitis above it should be recorded. The absence of esophagitis above stricture suggests that a chemical induced injury or a neoplasm caused the stricture. Neoplasia should always be considered and is ruled out only by tissue biopsies ***(Clement and Hiebert, 1991).***

***C) Barrett's esophagus***

Barrett's esophagus is suspected at endoscopy when there is difficulty in visualizing the squamocolumnar junction at its normal location and when a red, more luxuriant mucosa than normally seen in the lower esophagus is present. Its presence is confirmed by biopsy ***(Clement and Hiebert, 1991).***

Multiple biopsies should be taken in a cephalic direction to determine the level at which the junction of Barrett's epithelium with normal squamous mucosa occurs. When the junction is less than 3 cm above the crura, the diagnosis of Barrett's esophagus is questionable, and the possibility that the biopsies were taken from a herniated stomach is considered. Barrett's esophagus is susceptible to ulceration, bleeding, stricture formation, and most important, malignant degeneration. The earliest sign of the latter is severe dysplasia or intra-mucosa adenocarcinoma. Because these neoplastic changes have a patchy distribution, a minimum of five biopsies should be taken from the Barrett's lined portion of the esophagus. Histological changes seen in one biopsy specimen are significant ***(Clement and Hiebert, 1991).***

## II. Manometry

Esophageal manometry is the most accurate method for assessing the function of the lower esophageal sphincter. Manometry is the technique used for recording mechanical activity of the bowel by detecting and measuring changes in the intra-luminal pressure caused by contractions of the gut wall ***(Jeffery and DeMeester, 1999).***

Esophageal manometry is not a mean of diagnosing of gastro-esophageal reflux, but it can give useful information about esophageal motor function, which is relevant to the management. So, the principal use of esophageal manometry is localization of the position of lower esophageal sphincter (LES) and esophageal motor function.

Lower esophageal sphincter competency is determined from manometric assessment of lower sphincter pressure and intra-abdominal and overall sphincter length ***(Watson, 1998).***

This examination gives information relating to the propensity for reflux to occur and information of the precise patho-physiological defect as regards lower esophageal sphincter and peristaltic function. However, it is not a test to determine the presence of reflux and, because of its expense, is not performed routinely in every centre in the assessment of patients with suspected gastro-esophageal reflux disease, although it is performed routinely in most centers which specialized in the investigation and management of such patients ***(Watson, 1998).***

Esophageal manometry is performed using electronic pressure sensitive transducers located within the catheter or water perfused catheters with lateral side holes attached to transducers outside the body which in turn is coupled to a recorder. The transducer is passed through the nose or mouth into esophagus and stomach and then pulled back stepwise across the gastro-esophageal junction at 1 cm intervals to obtain pressure profile of the distal esophageal sphincter. The catheter usually consists of a train of five pressure transducers or five or more water perfused tubes bound together. The transducers or lateral openings are placed at 5 cm intervals from the tip and oriented radially at 72 to each other, around the circumference of the catheter. A special catheter assembly consisting of four

lateral openings at the same level, oriented at 90° to each other, is of special use in measuring the three dimensional vector volumes of the LES. Other specially designed catheters can be used to assess the upper sphincter ***(Ogorek and Fisher, 1999).***

There are two methods for evaluation of the lower esophageal sphincter pressure, the stationary and the rapid pull-back (the catheter assembly is drawn back at a steady rate) techniques. The pull back is less accurate, because it fails to take into consideration the variation in the lower esophageal sphincter pressure owing to respiration and crural diaphragmatic contraction. The stationary pull back technique entails withdrawal of the recording catheter at 0.5 cm or 1 cm intervals through the length of lower esophageal sphincter, with a pause at each step for recording several respiratory cycles and the swallows induced relaxation at each station***(Ogorek and Fisher, 1999).***

As the catheter is pulled back, a high pressure zone is normally encountered in the distal esophageal segment, with further withdrawal, the pressure drops to less than atmospheric pressure, reflecting the pleural pressure ***(Ogorek and Fisher, 1999).***

As the pressure sensitive station is brought across the gastro-esophageal junction, a rise in pressure above the gastric baseline signals at the beginning of the LES. The respiratory inversion point is identified when the positive excursions that

occur in the abdominal cavity with breathing change to negative deflections in the thorax. The respiratory inversion point serves as a reference point at which the amplitude of LES pressure and the length of the sphincter exposed to abdominal pressure are measured. As the pressure sensitive station is withdrawn into the body of the esophagus, the upper border of the LES is identified by the drop in the pressure to the esophageal baseline. From these measurements the pressure, abdominal length, and overall length of the sphincter are determined. To account for the asymmetry of the five radially oriented transducers, and the average values for sphincter pressure above gastric baseline, overall sphincter length, and abdominal length of the sphincter are calculated ***(Peters and De Meester, 1999).***

The intra-abdominal length of the esophagus is defined as the distance in centimeters between the point where gastric baseline pressure is recorded and the respiratory inversion point (i.e., the position where inspiratory pressure changes from a positive to a negative deflection as the manometric catheter is brought back from the abdomen into the negative pressure environment of the chest). The difference between gastric baseline pressure and the pressure at the respiratory inversion point is the mean lower esophageal sphincter (Fig. 8). Finally the total length of the lower esophageal sphincter is determined by measuring the length in centimeters of the high pressure zone between zones of gastric baseline pressure and esophageal body pressure ***(Peters and De Meester, 1999).***

The diagnosis of mechanically defective lower esophageal sphincter depends on three functional variables of the sphincter which determine its competency, these are:

a) Lower sphincter pressure.

b) Intra-abdominal sphincter length

c) Overall sphincter length

***(De Meester, and Crooks., 1998)***

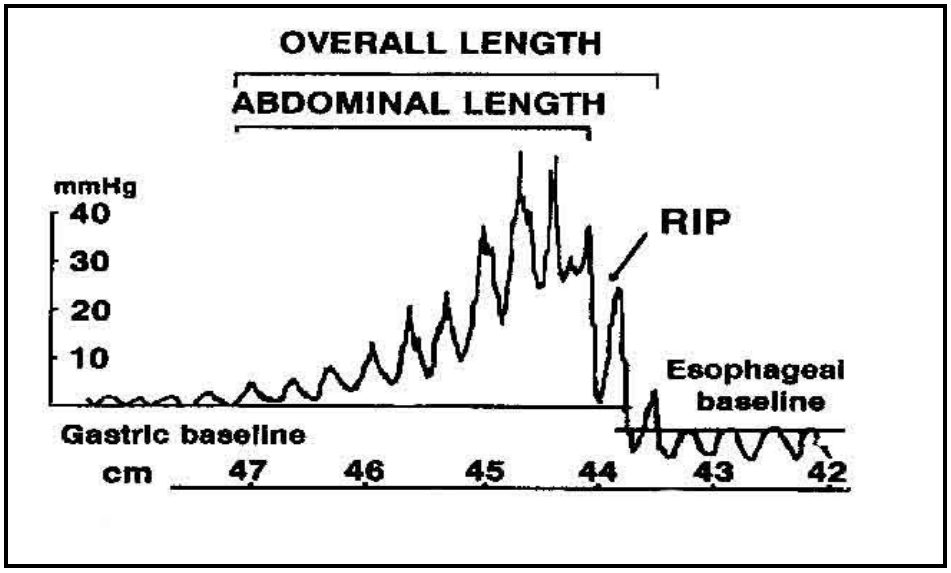



**Fig. (8):** Normal manometry tracing obtained by pulling the transducer through the gastro-esophageal junction from the stomach into the esophagus ***(Johnsson et al., 1993)***.

The principal uses of esophageal manometry in the contest of gastro-esophageal reflux disease include the accurate localization of the position of the lower esophageal sphincter

(LES) prior to pH monitoring, the exclusion of a primary motility disorder, such as achalasia and to provide information on lower esophageal sphincter and peristaltic function which may aid management. It is recognized that esophageal manometry is the most accurate way of identifying the position of the lower esophageal sphincter, in order that a pH probe may be placed 5 cm above it for ambulatory pH monitoring. The distinction between gastro-esophageal reflux disease and a primary motility disorder is usually relatively straight forward although, because of the high incidence of associated motility disturbances in gastro-esophageal reflux disease, dysphagia may be a symptom in up to 50% of cases referred for anti-reflux surgery. Furthermore, patients with achalasia frequently complain of reflux type symptoms and, indeed pH monitoring has shown that 40% patients with achalasia have pathological acid exposure. Manometry is the only reliable means of diagnosing achalasia and although the incidence of this in patients referred for anti-reflux surgery is relatively low, an inappropriate operation in such circumstances will inevitably lead to the need for revisional surgery and a dissatisfied patient ***(Farraro et al., 1995).***

While many patients with pathological reflux may have a low normal lower esophageal sphincter pressure (LESP), many such patients not required surgical treatment. Patients with most severe form of reflux have very low resting values of LESP of 6 mmHg or less, and a greater proportion of these patients will

require anti-reflux surgery as acid suppression is less effective in such patients, particularly if regurgitation is a predominant symptom. Impaired esophageal peristaltic function, as manifest either by low amplitude contractions or with greater than 50% of contractions being simultaneous and none propagated, is also associated with the more severe forms of reflux. Multiple regression analysis has shown a significant correlation between pre-operative manometric evidence of failed primary peristalsis and the incidence of mechanical complications, particularly dysphagia, following Nissen fundoplication. A subsequent study from the same centre conducted during the laparoscopic era showed that Nissen fundoplication in such circumstances was associated with an incidence of troublesome dysphagia of 6% one year postoperatively. As a results of these factors, many workers have advocated a "tailored" approach to anti reflux surgery in which the approximately 60% of patients with normal esophageal body motility undergo Nissen fundoplication, and the remainder undergo a partial fundoplication procedure ***(Hunter et al., 1996).***

Esophageal manometry is of great value in the assessment of patients with complex esophageal problems, particularly those who have undergone previous surgery. Such problems include recurrent reflux or obstructive symptoms following anti-reflux surgery and reflux symptoms or persistent obstructive symptoms following cardiomyotomy. In these circumstances, knowledge of whether the lower esophageal sphincter is hypertensive or has impaired relaxation on

swallowing and whether esophageal body function shows impaired peristalsis are crucial to the planning of corrective treatment ***(Baigrie et al., 1997).***

Esophageal manometry is however useful in the assessment of the propulsive activity of the body of the esophagus and in assessment of the results of anti-reflux surgical procedures ***(Cuschieri, 1995).***

## III. Evaluation of esophageal motility (Fluoroscopy)

The patients are studied using video fluoroscopy with an alternate 5c.c and 10c.c boluses of low viscosity barium suspension. These are injected orally and individual swallows are recorded. A total of 10 or more separate swallows are obtained. The radiology can accurately assess normal and abnormal esophageal motility when at least single swallows are used and careful fluoroscopic observations are performed. So the fluoroscopy and manometery are both excellent studies for identifying abnormal peristalsis. These tests provide complementary information since radiology assess bolus movement and the manometery provides quantitative pressure data ***(Baigrie et al., 1997).***

## IV. PH monitoring

### *1. Standard acid reflux test*

In this test an acid load (300 c.c. of 0.1 NHCl3) for an average sized adult is placed into the stomach. This is done when manometry is complete and the distal manometry catheter

is placed into the stomach with infusion of the acid. After flushing with water, the manometry catheter is withdrawn into the body of the esophagus and the pH electrode is positioned 5 cm above the top of high pressure zone as determined from the manometry tracing. PH is recorded continuously at this level while the patient is asked to perform a series of postural and respiratory maneuvers in sequence. These are performed in an effort to provide a uniform challenge to the cardia that can be compared from one patient to another ***(Jeffery and DeMeester, 1999).***

In comparison with the more extensive 24 hour pH monitoring test, the standard acid reflux test is some what less sensitive in detecting minor degrees of esophageal reflux. It proves more accurate than 24 hour pH monitoring in diagnosing reflux in patients with achlorohydria because the acid load in the stomach provides the necessary marker for recording reflux, but acid is absent during prolonged esophageal pH monitoring in patient with achlorohydria. This test remains valuable and in wide use as a short out patient maneuvers for detecting abnormal reflux as a possible cause of symptoms ***(Skinner and Belsey, 1988).***

### *2. 24 hour pH monitoring*

24 hour ambulatory pH monitoring is considered to be the most accurate means of diagnosing and quantifying GERD ***(Watson, 1997).***

Twenty four hours ambulatory pH monitoring has an important role to play in the assessment of atypical symptoms such as chest pain or respiratory symptoms (e.g. nocturnal wheezing and hoarseness), where reflux is suspected to be the cause of these symptoms ***(Koufman, 1991).***

The most direct method of measuring increased esophageal exposure to gastric juice is by an indwelling pH electrode. Prolonged monitoring of esophageal pH is performed by placing a pH probe 5 cm above the manometrically measured upper border of the distal sphincter for 24 hours ***(Jeffery and DeMeester, 1999).***

It is performed by passing a glass or antimony pH probe trans-nasally, so that it comes to lie 5 cm above the upper margin of the lower esophageal sphincter which has been identified manometrically. The pH probe is connected to a portable recorder, which the patient wears around the waist, which samples and records intra-esophageal pH over a 24 hours period. The patient is able to press an event marker in order to indicate the presence of symptoms, the time of consumption of meals and periods spent in the upright and supine postures. At the end of the 24 hours monitoring period, the data in the portable recorder are off loaded into a personal computer with appropriate software ***(Watson, 1997).***

The number of reflux episodes in which the pH dropped below 4, the time and duration of each episode and the proportion of overall time the pH was less than 4, subdivided into the upright and supine postures, is generated. These individual values may also be collated into an overall score, such as the DeMeester score, which gives greater weighting to parameters with more severe clinical consequences, such as reflux in the supine posture and prolonged reflux episodes, so that a single score is generated which facilitates clinical decision making for those less familiar with the details of pH monitoring ***(Johnsson et al., 1993).***

**The units used to express esophageal exposure to gastric juice are:**

1. Cumulative time the esophageal pH is below a chosen threshold, expressed as the percent of the total, upright, supine monitored time.
2. Frequency of reflux episodes below a chosen threshold, expressed as number of episodes per 24 hours.
3. Duration of the episodes, expressed as the number of episodes greater than 5 min/24 and the time in minutes of the longest episode recorded.

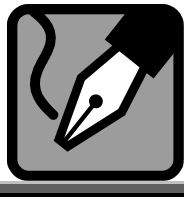
Although of all these, it is not 100% accurate and only detects a refluxate with a pH monitoring of 4 or less. the primary uses of prolonged pH monitoring are to evaluate

patients with atypical symptoms or an incomplete response to therapy and to assess patient before and after anti-reflux therapy ***(Jeffery and DeMeester, 1999).***

There is good correlation between the results of the prolonged ambulatory pH monitoring and severity of esophagitis at endoscopy. In addition, the results of the two investigations are complementary ***(Cuschieri, 2002).***

## V. Acid clearance test

This test is designed to assess the ability of the distal esophagus to clear an acid load by repeated swallowing; it correlates well with the presence of esophagitis. Motility of the esophagus is impaired with the onset of the esophagitis and a vicious circle is established with prolongation of the contact time between the acid and the inflamed esophageal epithelium. In performing this test, a PH probe is positioned 5 cm above the high pressure zone (HPZ) and a load of acid (15 ml of 0.1 NHCl3) is then instilled above the tip of the probe. The PH drops to about 1.4. Normal individuals are able to clear the acid in 3-10 swallows. Delayed clearance is diagnosed if the patient requires more than 10 swallows to achieve return to the esophageal PH to the pre-infusion level ***(Cuschierri, 1995).***



# TREATMENT OF GERD

# TREATMENT OF GERD

The primary goal in the management of gastro-esophageal reflux is to improve the quality of life in these patients by complete symptoms resolution and maintenance of symptoms control. Additional therapeutic end points include the healing of damage already incurred and prevent of long term complications of reflux ***(Sami, 2002).***

Effective treatment requires an awareness of the clinical spectrum of GERD, its varied symptomatology and potential complication and the many treatment options available. Every patient should be treated with the goal of achieving complete, long term symptoms relief ***(Kahriias, 2001).***

The treatment of GERD encompasses three phases dependent on the severity of the disease. These comprise general measures, specific medical treatment and surgical treatment ***(Watson, 1998).***

## I. General measures

Most patients with gastro-esophageal reflux have mild disease and are never referred to hospital. In these patients symptomatic relief is usually achieved by modification of lifestyle, which is an important adjuvant to all forms of therapy for GERD ***(Holloway et al., 1997)***.

Modification of life style include weight reduction if the patient is overweight, elevation the head of the bed if nocturnal symptoms are evident, stop smoking, avoid alcoholics, and the avoidance of excessive bending and stooping ***(Watson, 1998).***

Patients with gastro-esophageal reflux should be advised to take small frequent dry meals in order to minimize gastric distension and therefore, decrease transient lower esophageal sphincter relaxation. Patients should not eat any appreciable quantity of food before going to bed, and before engaging in activities such as bending and lifting that increase intra-abdominal pressure ***(Holloway et al., 1997).***

Specific dietary advises include the avoidance of fatty, spicy and acidic foods, fruit juices and spirits. Patients also must avoid certain foods which impair lower esophageal sphincter tone such as chocolate and coffee, tea, cola beverages ***(Holloway et al., 1997).***

## II. Medical treatment

The gastro-esophageal reflux disease is the most frequent problem seen in an esophageal clinic, so there are various medical regimens which are usually tried on a patient with symptoms of GERD before referral to a surgeon ***(DeMeester and Crooks, 1998).***

The basic aim of therapy is to relieve the symptoms and to prevent complications arising from the gastro-esophageal reflux. In patient with reflux symptoms but no macroscopic esophagitis the treatment is directed towards symptoms relief but in patients with macroscopic esophagitis however, the aim of treatment should be to heal esophagitis as well as to relief symptoms ***(Brain and Sampliner, 1997)***.

The medical treatment can be approached in tow different fashions. The correction of mechanisms involved in the pathogenesis of reflux includes a decrease in the frequency of transient lower esophageal sphincter relaxations and improvement of the esophageal clearance of the refluxate to minimize the esophageal exposure time. The more popular approach employs neutralization or suppression of intra-gastric acidity to render the refluxate less harmful to the esophageal mucosa. This approach provides a symptomatic relief and prevents mucosal injury in the majority of the patients ***(Sami, 2002).***

## Antacid:

Antacids have been the primary mode of therapy for GERD symptoms for centuries, but they have little impact on neutralizing the volume of gastric acid usually present in the stomach. Although, antacids are effective in neutralizing residual acid coating the esophageal mucosa, a rapid return of symptoms usually occur due to continued reflux of gastric acid.

Additionally, antacids fail to heal esophagitis, prevent strictures, erosions, or maintain the patient in remission. Thus, the role of antacids in GERD is limited to short term relief as an adjunctive therapeutic agent ***(Brain and Sampliner, 1997).***

## Mucosal coating drugs (esophageal cytoprotectors)

### *1. Sucralfate:*

Sucralfate is an aluminum hydroxide salt of sucrose octasulfate, which has been shown to form adherent complex with proteinacious material to act as an effective barrier against the action of acid, pepsin and bile. Such coating may occur during passage through the esophagus after swallowing or upon reflux. In addition, once sucralfate enters the stomach, the low intra-gastric pH may release sucrose sulphate by dissolution, which may also coat and protect the mucosa after episodes of reflux. Recently, sucralfate has been shown to stimulate endogenous gastric mucosal prostaglandins synthesis and may have a cytoprotective effect by augmentation of mucosal blood flow, mucous secretion and bicarbonate secretion. Mild constipation seems to be most common adverse effect experienced by patients receiving sucralfate treatment. Sucralfate has been administered in a dose of 1 gm four times a day ***(Holloway et al., 1998).***

### *2. Alginic acid:*

Alginate is a combination of alginic acid and antacid which is a popular agent used for treatment of heart burn.

Alginic acid reacts with sodium bicarbonate in saliva to form a highly viscous solution that floats on the surface of gastric pool, acting as a mechanical barrier. Alginic acid however, does not affect lower esophageal sphincter pressure. The symptomatic relief is greater than with antacid alone. Studies using intra-esophageal pH monitoring have demonstrated a significant decrease in the number of episodes and quantity of reflux after administration of alginic acid in dose of one tablet four times daily ***(Watson, 1998).***

## Acid suppressing drugs:

The currently most widely used principle of drug treatment in GERD is inhibition of acid secretion with $H_2$ receptor antagonist and proton pump inhibitor. The predominance of secretory inhibitors is based on the unsurpassed efficacy as well as rarity of clinically relevant adverse effect in short or long term treatment of all degrees of GERD ***(KlinkenbergKnol et al., 1995).***

### *1. $H_2$-Receptor blocker:*

$H_2$-blocking agents reduce both day time and nocturnal gastric acid secretion. They block the acid secretion induced by histamine, gastrin, cholinergic drugs and vagal stimulation. They have no effect on the lower esophageal sphincter pressure or the rate of gastric emptying. Mucosal lesions heal or improve in about 65% of patients after 6 weeks of therapy ***(Altman, 1998).***

$H_2$-receptors antagonists, such as cimetidine, ranitidine and famotidine were the main stay of pharmacological treatment for severe degrees of GERD prior to introduction of proton pump inhibitors. The effects on intra-gastric and intra-esophageal acidity and the healing rate of endoscopic esophagitis are dose dependent. Healing of endoscopic esophagitis at 2 months with ranitidine 300 mg twice daily is approximately 50%, but increasing the dose increase this proportion to 75%. Many patients with mild disease can achieve symptomatic control and endoscopic healing on appropriate dose of $H_2$-receptors antagonist ***(Watson, 1998).***

### *2. Proton pumps inhibitor (PPI):*

Proton pump inhibitors are the drug of choice in the treatment of GERD. They are effective in healing of acute esophagitis and may relieve patients from heartburn. However, due to complex patho-physiology of GERD, proton pump inhibitors fail to control it for long term, especially in the presence of large hiatal hernia, poor esophageal peristalsis, regurgitation of large volume, dysphagia and atypical or respiratory symptoms. Moreover, medical treatment does not prevent the development of Barrett's metaplasia effectively. Therefore, there is still the need for anti-reflux surgery in many GERD patients ***(Wykypiel et al., 2005).***

Proton pump inhibitors, such as omeprazole and lansoprazole, are much more effective agents for symptomatic

relief and for healing of esophagitis. Although, they are best in reducing heart burn, up to 20% of treated patient will have persistent esophagitis ***(DeMeester and Crooks, 1998).***

The proton pump inhibitors are the most potent inhibitors of gastric acid secretion which act by specifically inhibit the enzyme $H^+/K^+$-ATPase which is the final step in the formation of hydrochloric acid in the parietal cells. They produce more profound and long lasting suppression of acid secretion than $H_2$ receptors antagonist. Healing of endoscopic esophagitis occurs in 80-90% of cases using omeprazole 20-40mg daily for one month. The percentage of healing of low grade esophagitis (grad II) on 20 mg omeprazole daily, for 4 weeks was 81%, and with more severe esophagitis (grad III), healing at 8 week with 40 mg of omeprazole daily was only 56% ***(Sami, 2002).***

Long term administration of proton pump inhibitors in high doses is very expensive, whereas the cost of laparoscopic procedures has decreased as they have gained more wide spread acceptance ***(DeMeester and Crooks, 1998).***

## Efficacy of acid suppressing drugs:

Potent acid inhibitory drugs almost always lead to symptomatic and endoscopic healing of GERD. The major problem is keeping GERD in remission. Depending on the severity of GERD recurrence rates after cessation of the

treatment vary between approximately 30 and 80% within 6 month ***(Wilder-Smith et al., 1999).***

Healing of reflux esophagitis correlates directly with the acid inhibitory capacity of the drug used. Inhibition of intra-gastric acidity to a pH greater than 4 during approximately 22-24 hours leads to healing in nearly 90% of cases. Insufficient acid inhibition is the main cause of therapy refractory GERD ***(Holloway et al., 1996).***

On a given treatment, endoscopic severity of esophagitis prior to treatment is an important factor that has repeatedly been shown to predict slow healing. More extensive erosions need more time to heal than minor lesions. This negative influence can be contracted in most cases by adjusting the initial treatment according to the endoscopic severity of GERD ***(Barbier et al., 1999).***

It is difficult to achieve prolonged and profound acid inhibition with $H_2$ receptors antagonists because they have a short duration of action, the development of tolerance and incomplete inhibition of acid secretion in response to meal ***(Wilder-Smith and Merki, 1999).***

Recent trials indicate that, proton pump inhibitors e.g. Omeprazole are more efficient as a maintenance therapy for GERD. Repeated relapses during maintenance treatment can almost always be treated successfully with a temporary increase

of the daily dose. Patients with particularly severe esophagitis need 40-60 mg daily of omperazole for maintenance treatment, but the majority can be maintained with 20 mg daily for many years ***(KlinkenbergKnol et al., 1995).***

Symptoms relief usually accompanies healing of esophagitis; therefore, patients who respond well to treatment in general don't need re-endoscopy. Re-endoscopy is recommended, however, if the symptomatic response is insufficient and if complications have occurred ***(Robinsn et al., 1995).***

## Adverse effects:

Considering the tolerance in short and long term use, omperazole has an excellent record. The incidences of general adverse drug events with proton pump inhibitors are similar to those of the $H_2$ receptor antagonist. Side effects are almost due to acid inhibition**.**

As in pernicious anemia with achlorohydria, or after total gastrectomy, malabsorption of protein, iron and calcium are of no concern even during high dose omperazole treatment for extended periods. There is slightly decrease of vitamin $B_{12}$ absorption during omperazole therapy, probably due to insufficient release of food bound vitamin $B_{12}$ in the absence of acid, but the serum levels remained within normal limits ***(Koop and Bachem, 1998).***

One of the main important functions of gastric acid is its action as disinfectant of bacteria and parasites in contaminated food. Hydrochloric acid at a pH below 3 is bactericidal in vitro to many pathogenic bacteria. Not unexpectedly, omperazole treatment leads to gastric and duodenal bacterial overgrowth, but so far there is no evidence of an increased risk during treatment with acid inhibitors ***(Bompton et al., 1996).***

## Prokinetic agents (Motility modulating drugs):

The prokinetic agents have been used in the management of GERD on the basis that many patients have impaired peristaltic function and LES function. These agents are metochlopromide, domperidone, Bethanechol**.** They were shown to produce some symptomatic improvement but little firm evidence of healing of endoscopic esophagitis. The prokinetic agents aim in increase LES tone and esophageal body peristalsis or improving gastric emptying ***(Vignerie et al., 1995)***.

### *1. Dompridone:*

Domperidone is a dopamine antagonist. It stimulates the esophageal peristalsis, stimulates elevation of lower esophageal sphincter pressure and increase gastric emptying. As dompridone does not pass the blood brain barrier, the risk of extra-pyramidal side effects is low. Dompridone may produce galactorrhea and amenorrhea. Symptomatic but not objective

improvement has been shown in some patients with gastro-esophageal reflux who is treated by dompridone ***(Vigneri et al., 1995).***

***2. Metochlopromide:***

Metochlopromide is also a dopamine antagonist. It has shown to increase the amplitude of esophageal contractions and also increase the lower esophageal sphincter and improve abnormal gastric emptying in reflux patients. Symptoms improved but not the endoscopic findings. It has little therapeutic effect in GERD. Side effects such as, fatigue, anxiety, confusion, hallucination may occur and extra pyramidal side effect is present ***(Altman, 1998).***

***3. Bethanechol:***

Bethanechol is a cholinergic drug which tightens the lower esophageal sphincter and improves esophageal clearing function. Prolonged pH studies have shown that Bethanechol is effectively improving acid exposure in recumbent position and the drug may increase salivary flow and increase the amplitude of esophageal peristalsis. It is usually well tolerated thorough some patients complain of side effects such as, abdominal cramps, diarrhea, urinary frequency and blurred vision ***(Altman, 1998).***

## II. Surgical Management:

Surgical therapy, which addresses the functional nature of this condition, is curative in 85%-93% of patients and, thus, is more effective than medical therapy and also provides good long term results ***(Bammer et al., 2001).***

The aim of the technical procedure in antireflux surgery is to restore and anchor the esophago-gastric junction below the diaphragm to achieve an adequate length of intra-abdominal esophagus. In addition, mechanism must be created at the esophago-gastric junction to restore competency of gastro-esophageal/ lower esophageal sphincter mechanism. In this respect closure of the hiatus is not always needed, but hiatal repair and crural approximation is indicated if there is an associated hiatus hernia ***(Sami, 2002).***

Antireflux surgery may restore the antireflux barrier, may strengthens esophageal peristalsis, speeds gastric emptying and may improve gastric clearance function, thus counteracting duodeno-gastric reflux ***(Wykypiel et al., 2005).***

Therefore, antireflux surgery is effective in control of GERD in the long term and in the prevention of the development of Barrett's metaplasia and Barrett's carcinoma, consequently ***(Bammer et al., 2001).***

**Indications for antireflux surgery:**

The majority of patients with gastro-esophageal reflux disease will have limited periods plagued with episodes of reflux controlled by life style adjustments and a pharmacological agents with or without maintenance therapy. Approximately 10-15% of patients will be referred for consideration to have antireflux surgery. The indications for surgical treatment are as follows:

- Failure of medical therapy. This should be considered when the patient has received the appropriate medications at the appropriate dosage with the development of the side effects of the medication, persistence of symptoms, or inability to comply with the medications or life style adjustments. Disease progression despite appropriate medical therapy also falls under this category. The persistence of vomiting or regurgitation in patients with an incompetent gastro-esophageal sphincter and usually with a hiatus hernia is a strong indication for surgical intervention.
- Development of complications. This include ulcer, strictures, columnar metaplasia of the lower esophagus and secondary motility disorders of the esophagus. Many of these patients may be elderly and frail with comorbid disease and surgical intervention in this group is inappropriate. Young and fit patients who developed these complications should be considered for surgery. Patients

with high grade esophagitis should be included in this category from the outset although this is controversial.

***(Sami, 2002)***

- Persistence of reflux in children beyond the age of 2 years.
- Reflux after previous upper abdominal surgery, particularly alkaline reflux.
- Atypical symptoms of gastro-esophageal reflux including respiratory, pharyngeal and dental problems.
- Individualized assessment. This includes young patients who can only be maintained in remission by the administration of continuous and life long medications. While the consequences of this approach may have been overstated, the prospect of life long medication in young patients should not be underrated. Within this category, socioeconomic considerations are also important for both the state and the individual. It is estimated that the cost for maintenance therapy for 10 years using proton pump inhibitors far exceeds the costs of an anti-reflux procedure.

***(Sami, 2002)***

- Patients with large hiatus hernias (>4 cm) often complain of atypical symptoms, and moreover, these patients have a high risk for development of Barrett's metaplasia. Giant hernias (>50% of stomach herniated) are being discussed as

presenting an absolute indication for antireflux surgery, since there is a risk of gastric volvulus and incarceration with lethal outcome in these patients ***(Luketich et al., 2000).***

It is important to note that patients with good preoperative symptoms response to proton pump inhibitors (PPI) thereby were also more likely to respond well to antireflux surgery ***(Anvari and Allen, 2003).***

**Intractable esophagitis:**

The persistent endoscopic esophagitis despite of symptomatic improvement or in the presence of a mechanically defective lower esophageal sphincter is an indication for an anti-reflux operation to prevent stricture formation or the development of Barrett's esophagus and bring about healing of the esophagitis ***(McKcrnan and Champion, 1998).***

**Esophageal stricture:**

The development of a stricture in a patient with a mechanically defective lower esophageal sphincter represents a failure of medical therapy. There are two types of esophageal strictures. Mild strictures in which the transmural damage are relatively slight. Severe strictures which are characterized by transmural fibrosis, and often have poor distal motility and esophageal shortening**.**

Mild strictures respond well to dilation and laparoscopic fundoplication and are less to require subsequent dilation. Severe strictures usually require transthoracic approach to perform a combined esophageal lengthening and partial (Belsey) fundoplication ***(DeMeester and Crooks, 1998).***

**Barrett's esophagus:**

The presence of Barrett's esophagus as an indication for surgery by itself is controversial. Barrett's esophagus is associated with the most severe form of GERD. In these patients there are strong grounds for operating regardless of the presence of Barrett's epithelium. Recommending surgery purely because Barrett's esophagus is present to diminish the risk of subsequent malignancy continues to generate controversy ***(Flesher, 1992).***

Medical treatment of Barrett's esophagus without dysplasia using proton pump inhibitors reduces the acid exposure to the esophagus, and by inhibiting acid production reduce the volume of reflux material that include the bile. However, both acid and bile reflux into the esophagus while patients are on maintenance therapy, and in some patients the degree of esophageal exposure to gastric juice remains abnormal. There is also concern that raising the pH of the gastric environment allows the bile acids to remains in solution in a non polar form that is noxious to the esophageal mucosa**.**

Surgical treatment of these patients by fundoplication will correct pathological esophageal reflux of both acid and duodenal content. There is evidence that surgically treated patients are protected from the development of dysplasia and esophageal cancer. There is also evidence that an antireflux procedure in patients with Barrett's esophagus may induce partial or even complete regression of Barrett' epithelium but the impact of this regression on the subsequent development of adenocarcinoma is not clear. There is further evidence that de novo development of Barrett's esophagus is exceeding rare in patients who have had effective antireflux surgery. This is marked contrast to reports of long term medical treatment where up to 34% of patients on long term acid suppression developed Barrett's esophagus while on therapy ***(Cushieri, 2002).***

Nissen fundoplication effectively controls intestinal metaplasia and clinical symptoms in the majority of patients with Barrett's esophagus. Endoscopic surveillance showed regression of the macroscopic columnar segment in 62% of patients with Barrett's esophagus. Regression at a histo-pathological level occurred in 40% of patients ***(Ozmen et al., 2006).***

In a recent study it has been demonstrated that the progression of Barrett's metaplasia to carcinoma in patients

with continuous PPI treatment is twice as fast as antireflux surgery ***(Bammer et al., 2001)***.

**Pulmonary complications:**

The presence of pulmonary complication may indicate the need for antireflux surgery even in the presence of normal esophageal mucosa. The chronic atypical symptoms of reflux namely chest pain, chronic cough, recurrent pneumonia or episodes of nocturnal chocking, regurgitation of gastric contents in the mouth or soilage of pillow may also indicate the need for the surgical therapy ***(DeMeester and Crooks, 1998).***

## Preoperative evaluation:

Before proceeding with an anti-reflux operation several factors should be evaluated and these may lead to selection of a particular antireflux procedure and/or addition of an operation to the later in order to achieve best results these are:

1- Documentation of a mechanically defective esophageal sphincter on manometry.

2- The presence or absence of anatomical shortening esophagus and its degree.

3- Excessive duodeno-gastric reflux.

4- The presence of delayed gastric emptying.

5- Gastric acid hypersecretion.

6- Increased esophageal exposure to gastric juice evident on 24-hour esophageal pH monitoring.

7- Persistent or recurrent symptoms or complication after 8 to 12 weeks of intensive acid suppression.

The propulsive force of the body of the esophagus should be evaluated by esophageal manometry to determine if it has sufficient power to propel a bolus of food through a newly reconstructive valve. The patient with normal peristaltic contractions does well with a (360) ° Nissen fundoplication. When peristalsis is absent or severally disordered or of low amplitude, the partial fundoplication is the operation of choice and is better than a 360° Nissen fundoplication as the latter may lead to sever post operative dysphagia ***(Peters and DeMeester, 1999).***

**Anatomical shortening of the esophagus:**

This may lead to tension on the repair with a higher incidence of breakdown or thoracic displacement of the repair. Esophageal shortening is identified on a barium swallow by a sliding hiatal hernia that will not reduce in the upright position or that measure larger than 5 cm between the diaphragmatic crura and gastro-esophageal junction on endoscopy.

When esophageal shortening is present the motility of the esophageal body must be evaluated and if inadequate a gastroplasty should be performed but in patient who have a

global absence of contractility more than 5% interrupted or dropped contraction or there is a history of several failed previous antireflux surgery the esophageal resection is an alternative ***(Peters and DeMeester, 1999).***

**Excessive duodeno-gastric reflux:**

It is found in about one third of patient with GERD and may be responsible for complaints like epigastric pain, anorexia, nausea, and vomiting. This problem is most obvious in patients who have had previous upper gastro intestinal surgery, particularly cholecystectomy ***(Peter and DeMeester, 1997).***

This diagnosis of abnormal duodeno-gastric reflux is documented by 24-hour pH monitoring of the stomach and also by $TC_{99m}$-HIDA-scan. Antireflux surgery may reduce duodeno-gastric reflux by improving the efficiency of gastric emptying ***(Peter and DeMeester, 1997).***

**Delayed gastric emptying:**

Delayed gastric emptying is found in approximately 40% of patients with GERD and can contribute to symptoms after an antireflux repair. Usually, however, mild degrees of this problem are corrected by the antireflux procedure and only in patients with severe emptying disorders there is a need for an additional drainage procedure like pyloroplasty or anterior gastroenterostomy ***(Cushieri, 1995).***

**Gastric acid hypersecretion:**

Approximately 30% of patients with proven gastro-esophageal reflux on 24-hour pH monitoring will have hypersecretion on gastric analysis and 2-3% of patients who have antireflux operation will develop a gastric or duodenal ulcer.

The additional of highly selective vagotomy may be required in patients with documentation of active ulcer disease or those with previous ulceration ***(Peters and DeMecster, 1999).***

The success of antireflux procedure depends on achieving the combination of:

1. Relief of symptoms.
2. Objective evidence on 24 hour esophageal pH monitoring that reflux has been reduced to physiological level.
3. Evidence on esophageal manometry that the mechanical defect of the LES has been corrected.

Thus, patients should be encouraged to undergo esophageal manometry and pH monitoring 6 months, 1 year and 3 years after an antireflux procedure ***(Stein and DeMeester, 1993).***

## Antireflux operations available for treatment of GERD

**Total fundoplication procedure:**

- Nissen fundoplication.
- Floppy Nissen fundoplication.
- Rossetti-Hell fundoplication.

**Partial fundoplication procedure:**

- Toupet fundoplication.
- Hill posterior gastropexy.
- Watson fundoplication.
- Belsey Mark IV procedure.
- Collis gastroplasty.

**Prosthetic insertion:**

- Angelchick prosthesis.

***(Attila et al., 2005)***

Total fundoplication procedures include the Nissen fundoplication, devised by Rudolph Nissen in Basle in 1956 and the Rossetti-Hell modification. There are several partial fundoplication procedures including the Belsey, Hill, Toupet, Watson and Lind procedures. Prosthetic insertion, principally using the Angelchick silicon prosthesis, is now so rarely performed because of the unacceptable high re-operation rate

for complications, predominantly dysphagia and the migration of the prosthesis ***(Sami, 2002).***

***Principles of Surgical Therapy***

The primary goal of antireflux surgery is to safely re-establish the competency of the cardia by mechanically improving its function while preserving the ability of the patients to swallow normally, to belch to relieve gaseous distention and to vomit when necessary. Regardless of the choice of the procedure this goal can be achieved if attention is paid to five principles in reconstructing the cardia:

***First:*** The operation should restore the pressure of the distal esophageal sphincter to a level twice resting gastric pressure, and its length to at least 3 cm.

***Second:*** The operation should place an adequate length of the distal esophageal sphincter in the positive pressure environment of the abdomen by a method that ensures its response to changes in intra-abdominal pressure. The permanent restoration of 1.5 to 2cm of abdominal esophagus in a patient whose sphincter pressure has been augmented to twice resting gastric pressure will maintain the competency of the cardia over various challenges of intra-abdominal pressure, thus the creation of a conduit that will ensure the transmission of intra abdominal pressure changes around the abdominal portion of the

sphincter is a necessary aspect of surgical repair. The fundoplication in the Nissen and Belsey repairs serves this purpose**.**

***Third:*** The operation should allow the reconstructed cardia to relax on deglutition. In normal swallowing a vagally mediated relaxation of the distal esophageal sphincter and the gastric fundus occurs. The relaxation lasts for approximately 10 seconds and is followed by a rapid recovery to the former tonicity**.**

***To ensure relaxation of the sphincter three factors are important:***

(a) Only the fundus of the stomach should be used to buttress the sphincter since it is known to relax in concert with the sphincter.

(b) The gastric wrap should be properly placed around the sphincter and not to incorporate a portion of the stomach, or no to be placed around the stomach, otherwise the stomach does not relax with swallowing.

(c) Damage to vagal nerves during dissection of the thoracic esophagus should be avoided because it may result in failure of the sphincter to relax**.**

***Fourth:*** The fundoplication should not increase the resistance of the relaxed sphincter to a level that exceeds the peristaltic power of the body of the esophagus.

***Fifth:*** The operation should ensure that the fundoplication can be placed in the abdomen without tension and maintained by approximating the crura of the diaphragm above the repair. Leaving the fundoplication in the thorax converts a sliding hernia into a para-esophageal hernia with all the complications associated with that condition. Maintaining the repair in the abdomen under tension predisposes to an increase incidence of recurrence; this occurs in patients who have a stricture or Barrett's esophagus and is probably due to shortening of the esophagus from the inflammatory process. This problem can be resolved by lengthening the esophagus by gastroplasty and constructing a partial fundoplication.

***(Jeffery and DeMeester, 1999)***

**Procedure Selection:**

Selection of the surgical procedure and approach is based upon an assessment of esophageal contractility and length. A transabdominal approach is used in patients with normal esophageal contractility and length. Patients with poor contractility or questionable esophageal length are approached transthoracically. Those with weak esophageal contractions and/or abnormal wave progression are treated with a partial fundoplication in order to avoid the increased outflow resistance associated with a complete fundoplication

***(DeMeester and Crooks, 1998).***

Nissen fundoplication produces a significant long lasting increase in esophageal contractile pressure in patients with a preoperative esophageal dysmotility. Preoperative esophageal dysmotility is therefore not an absolute contraindication to laparoscopic Nissen fundoplication ***(Biertho et al., 2006).***

If the esophagus is short after it is mobilized from diaphragm to aortic arch, a Colli's gastroplasty is done to provide additional length and avoid placing the repair under tension**.**

In the majority of patients who have good esophageal contractility and normal esophageal length the laparoscopic Nissen fundoplication is the procedure of choice for a primary antireflux repair**.** Experience and randomized studies have shown that the Nissen fundoplication is an effective durable antireflux repair with minimal side effects that provides long-lasting relief of reflux symptoms in 90 percent of patients ***(DeMeester and Crooks, 1998).***

A transthoracic approach is preferred for obese patients with a large hiatal hernia, significant esophageal shortening or stricture and patients who require repeated antireflux surgery ***(Henderson , 1998).***

The improved esophageal exposure afforded by this

approach facilitates creation of tension-free repair if significant peri-esophagitis or shortening is present. A Collis esophageal lengthening procedure can be done along with fundoplication to restore a tension-free intra-abdominal length of esophagus and to create a wrap around non inflamed tissue ***(Henderson, 1998).***

## Nissen fundoplication

It is the most common antireflux procedure which is usually done via the abdominal route, although it can be done via thoracotomy, and more recently through a laparoscope. It is a 360° wrap of the gastric fundus around the distal esophagus for a distance of 2-3 cm. Although this provides a good control of reflux but, it was associated with a number of side effects which have encouraged modifications of the procedure as originally described ***(Attila et al., 2005).***

Classical Nissen fundoplication, as described in 1956, has undergone several technical modifications in the past 48 years. However, the main principles of this operation have been maintained:

1. Preparation of the hiatal and fundic region by dissection of the esophago-gastric junction better through right posterior approach.
2. Preservation of vagal branches.
3. Division of the proximal short gastric vessels.
4. Closure of the hiatus.

5. Construction of a total $360^0$ symmetric fundoplication.

If these principles are adhered, late clinical results will be favorable ***(Attila et al., 2005).***

**Transabdominal open Nissen fundoplication (Fig. 9, 10):**

It is performed through an upper midline abdominal incision. Excellent exposure of the esophageal hiatus is paramount performing an open procedure. This can be achieved by utilizing a specialized retractor constructed by welding a weinbergy retractor to a Balfour handle. This retractor is placed under the liver down to the esophageal hiatus**.**

The operating table is placed in an anti-trendlenberg position (Fig. 9). This elevates chest wall and lifts the liver out of the way. The wound is retracted laterally with a Balfour retractor. Without this exposure, careful dissection of the hiatus is difficult, time consuming and dangerous ***(Jeffery and DeMecster, 1997).***

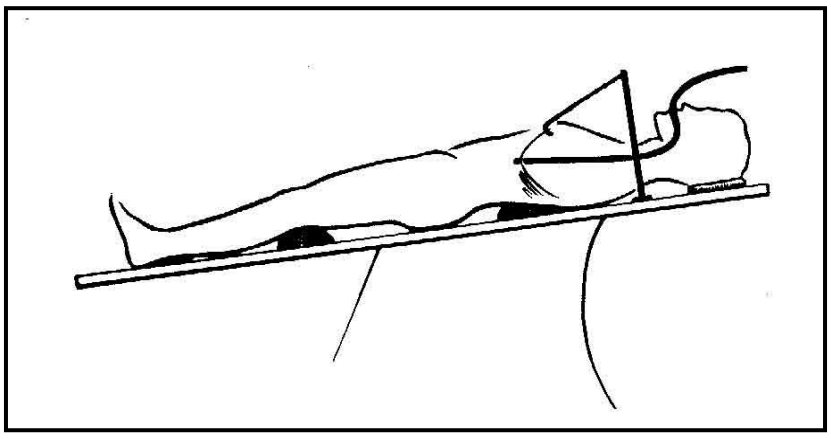


**Fig. (9):** Position of the reflux operations ***(Jeffery and DeMecster, 1997)***

The esophageal hiatus is approached by dividing the gastro-hepatic ligament in the area where it is thin and transparent. The esophagus is dissected circumferentially within the posterior mediastinum by blunt finger dissection. The procedure is continues with mobilization of the gastric fundus by dividing the short gastric vessels. The proximal third of the greater curvature is freed ***(Jeffery and DeMecster, 1997)***.

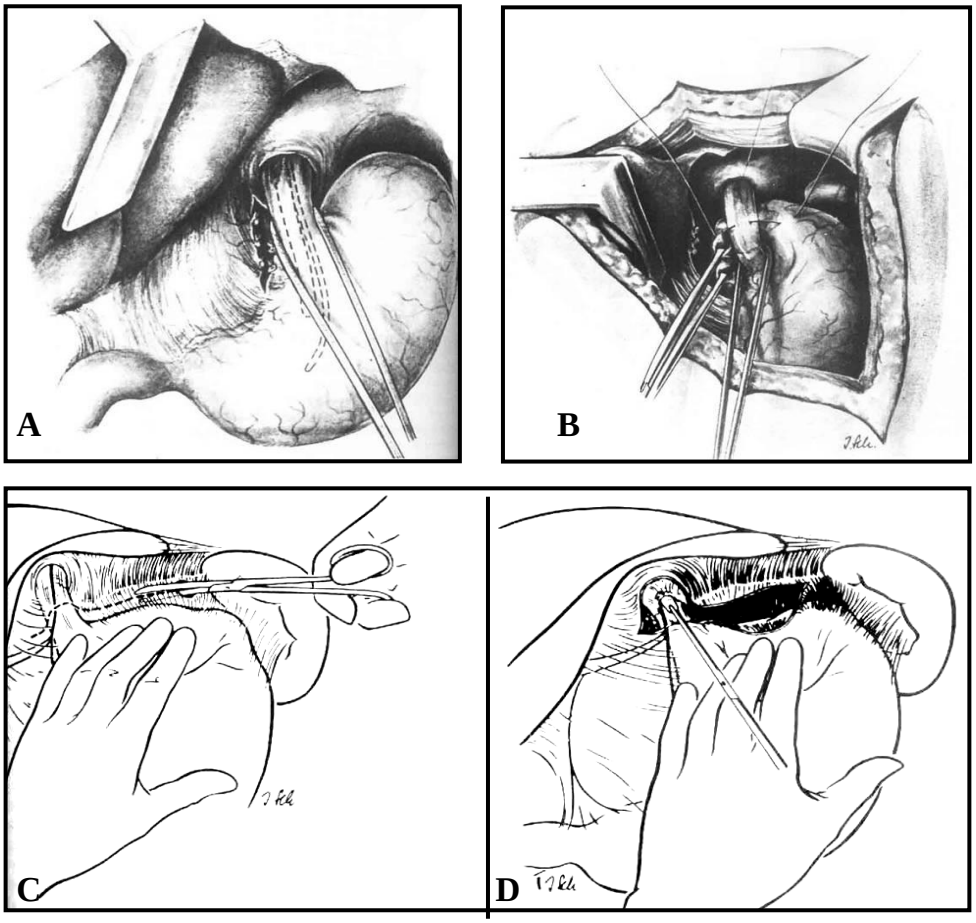
Construction of a satisfactory loose Nissen fundoplication is feasible in most patients without short gastric vessels division. Despite prolongation of the operative time short gastric vessels division provides a better symptomatic and physiologic outcome ***(Gad El-Hak et al., 2005).***

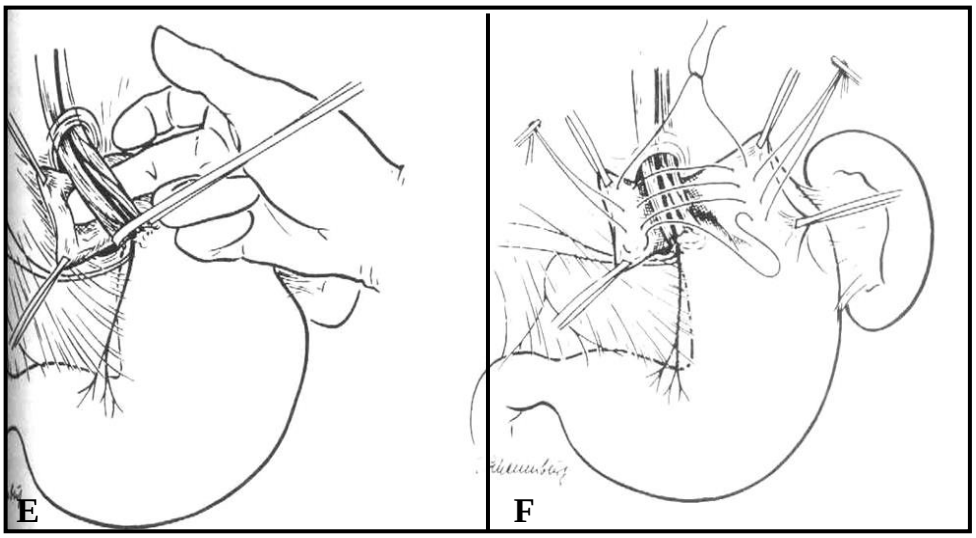
The esophageal hiatus is narrowed by retracting the esophagus to the left, and approximating the right and left crura with interrupted nonabsorbable sutures. Care is taken not to place the upper most sutures on the right side in the fascia of the diaphragm, since this will result n a constriction of the hiatus and dysphagia. When complete, the hiatus should freely admit a fingertip adjacent to the esophagus**.**

The freed posterior wall of the fundus is pulled between the right vagal trunk and the posterior wall of the esophagus. The anesthesiologist passes a 60-French bougie into the stomach and the anterior wall of the fundus is pulled a cross the anterior wall of the esophagus.

This results in enveloping the distal esophagus between the anterior and posterior fundic wall. The suture is tied with a

single throw to approximate the two lips of the fundic wrap around the esophagus constraining the 60-French bougie. If the surgeon is unable to insert his finger, the wrap is too tight, and the left end of horizontal U stitch must be replaced more laterally and inferiority on the anterior of the fundus. If there is excessive space, the wrap is too floppy and the left end of the U stitch must be replaced more medially and superiorly on the anterior wall of the fundus. When the wrap is of proper size the limbs of U-stitches are tied and the bougie is removed ***(Jeffery and DeMeester, 1997).***





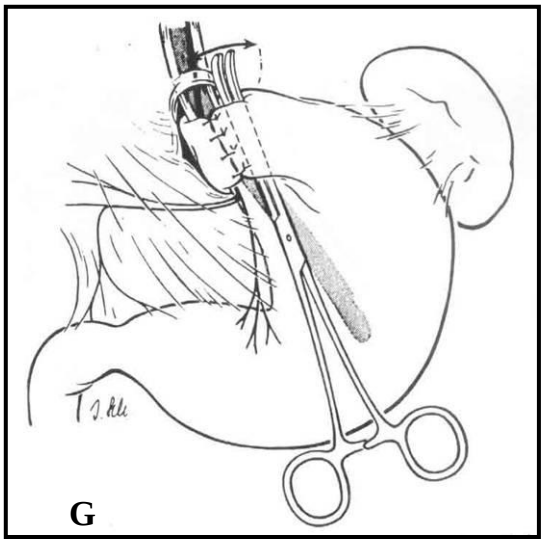




**Fig. (10):** Transabdominal open Nissen fundoplication
***(DeMeester and Jeffery, 1999)***

## Laparoscopic Nissen fundoplication:

Laparoscopic Nissen fundoplication has in the last few years been compared with open fundoplication, and several

studies have shown them to have similar patient outcome, both short term and long term. However, some reports have demonstrated better outcomes with laparoscopic procedure in terms of lower morbidity and better patient satisfaction ***(Raphael et al., 2005).***

Minimal access antireflux and hiatal hernia surgery has become increasingly established throughout the world. The minimal access rout confers the benefits of reduced post-operative pain, early recovery and discharge from the hospital, and minimum morbidity ***(Sami, 2002).***

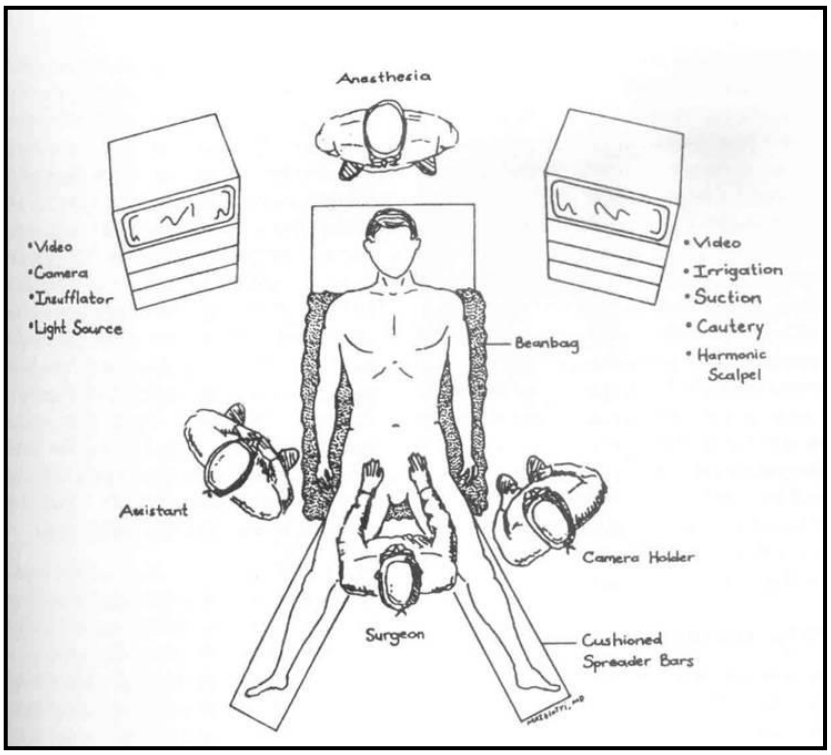



**Fig. (11):** Operating room layout for laparoscopic antireflux surgery ***(Williams et al., 1999).***

**Technique for laparoscopic Nissen fundoplication:**

The laparoscopic approach has become the standard procedure in anti-reflux surgery. For this purpose the patients is placed in the lithotomy position and brought into a reverse trendlenberg position with an angle of approximately $25^0$. The surgeon stands between the legs of the patient (Fig. 11). We place five trocars into the abdominal cavity (Fig. 12). The hepatogastric ligament is then divided along the upper part of lesser curvature of the stomach, which allows for the access to the right crus of the hiatus. The peritoneal layer over the right and over the left crura is dissected. Both crura are then gently dissected off the esophagus. After this procedure, it is possible to gain access into the mediastinum mainly by blunt dissection. The esophagus is mobilized far up into the mediastinum so that the gastro-esophageal junction comes to lie in the abdominal cavity without any tension. Usually approximately 5 cm of intra-abdominal length of the esophagus can be obtained. Then the hiatal crura are approximated. We do that with a 12 mm bougie in the esophagus and close the hiatus so that the hiatal crura are snugly attached to the esophagus ***(Wykypiel et al., 2005).***

The short gastric vessels and all attachment of the gastric fundus to the left crus are usually divided and then a contiguous parts of the gastric fundus is pulled behind the esophagus and brought to its right side for construction the fundic wrap. Suture of

the fundic wrap is accomplished with a sex points U shaped stitch buttressed by one Teflon bledge on each limb of the wrap. This stitch incorporates the esophagus, which may prevent the slippage of the stomach through the fundic wrap. Two additional simple fundo-fundic sutures that do not include the esophagus are placed above and below U stitch in order to obtain accurate approximation of both limbs of the fundic wrap. For all suturing 2-0 Prolene or 2-0 Ethibond is usually applied. The use absorbable sutures should be avoided, since it may result in a breakdown of the fundic wrap ***(Wykypiel et al., 2005).***

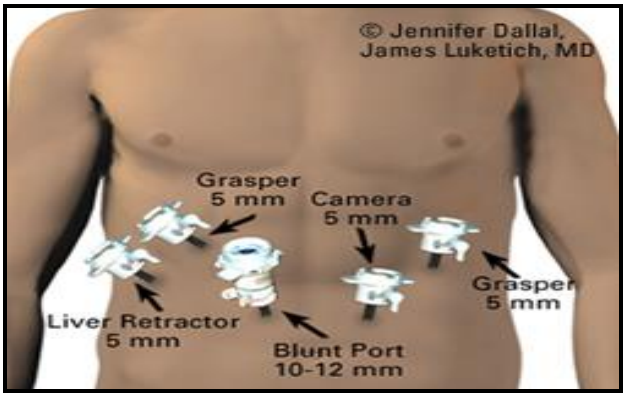
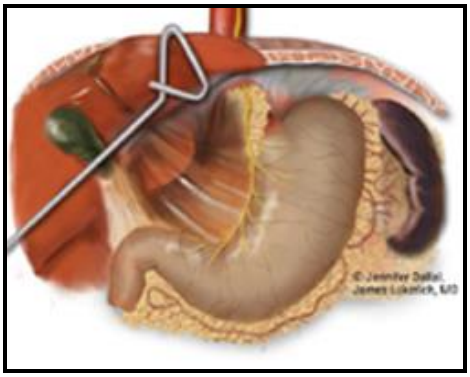
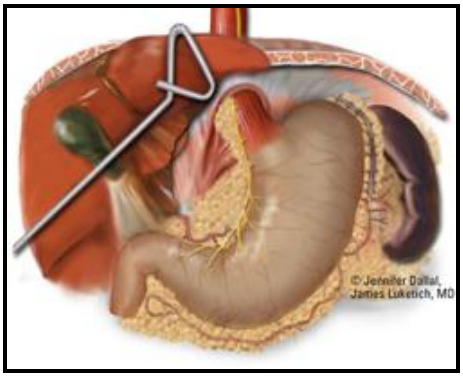


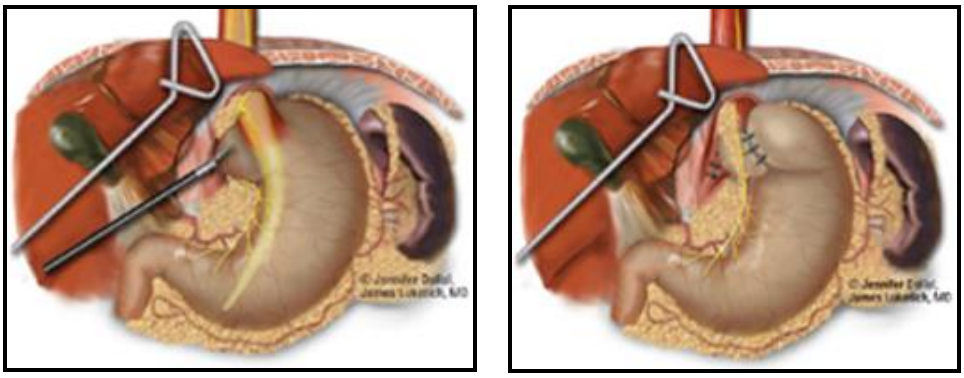

**Fig. (12):** Ports locations for laparoscopic antireflux surgery ***(Williams et al., 1999).***



**A**

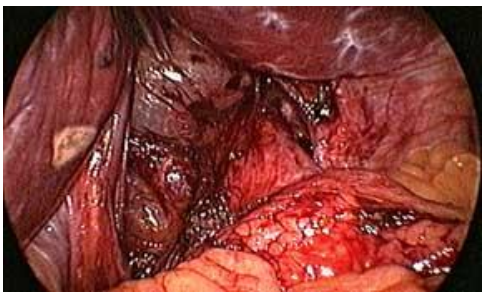


**B**

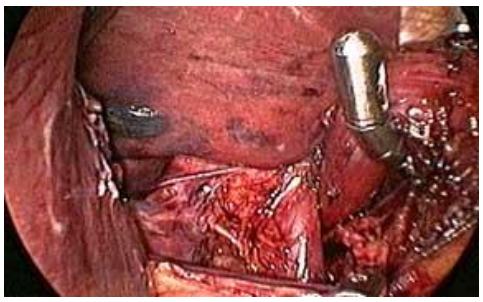


**C** **D**

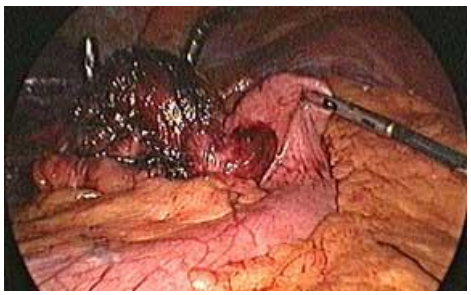
**Fig. (13):** Laparoscopic Nissen fundoplication
***(Jeffer and DeMeester, 1999)***



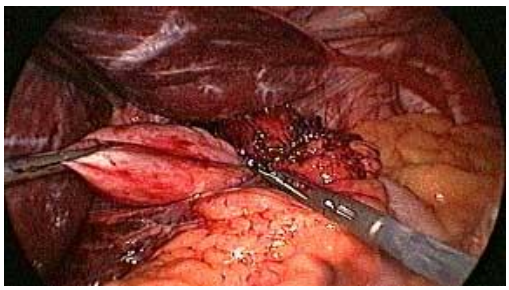
**A:** The oesophagus has been completely freed from the crura of the diaphragm. The vagus nerves have been isolated and identified



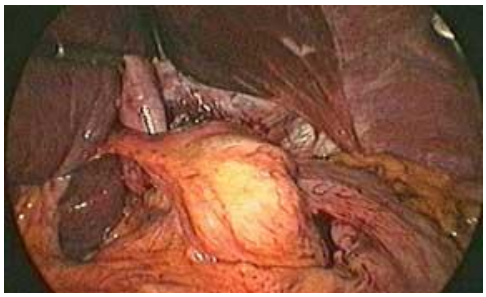
**B:** The malleable hook has loaded the oesophagus from left to right, passing in front of the posterior vagus nerve. This window, through which the stomach valve has to pass, can be suitably enlarged by inserting another hook from the right as well and opposing it to the first. The gastric fundus has already been fully mobilized



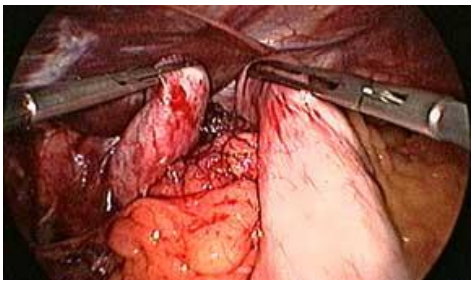
**C:** While the hook holds the oesophagus upwards, the operator passes the forceps in his left hand behind the oesophagus to grasp the already mobilised gastric fundus (phreno-fundal reflection and, generally, 1-2 short vessels divided), which is brought in by another forceps operated by the assistant.



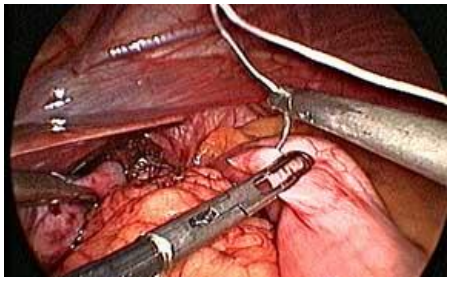
**D:** The forceps has withdrawn the gastric fundus behind and to the right of the oesophagus. This passage must take place without exerting force and the fundus must stay in position



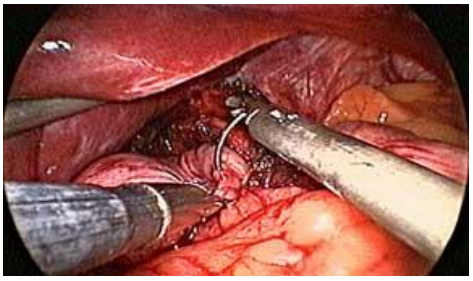
**E:** A probe is pushed into the stomach to calibrate the cuff. The authors say that the calibre ranges from 40 to 60 Fr



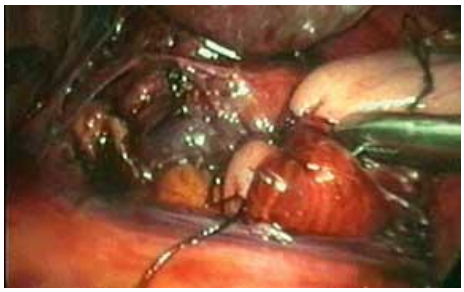
**F:** With the probe in position, correct gastro-gastric alignment is verified and, if required, mobilisation of the fundus is completed.



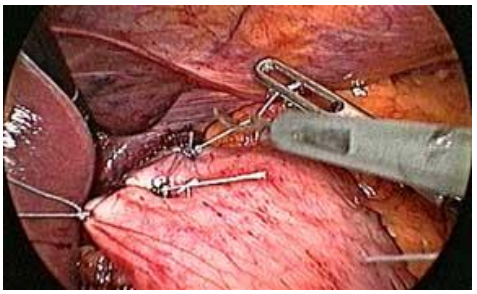
**G:** The most cranial stitch in the plasty is placed first. The more stitches there are on the outside part of the cuff, the wider the fundoplication will be, the stomach wall in direct contact with the oesophagus remaining abundant. The left edge of the stomach valve is loaded by the stitch



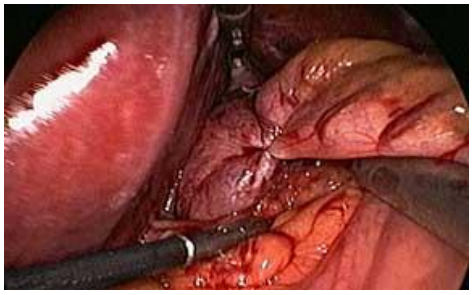
**H:** The stitch, as envisaged in the original technique, is about to transfix the muscle wall of the oesophagus. Care must be taken not to damage the anterior vagus nerve.



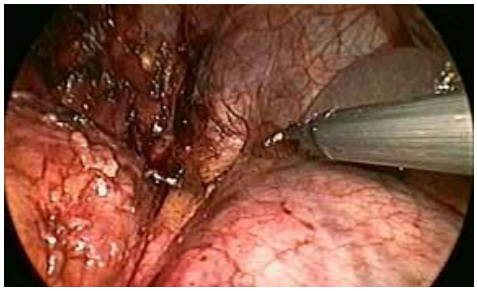
**I:** In particular, one notes the grip on the oesophageal muscle system. This is to prevent the oesophagus and the stomach from rising inside the gastric cuff



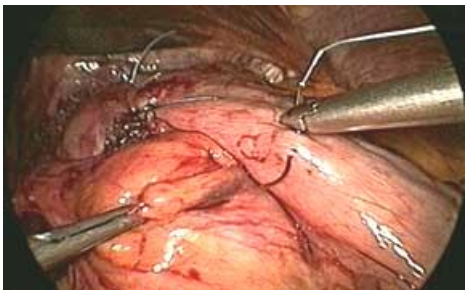
**J:** The plasty must be floppy and short. Generally speaking, three stitches one centimetre apart are sufficient. Indeed, a rigid, unidirectional valve should not be made, but rather, a dynamic anti-reflux mechanism which hampers reflux as intra-gastric and intra-abdominal pressure increases but does not prevent vomiting and belching



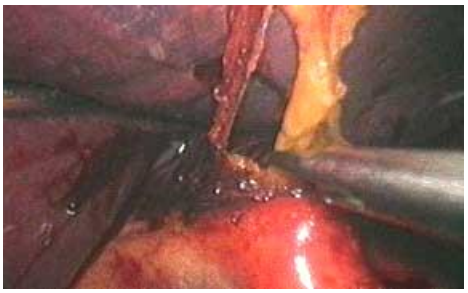
**K:** Further calibration consists in passing a 10-mm exploratory probe inside the cuff with the endo-oesophageal tube still in place. The probe should be inserted without force being exerted.



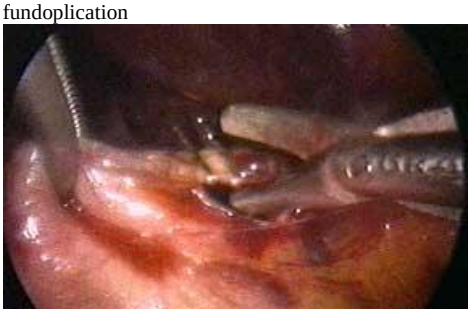
**L:** Some details and variations in the technique: incision of the peritoneal phreno-fundal reflection as far as penetration into the retroperitoneum in order to mobilise the gastric fundus. This stage is essential in the Rossetti technique, which envisages respect of the short vessels.



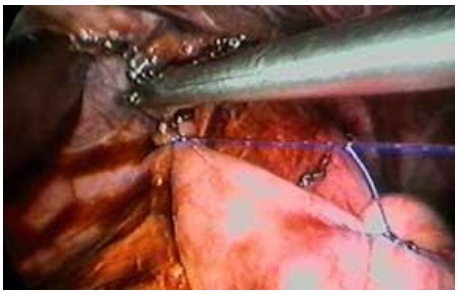
**M:** Some details and variations in the technique: the so-called Rossetti stitch, which fixes the anterior wall of the stomach to the inferior edge of the left part of the anti-reflux cuff. This stitch, if the oesophagus is not transfixed with the cuff sutures, is indispensable for preventing the stomach from slipping into the fundoplication



**N:** Some details and variations in the technique: in the case of voluminous hernias, one frequently encounters, generally between the right crus and the oesophagus, hernial lipomas, even large ones, which should be resected before the plasty is created



**O:** Some details and variations in the technique: a short vessel has been isolated, grasped between clips and is about to be divided. Freeing and dividing the short vessels is more convenient if one starts from the point chosen on the greater curvature and proceeds towards the fundus.



**P:** Some details and variations in the technique: some authors suggest that the most cranial part of the right part of the anti-reflux valve should also be fixed to the diaphragm so as to avoid the entire cuff from slipping into the mediastinum.

**Fig (14):** Technique of laparoscopic antireflux surgery
***(Jeffer and DeMeester, 1999)***

**Transthoracic open Nissen fundoplication:**

The esophageal hiatus is approached transthoracically through a left posterolateral thoracotomy incision in the sixth intercostal space. This approach allows better exposure of the abdomen through the diaphragm incision. When necessary the diaphragm is incised circumferentially 2-3 cm from the chest wall for a distance of approximately 10-15 cm an adequate rim of diaphragm must be preserved for approximation of the muscle ***(Brian and Sampliner, 1997).***

**The first step:**

Mobilize of the esophagus from the level of the diaphragm to underneath the aortic arch. Care for vagal nerves must be taken. Mobilization up to the aortic arch is usually necessary to place the reconstructed cardia of a shortened esophagus into the abdomen without tension (Fig. 15) ***(Peters and DeMeester, 1997).***

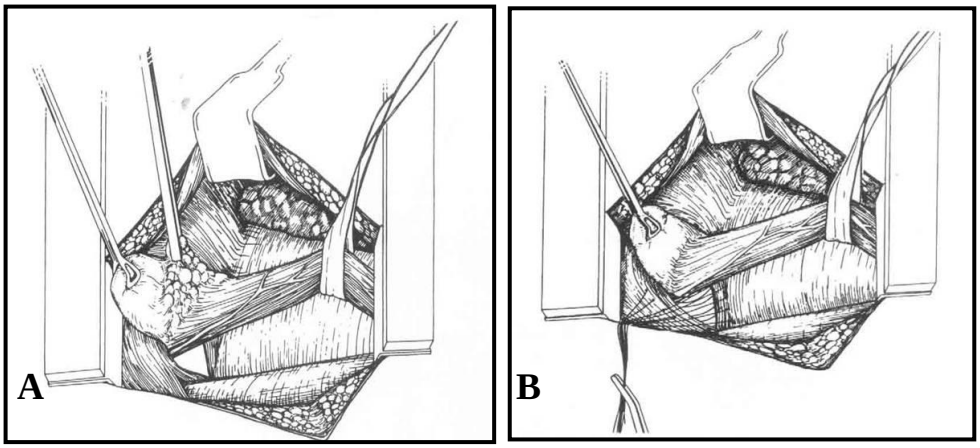



**Fig. (15):** A transthoracic mobilization of the stomach and esophagus ***(Peters and DeMeester, 1997).***

**The second step (Fig. 16A):**

Free the cardia from the diaphragm. When all the attachments between the cardia and diaphragmatic hiatus are divided, the fundus and part of the body of the stomach are drown up through the hiatus into the chest**.**

**The third step (Fig. 16B,C):**

The fundoplication constructed by enveloping the fundus around the distal esophagus in a manner similar to abdominal approach. Crural sutures are then placed to close the hiatus.

**Fourth step (Fig. 16D):**

The fundoplication is placed into the abdomen by compressing the fundic ball with the hand and manually maneuvering it through the hiatus and the last step is to tie the crural stitches.

***(Peter and DeMeester, 1999)***

The Nissen fundoplication gives good cure of symptoms of gastro-esophageal reflux and heals esophagitis effectively. To decrease the postoperative symptoms and also to increase the durability of the wrap, total fundoplication has been modified to the floppy Nissen fundoplication with mobilization of the fundus with a large-bore dilator within the esophagus during plication has been suggested ***(Luostarinenc and Isoluri, 1999).***

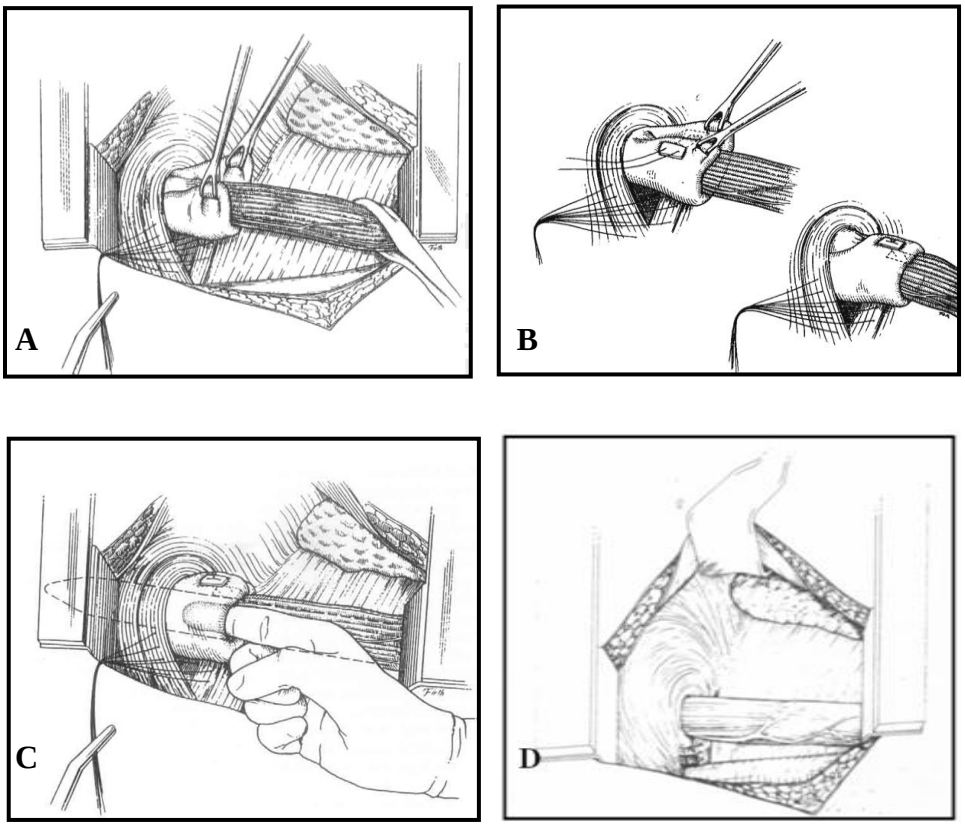



**Fig (16):** Transthoracic open Nissen fundoplication
***(Peter and DeMeester, 1999)***

## Nissen-Rossetti fundoplication:

In most large series reported, laparoscopic Nissen fundoplication was the most common performed procedure, although there have been various modifications, most notably the Rossetti modification where the short gastric vessels are not routinely divided. Laparoscopic Nissen Rossetti fundoplication theoretically ease dissection, and minimize the risk of intra- and postoperative complications, such as hemorrhage ***(Donald et al., 2006).***

A common criticism of the Rossetti technique however is that by avoiding division of short gastric vessels, an adequate, floppy fundic wrap could not be reliable created, which would result in increased incidence of postoperative dysphagia, gas blot, and/or recurrent GERD secondary to undue wrap tension with early breakdown. Although some retrospective studies claim a lower incidence of above complications after division as opposed to preservation of short gastric vessels, prospective randomized trials have failed to show such a difference ***(Donald et al., 2006).***

## Floppy Nissen fundoplication:

This procedure is a modification of Nissen and is described in order to solve the problem of supercompetent lower esophageal sphincter causing gas bloating and dysphagia after Nissen fundoplication. The procedure is designed to create a circumferential 1-2 cm loose wrap of gastric fundus around the mobilized abdominal esophagus ***(Bancewicz, 2000).***

The length of fundoplication is near 4-5 cm, because that it is the normal length of the lower esophageal sphincter. Many surgeons prefer to perform a wrap of 2 to 3 cm length, which decrease the incidence of post operative dysphagia ***(Attila et al., 2005)***.

### Operative technique:

After the right crus of the diaphragm is dissected, the short gastric vessels, including posterior short gastric vessels, are divided (Fig. 18). The pancreatico-gastric and phreno-

gastric ligaments are divided (Fig. 19). Further mobilization of the stomach off the diaphragm allows a creation of a window behind the esophagus. A penrose drain is passed around the distal esophagus to facilitate mobilization of the posterior stomach during subsequent maneuvers. The crural defect is closed using interrupted 0 Ethibond sutures. A 60 Fr bougie is passed into the stomach to assure the proper tightness of the crural closure and then pulled back into the distal esophagus**.**

A Babcock clamp is placed through the window behind the gastro-esophageal junction to the left of the esophagus. An assistant then grasp the fundus 4.5 cm distal to the angle of His and 2 cm posterior to the stumps of the ligated short gastric vessels. This portion of the fundus is given to the Babcock clamp and pulled behind the esophagus as the right posterior wing. It is then released to see whether the tension on it is sufficient to return it to its former location. We refer to this maneuver as the "drop test". If the grasped portion of the fundus remains in place after being released, it is considered appropriate for formation of the fundoplication.

The left anterior fundus is grasped, and both expected limbs of the fundoplication are pulled sequentially to determine the continuity. This is the "shoe shine" maneuver (Fig. 20). If continuity is not established, further fundic mobilization is necessary. In this case, the usual problem is that the pancreatico-gastric ligament has not been divided ***(Richard et al., 2004).***

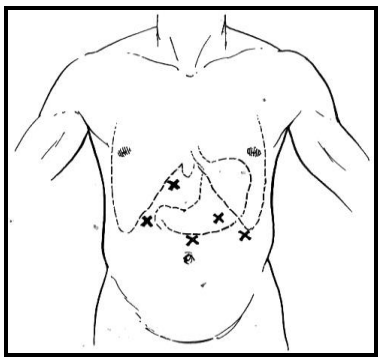


**Fig. (17):** 5 trochars for laparoscopic floppy Nissen fundoplication

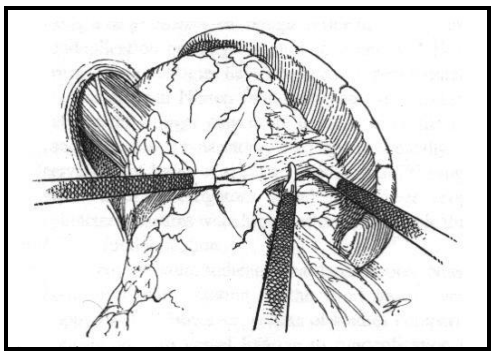


**Fig. (18):** The short gastric vessels, including the posterior vessels, are completely divided using a harmonic scalpel

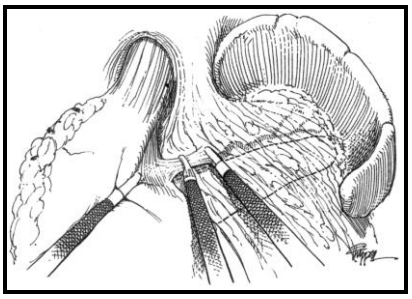


**Fig. (19):** The posterior attachments of the stomach including the pancreaticogastric and phrenogastric ligaments are divided

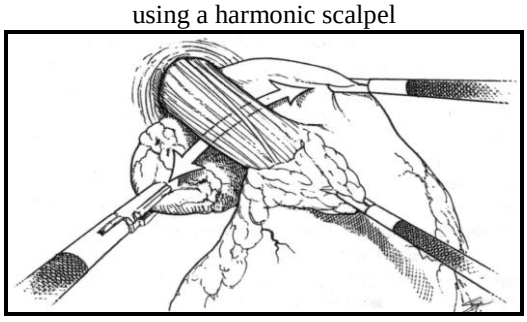


**Fig. (20):** The Shoe Shine maneuver

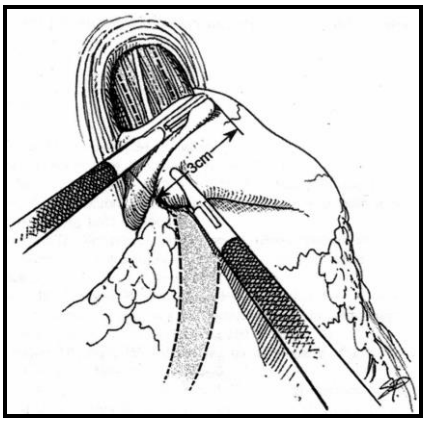



**Fig. (21):** The left wing of the fundoplication is passed to the right to overlap the right wing by 3 cm.

***(Richard et al., 2004)***

The penrose drain is then removed, and a 60 Fr bougie is passed into the stomach with anterior and caudal traction on the esophageal fat pad to prevent perforation. The final step is to pass the anterior left wing of the plication to the right, to overlap the right wing by 3 cm, while simultaneously applying pressure to grasping forceps holding the lead points (Fig. 21). If this step is not possible, a different portion of the anterior fundus is selected for the left wing of the plication. Care is taken to use only fundus because a wing created too low on the anterior body will lead to the "two compartment" stomach and a poorly relaxing fundoplication. A 2-0 prolene U stitch is used for the fundoplication ***(Richard et al., 2004).***

## Toupet fundoplication:

It comprises a 180-270° posterior partial fundoplication with fixation of the wrap to each limb of the crural sling over the length of 2-3 cm to achieve maintenance of the cardia within the abdomen, distal fixation of the cardia and to ensure an acute angle of implantation (Fig. 22). The partial fundoplication Ensures the LES function during raised intra-gastric pressure ***(Watson, 1998).***

It is performed by full mobilization of the abdominal esophagus and esophago-gastric junction. After the right vagus is freed from the esophagus, the fundus and upper part of the posterior wall of the stomach are first anchored to the right and left crura by interrupted sutures before the partial ($270^0$) fundoplication is fashioned by suturing the stomach to the

anterior wall of the esophagus on either side of anterior vagus nerve. The fixation of the partial wrap to the crura is important and serves two objectives. It prevents herniation through the hiatus and abolishes any drag on esophageal sutures ***(Sami, 2002).***

The disadvantage of this operation lies in the two rows of sutures placed longitudinally on the anterior wall of the esophagus which pulls in opposite directions favoring disruption especially in the presence of significant peri-esophagitis where there is a tendency for suture to cut out ***(Hennessy, 2000).***

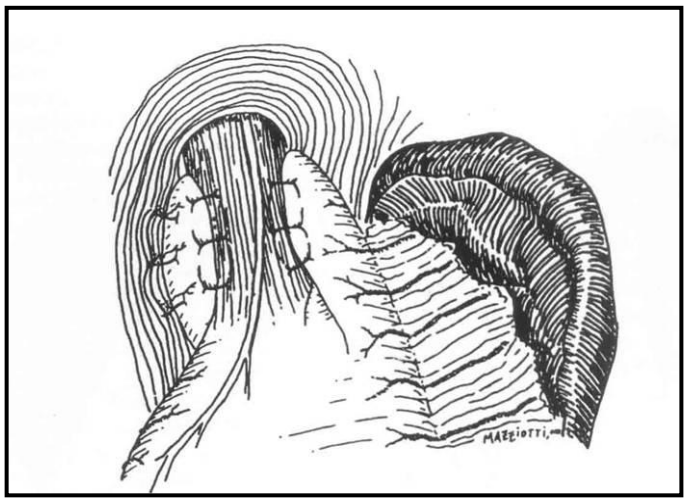



**Fig (22):** Toupet fundoplication ***(Hennessy, 2000).***

## Belsey Mark IV procedure (Fig. 23):

Aside from individual preferences and experiences, most general surgeons prefer the abdominal route and use the Nissen

procedure or one of its modifications. Fundoplication may, however, be difficult in obese patients, in those with dense adhesions due to previous upper abdominal surgery and those with a narrow subcostal angle and the Barrel shaped chest with a deep subdiaphragmatic region. In these patients, thoracic approach using Belsey mark IV procedure is a better and save procedure ***(Sami, 2002).***

Belsey mark IV operation is indicated in the presence of altered esophageal motility, where the propulsive force of the esophagus is not sufficient to overcome the outflow obstruction of complete fundoplication. It consists of a 270° anterior fundoplication around the distal 4 cm of the esophagus performed through a left thoracotomy incision. To perform the Belsey mark IV partial fundoplication the esophagus is mobilized up to the aortic arch, the cardia is dissected free of the hiatus, and the fundus of the stomach is brought up through the hiatus. The partial fundoplication is held in place by two rows of three horizontal matters sutures placed between the seromuscular layer of the stomach and the muscular layer of the esophagus. A second row of sutures is placed 1.5 to 2.0 cm above the first row. The diaphragmatic sutures are placed at the 4, 8 and 12 O'clock position on a lock face. The reconstructed cardia is gently pushed through the hiatus and placed in the abdomen. Once in the abdomen, the cardia should remain there without tension. The diaphragmatic and crural sutures are then tied, anchoring the wrap in the abdomen ***(Peters and DeMeester, 1997)***.

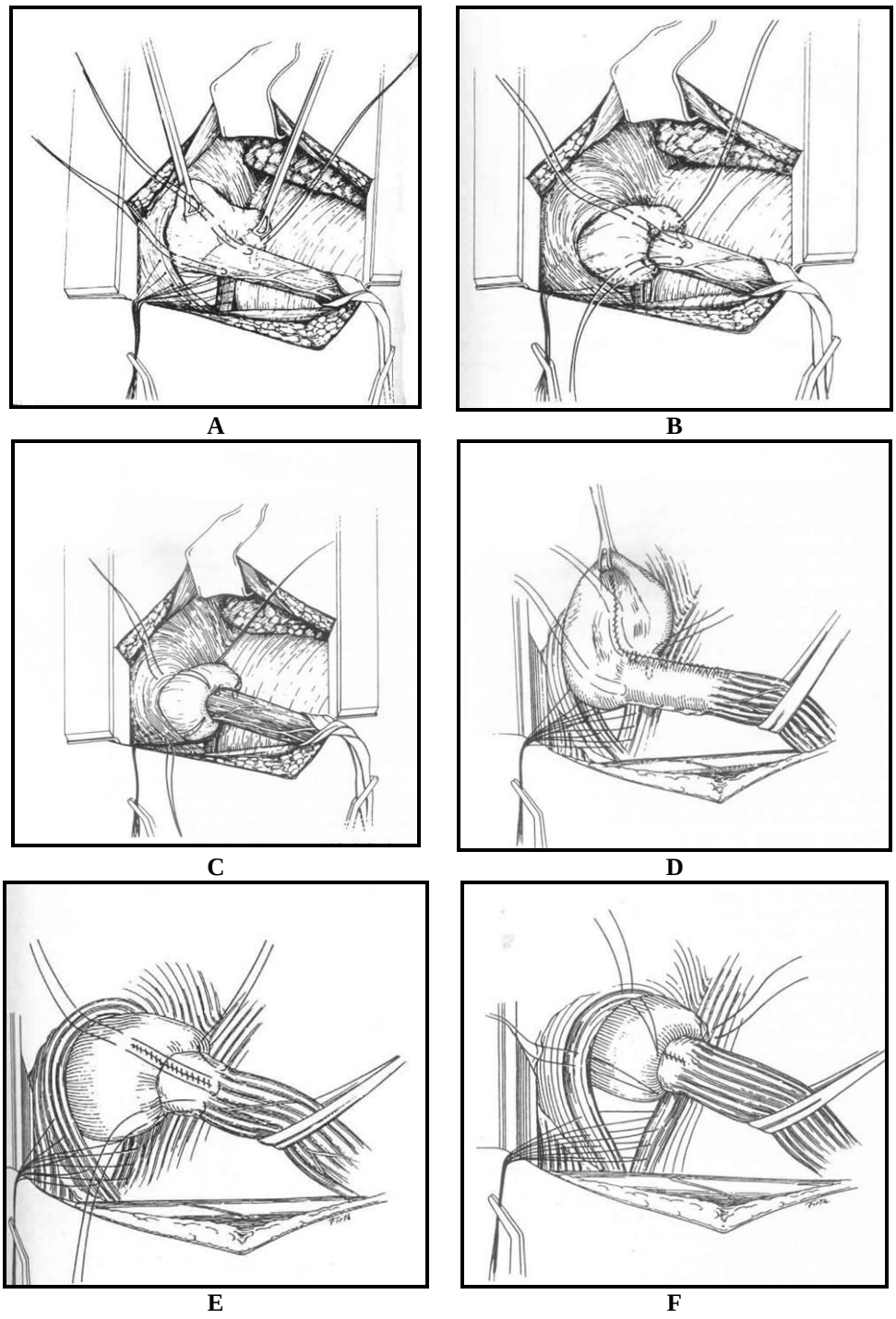



**Fig (23)**: Belsy Mark IV procedure ***(Peters and DeMeester, 1997).***

The disadvantage of the Belsey mark IV procedure is that it is performed through a left thoracotomy, as its originator believed that this was the only way of achieving a long intra-abdominal segment of esophagus. As a consequence, it is not a popular procedure now because of the greater metabolic insult, increased incidence of respiratory complications, prolonged hospital stay and convalescence and the risk of post-thoracotomy pain, together with the availability of other effective transabdominal procedures ***(Watson, 1998).***

## Collis gastroplasty:

This operation was devised for patients with early stricture and shortening of the esophagus such that its lower end cannot be replaced within the abdomen despite adequate mobilization, also performed for Barrett's esophagus or a large hiatus hernia ***(Cuschieri, 2002)***.

The gastroplasty will lengthen the tubular esophagus (Fig. 24), followed by construction of a tension free partial or complete fundoplication around the gastric tube with placement of the repair in the abdomen. It should be combined with a fundoplication and the Belsey Mark IV partial fundoplication is better because of the absence of peristalsis in the gastric tube ***(De Meester and Crooks, 1998).***

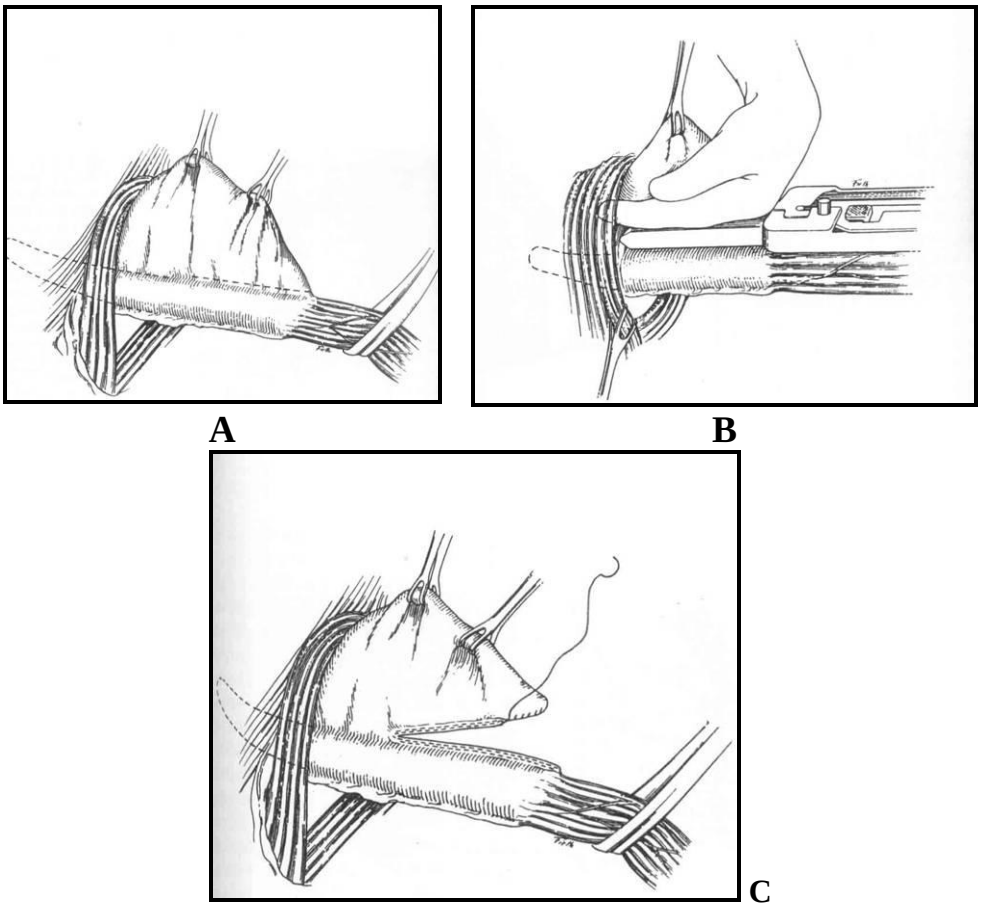



**Fig (24):** Collis gastroplasty ***(Cuschieri, 2002)***

## Hill posterior gastropexy (Fig. 25):

Laparoscopic posterior gastropexy presents comparable results to those reported after Nissen fundoplication and therefore could be another excellent therapeutic option in patients with reflux esophagitis ***(Braghetto et al., 2005).***

The principal of the operation is to create a good length of intra-abdominal esophagus by securing the uppermost portion of the lesser curve to the median arcuate ligament ***(Kramer et al., 1997).***

After mobilization of the lower esophagus, the celiac axis is identified and the median arcuate ligament overlying the aorta is dissected. Crural repair is performed with non-absorbable sutures so that hiatus is narrowed to an orifice, which admits one finger along with the esophagus**.**

The gastropexy is achieved by 2-3 plicating sutures, which pick cardio-esophageal junction in front, and behind the esophagus on the medial side, to the median arcuate ligament. When these sutures are tied, approximately $180^0$ of the distal esophagus is included in a partial gastric wrap, which is fixed firmly to the arcuate ligament and preaortic fascia. Hill employs an esophageal manometry catheter and advocates the intraoperative monitoring of pressures during the repair to assure that a satisfactory narrowing of the cardia has been achieved ***(Sami, 2002)***.

The hill procedure is an effective transabdominal procedure but its principal disadvantages are that it is technically difficult to perform with risk of damage to the celiac axis and celiac plexus and the intra operative manometry is necessary ***(Watson, 1998).***

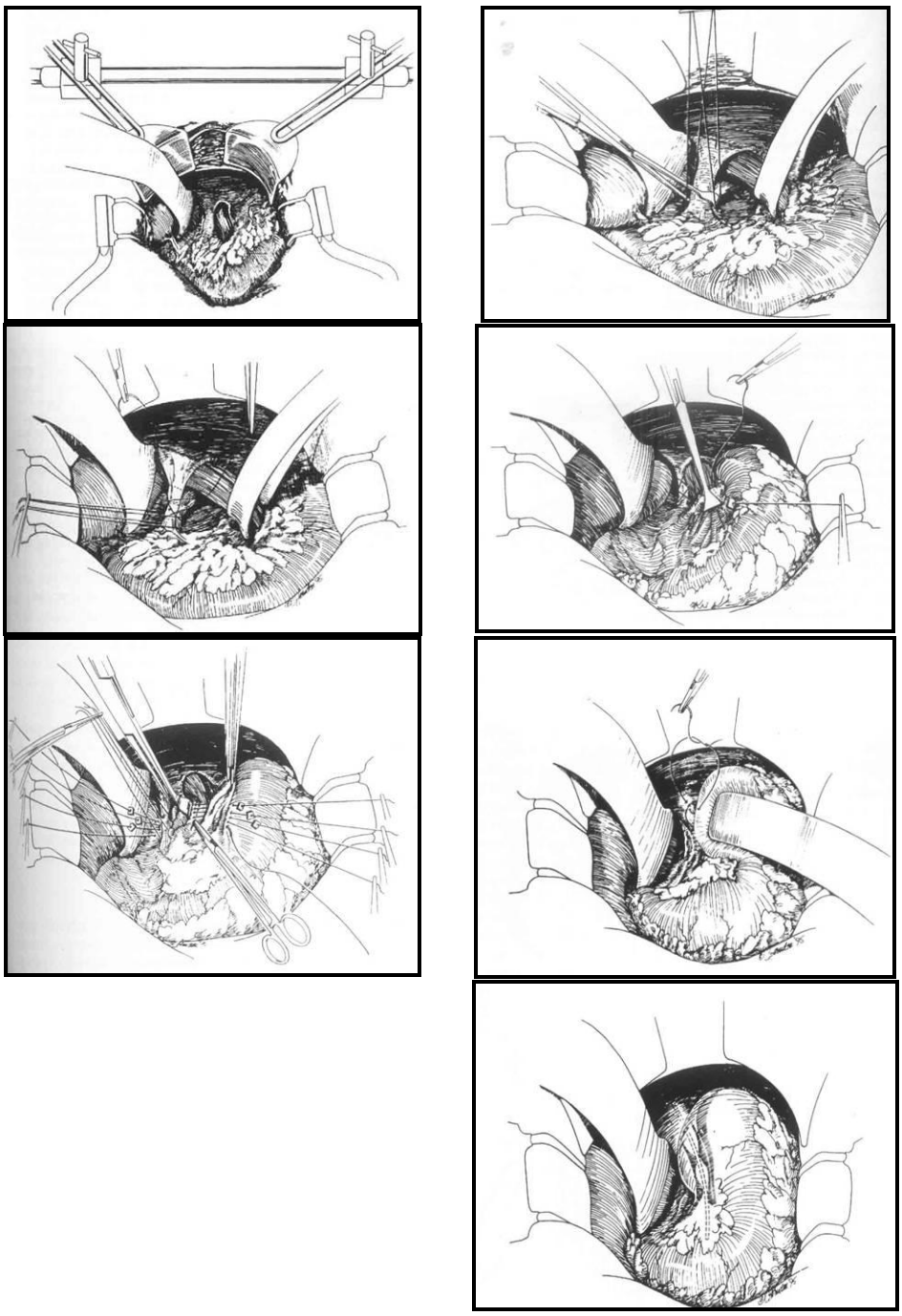


**Fig (25):** Hill posterior gastropexy ***(Watson, 1998)***

## Watson-Dor "physiological anterior-lateral fundoplication":

This procedure was designed with the specific aim of correcting and accentuating each of the known physical factors relevant to the anti-reflux mechanisms and minimizing the fundoplication to 120° in an attempt to provide effective reflux control whilst preserving physiological LES function, in order to obviate mechanical complications ***(Watson and Norris, 1994).***

The procedure involves firstly creation of a 5 cm segment of intra-abdominal esophagus by blunt transhiatal dissection. The limbs of the crural sling are then opposed routinely for three reasons. **First:** Because of the known influence of the crural sling on competence, its diminished influence in reflux patients and its restorability by crural repair, **Second**: It provides an easily accessible structure to which the long intra-abdominal segment can be fixed and maintained. **Third:** To provide a posterior buttress for the intra-abdominal segment during increased intra-gastric pressure. The angle of implantation is rendered extremely acute to enhance the valvuloplasty effect, and finally the 120° anterolateral fundoplication procedures augmentation of LES function during gastric distension ***(Peck et al., 1995)***.

This procedure has been studied in considerable detail and, at operation, when the stomach is distended by insufflating air down a nasogastric tube, the fundus is seen to distend so

that the whole anterior aspect of the intra-abdominal segment is covered by gastric fundus and is buttressed against the crural repair. When studied manometrically, LES characteristics including resting pressure, intra-abdominal length and, particularly, relaxation on swallowing are comparable to physiological levels, unlike after Nissen fundoplication, which is believed to account for the extremely low incidence of mechanical complications associated with the "physiological" repair. Because of the limited nature of the fundoplication, neither insertion of a bougie nor division of the short gastric vessels is required ***(Watson, 1998).***

## Angelchick prosthesis:

The prosthesis consists of an incomplete annualr silicon gel-filled implant with a Dacron tape at either end. These are tied together after insertion and a clip is applied to the ligated tapes to prevent slippage. A radiopaque marker within the tape allows postoperative radiological localization of the device. The procedure requires minimal mobilization of the esophagus, just enough to enable insertion of the implant and indeed, excessive mobilization favors migration of the prosthesis which weights some 45 gm ***(Stuart et al., 2000).***

The most common displacement is distal displacement which results in gastric obstruction and erosion through the wall of the gastro-esophageal junction is a serious complication but is fortunately rare. 8-10% of patients have initial dysphagia

with persistence of the distressing symptoms ***(Stuart et al., 2000).***

The advent of silicon in a medical graft brought about the manufacture of prosthesis in a material well tolerated by body tissues and fluids. The original prosthesis was followed by serious complications, probably made more common by unnecessary dissection and mobilization of the esophagus ***(Maxwell and Armastrong, 1997).***

The prosthesis is a foreign body and should not be used in the presence of sepsis or where there is a possibility of contamination from gastro-intestinal tract. Sepsis may lead to abscess formation and erosion of the prosthesis into the lumen of the stomach or esophagus, this is an extremely rare, but well documented complication ***(Angelchick, 1993).***

## COMPLICATIONS OF ANTI-REFLUX SURGERY

1- Dysphagia and gas bloat syndrome (result of super competent fundoplication).

2- Inappropriate position of fundic wrap (slipped Nissen).

3- Vagal nerve injury.

4- Splenic or pancreatic injury.

5- Gastric ulceration and ischemia due to iatrogenic para-esophageal hernia.

6- Recurrent gastro-esophageal reflux.

Antireflux surgeries are currently associated with low mortality of 1 % or less, which is mainly related to comorbid factors, but variable incidence of complications, although this is higher if a transthoracic procedure is performed ***(Pope, 1999).***

***I) perioperative complications:***

These include, splenic injury which may necessitate spleenectomy in about 10% following total fundoplication. Wound infection, wound dehiscence, respiratory complications, gastric stasis after injury of the vagus nerve, deep vein thrombosis and pulmonary embolism. Esophageal perforation may occur when encircling the esophagus is attempted by blunt dissection.

Nissen fundoplication is most frequently associated with mechanical complications because of the completely circumferential wrap, the degree of tension which may be associated with this and its effect on lower esophageal sphincter, particularly the ability to relax on swallowing ***(Rogers et al., 1999).***

Incarceration of the wrap may occur either because the crura are approximated too tightly or when vigorous attempts are made to bring the wrap bellow the diaphragm ***(Shirazzi et al., 1997).***

### *II) Delayed (Functional) complications:*

Most patients who have undergone Nissen fundoplication for gastro-esophageal reflux disease will have gastrointestinal complaints during the initial 3 postoperative months. Early satiety, bloating\ flatulence and dysphagia were the most common. Nearly all of these patients will have resolved their symptomatology after 3 months. Those with persistent symptoms after 3 months warrant evaluation of operative failure ***(Frantzides et al., 2006).***

### 1. Dysphagia:

Transient dysphagia occurs in 40% to 70% of patients after Nissen fundoplication. This is though to be secondary to edema at the gastro-esophageal junction (GEJ) or transient esophageal hypomotility. Fortunately, dysphagia usually resolves spontaneously within 2 to 3 months. Persistent dysphagia (PD), however, occurs in 3% to 24% of patients after Nissen fundoplication. The laparoscopic approach may be associated with a higher rate of Persistent dysphagia than its open counterpart**.**

The precise etiology of persistent dysphagia is unclear. The construction of tight, slipped or displaced fundoplication, peptic stricture, impaired esophageal clearance, and unrecognized achalasia are believed to be the primary causes of persistent dysphagia. The role of short gastric vessels division

and fundic mobilization in the avoidance of persistent dysphagia remains controversial ***(Kazuyoshi et al., 2002).***

Anvari et al demonstrated that chronic persistent dysphagia patients had a higher post-operative lower esophageal sphincter (LES) basal and nadir pressure than patients without post-operative dysphagia ***(Anvari and Allen, 1998).***

Herron et al found that pre-operative manometric analysis was similar among patients with persistent dysphagia (PD) and their control group ***(Herron et al., 1999).***

Some authors identified pre-operative difficulty of swallowing as the sole risk factor for PD. Others, however, found that 68% of laparoscopic Nissen fundoplication patients with pre-operative dysphagia noted an improvement in swallowing function post-operatively ***(Kazuyoshi et al., 2002).***

It is important to note that postoperative dysphagia occurred just as often in patients with preoperative proven motility disorders as in patients with normal preoperative esophageal motility. Patients with preoperative motility disorders who underwent a Nissen procedure did not develop dysphagia symptoms any more often than patients with normal preoperative motility. Thus the hypothesis of the tailored concept that antireflux surgery should be tailored to the individual based on the status of his or her preoperative

esophageal motility so as to avoid postoperative dysphagia could not be proven ***(Zoring et al., 2002).***

**2. The gas bloat syndrome:**

The gas bloat syndrome characterized by postcibal epigastric discomfort and distension, More common after total fundoplication due to supercompetent wrap which traps swallowed air in the stomach**.**

It occurred in up to 25% of patients post operative and can be diminished by placing esophageal dilator in position during performing the procedure ***(Dallemague et al., 1996).***

Gas bloat is the uncomfortable sensation caused by the inability to belch or vomit following antireflux surgery. It can result when the fundus is wrapped too tightly around the distal esophagus, when undue tension exists on the wrap because of failure to completely mobilize the greater curvature, or when inadvertent injury to vagus nerve occurs. The problem may be magnified by frequent aerophagia, a primary or secondary disorder of gastric employing, spasticity or scoliosis. Symptoms of gas bloat include nausea, retching gagging, and abdominal fullness that may or may not be temporarily related to eating. In most patients, the problem is self limiting improving in several weeks to a few months. Rarely, however, the onset of symptoms can occur several months postoperative. It has been reported that one third of patients continue to manifest

symptoms 4 to 9 months after surgery suggesting that the problem may never completely resolve ***(Pope, 1999).***

The occurrence of the mechanical complications, predominantly associated with Nissen fundoplication, have resulted in a series of modifications to the technique as described previously. The incorporation of a very loose, short wrap with adequate mobilization of the short gastric vessels resulted in reduction of the incidence of gas bloat from 15% to 11% and a troublesome dysphagia from 14% to 3%, but inability to belch or vomit remained relatively unchanged. These complications are extremely uncommon with partial fundoplication procedures. It should be borne in mind that the quality of life following anti-reflux surgery can only be improved when the symptoms of reflux are eliminated without the production of post operative symptoms created by the procedure itself ***(Pope, 1999).***

**3. Complications of angelchick prosthesis:**

It was found that more than half of the patients experience dysphagia after insertion of the prosthesis and persistence of this distressing symptom in 8-10% of patients. This requires removal of prosthesis, as the dysphagia is unresponsive to dilatation. Erosion through the wall of the gastro-esophageal junction is a serious complication but is fortunately rare ***(Cuscheri, 2002).***

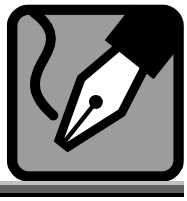
One of the most serious complication of the prosthesis is migration of the prosthesis either proximal into the chest, where angulation of the prosthesis may cause acute dysphagia, or detachment of the tapes, which allows displacement into the peritoneal cavity. The risk of proximal migration may be minimized by crural repair ***(Stuart et al., 2000).***

**4. Recurrence:**

Recurrent reflux following fundoplication may be secondary to a variety of anatomic and functional abnormalities which include: a patulous or incompetent initial repair, a slipped repair in which the wrap slips to encircle the stomach, complete or partial disruption of the wrap, intussusception of the gastric mucosa cephaled to the fundic wrap, and in addition to several technical points have been implicated as contributing to fundoplication failure**.**

Inadequate mobilization of the gastro-esophageal Junction, fundus and cardia is thought to increase the risk of wrap disruption. There is evidence suggesting that delayed gastric emptying may predispose to postoperative wrap failure ***(Wheatley et al., 2001).***

Patients with post operative gagging and an intra-operative hiatal hernia greater than 3 cm have a poor outcome, whereas patients with postoperative belching have a better long term outcome ***(Iqbal et al., 2006)***.



# PATIENTS AND METHODS

# PATIENTS AND METHOD

## Patients:

This prospective controlled randomized study was done in the period between August 2005 and June 2007. This study include 40 patients with symptomatic gastro-esophageal reflux disease underwent laparoscopic antireflux fundoplication at Ain Shams University Hospitals. These patients were randomly divided into two groups, each group formed of 20 patients. The first group had laparoscopic Nissen fundoplication and the second group had Laparoscopic Nissen Rossetti fundoplication.

Four patients, two of the Laparoscopic Nissen fundoplication group and two of the laparoscopic Nissen Rossetti fundoplication group, failed to attend postoperative follow up. Therefore, they were excluded from further consideration. Eventually, 36 patients were included in this study (18 patients in the group of Nissen fundoplication, and 18 patients in the group of Nissen Rossetti fundoplication).

### Age and Sex:

The group of Laparoscopic Nissen Fundoplication formed of 18 patients (12 male; 66.7% and 6 female, 33.3%). The mean age was 45 years (range from 27-62 years).

The group of Laparoscopic Nissen Rossetti fundoplication formed of 18 patients (13 male; 72% and 5

female; 28%). The mean age was 42.7 years (range from 24-55 years).

## Inclusion criteria:

- Failure of medical therapy. This should be considered when the patient has received the appropriate medications at the appropriate dosage for at least 12 weeks with persistence of symptoms, or inability to comply with the medications or life style adjustments.
- Atypical symptoms of gastro-esophageal reflux including respiratory, pharyngeal and dental problems.
- Individualized assessment. This includes patients who can only be maintained in remission by the administration of continuous and life long medications. Within this category, socioeconomic considerations are also important for the individual.
- Patients with symptomatic gastro-esophageal reflux and associated with large sliding hiatus hernias.

## Exclusion criteria:

- Esophageal stricture.
- Impaired distal esophageal peristalsis.
- Preoperative dysphagia.
- Shortened esophagus.

- Barrett's esophagus.
- Previous gastric or esophageal surgery.
- Previous upper abdominal surgery.

All those patients were excluded from the study to facilitate the comparison between the two groups without preoperative morbidity.

## Preoperative assessment:

All patients included in this study presented with symptomatic gastroesophageal reflux. Heartburn and regugitation were the commonest symptoms. Other symptoms like chocking episodes, dry cough, other respiratory symptoms and atypical chest pain were less common.

Preoperative assessment of all patients included upper alimentary endoscopy, standard esophageal manometry, ambulatory 24-hour pH monitoring and barium swallow and meal.

At esophagoscopy, the presence of esophagitis was a common finding. The severity of esophagitis was assessed according to the Los Angeles classification of esophagitis. Biopsy was taken from all cases to confirm the presense of reflux esophagitis and to exclude the presence of Barrett's esophagus.

**Los Angeles classification of esophagitis:**

Grade A : one (or more) mucosal break no longer than 5 mm, that does not extend between the tops of two mucosal folds

Grade B : one (or more) mucosal break more than 5 mm long that does not extend between the tops of two mucosal folds

Grade C : One (or more) mucosal break that is continuous between the tops of two or more mucosal folds but which involve less than 75% of the circumference

Grade D : One (or more) mucosal break which involves at least 75% of the esophageal circumference

***(Cuschieri, 2002)***

At esophagoscopy, the presence and axial length of a hiatal hernia was also estimated. A non-reducing hiatal hernia was defined as a pouch of gastric mucosa longer than 2 cm, confined between the endoscopic squamo-columnar junction and the diaphragmatic hiatal dentation on the stomach. Preoperative barium swallow served to image also the presence and length of hiatal hernia.

Esophageal manometry was done for all patients preoperatively. Prior to manometry any medication with proven or likely gastrokinetic properties was discontinued for at least 3

days. Esophageal manometry was performed with an eight lumen water perfused poly-vinyl catheter. Each tube was constantly perfused with distilled water at a rate of 0.6 ml/ min by a low compliance perfusion system. A pressure transducer, incorporated to each perfusion line, was connected to a polygraph device amplifier.

The probe was introduced through the nose in subjects being fasted overnight. A station pull-through technique was used and the probe was withdrawn 0.5 cm each time and kept at each level for at least 30 seconds or until the recording became stable. The high pressure zone was defined as the mean of the highest pressure plateau recorded by each of the ports minus the mean pressure of the gastric fundus measured at the end of expiration. The total length of high pressure zone was easily measured. The length of the intra-abdominal part of the high pressure zone was calculated from the pressure profile as the distance from the point of the first stable pressure increase above fundus pressure to the first point of negative pressure to change at inspiration (respiratory inversion point).

Ambulatory 24 hour esophageal hydrogen monitoring was performed as an outpatient investigation after discontinuation of any medication with proven or likely gastrokinetic properties for at least 3 days. Also, $H_2$ receptor antagonist and proton pump inhibitor must be discontinued for 5 days before the test. The device consisted of an electrode,

which was transnasally introduced and placed 5 cm proximal to the upper limit of the manometrically defined lower esophageal sphincter (LES), and a portable digital recorder.

Patients were instructed to follow their usual pattern of living. Reflux symptoms, meals, smoking and resting periods were recorded in a diary by the patients. The original scoring system devised by Johnson and DeMeester examined six variables which are percent total time pH was <4, percent upright time pH was <4, percent recumbent time pH was <4, number of reflux episodes, number of reflux episodes with pH < 4 for more than 5 minutes, and the period of the longest single acid exposure episode, and calculated a composite score according to a formula dependent on the deviation of each of these variables from normal values.

## Operative technique: **(Figs. 26-39)**

In this study both types of fundoplication were done simultaneously to avoid bias of learning curve if one type of fundoplication was done then shifting to the other type.

The operating room is set up with one or two video monitors placed near the head of the operating table. Patients was placed in modified lithotomy position (the thigh flexed no more than 20-25 degree) and brought into a reverse trendlenberg position with an angel of approximately $25^0$. The main surgeon stands between the patients legs. The main

assistant stands on the left of the patient and the second assistant stands on the right of the patient **(fig. 26)**. Nasogastric tube (16F) was routinely introduced once patient anesthethized. Routine application of compression bandage of both legs was done to avoid deep venous thrombosis which may be precipitated by laparoscopic surgery and lithotomy position.

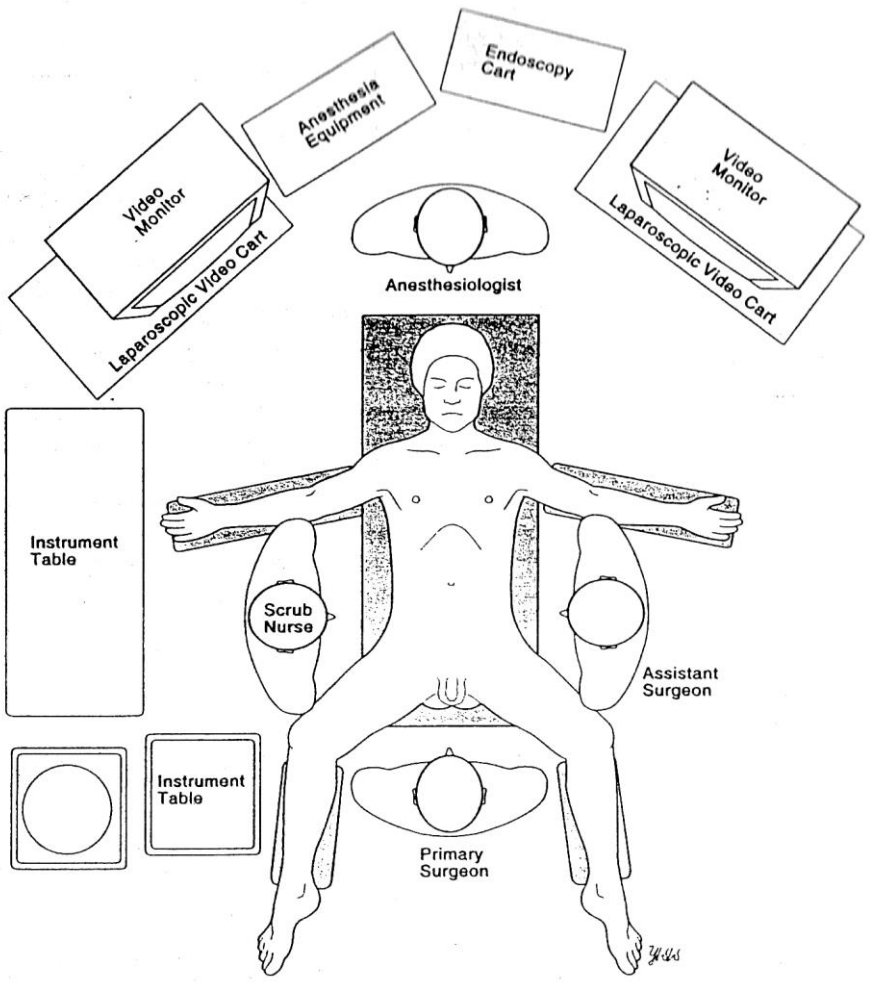



**Fig (26):** Patient in modified lithotomy position with the primary surgeon standing between the legs

Several different schemes have been described for position of laparoscopic cannulas. In this study the preferred sites for trocar placement are shown in the **fig (27)**.

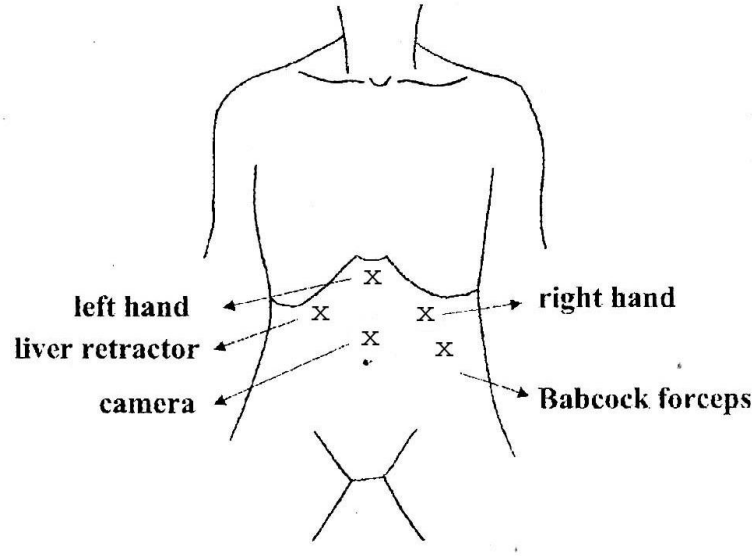



**Fig. (27):** Trocar placement for laparoscopic Nissen fundoplication or its Rossetti modification

In both groups of patients, the first step of the procedure was to completely divide the gastro-hepatic omentum and phreno-esophageal membrane. The hiatal pillars were fully exposed anterior, lateral and posterior to the lower esophagus. The posterior vagus was always left behind, while the anterior one was covered by the fundoplication.

A window at least 5 cm in length was created behind the lower esophagus. Even in the Nissen Rossetti group, a minimal dissection of the posterior upper part of the gastric fundus was carried out. In all cases, the diaphragmatic crurae were

approximated behind the esophagus with 2 - 3 nonabsorbable sutures (prolene 0, rounded needle).

Fundoplication in the Nissen Rossetti group was constructed by using the anterior wall of the gastric fundus without division of short gastric vessels.

In the Nissen group short gastric vessels were divided with the use of harmonic scalpel. Both the anterior and the posterior aspect of the gastric fundus were used to construct the fundoplication in the Nissen group.

In both groups, three interrupted nonabsorbable stitches were applied to the gastric fundus to construct 2 cm long fundoplication. The anterior wall of the esophagus was incorporated in two of the three stitches.

Closed suction drain was routinely used in all patients. All port sites closed with interrupted stitches.

## Postoperative care:

Analgesics were given for the first postoperative day, then at the patient's request for the next 24-48 hours. Nasogastric tubes were usually removed and intravenous fluid administration was discontinued on the second postoperative day, when oral fluid intake started. Drain removed after oral intake was started and minimal fluid was in the drain. Discharge from the hospital was encouraged from the third postoperative day.

Patients were instructed to remain on soft solid food for 2-4 weeks postoperatively. Normal physical and social activities were allowed 1 week after discharge, at the patient convenience. No objective tests were obtained immediately postoperatively.

## Postoperative assessment:

Assessment of the patients was made two weeks, one month, three month and 6 months after the operation. Early postoperative assessment (during the first three months) was mainly subjective; including symptoms control and occurrence of the postoperative complications (early dysphagia, gas bloating syndrome, nausea, diarrhea and recurrence of symptoms).

Late postoperative assessment (after $6^{th}$ month) was subjective and objective. Subjective assessment included disappearance of any of the previous complication or recurrence of symptoms. Objective assessment included the upper alimentary endoscopy, esophageal manometry and ambulatory pH monitoring.

Comparison between both groups of Nissen and Nissen Rossetti fundoplication was done as regards control of symptoms (subjectively and objectively by comparison of preoperative and postoperative investigations results), occurrence of the complications and different managements for these complications.

## Statistical analysis of the results:

All the results of this study were analysed using SPSS program version 12 (T test).

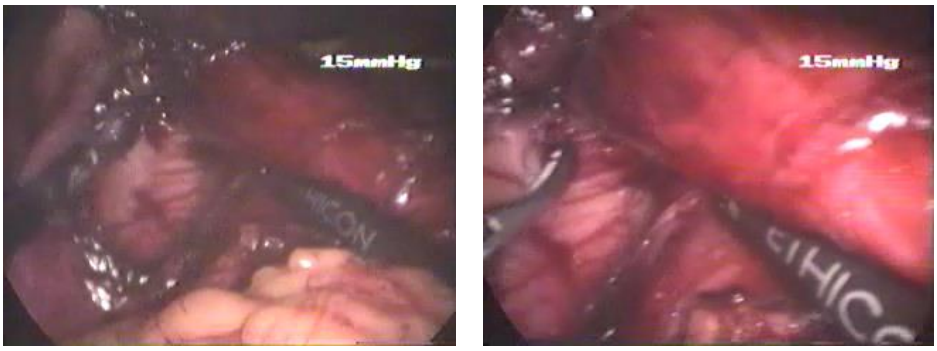



**Fig (28):** Complete dissection of the esophagus from diaphragmatic crura.

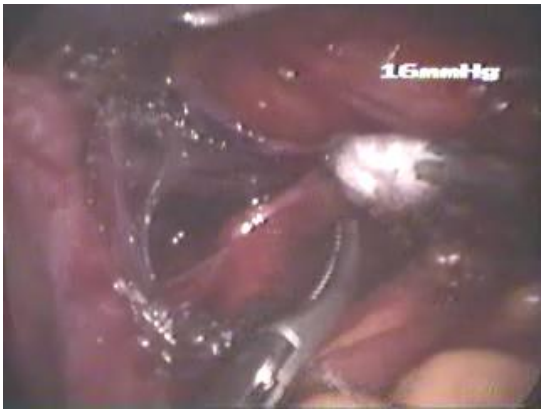



**Fig (29):** Retroesophageal mobilization (blunt dissection).

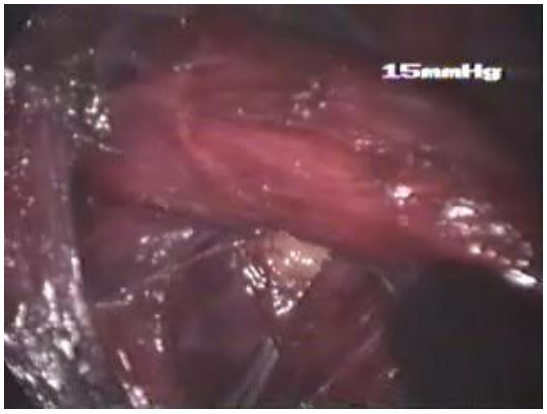



**Fig (30):** Creation of retroesophageal window.

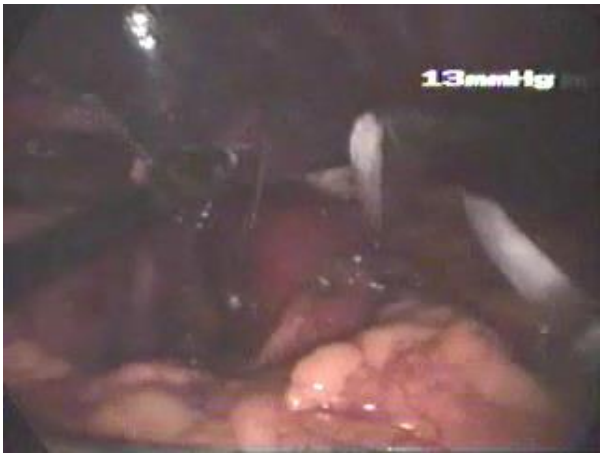



**Fig (31):** Tape around esophagus for traction.

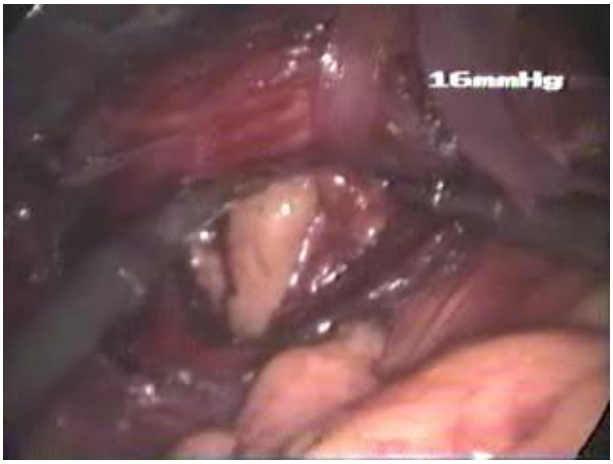



**Fig (32):** Complete dissection of the window to be more than 5 cm.

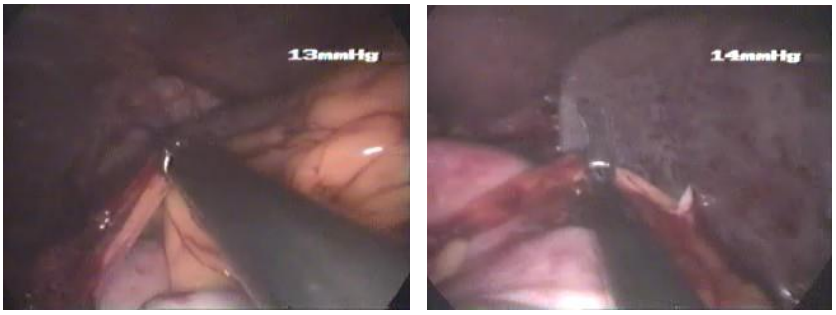



**Fig (33):** Division of short gastric vessels using Harmonic scalpel.

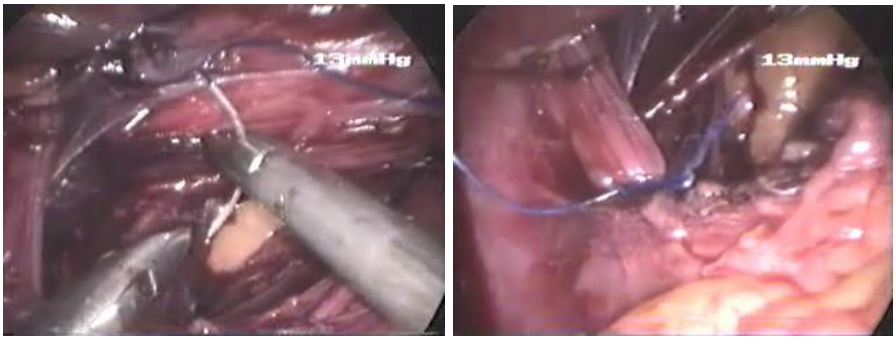



**Fig (34):** Crural repair with 2 prolene 0 stitches.


**Fig (35):** Delivery of the gastric fundus through retroesophageal window.

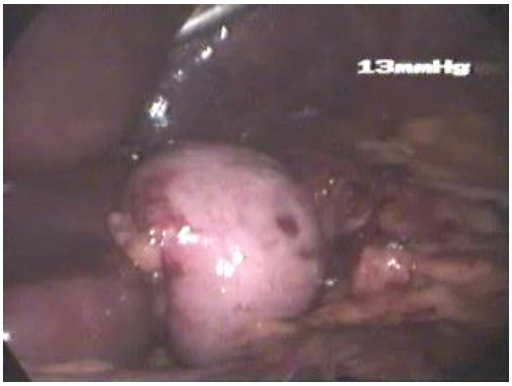
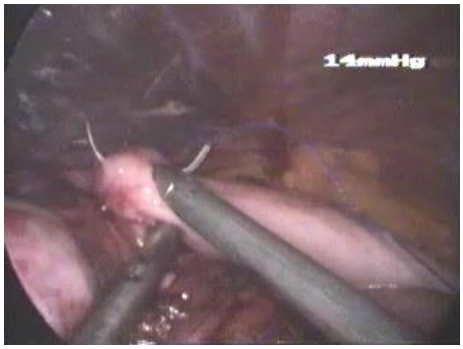



**Fig (36):** persistence of stomach without retraction indicating no tension "drop test".



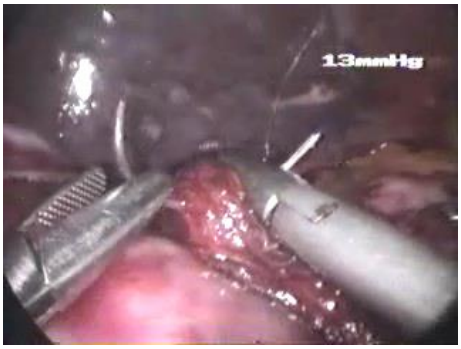




**Fig (37):** Upper two stitches include the wall of the esophagus.

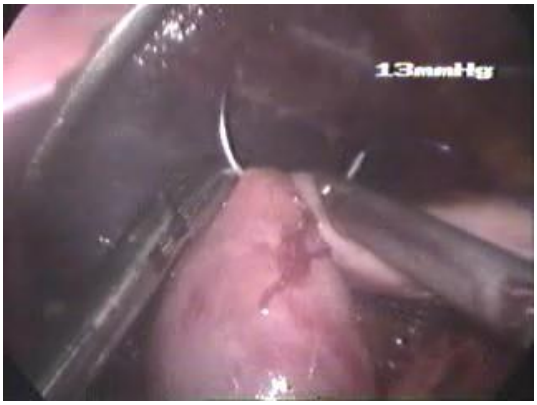



**Fig (38):** Last stitch includes only stomach wall.

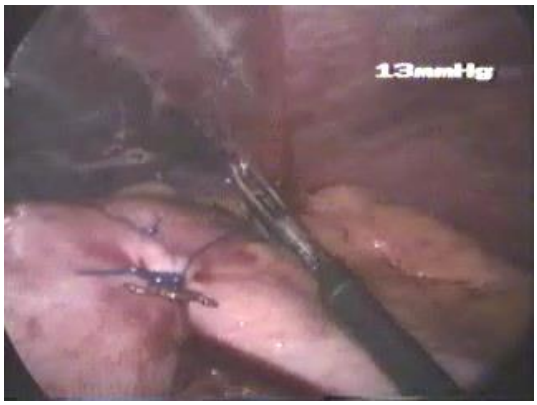
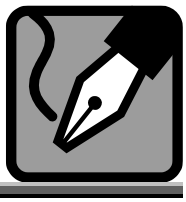



**Fig (39):** Complete fundal wrap (2 cm) around lower esophagus.



# RESULTS

# RESULTS

Initially 40 patients entered the study. Four patients, two of the Nissen group and two of the Nissen Rossetti group, failed to attend postoperative follow up. Therefore, they were excluded from further consideration, and eventually, 36 patients were included in this study. Eighteen of them (12 male and 6 female) were in the group of Nissen fundoplication. Their ages ranged from 27 to 62 years (mean age was 45 years). The remaining eighteen patients (13 male and 5 female) were in the group of Nissen Rossetti fundoplication. Their ages ranged from 24 to 55 years (mean age was 42.7 years).

## Presentation of the patients:

All patients included in this study presented with symptomatic gastro-esophageal reflux. Hearburn and regurgitation were graded according to DeMeester's scoring system, which is as follow:

| Symptoms | Grade | Description |
|---|---|---|
| **Heartburn** | | |
| None | 0 | No heartburn |
| Minimal | 1 | Occasional episodes |
| Moderate | 2 | Reason for medical visit |
| Severe | 3 | Interference with daily activities |
| **Regurgitation** | | |
| None | 0 | No Regurgitation |
| Minimal | 1 | Occasional episodes |
| Moderate | 2 | Predictable on position or straining |
| Severe | 3 | episodes of pulmonary aspiration with nocturnal cough or recurrent pneumonia |

***(Cuschieri, 2002)***

All patients included in this study presented with symptomatic gastro-esophageal reflux. Incidence and severity of symptoms related to GERD, such as heartburn, regurgitation, chest pain and respiratory complaints were nearly similar for both groups. All patients included in this study had received medical treatment of GERD for variable duration ranged from 3 months to 5 years (the mean duration was 14 months) with variable degree of response ranged from no response at all to

complete remission but with repeated recurrence after stoppage of the treatment.

Heartburn was the commonest symptom. All the patients (100%) in both groups complained of moderate or severe degree of heartburn. Regurgitation was the second most common symptom. Only one patient in the group of Nissen Rossetti fundoplication did not complain of significant regurgitation, while all other patients (97.2%) had minimal, moderate or even severe degree of regurgitation. Chocking episodes occurred in 7 patients 19.4% (3 patients [16.7%] in the group of Nissen fundoplication, and 4 patients [22.2%] in the group of Nissen Rossetti fundoplication). Dry cough and recurrent respiratory symptoms occurred in only 3 patients 8.3% (2 patients [11.1%] in the group of Nissen, and one patient [5.5%] in the group of Nissen Rossetti). Chest pain occurred in only one patient (5.5%) in the group of Nissen fundoplication.

Table 1 shows the symptoms of GERD and the duration of medical treatment at presentation in both groups.

**Table (1): Symptoms at presentation and duration of medical treatment**

| Symptoms | Group of Nissen fundoplication | Group of Nissen Rossetti fundoplication |
|---|---|---|
| Duration of medical treatment | 3-48 months<br>Mean 11 months | 4-60 months<br>Mean 17 months |
| Heartburn | | |
| None | 0 | 0 |
| Minimal | 0 | 0 |
| Moderate | 6 (33.3%) | 7 (39%) |
| Severe | 12 (66.7%) | 11 (61%) |
| Regurgitation | | |
| None | 0 | 1 (5.6%) |
| Minimal | 5 (27.8%) | 4 (22.2%) |
| Moderate | 9 (50.0%) | 9 (50%) |
| Severe | 4 (22.2%) | 4 (22.2%) |
| Chocking episodes | 3 (16.7%) | 4 (22.2%) |
| Dry cough and other respiratory symptoms | 2 (11.1%) | 1 (5.6%) |
| Chest pain | 1 (5.6%) | 0 |

## Objective preoperative assessment:

As regards upper alimentary endoscopy, the grades of esophagitis in both groups were nearly similar. In the group of Nissen fundoplication 4 patients (22.2%) had grade A esophagitis, 8 patients (44.4%) had grade B esophagitis, 4 patients (22.2%) had grad C esophagitis and 2 patients (11.1%) had grade D esophagitis. In the group of Nissen Rossetti fundoplication 2 patients (11.1%) had grade A esophagitis, 9 patients (50%) had grade B esophagitis, 5 patients (27.8%) had grade C esophagitis and 2 patients (11.1%) had grade D esophagitis.

In the group of Nissen fundoplication 12 patients (66.7%) had sliding hiatus hernia. In the group of Nissen Rossetti fundoplication 10 patients (55.6%) had sliding hiatus hernia. Hiatus hernia diagnosed with esophagoscope and confirmed by barium study.

At esophageal manometry, amplitude of esophageal peristalsis and esophageal body pressure were within normal in both groups as patients with abnormal results were excluded. The lower esophageal sphincter pressure in the group of Nissen fundoplication were ranged from 6 mmHg to 12 mmHg (the mean pressure was 8.6 mmHg), while the lower esophageal sphincter pressure of the group of Nissen Rossetti

fundoplication ranged from 7 to 11 mmHg (the mean pressure was 9 mmHg).

At ambulatory pH monitoring diagnosis of GERD confirmed in all patients of both groups. In the group of Nissen fundoplication the DeMeester score ranged from 39 to 51 with the mean score was 42.2, while in the group of Nissen Rossetti fundoplication the DeMeester score ranged from 41 to 50 with the mean score was 45.4.

The table 2 shows the results of all preoperative investigation.

**Table (2): Results of preoperative investigation**

| Preoperative investigation | Group of Nissen fundoplication | Group of Nissen Rossetti fundoplication |
|---|---|---|
| **Upper alimentary endoscopy:** | | |
| Grade A | 4 (22.2%) | 2 (11.1%) |
| Grade B | 8 (44.4%) | 9 (50.0%) |
| Grade C | 4 (22.2%) | 5 (27.8%) |
| Grade D | 2 (11.1%) | 2 (11.1%) |
| Manometry: | | |
| LES | Range from 6-12 mmHg<br>Mean 8.6 mmHg | Range from 7-11 mmHg<br>Mean 9 mmHg |
| Esophageal motility | All had normal motility | All had normal motility |
| Ambulatory pH monitoring | DeMeester score ranged from 39 to 51<br>Mean score 42.2 | DeMeester score ranged from 41 to 50<br>Mean score 45.4 |
| Barium swallow and meal:<br>Sliding hiatus hernia | 12 (66.7%) | 10 (55.6%) |

N.B. In upper alimentary endoscopy and barium swallow, the number of the patients and their percentage in their groups are documented.

## Operative time:

Operative time was shorter in the group of Nissen Rossetti fundoplication. In the group of Nissen fundoplication the mean operative time was 108 minutes (ranged from 80 - 150 minutes). In the group of Nissen Rossetti fundoplication the mean operative time was 97 minutes (ranged from 75 – 125 minutes).

The mean operative time in the first 12 patients in the group of Nissen fundoplication was 111.4 minutes and the mean operative time in the last 6 patients was 99 minutes. The mean operative time in the first 12 patients in the group of Nissen Rossetti fundoplication was 106.5 minutes and the mean operative time in the last 6 patients was 90 minutes.

Table 3 shows the differences in operative time in both groups.

**Table (3): Operative time**

| | Group of Nissen fundoplication | Group of Nissen Rossetti fundoplication |
|---|---|---|
| Operative time | 80 – 150 min<br>Mean 108 min | 75 – 125 min<br>Mean 97 min |

## Perioperative complications:

Perioperative complications were either intra-operative or post-operative complications.

### Intra-operative complications:

The most common intra-operative complication was intra-operative bleeding. Intraoperative bleeding occurred in 7 patients (19.4%) in this study. In the group of Nissen fundoplication 4 patients (22.2%) had intra-operative bleeding. one patient (5.6%) had bleeding due to splenic laceration. Two patients (11.1%) had bleeding from left lobe of the liver, and another one patient (5.6%) had bleeding from retro-esophageal mobilization and dissection of the crurae. In all cases bleeding was managed by the usual haemostatic measures. Non of the cases needed blood transfusion.

In the group of Nissen Rossetti fundoplication intra-operative bleeding occurred in three patients (16.7%). One case (5.5%) had bleeding from splenic laceration due to traction on inadequately mobilized gastric fundus. Laparotomy needed to stop bleeding and splenectomy was done. In other two cases (11.1%) bleeding were due to laceration in the left lobe of the liver and stopped by the usual haemostatic measures. Blood transfusion was not required, either Intraoperative or immediate postoperatively in both groups.

Laparotomy occurred, after completion of the wrap, in one patient (5.6%) in the group of Nissen Rossetti fundoplication due to uncontrolled bleeding from splenic laceration and Splenectomy was mandatory. Removal of Nasogastric tube and oral intake delayed to the third day. Discharge of the patient delayed to the fifth day due to delayed oral intake, need of more analgesic and delayed restoration of normal physical activity.

The most serious complication was gastric perforation which occurred in one patient (5.6%) in the group on Nissen Rossetti fundoplication. It was detected intra-operatively and was mainly disruption of the seromuscular stitch of the fundoplication. Repair was done laparoscopically, and fundoplication completed with intra-abdominal drain. Removal of Nasogastric tube and oral intake delayed to the fourth day. Discharge of the patient delayed to the $6^{th}$ day due to delayed oral intake, need of more analgesic and delayed restoration of normal physical activity.

**Postoperative complications:**

The most common postoperative complication was early postoperative dysphagia, which occurred in the first 3 months postoperatively. Early postoperative dysphagia occurred in 5 patients (27.8%) in the group of Nissen fundoplication (3 from 12 patients [25%] had GERD and hiatus hernia (H.H), and 2

from 6 patients [33.3%] had GERD without H.H). All of these patients had dysphagia to the solid diet only.

Early dysphagia also occurred in 8 patients (44.4%) in the group of Nissen Rossetti fundoplication (3 from 10 patients [30%] had GERD and H.H, and 5 from 8 patients [62.5%] had GERD without H.H). Six from these eight patients had dysphagia to the solid diet only, while the remaining two patients had dysphagia to the solid and soft diet.

Persistent postoperative dysphagia is the dysphagia occurred and persisted for more than 3 months. It occurred in two patients (11.1%) in the group of Nissen Rossetti fundoplication and it was to the solid diet in both cases. Esophageal dilatation was a safe and easy management of this complication. Multiple sessions of esophageal dilatation were done (two sessions in the first patient and three sessions for the other). Esophageal dilatation was done as an outpatient procedure without need to hospital admission. Dysphagia responded well for dilatation without any symptoms of recurrence.

Gas bloating syndrome was an important postoperative complication. It is a syndrome characterized by a wide variety of symptoms like epigastric discomfort, epigastric fullness or rarely epigastric pain, early satiety and inability to belch or vomit. This syndrome occurred in 4 patients (22.2%) in the

group of Nissen fundoplication (3 from 12 patients [25%] with GERD and H.H., and 1 from 6 patients [16.7%] with GERD without H.H.). it occurred in 6 patients (33.3%) in the group of Nissen Rossetti fundoplication (2 from 10 patients [20%] with GERD and H.H., and 4 from 8 patients [50%] with GERD without H.H.). All cases of both groups improved within 2 to 3 months without need to any further management.

Other rare complications include nausea which occurred in one patient (5.6%) in the group of Nissen fundoplication and two patients (11.1%) in the group of Nissen Rossetti fundoplication. Diarrhea occurred in only one patient (5.6%) in the group of Nissen Rossetti fundoplication. in all cases nausea and diarrhea disappeared within two weeks.

Recurrence of symptoms occurred in one patient (5.6%) in the Nissen Rossetti fundoplication group. This patient had only GERD without HH. This patient had a transient relieve of symptoms followed by recurrence of symptoms of heartburn and regurgitation 4 months postoperatively. Diagnosis of recurrence was confirmed by 24 hours pH monitoring and upper alimentary endoscopy was done and grade A esophagitis was found. On reoperation disruption of the wrap was the cause of recurrence, due to inadequate gastric fundus mobilization with tension on the wrap. Short gastric vessels division was done with reconstruction of the wrap without any tension.

Follow up of the patient for more than 6 months with no recurrence of the symptoms. Table 4 shows the perioperative complications.

**Table (4): Perioperative complications**

| Complication | Group of Nissen fundoplication | Group of Nissen Rossetti fundoplication |
|---|---|---|
| Intraoperative bleeding: | | |
| Spleen | 1 (5.6%) | 1 (5.6%) |
| Liver (left lobe) | 2 (11.1%) | 2 (11.1%) |
| Retroesophageal & crurae | 1 (5.6%) | 0 |
| **Total** | **4 (22.2%)** | **3 (16.7%)** |
| Gastric perforation | 0 | 1 (5.6%) |
| Esophageal perforation | 0 | 0 |
| Conversion to open | 0 | 1 (5.6%) |
| Early postoperative dysphagia | | |
| Solid | 5 (27.8%) | 6 (33.3%) |
| Liquid | 0 | 2 (11.1%) |
| **Total** | **5 (27.8%)** | **8 (44.4%)** |
| Persistent dysphagia | 0 | 2 (11.1%) |
| Gas bloating syndrome | 4 (22.2%) | 6 (33.3%) |
| Recurrence | 0 | 1 (5.6%) |
| Nausea | 1 (5.6%) | 2 (11.1%) |
| Diarrhea | 0 | 1 (5.6%) |

The incidence of postoperative complications especially dysphagia and gas bloating syndrome were strongly related to the presence or absence of the hiatus hernia with the GERD in the group of Nissen Rossetti fundoplication. The table 5 demonstrates the number and percentage of the patients in each group which had the complication in relation to the presence or absence of preoperative sliding hiatus hernia.

**Table (5): Relation of post operative complication to preoperative HH**

| Complication | Group of Nissen fundoplication | | | Group of Nissen Rossetti fundoplication | | |
|---|---|---|---|---|---|---|
| | GERD + HH | GERD + no HH | Total | GERD+ HH | GERD + no HH | Total |
| Early postoperative dysphagia | 3/12 (25%) | 2/6 (33.3%) | 5/18 (27.8%) | 3/10 (30%) | 5/8 (62.5%) | 8/18 (44.4%) |
| Persistent dysphagia | 0 | 0 | 0 | 0 | 2/8 (25%) | 2/18 (11.1%) |
| Gas bloating syndrome | 3/12 (25%) | 1/6 (16.7%) | 4/18 (22.2%) | 2/10 (20%) | 4/8 (50%) | 6/18 (33.3%) |
| Recurrence | 0 | 0 | 0 | 0 | 1/8 (12.5%) | 1/18 (5.5%) |

## Postoperative hospital stay:

Postoperative hospital stay of the patients in both groups was relatively short. In the group of Nissen fundoplication the hospital stay ranged from 2 to 5 days with a mean duration of 3 days. In the group of Nissen Rossetti fundoplication the hospital stay ranged from 2 to 6 days with a mean duration of 4 days.

## Postoperative symptomatic control:

Postoperative symptomatic control occurred in all patients of the Nissen fundoplication group, while only one patient (5.6%) in the group of Nissen Rossetti fundoplication had a recurrence of symptoms of regurgitation and heartburn as mention above.

## Postoperative objective assessment:

At Upper alimentary endoscopy esophagitis was markedly improved and disappeared in all patients of Nissen fundoplication group. In the group of Nissen Rossetti fundoplication only one patient (5.6%) had esophagitis grade A.

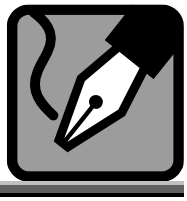
At postoperative esophageal manometry study, the lower esophageal sphincter pressures were ranged from 22 to 29 mmHg (the mean pressure was 24 mmHg) in the group of Nissen fundoplication. In the group of Nissen Rossetti fundoplication the lower esophageal sphincter pressures were

ranged from 17 to 33 mmHg (the mean pressure was 28 mmHg).

At postoperative pH monitoring, DeMeester score dropped significantly in both group. In the group of Nissen fundoplication DeMeester score ranged from 7 to 12 with the mean score was 8.6. In the group of Nissen Rossetti fundoplication the score ranged from 9 to 21 with the mean score was 10.8. Table 6 shows the results of preoperative and postoperative investigations.

**Table (6): Preoperative versus postoperative investigations results**

| Investigation | Group of Nissen fundoplication | | Group of Nissen Rossetti fundoplication | |
|---|---|---|---|---|
| | **Preoperative** | **Postoperative** | **Preoperative** | **Postoperative** |
| Upper alimentary endoscopy | 100% esophagitis | 0% esophagitis | 100% esophagitis | 5.5% esophagitis |
| Esophageal manometry | 6–12 mmHg<br>Mean 8.6 mmHg | 22-29 mmHg<br>Mean 24.3 mmHg | 7-11 mmHg<br>Mean 9 mmHg | 17-33mmHg<br>Mean 28 mmHg |
| pH monitoring | 39 – 51<br>Mean 42.2 | 7 – 12<br>Mean 8.6 | 41 – 50<br>Mean 45.4 | 9 – 21<br>Mean 10.8 |



# DISCUSSION

# DISCUSSION

Nissen fundoplication has proved to be a successful means of resolving the distress experienced by patients whose gastro-esophageal reflux disease does not respond to medical therapy ***(Leggett et al., 2000).***

Antireflux surgery is entering a new era with the advent of minimally invasive techniques ***(Spivak et al., 1999).*** Minimal access antireflux and hiatal hernia surgery has become increasingly established throughout the world. The minimal access route confers the benefits of reduced post-operative pain, early recovery and discharge from the hospital, and minimum morbidity ***(Sami, 2002).***

Laparoscopic Nissen fundoplication has in the last few years been compared with open fundoplication, and several studies have shown them to have similar patient outcome, both short term and long term. However, some reports have demonstrated better outcomes with laparoscopic procedure in terms of lower morbidity and better patient satisfaction ***(Raphael et al., 2005).***

In this study 36 patients underwent laparoscopic antireflux surgery in the form of laparoscopic Nissen fundoplication or laparoscopic Nissen Rossetti fundoplication with complete resolution of symptoms in 35 patients (97.2%).

Also, objective assessment of the patients after 6 months postoperatively showed marked improvement in the lower esophageal sphincter pressure, DeMeester score in pH monitoring and grade of esophagitis in upper gastrointestinal endoscopy. Only one patient (5.6%) in the group of Nissen Rossetti fundoplication had recurrent symptoms due to disruption of the wrap which was corrected in the second operation (open Nissen fundoplication) with resolution of the symptoms and objective improvement after 6 months from the second operation was confirmed.

Although, one and 6 months postoperative assessments are still short term follow up, but the short term results were very satisfactory and promising indicating efficacy of the laparoscopic antireflux surgery for control symptoms like open antireflux surgery.

Nissen fundoplication involves the creation of a circumferential fundal wrap around the distal esophagus. To reduce the risk of postoperative problems, it is thought that this should be constructed loosely, with many surgeons advocating routine division of the short gastric vessels to fully mobilize the gastric fundus, thereby facilitating the formation of a loose floppy wrap ***(Levy et al., 1998& Blomqvist et al., 2000).***

However, Nissen's original procedure and Rossetti's subsequent modification achieve similar effect without routine

division of the short gastric vessels ***(Loustrainen and Isolauri, 1999& O'Boyle et al., 2002).***

Short gastric vessels division provided no clinical benefit in term of complications, reoperation rate, or dysphagia at various time intervals up to 2 years after surgery. Many authors reported good outcomes from extensive experiences with Nissen fundoplication without division of short gastric vessels ***(Anvari and Allen., 1998& Zaninotto et al., 2000).***

In this study, a comparison was made between Nissen fundoplication and Nissen Rossetti fundoplication as regard operative time, resolution of symptoms and incidence of complications.

Operative time in all studies was shorter in the group of Nissen Rossetti fundoplication than in the group of Nissen fundoplication. Operative time in the group of Nissen Rossetti fundoplication was 60 $\pm$ 12 minutes, while operative time in the group of Nissen fundoplication was 100 $\pm$ 22 minutes. Technical difficulties, although not scored, were greater in the division group than in the non division group, and that was reflected in the significantly increased operating time in the former group ***(Watson et al., 1997 & Chrysos et al., 2001).***

Paradoxically, the average operative time for the laparoscopic Nissen was shorter than that for the Rossetti by 25 minutes. There were several factors that contributed to this

result. The influencing of the learning curves for both the surgeons and the operating room team should not be underestimated. Advances in equipment have contributed to shorter operative times especially the laparoscopic coagulation shears (LCS) which facilitates division of gastro-splenic omentum and short gastric vessels ***(Leggett et al., 2000).***

In this study, the operative time in the group of Nissen fundoplication was ranged from 80 to 150 minutes (the mean time was 108 minutes). The operative time in the group of Nissen Rossetti fundoplication was ranged from 75 to 125 minutes (the mean time was 97 minutes). The mean operative time in Nissen group was not significantly longer than in the Nissen Rossetti group **(P=0.151)**. The longer operative time in the group of Nissen than in the group of Nissen Rossetti was due to the more technical steps done (division of short gastric vessels).

The mean operative time in the first 12 patients in the group of Nissen fundoplication was 111.4 minutes and the mean operative time in the last 6 patients was 99 minutes. The mean operative time in the first 12 patients in the group of Nissen Rossetti fundoplication was 106.5 minutes and the mean operative time in the last 6 patients was 90 minutes.

The mean operative time in both groups in this study was longer than the mean operative time in other studies of the more

specialized centers. Also, the mean operative time in our study decreased in the last cases more than the former cases in the same group. The only explanation for these observations is the effect of the experience in the improvement of the learning curve with the decrease of the operative time needed.

There are reports suggesting that resolution of reflux symptoms decreased in patients undergoing total fundic wrap without division of short gastric vessels (Nissen Rossetti fundoplication) ***(Patti et al., 1998& Gotely et al., 2001)***

It is rather generally accepted that sufficient control of reflux and cure of symptoms related to reflux are accomplished after Nissen fundoplication either with or without division of the short gastric vessels. All patients experienced relief from symptomatic gastro-esophageal reflux, whether they received the Rossetti modification or the Nissen fundoplication ***(Peters et al., 1998& Leggett et al., 2000).***

In this study, all patients in the group of Nissen fundoplication had complete relief of all symptoms of gastroesophageal reflux. In the group of Nissen Rossetti fundoplication only one patient (5.6%) had recurrence of reflux symptoms. Objective assessment, 6 months after the procedure, confirmed complete improvement in all patients except the patient who had the recurrence, so the Nissen fundoplication

was significantly **(P=0.041)** more effective in control of reflux symptom.

Intraoperative bleeding in all studies was less common in the group of Nissen Rossetti fundoplication than in the group of Nissen fundoplication. Intraoperative bleeding in the group of Nissen fundoplication was mainly during division of short gastric vessels. In the group of Nissen Rossetti fundoplication bleeding mainly was due to injury to the left lobe of liver during retraction ***(Watson et al., 1997 & Kazuyoshi et al., 2002).***

The incidence of bleeding while attempting to divide the short gastric vessels between clips was reported to be (25%). Division of the short gastric vessel with the use of harmonic scalpel was uneventful. No Intraoperative bleeding in the group of Nissen Rossetti fundoplication. Blood transfusion were not required, either Intraoperative or immediate postoperatively ***(Chrysos et al., 2001).***

In this study only minor insignificant difference was noticed in the occurrence of intraoperative bleeding between both groups **(P=0.161)**. In the group of Nissen fundoplication the incidence of intraoperative bleeding was higher (22.2%), but bleeding was minor and easily controlled. While in the group of Nissen Rossetti fundoplication the incidence of intraoperative bleeding was lower (16.7%). In one case (5.6%)

bleeding persisted due to splenic laceration due to traction on inadequately mobilized gastric fundus and Laparotomy needed with splenectomy was done.

Division of the short gastric vessels (SGV) with harmonic scalpel associated with no bleeding from the vessels. So this technique is a very safe technique and makes the step of division of the short gastric vessels safe, easy and more rapid.

In both groups 4 patients had bleeding from laceration of the left lobe of a healthy liver during retraction which was the commonest cause for intraoperative bleeding. In all cases bleeding stopped with usual haemostatic measures. So minor bleeding from healthy liver usually controllable and unnecessary conversion to open must be avoided.

Gastric perforation occurred in only one patient (5.6%) in the group of Nissen Rossetti fundoplication which is significant **(P=0.041)**. it was mainly due to disruption of seromuscular stitch indicating more tension on the wrap with Nissen Rossetti modification. Post-operative removal of Nasogastric tube, and oral intake were delayed for the 4$^{th}$ postoperative day. The drain was not removed until oral intake became satisfactory with no evidence of gastric leak. Discharge of the patient was delayed to the 6$^{th}$ postoperative day.

Even though there was no statistical difference in the complications between Nissen fundoplication and Rossetti's modification, it is notable that conversion was more with the Nissen fundoplication than with Rossetti's modification ***(Watson et al., 1997& Leggett et al., 2000).***

In this study, conversion to laparotomy occurred only in one patient in the group of Nissen Rossetti fundoplication due to bleeding from splenic laceration. This conversion considered statistically significant **(P=0.041)**.

Transient dysphagia occurs in 40% to 70% of patients after Nissen fundoplication. Fortunately, dysphagia usually resolves spontaneously within 2 to 3 months. Persistent dysphagia, however, occurs in 3% to 24%of patients after Nissen fundoplication. The role of short gastric vessels division and fundic mobilization in the avoidance of persistent dysphagia remains controversial ***(Kazuyoshi et al., 2002).***

Dysphagia is a common sequel of a total fundic wrap. Even from the era of open approach to Nissen fundoplication, division of the short gastric vessels has been advocated as a mandatory operative step because it offers full mobilization of the posterior aspect of the gastric fundus. So the wrap is constructed by pulling the posterior fundic wall behind the esophagus and suturing it to the anterior fundic wall in front of abdominal aspect of the esophagus. This step allows the

construction of floppy fundic wrap and minimize postoperative incidence of dysphagia ***(Rantanen et al., 1999 &* Zoring et al., 2002).**

Other studies showed that division of the short gastric vessels did not improve incidence and severity of dysphagia after Nissen fundoplication ***(Patti et al., 1998& Chrysos et al., 2001).***

From a technical standpoint, it was found that routine division of the short gastric vessels was not necessary to create a loose, floppy wrap. Full division of the peritoneal attachment of the left lateral aspect of the esophagus deep to the left pillar of the diaphragmatic crus and partial dissection of the posterior fundic wall offered adequate mobilization to create a large posterior esophageal window, thus facilitating the construction of a loose wrap. Furthermore, the initial piece of the fundus grasped behind the esophagus was usually quit tight, but by repositioning instruments and grasping an adjacent piece of the fundus, one is able to mobilize a much floppier segment to create an adequate fundic wrap ***(Levy et al., 1998& Donald et al., 2006).***

Division of the short gastric vessels should only be done when necessary and the results without division will be just as good so long as the wrap is short and loose ***(Jamieson et al., 1994).***

In cases with good esophageal peristalsis, lower esophageal sphincter (LES) relaxes promptly and completely on swallowing, and the wrap is loose after Nissen fundoplication with or without division of the short gastric vessels, it could be speculated that dysphagia is the result of outlet partial mechanical obstruction of he esophagus. Incomplete LES relaxation on swallow might contribute to dysphagia, although not as the main mechanism. Patients with dysphagia may present either with complete or incomplete LES postdeglutition relaxation. Furthermore, some patients without dysphagia may equally exhibit incomplete LES relaxation on swallowing. Incomplete LES relaxation on swallowing has not been implicated as the main mechanism of dysphagia after fundoplication although it might contribute to it ***(Wills and Hunt, 2001& Bais et al., 2001).***

In this study, the incidence of postoperative dysphagia was higher in the group of Nissen Rossetti fundoplication than in the group of Nissen fundoplication. Early dysphagia was 44.4% in the group of Nissen Rossetti fundoplication versus 27.8% in the group of Nissen fundoplication. This difference between both groups considered insignificant (P=0.073). In the group of Nissen fundoplication the dysphagia was to the solid diet only, while in the group of Nissen Rossetti fundoplication 6 patients (33.3%) had dysphagia to the solid diet and 2 patients (11.1%) had dysphagia to soft diet.

Persistent dysphagia occurred only in two patients of the group of Nissen Rossetti fundoplication (11.1%) and this considered a significant difference between both groups **(P=0.02)**. Esophageal dilatation was a safe and effective treatment for both cases.

In the group of Nissen Rossetti fundoplication it was noticed that, the incidence of early postoperative dysphagia was markedly lower (30% vs 62.5%) in the presence of preoperative hiatus hernia in association to GERD. The incidence of early postoperative dysphagia in the group of Nissen Rossetti fundoplication in patients with hiatus hernia was nearly similar the incidence of early dysphagia in the group of Nissen fundoplication (30% versus 27.8%). Persistent dysphagia in the group of Nissen Rossetti fundoplication did not occur in patients with GERD and hiatus hernia.

The overall incidence of postfundoplication gas-bloating syndrome varies between 10% and 76%, depending on the duration of the follow up and the subjectiveness of clinical assessment, and tends to improve with time ***(Lundell et al., 2000& Tew et al., 2000).***

Regarding the etiology of the syndrome, several anatomical or functional causes have been proposed, such as vagus nerve injury, slipping, dislocation or disruption of the wrap, incomplete lower esophageal sphincter relaxation and

preexisting gastric motility disorders ***(Lundell et al., 2000& Tew et al., 2000).***

A recent study showed that postfundoplication exhibits normal compliance but impaired relaxation of the proximal stomach. At the same time, postprandial sensation of fullness in those patients is significantly increased. That it could be speculated that division of the short gastric vessel may result in alteration of proximal gastric motility pattern, which, in turn leads to increased incidence of epigastric fullness. Undoubtedly, this speculation should be explored with further studies ***(Vu et al., 1999).***

A recent study randomized 99 patients undergoing laparoscopic fundoplication to have their short gastric vessels either divided or left intact. At a median follow up of 1 year, no clinical outcome differences were seen between the two techniques for the problem of dysphagia, ability to belch, amount of flatus passed and gas-bloat syndrome ***(Blomqvist et al.,2000).***

In this study the incidence of gas bloat syndrome was insignificant **(P=0.154)** more common in the group of Nissen Rossetti fundoplication than in the group of Nissen fundoplication (33.3% versus 22.2%). This syndrome improved within the next 2 to 3 months in all cases without need to any further management.

The incidence of gas bloat syndrome in the group of Nissen Rossetti fundoplication varies also with the existence of preoperative hiatus hernia in association with the GERD in the same picture like the postoperative dysphagia. The incidence of gas bloat syndrome significantly lower **(P=0.031)** in the presence of preoperative association of GERD with hiatus hernia than that in the patients with GERD without hiatus hernia (20% versus 50%).

The incidence of gas bloat syndrome in the group of Nissen Rossetti fundoplication in patients had hiatus hernia in association with the GERD was nearly similar to that which occurred with the group of Nissen fundoplication.

These differences of both dysphagia and gas bloat syndrome in the group of Nissen Rossetti fundoplication and their relation to the preoperative hiatus hernia may indicate that the presence of hiatus hernia may help in construction of a more floppy wrap without short gastric vessels division.

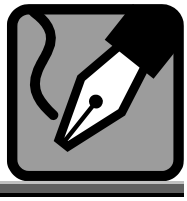
Reflux symptoms resolved without significant differences between both laparoscopic Nissen fundoplication and laparoscopic Nissen Rossetti fundoplication. Follow up for the Rossetti group extends from 36 to 82 months and that for Nissen group from 17 to 35 months. Postoperatively, no patient in either group complained of persistent reflux ***(Leggett et al., 2000).***

At 2 years follow up of patients underwent laparoscopic Nissen and Nissen Rossetti fundoplication the recurrence rate of reflux symptoms was nearly similar. The rate of recurrence in group of Nissen was 5.1%, while in the Nissen Rossetti group was 5.2% ***(Pessaux et al., 2000).***

In this study the total incidence of recurrence of reflux symptoms was very low, only one patient (2.7%) in both groups. The only reccurrence was in the group of Nissen Rossetti fundoplication which considered significant **(P=0.041)** in comparison to the group of Nissen fundoplication.

This patient had a laparoscopic Nissen Rossetti fundoplication with primarily control of symptoms for short time then progressive increase of symptoms mainly heartburn and regurgitation with the maximum symptoms was at 4$^{th}$ month postoperative. 24 Hour pH monitoring confirm the diagnosis of recurrence with grade A esophagitis on upper alimentary endoscopy. Reoperation showed disruption of the wrap. The explanation was that, inadequately mobilized gastric fundus with more tension on the wrap, and wrap disruption. For adequate mobilization division of the proximal short gastric vessels was mandatory and total wrap done. Follow up of the patient showed improvement of the symptoms of reflux both early and 6 months after the second operation. Objective assessment was done after 6 months of the second operation showed healing of the esophagitis and normal pH monitoring.

Other complications either intraoperative like esophageal perforation, pleural injury and complications related to the trocars e.g. bleeding from the site of the trocar or injury to internal viscus or postoperative complications like wound infection, atelactasis and incisional hernia from the site of camera insertion did not occurred in this study.



# SUMMARY AND CONCLUSION

# SUMMARY AND CONCLUSION

Gastroesophageal reflux disease is a common condition with a very distressing symptoms of heartburn, regurgitation, dysphagia and atypical symptoms like respiratory symptoms. The condition become more common due to life style modification with increase weight and bad habit like excess cigarettes smoking and repeated intake of coffee, tea, cola beverages which impair lower esophageal sphincter tone.

Most patients with GERD complain from mild and intermittent symptoms. In small percentage (~10%) symptoms become severe enough to push the patient for medical consultation. With the development of H2 receptors blocker and proton pump inhibitors the reflux symptoms could be controlled and the incidence of the complication like Barrett's esophagus decreased. The problem was the recurrence of symptoms after stoppage of the medical treatment which was a very common event in up to 80% of the patients with the need for long life treatment. The second problem was that with the long term follow up of the patients on the medical treatment the development of the Barrett's esophagus decreased but still occurs.

Surgical therapy, which addresses the functional nature of this condition, is curative of the patients and, thus, is more

effective than medical therapy and also provides good long term results. Antireflux surgery is entering a new era with the advent of minimally invasive techniques

Different types of antireflux surgery developed. Nissen fundoplication has proved to be a successful antireflux surgery with both open and laparoscopic techniques. Various modifications of Nissen had been done to improve efficacy and to decrease postoperative complications. Nissen Rossetti fundoplication is a modified Nissen fundoplication in which short gastric vessels are not divided and use only the anterior wall of the gastric fundus to create the valve. A long time of debate was taken about the advantage and disadvantage of this modification.

In this study comparison was made between laparoscopic Nissen fundoplication and laparoscopic Nissen Rossetti fundoplication as regards operative time, intraoperative and postoperative complications, and control of symptoms of reflux

Thirty six patients with symptomatic gastroesophageal reflux disease and after failure of medical treatment were included in this study and randomly divided into two groups. Eighteen patients were in the group of laparoscopic Nissen fundoplication and eighteen patients in the group of laparoscopic Nissen Rossetti's fundoplication.

This study shows that, there are no significant differences between laparoscopic Nissen fundoplication and laparoscopic Nissen Rossetti fundoplication as regards operative time, intraoperative bleeding, and postoperative gas bloating syndrome and early dysphagia. However, laparoscopic Nissen fundoplication is significantly better than laparoscopic Nissen Rossetti fundoplication as regards symptoms control, intraoperative incidence of gastric perforation, conversion to open, postoperative persistent dysphagia and recurrence of symptoms.

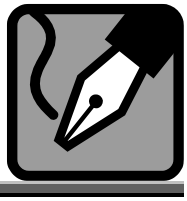
The previous advantages of laparoscopic Nissen Rossetti fundoplication were the shorter operative time and the lower incidence of bleeding because there was no short gastric vessels division. With the development of harmonic coagulating shears (HCS), which provides safe and rapid division of short gastric vessels, the difference in operative time and the incidence of intraoperative bleeding becomes insignificant.

In the group of laparoscopic Nissen Rossetti fundoplication, postoperative complications were significantly lower in patients who had sliding hiatus hernia in association with GERD than in patients who had GERD only.

# CONCLUSION

The laparoscopic Nissen fundoplication is more superiorly than laparoscopic Nissen Rossetti fundoplication as regards control of symptoms, intraoperative and postoperative complications.

However, the nearly similar results of laparoscopic Nissen Rossetti fundoplication and laparoscopic Nissen fundoplication in patients with associated sliding hiatus hernia make Rossetti's modification a safe alternative technique in those patients.



# ARABIC SUMMARY

# الملخص العربى

ارتجاع العصارة المعدية للمرئ من الأمراض الشائعة الحدوث ويتميز هذا المرض بأعراضه المؤلمة من حدوث آلام حادة أسفل منتصف الصدر وارتجاع العصارة الحمضية اللاذعة الطعم إلى الفم أو حدوث صعوبة فى ابتلاع الطعام وأعراض أخرى مثل التهاب الجهاز التنفسى.

ومما زاد انتشار هذا المرض انتشار السمنة والعادات السيئة مثل السجائر – كثرة تناول المشروبات التى تؤدى إلى ضعف فى الصمام السفلى للمرئ مثل (القهوة والمشروبات الغازية).

أعراض هذا المرض كثيرا ما تكون ضعيفة وغير مستمرة إلا أنها فى حوالى ١٠% من المرضى تكون الأعراض مستمرة وعنيفة مما يدفع المريض للبحث عن العلاج.

وقد نجحت العلاجات الطبية الحديثة فى تحسن أعراض المرض وعلاج بعضا لمضاعفات أيضا إلا أن المشكلة هو حدوث تكرر للأعراض فى حال توقف العلاج وهو ما يحدث فيما يقرب من ٨٠% من المرضى كما أنه ثبت على المدى الطويل لاستخدام هذه العلاجات نقص حدوث مضاعفات المرض إلا أنها لا تزال تحدث.

أما عن العلاجات الجراحية لهذا المرض فقد ثبت أنها تحدث شفاءً تاما لكل أعراض المرض كما تمنع حدوث المضاعفات نهائيا ربما زاد من انتشار هذه الجراحات إمكانية إجراء هذه الجراحات بالمنظار الجراحى مما أدى إلى سرعة تماثل المريض للشفاء وعودته إلى الأنشطة اليومية فى وقت محدد.

وهناك جراحات مختلفة لمرض ارتجاع العصارة المعدية للمرئ ومن أشهر هذه الجراحات عملية نيسن والتى تتم إما بالجراحة التقليدية أو باستخدام المنظار الجراح. وقد تم إدخال العديد من التعديلات على هذه العملية مما أدى إلى تحسن نتائج هذه العملية فى علاج المرض وعدم تكرار مضاعفات هذه العملية ومن أشهر هذه التعديلات عملية نيسن روزيت والتى تتميز بعدم قطع الأوعية الدموية التى من بين المعدة والطحال واستخدام الجدار الأمامى للمعدة فى عمل صمام جديد حول أسفل المرئ. وقد حدث جدال لفترات طويلة حول مميزات وعيوب هذه العملية.

وفى هذا البحث تم مقارنة كلا من عملية نيسن بالمنظار الجراحى وعملية نيسن روزيت بالمنظار الجراحى فيما يتعلق بمدة إجراء العملية وحدوث مضاعفات أثناء إجراء الجراحة أو بعدها وأيضا فى قدرة كلا منهم فى منع أعراض المرض.

وقد أثبت هذا البحث أن الميزة الوحيدة لعملية نيسن روزيت باستخدام المنظار الجراحى هو الانتهاء من الجراحة فى وقت أقل لعدم قطع الأوعية الدموية بين الطحال والمعدة. ولكن مع تقدم الوسائل المستخدمة لقطع الأوعية الدموية بالمنظار الجراحى مثل جهاز الهارمونيك أصبح قطع هذه الأوعية الدموية سريع وآمن. وأصبح الفارق الزمنى فى إجراء كلا من الجراحتين بسيط وغير مؤثر. كما أنه ثبت من خلال هذا البحث أن عملية نيسن بالمنظار الجراحى أكثر تفوقا من علمية نيسن روزيت فيما يتعلق بالتحكم فى أعراض المرض وأيضا من حيث تقليل حدوث المضاعفات أثناء وبعد الجراحة.

ومع تفوق عملية نيسن باستخدام المنظار الجراحى على عملية نيسن روزيت باستخدام المنظار إلا أن نتائج كلا منهما تكاد تكون متماثلة فى حالة واحدة وهو وجود فتق بالحجاب الحاجز مع ارتجاع العصارة المعدية للمرئ.

ولهذا فإن هذا البحث يفضل استخدام جراحة نيسن بالمنظار لعلاج مرضى ارتجاع العصارة المعدية للمرض فى كل الحالات مع إمكانية استخدام عملية نيسن روزيت فى حالة وجود فتق بالحجاب الحاجز مع ارتجاع العصارة المعدية للمرئ.

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# دراسة مقارنة بين أسلوب نيسن وأسلوب نيسن روزيت لعلاج إرتجاع عصارة المعدة للمريء باستخدام المنظار الجراحى

## رسالة

توطئة للحصول على درجة الدكتوراه
فى **الجراحة العامة**

## مقدمة من

الطبيب/ هانى سعيد عبد الباسط
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