INTRODUCTION

Gastroesophageal reflux is a normal physiological phenomenon experienced intermittently by most people, particularly after a meal. Gastroesophageal reflux disease occurs when the amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury (i.e., esophagitis) *(Fisichella et al., 2007).*

Studies of the natural history of gastroesophageal reflux disease indicate that most patients have a relatively benign form of the disease that is responsive to lifestyle changes and dietary and medical therapy and do not need surgical treatment. Approximately 25% to 50% of the patients with gastroesophageal reflux disease have persistent or progressive disease, and it is this patient population that is best suited to surgical therapy. Fundoplication is indicated in operable patients with objectively documented gastroesophageal reflux disease whose symptoms/signs have not been controlled by medical management with proton pump inhibitor (PPI) therapy and other measures *(Jeffrey, 2005).*

Better understanding of the pathogenesis of gastroesophageal reflux disease in recent years has not been accompanied by appreciable advances in the design of antireflux operations. In many cases, operations are still being performed just as they were described 30 years ago. It is important now to go beyond the eponymous procedures traditionally associated with antireflux operations and to identify the technical elements that contribute to effective and durable fundoplications *(Patti et al., 1998).*

There are many operations for gastroesophageal reflux disease. The major types of antireflux operations were all developed in the 1950. Nissen described total fundoplication in which the fundus of the stomach is wrapped completely around the lower oesophagus. This procedure increases the pressure at the lower end of the esophagus and thereby reduces acid reflux. Laparoscopic fundoplication has become commonplace and has replaced the open abdominal Nissen fundoplication as the procedure of choice. Nearly all published reports of laparoscopic fundoplication show that this procedure relieves the typical symptoms of gastroesophageal reflux in greater than 90% of patients *(Baily and Love, 2004).*

Laparoscopic Nissen fundoplication is currently the most commonly practiced antireflux operation. Some adverse consequences of the operation remain in the form of mechanical side effects, labeled postfundoplication complaints, of which dysphagia and gas bloat seem to predominate. Measures have been suggested to counteract some of these and one frequently advocated has been division of the short gastric vessels to create a short-floppy wrap. The advantages of this are still debated *(Engström et al., 2004).*

There are today a significant greater number of laparoscopic antireflux procedures for the surgical treatment of gastroesophageal reflux disease and there are yet controversies about the necessity of division of the short gastric vessels and full mobilization of the gastric fundus to perform an adequate fundoplication *(Felix et al., 2002)*.

To divide or not the short gastric vessels during Laparoscopic Fundoplication is still controversial. It has been suggested that routine division of short gastric vessels results in a more "floppy" Nissen fundoplication leading to improved outcomes, that is, less dysphagia and lower incidences of recurrent gastroesophageal reflux disease *(Liu et al., 2006)*.

AIM OF THE WORK

The aim of this prospective study is to assess whether laparoscopic Nissen fundoplication without division of short gastric vessels (Rossetti modification) (laparoscopic Nissen-Rossetti fundoplication) is associated with acceptable clinical outcome in patients with gastroesophageal reflux disease in comparison with the original laparoscopic Nissen fundoplication with division of short gastric vessels.

ANATOMICAL CONSIDERATIONS

Embryology:

The esophagus comes from two sources of the primitive gut. The cranial portion is derived from the pharyngeal gut or pharynx, and the caudal part from the pregastric segment of the foregut. With the growth of the embryo, the primitive gut lumen becomes almost filled but later, due to a process of epithelial layer vacuolization hollows out again. At about 4 weeks of embryonic development, the laryngotracheal groove appears, subsequently forming the tracheobronchial diverticulum on the ventral surface of the foregut, at the level of the fourth pharyngeal pouches. The diverticulum is gradually closed by the tracheoesophageal folds (internal ridges of the lateral esophageal groove), caudally first, forming the tracheoesophageal septum *(Lerut et al., 2007)*.

The endoderm forms the mucosal epithelium and associated ducts and glands. The mesoderm forms the lamina propria, muscularis mucosa, and muscular coat; the branchial arches form the striated muscle; and the visceral splanchnic mesoderm forms the

smooth muscle coat. Arterial and venous supply of the esophagus is segmental. The cranial arteries are derived from the branchial arches and the caudal arteries from branches of the aorta. With the unfolding and lengthening of the embryo, the esophagus also lengthens. The original cell lining of the esophagus changes from a two- to three-layer pseudostratified columnar epithelium via a stratified columnar stage to a stratified squamous epithelium by 90 to 130 mm embryo length *(Lerut et al., 2007)*.

Anatomy of lower esophageal sphincter:

The lower esophageal sphincter (LES) is located at the terminal end of the esophagus and represents the distal 3 to 5 cm of the organ (Figure 1). Most of the LES lies within the abdominal cavity, but a short segment often extends above the diaphragm. A small condensation of circular smooth-muscle fibers resides in this region, too indefinite to constitute an anatomical sphincter *(Haile, 2004)*.

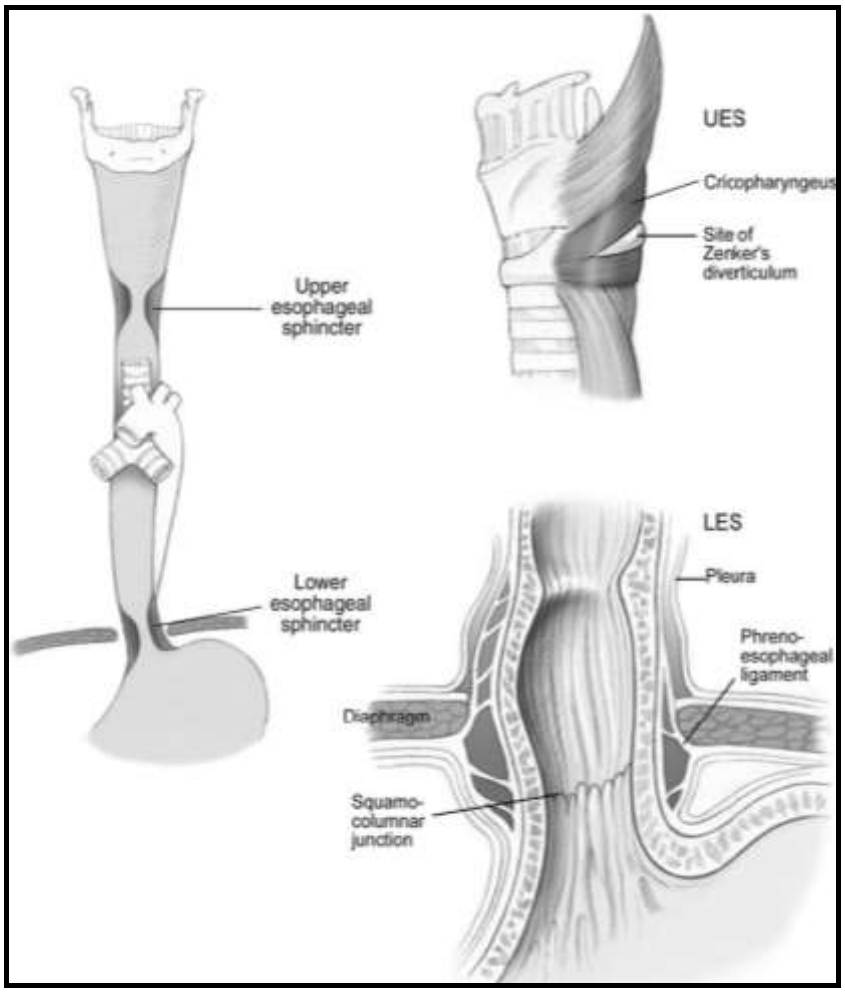

Fig. (1): The upper esophageal sphincter (UES) and lower esophageal sphincter (LES) ***(from Rothberg et al, 1989)***.

Below the tracheal bifurcation, the esophagus runs to the right of the descending aorta, posterior to the fibrous pericardium, and in between the left and right mediastinal pleura. The anterior and posterior vagal trunks run longitudinally on the esophageal surface, and the esophagus passes through the diaphragm to the left

of the midline into the abdomen. As it does so, there is a fat pad that marks the position of the superior margin of the phrenoesophageal ligament or membrane, a condensation of pleura, endothoracic fascia, transversalis fascia, and peritoneum, and consists of two sheaths *(Hagen et al., 2005)*.

The upper sheath of the phrenoesophageal ligament is inserted into the lower 1 to 3cm of the thoracic esophagus. Although the fibers of this sheath are mainly inserted into the outer adventitial layer of the esophagus, some penetrate the wall as far as the submucosa. The lower sheath is inserted into the cardia but is separated from the gastroesophageal junction by a fat pad and loose areolar connective tissue. At the level of the gastric fundus below the cardia the fibers of this lower sheath disappear into the serosa and the gastrohepatic and gastrosplenic ligaments. As a result, the phrenoesophageal membrane is well developed anteriorly but weak posteriorly. Elastic and collagenic fibers are its main elements at histologic examination. They are arranged in bundles forming a meshwork resulting in the necessary strength and plasticity to cope with the continuous respiratory movements of the diaphragm *(Hagen et al., 2005).*

The phrenoesophageal segment (figure 2) acts as a barrier between the negative intrathoracic pressure and the positive abdominal pressure. It provides at the same time an air-tight seal between the thoracic and abdominal cavities. Studies of patients of different ages have suggested that the inferior leaf of the phrenoesophageal ligament is weaker than the superior, and with advancing age the ligament ascends into the thorax. The esophagus parts accompany the aorta as it descends into the abdomen, moving anteriorly to reach the esophageal hiatus, the aorta passing posteriorly to the median arcuate ligament *(Lerut et al, 2007).*

The crural arrangement is complex and variable. The crura have an origin from the first four lumbar vertebrae. The left crus does not divide but the right crus has superficial and deep components, the latter forming a sling around the esophagus.

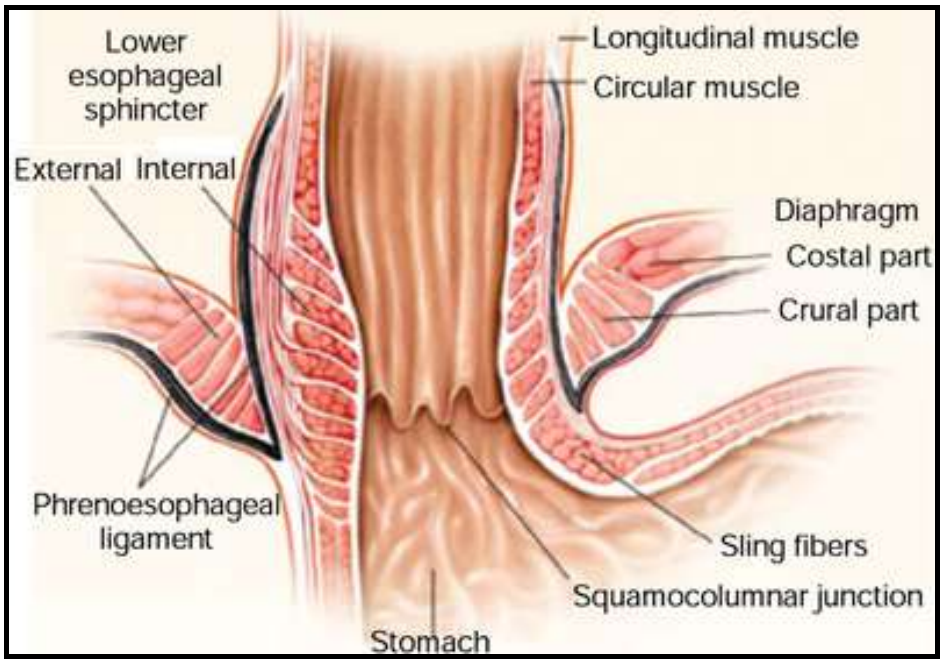

Fig. (2): Diagram showing the distal esophagus, gastroesophageal junction, and formation of the phrenoesophageal ligament. Attachment and structure of the phrenoesophageal membrane ***(From Hagen et al, 2005)***.

In the most common arrangement the superficial fibers of the right crus pass anterosuperiorly in a lazy curve to insert into the central tendon of the diaphragm, forming the right pillar; the deeper fibers twist acutely to wrap around the left side of the esophagus, forming the left pillar. The left crus lies to the left of this, passing to the left of the aorta, and therefore is not an immediate relation to the esophagus. However, in 40% of patients the sling does include components of the left crus. Where the right

and left crura cross, the median arcuate ligament forms. Posterior to the median arcuate ligament is the descending thoracic aorta as it passes through the diaphragm at the level of the 12th thoracic vertebra. As the esophagus passes the hiatus, a concentric narrowing of the lumen occurs (which, however, is not synonymous with the high-pressure zone, although the high-pressure zone extends into this area) *(Lerut et al., 2007)*.

The length of the intra-abdominal segment is said to be from 0.5 cm to 2.5cm. The angle formed between the left margin of the intra-abdominal segment and the gastric fundus is referred to as the angle of His. The intra-abdominal esophagus passes into the cardia of the stomach. At this point the density of the circular muscle fibers of the esophagus is the greatest, particularly on the lesser curve side, which, combined with the similarly dense oblique muscle fibers of the stomach, contribute to the antireflux mechanism of the lower esophageal sphincter, together with the anatomic relation between the esophagus and the area of the hiatus. The abdominal esophagus is invested in a peritoneal covering consisting of the two layers of the hepatogastric ligament. The hepatogastric ligament

extends from the porta hepatis to the lesser curvature of the stomach and abdominal esophagus. The hepatogastric ligament encloses the abdominal esophagus on the right and it comes together on the left side to form the gastro-splenic ligament. The lesser sac lies behind these two ligaments so that there is a bare area in the posterior wall of the stomach *(Duranceau et al, 1991).*

From an anatomical surgical point the antireflux mechanism is based on the following elements:

- Thickening of the gastric oblique sling fibers and the clasp fibers that form the lower esophageal sphincter.
- Intra-abdominal pressure.
- Rosette-like configuration of the top end of the gastric mucosal folds.
- The sharp angle of the fundus (angle of His).
- The phrenoesophageal membrane and its insertion into the esophagus.
- The pinching of the diaphragmatic crura.

(Lerut et al, 2007)

Physiological basis:

The specialized nature and unique innervation of the smooth muscle of the terminal esophagus provide the basis for a physiological sphincter. These specialized smooth-muscled fibers contain receptors for a number of peptide (e.g., vasoactive intestinal peptide, substance P) and nonpeptide neurotrans-mitters that are released locally from peptidergic and nonpeptidergic neurons. The LES is a high-pressure zone interposed between the body of the esophagus and the cardia of the stomach (Fig. 3) *(Haile, 2004).*

Normally, a mean pressure of 20±5mmHg is maintained at all times except during swallowing, when the pressure falls to 0mmHg, allowing esophageal peristalsis to empty the swallowed food into the stomach. In its resting state, therefore, the high pressure of the LES prevents reflux of gastric contents into the esophagus. The LES contracts and increases its pressure in response to sudden increases in abdominal pressure and to alkalinization of the gastric lumen. As mentioned above, the LES relaxes in response to swallowing, a function mediated by local release from neurons in the LES musculature of vasoactive intestinalpeptide (VIP) and nitric oxide

(NO). Other factors contributing to the lowering of LES pressure are nicotine, gastric acidification, ingestion of fats, and release of cholecystokinin (CCK) *(Haile, 2004).*

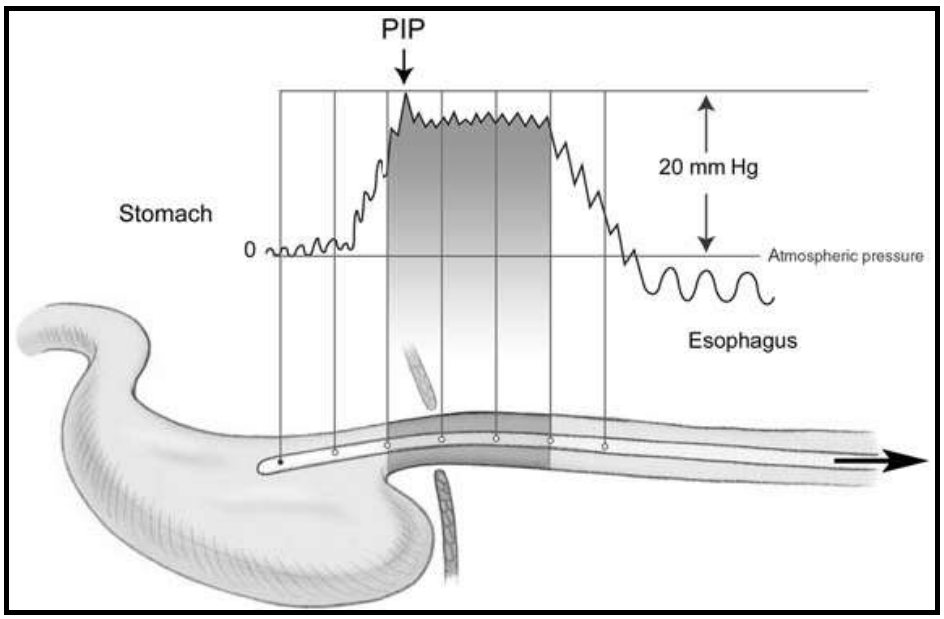

Fig. (3): The lower esophageal sphincter (LES) can be identified by measuring the resting pressure of the distal esophagus at various points. It is 3.0-5.0cm in length, with a mean resting pressure of 20±5mmHg. When the sensing device is below the diaphragm, inspiration causes a positive pressure inflection. The opposite is true above the diaphragm. The pressure inversion point (PIP) indicates the level of the diaphragm *(From Haile, 2004)*.

Blood and nerve supply and lymphatics:

The esophagus is nourished by numerous segmental arteries, all of which contribute to the extensive capillary network. The cervical esophagus

receives blood from the superior thyroid artery as well as the inferior thyroid artery, with both sides communicating through collateral vessels. The major blood supply of the thoracic esophagus is from four to six aortic esophageal arteries, supplemented by collateral vessels from the inferior thyroid, intercostal and bronchial, inferior phrenic and left gastric arteries. The aortic esophageal arteries terminate in fine capillary networks before they actually penetrate the esophageal muscle layer. After penetrating and supplying the muscle layers of the esophagus, the esophageal capillary network runs longitudinally in the submucosa. The extensive venous drainage of the esophagus includes the hypopharyngeal, azygous, hemiazygous, intercostal, and gastric veins *(Joseph, 2004)*.

The nerve supply of the esophagus derives from the vagus and the cervical sympathetic trunk. The vagus provides the essential innervation of the body and sphincters of the esophagus. The cervical esophagus is innervated by branches of the recurrent laryngeal nerves, while the thoracic and abdominal portions are innervated directly from the vagal neural plexus surrounding the organ. At the hiatus, considerable variation exists from individual to individual. In about 60% of human subjects, the