HIATAL HERNIA AND GASTROESOPHAGEAL REFLUX DISEASE (GERD)

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BACKGROUND

1. HH found in 50% of the adult population.

2. Symptomatic GER is also common:

   11% → daily heartburn

   12-15% → weekly or monthly symptoms

3. ↑incidence of esophageal adenocarcinomas is related to ↑ incidence of Barrett's esophagus in this population

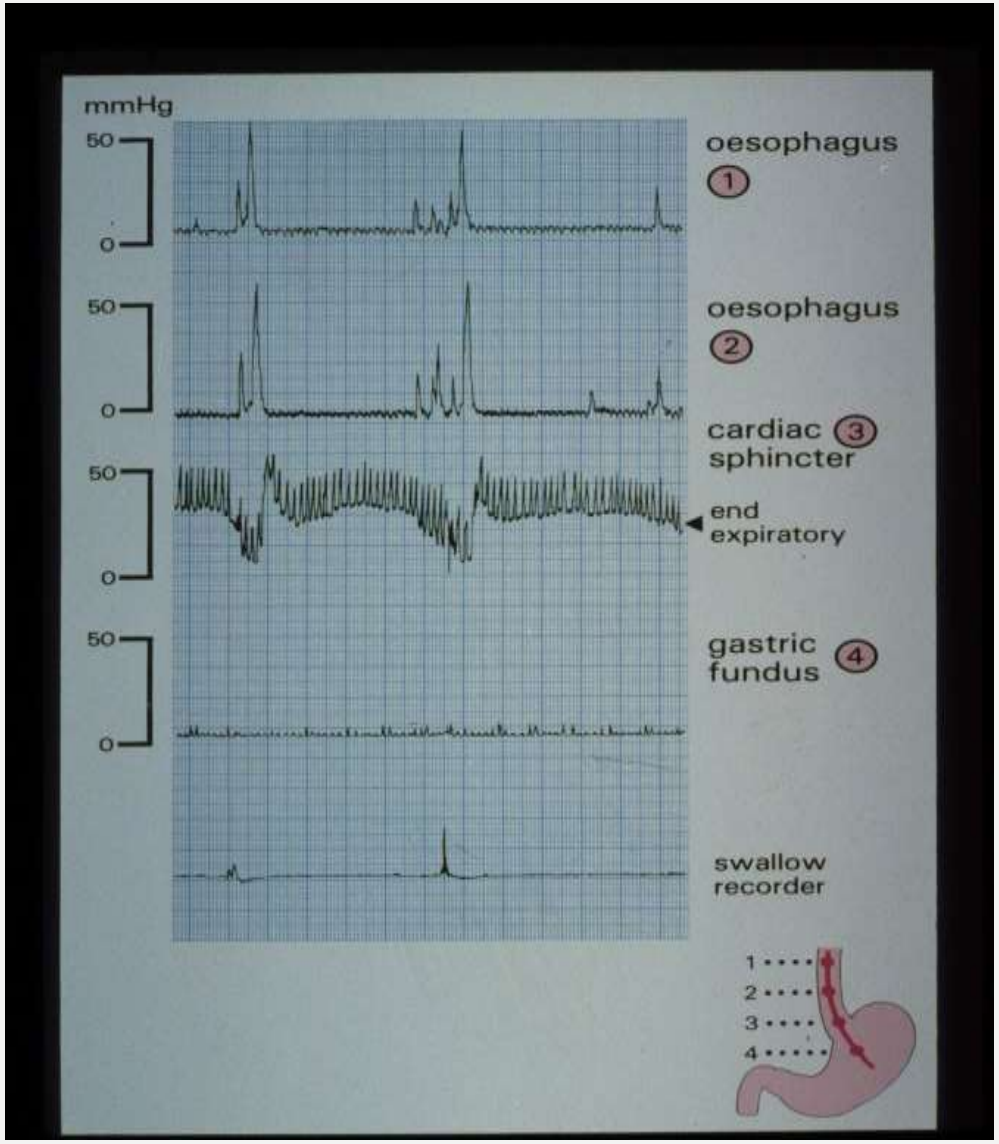
4. Medical liability:

   37% of liability problem (esophagus) → treatment of HH and GER.

   50% of the costs for liability problems (esophageal surgery) → HH and GER

ANATOMY OF THE ESOPHAGUS (1)

- **Muscle: (striated and smooth)**
  - Inner circular - high-pressure zone
  - Outer longitudinal
- **Submucosa:**
- **Mucosa:** squamous → columnar 2 cm. above the anatomic GEJ (Z line)
- **Esophageal hiatus:**
  - Right crus L1-L4
  - Left crus L1-L2
  - → central tendon of the diaphragm


mmHg
50
0
oesophagus
1
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2
cardiac
3
sphincter
end
expiratory
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4
fundus
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recorder
1
2
3
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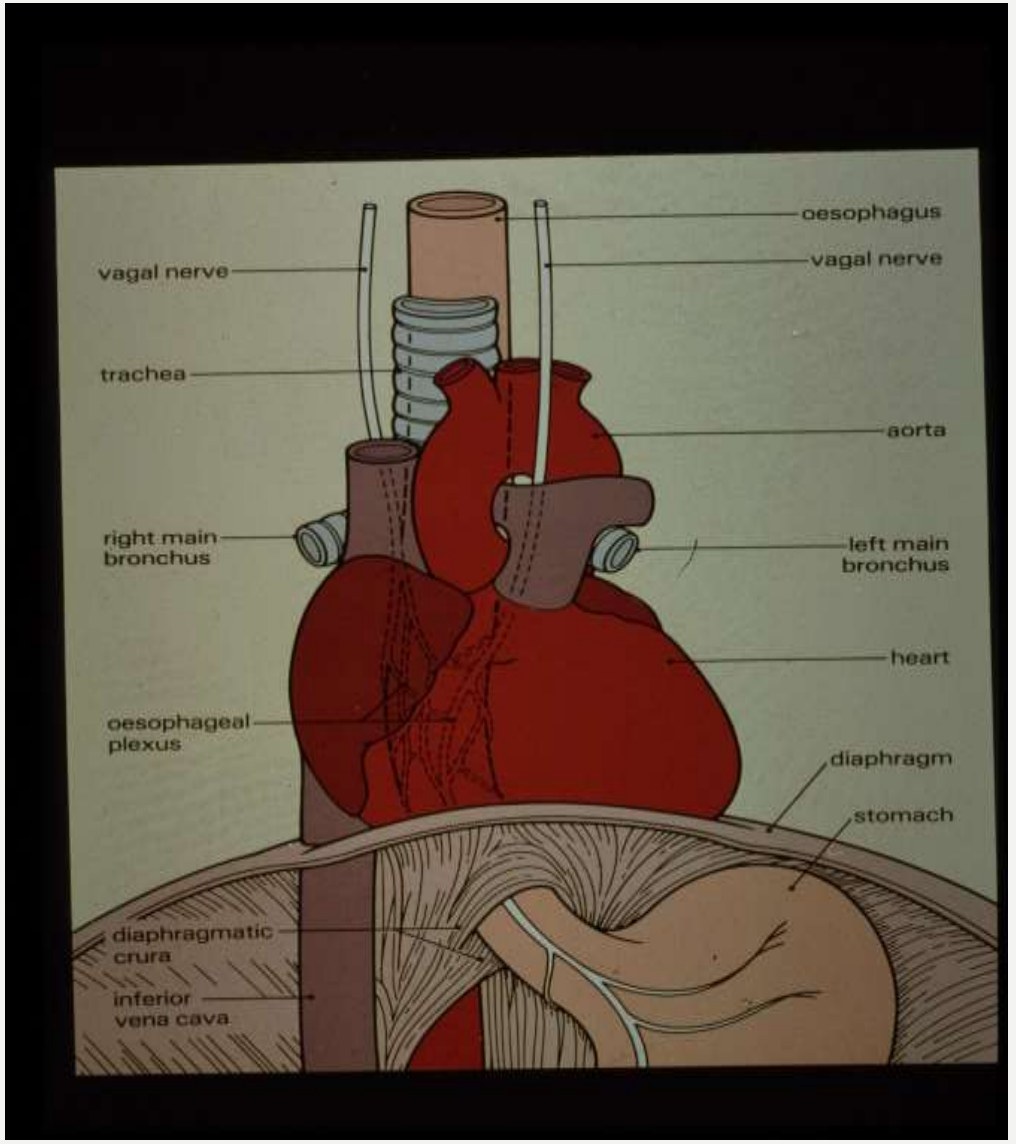
oesophagus
vagal nerve
vagal nerve
trachea
aorta
right main
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crura
inferior
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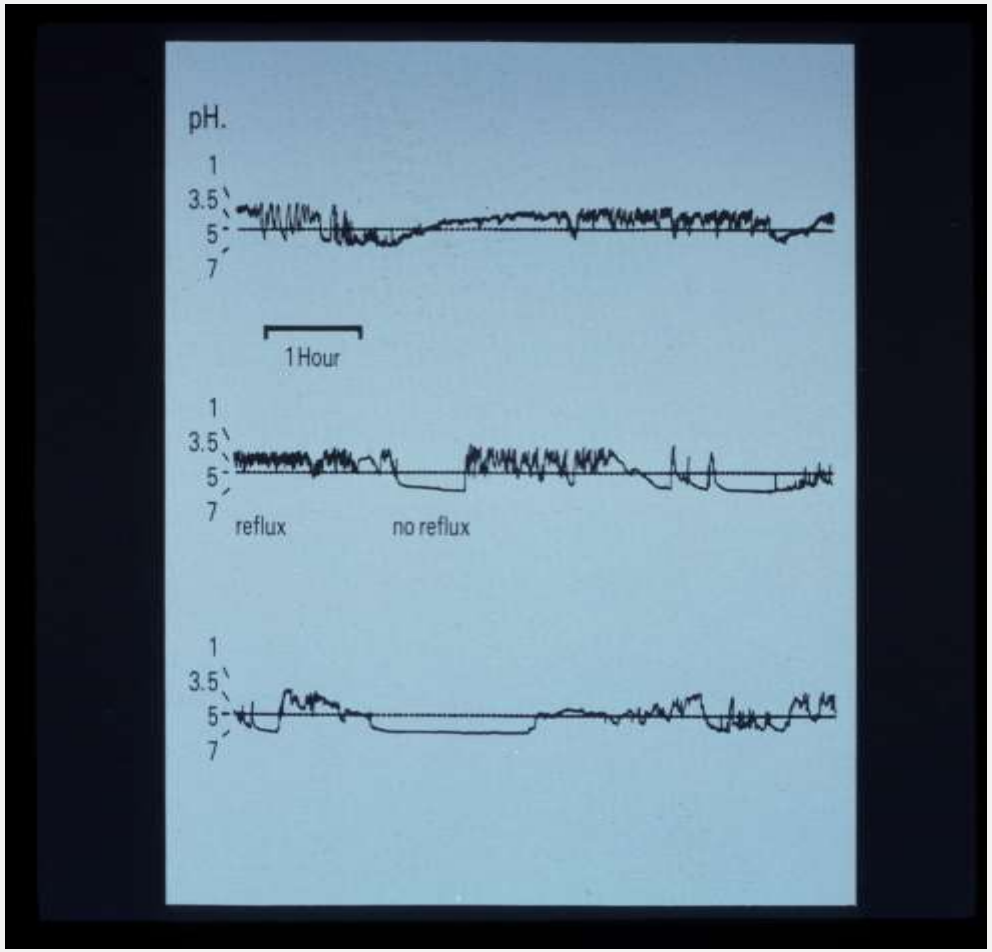
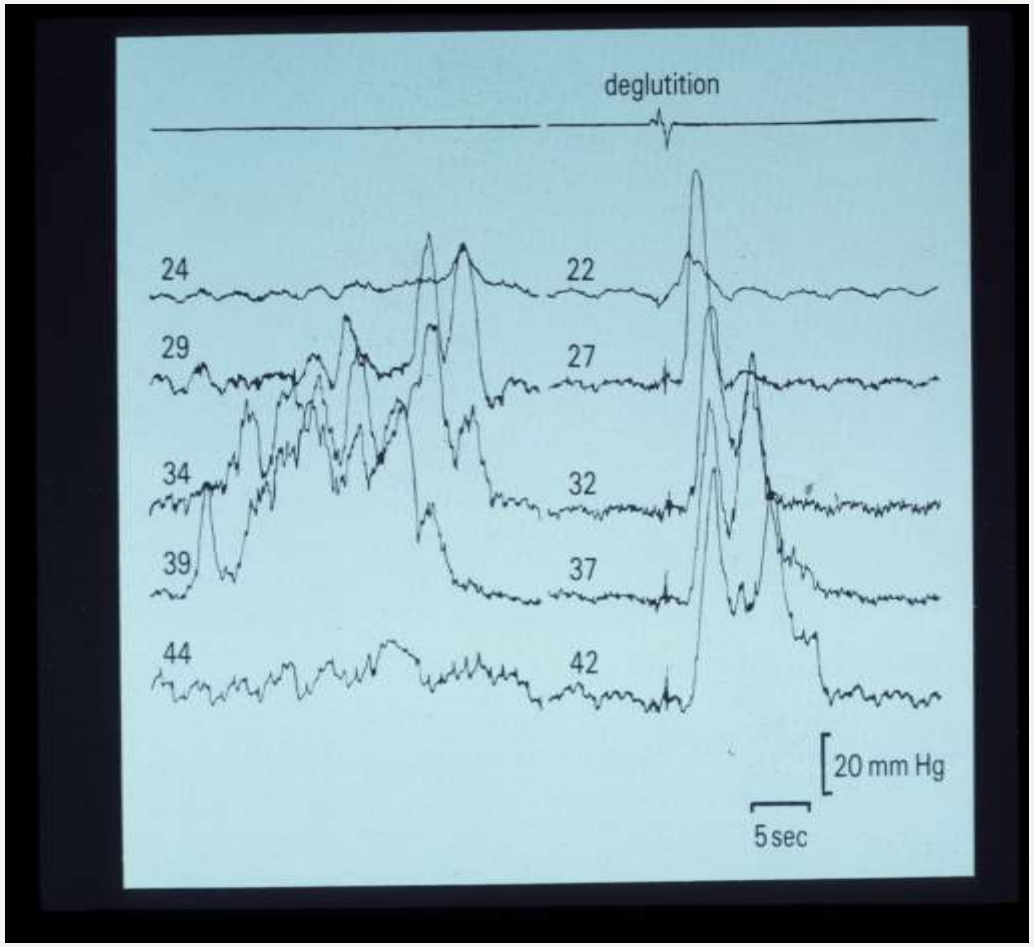
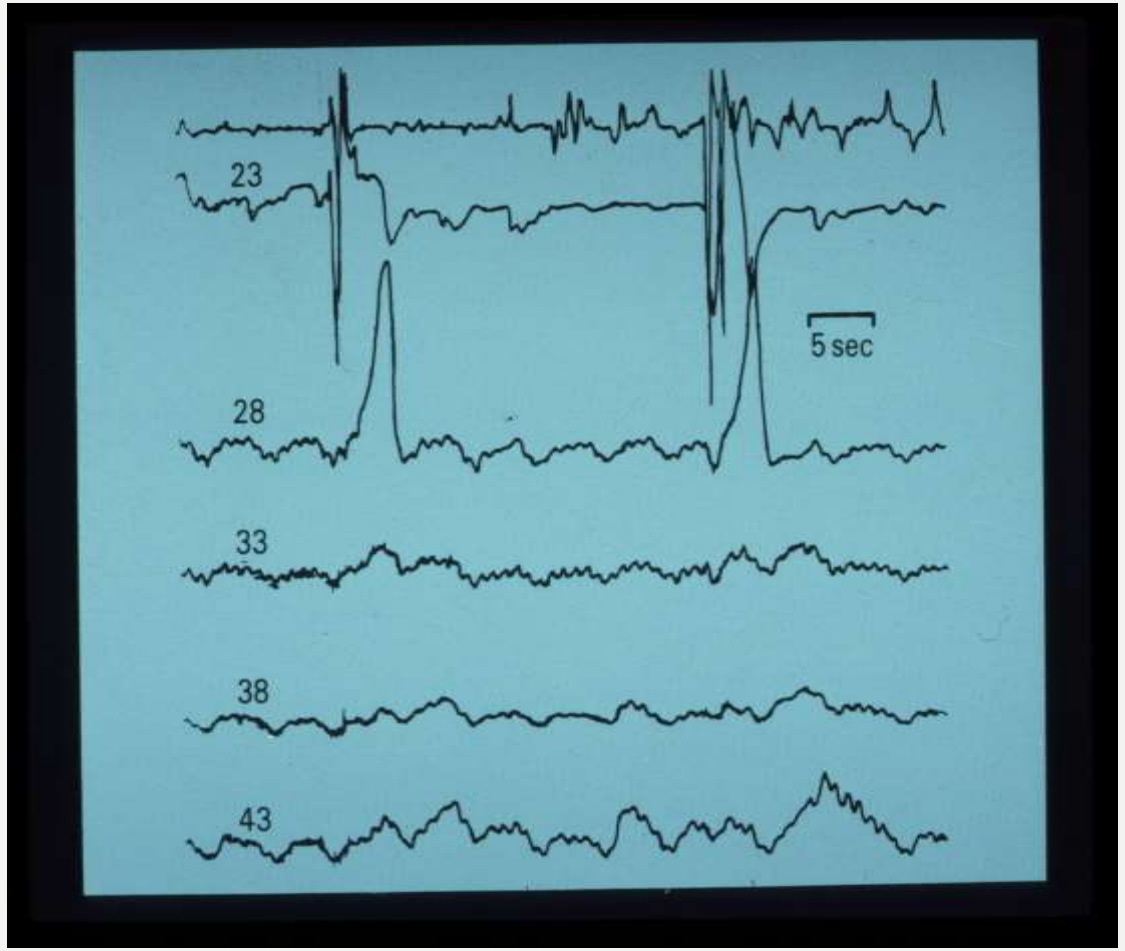

pH.
1
3.5
5
7
1 Hour
reflux
no reflux


deglutition
24
22
29
27
34
32
39
37
44
42
20 mm Hg
5 sec


23
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28
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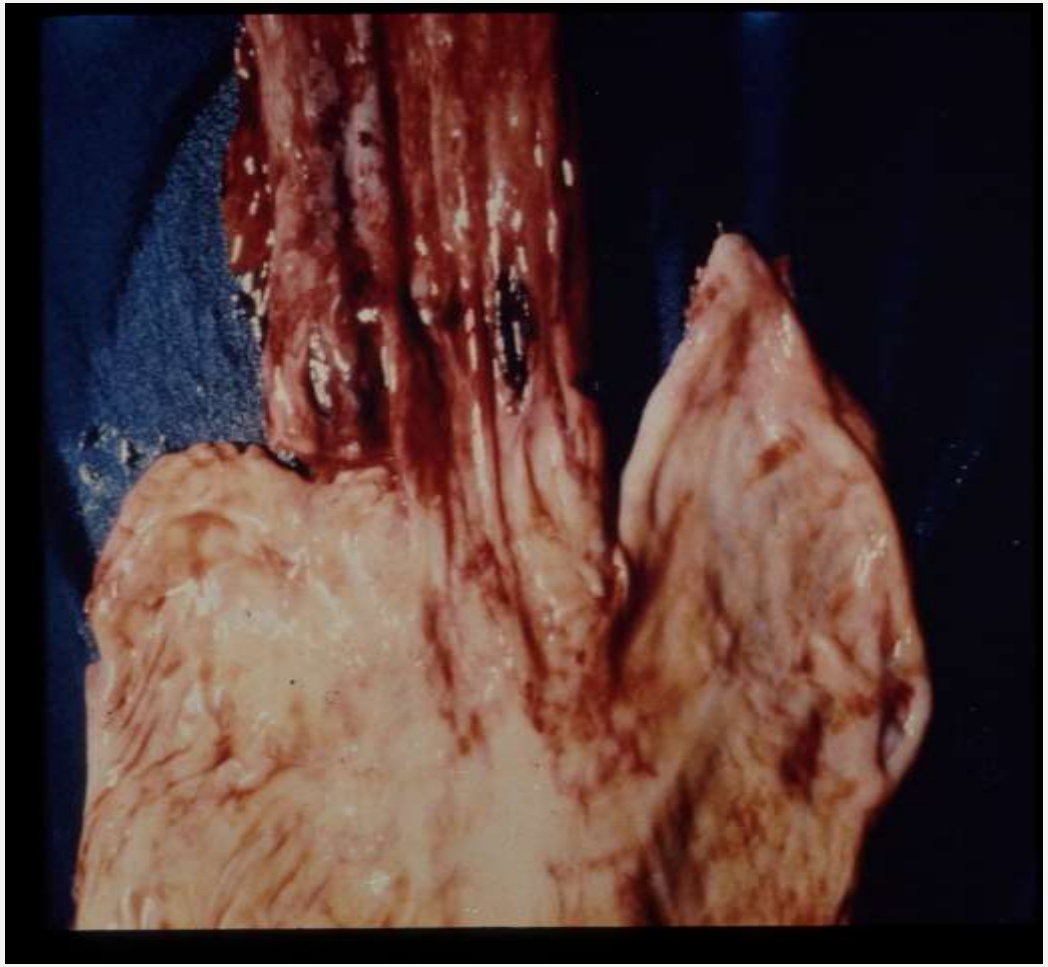
38
43



ANATOMY OF THE ESOPHAGUS (2)

The tissue maintaining the esophagus in the hiatus consists of :

a) Pleura
b) Subpleural endothoracic fascia
c) Phrenoesophageal proximal fascia
d) Transversalis endoabdominal fascia
e) Peritoneum

The GEJ is held in place in the diaphragmatic hiatus by the **phrenoesophageal membrane**

ESOPHAGEAL CONTRACTIONS

- Voluntary deglutition → pharyngeal contraction → close the upper esophageal sphincter → the swallowing wave start the **primary** wave of peristalsis → traverses both the striated and smooth muscle of the esophagus as a continuous wave.

Speed	3.5 cm/second
Amplitude	90-109 mm. Hg
Duration	5 seconds

- **Secondary** peristalsis is an esophageal wave **not** preceded by swallowing. It is stimulated by esophageal irritants (e.g., acid) (efficient defense mechanism)

- **Tertiary** contractions are nonpropulsive contractions in the esophageal body, and they are either spontaneous or may follow deglutition.

GEJ

- Is a high-pressure zone of **3-4 cm in length** located just above the junction of the esophagus with the stomach. The muscle arrangement at this level creates a radial asymmetry, with the highest pressures measured in the left posterior area.

- It is composed of smooth muscle and is under noncholinergic and nonadrenergic control mechanisms. The sphincter is maintained in a contracted state, mostly from intrinsic myogenic activity, but its resting tone is affected by numerous neural and hormonal factors.

Substances Influencing LES Pressure

Substance	Increase Pressure	Decrease Pressure
Hormones	Gastrin, Motilin , Substance P	Secretin, Cholecystokinin, Glucagon
		Somatostatin , VIP, Progesterone
Neural agents	α-Adrenergic agonists	α-Adrenergic antagonist
	β-Adrenergic antagonists	β-Adrenergic agonists
	Cholinergic agonists	Cholinergic antagonists
Foods	Protein	**Fat, Chocolate, Ethanol**
		Peppermint, Tobacco Smoking
Medication	Histamine, Antacids	Theophylline, Prostaglandins E2 and I2
	Metoclopramide, Domperidone	Morphine, Dopamine
	Prostaglandin F2a	Calcium channel blockers, Nitrates
		Diazepam, Oxazepam, Barbiturates

LOWER ESOPHAGEAL SPHINCTER (LES)

- The resting pressures in the LES is **24.8** mm. Hg
- The resting intragastric pressure is **7.3** mm. Hg
- A gradient of **17.5** mm. Hg between the esophagus and the stomach.
- The tone of the sphincter increases in response to increased intra-abdominal pressures and during gastric contractions.
- The LES relaxes at the same time as a peristaltic wave approaches it. Esophageal peristalsis pushes a bolus in the esophageal lumen toward the gastric cavity
- The bolus then traverses the relaxed sphincter, which closes once the bolus has passed through.
- Closing pressures of the sphincter are about double the resting values in the sphincter. Pressure within the sphincter then returns to resting levels. LES relaxation occurs similarly with secondary peristalsis
-

TRANSIENT LES RELAXATIONS

- After meals, when the stomach is full, **transient LES relaxations** unrelated to peristalsis occur, and these relaxations last from **5 to 30 seconds**.

- It is during such transient relaxations that much reflux occurs, both in normal individuals and in patients with reflux disease.

FACTORS PREVENTING GER

1. Intrinsic LES tone
2. Saliva (weak base)
3. Esophageal clearance (influenced by position, bolus consistency, and esophageal pH)
4. Squamous epithelium (normally impermeable to H^+)
5. Muscular organization of the sphincter
6. Length of the intra-abdominal esophagus
7. Acute angle of entry of the GEJ and its valve action
8. Integrity of the supporting structures to maintain proper positioning of the GEJ within the hiatus
9. Crural diaphragm (squeeze ability)

PATHOPHYSIOLOGY OF REFLUX DISEASE

- Normal individuals have daily physiologic reflux. These reflux episodes cause neither symptoms nor histologic changes.

- When reflux episodes cause symptoms, either by an increase in amount or change in composition or increase in frequency, then the pathologic entity of gastroesophageal reflux disease is present.

LES FUNCTION

- Low basal LES pressure in patients with HH and GER.
- Patients with resting LES pressures **< 6 mm. Hg** show excellent correlation with their symptoms.
- Histologic evidence of esophagitis is ↑ when the LES tone is weak
- A poor LES tone at the lower limits of normal (**6 to 10** mm. Hg) suggests the possibility of reflux.
- When the LES is **< 6 mm. Hg**, abnormal reflux is probable
- When there is **no** measurable sphincter tone, significant pathologic reflux is usually present.

ESOPHAGEAL BODY FUNCTION (1)

- Primary and secondary peristalsis represent the most useful defense mechanism of the esophagus.

- Abnormal motility affecting the esophageal function is observed in patients with symptomatic HH

- Only a minority of patients with GER presented with failure of peristalsis. There is, however, a significant ↑ in **failed peristalsis** and a **loss of peristalsis strength** with ↑ mucosal damage.

ESOPHAGEAL BODY FUNCTION (2)

- When extensive circumferential **CLE** is present, there is an ↑ in **failed peristalsis** and an ↑ in **tertiary contractions** in response to deglutition.

- Loss of function → **more prolonged exposure** to any refluxate with an impaired esophageal clearance and emptying capacity.

- Significant functional abnormalities can be detected when both acid and alkaline reflux were responsible for the esophageal damage.

- Esophageal emptying capacity: normal individuals take a mean of 2 swallows (range, 1-9) to clear their esophagus of a solid bolus while patients with documented reflux take a median of 5 swallows (range, 1-30) to achieve the same results.

OTHER FACTORS AFFECTING REFLUX: (1) TYPE OF REFLUXATE

- Unaltered gastric juice cause esophageal inflammation and ulceration.
- Bile alone does not cause esophagitis, but bile and pancreatic juice together cause significant damage
- If HCL is infused, erosive esophagitis can be produce, but the mucosal damage is increased when pepsin is added
- HCL causes further damage when mix with bile and bile salts.
- The digestive enzymes mixed with acid and bile salts induce loss of impermeability in the squamous epithelium.
- Back-diffusion of the H^+ causes damage at the level of the middle and basal layers of the mucosa
- Clinically, a mix of acid and bile is present in the more severe forms of reflux disease
- Patients with reflux esophagitis and the presence of Barrett's metaplasia show a greater exposure to both acid and alkaline reflux than patients with simple esophagitis.

(2) GASTRIC EMPTYING

- A significant number of patients with GER show abnormal liquid and/or solid emptying pattern. If delayed gastric emptying is a causative factor it is probably through the mechanism of prolonged **fundal distention**, which increases the frequency of transient LES relaxations.

(3) ANATOMIC ALTERATION OF THE GEJ

- Operations at the GEJ alter supporting structures at the hiatus
- Surgical alterations of the diaphragm or the phrenoesophageal membrane do not alter the physiologic characteristics of the GEJ, but they may expose the intra-abdominal esophagus and the hiatal structures to a modified response when intra-abdominal conditions are altered
- The presence of a HH in itself does not produce pathologic reflux, acute increases in intra-abdominal pressures, especially in obese patients, can decrease the GEJ resistance, favoring reflux when the LES barrier is weakened
- Resection of the GEJ is always followed by reflux esophagitis, especially when reanastomosis of esophagus and stomach is completed in the lower chest
- If total gastrectomy is not reconstructed with the use of a Roux-en-Y reconstruction to exclude biliary and pancreaticoduodenal secretions, esophageal mucosal damage invariably occurs
- Myotomy of the distal esophagus and GEJ in the treatment of motor disorders decreases substantially the muscle tone of the LES, and this also favors GER and its complications.

(4) OBESITY

- **It** is associated with abnormal reflux. The delayed gastric and esophageal emptying in these patients, with possible changes in the gastroesophageal resistance by the resulting intra-abdominal conditions, may represent major factors leading to reflux symptoms and esophageal mucosal damage

- The incidence of reflux episodes is directly related to the size of the diaphragmatic hiatus, herniation of the stomach with impairment of the circumferential squeeze ability of the crural diaphragm in these patients possibly facilitates reflux.

(5) SOCIAL HABITS AND MEDICATION

INVESTIGATION AND STAGING OF HH AND GERD

- HH and GERD are measurable conditions, and they should be staged objectively to assess severity and degree of reflux.

(1) SIGNS AND SYMPTOMS

- Three categories of clinical features in patients with reflux disease

Most Common	Regurgitations, Heartburn
Related to Complications of Reflux Disease	Dysphagia, Odynophagia, Hematemesis, Melena
Unrelated to Esophageal Damage	Oropharyngeal dysphagia, Asthma, Chest pain, Hoarseness, Pharyngitis

- Features present with larger hernias are caused mostly by the volume occupied by the hernia in the mediastinum.
- **Postprandial fullness**, **substernal and parasternal discomfort**, or **pain** and **vomiting** may occur after eating.
- **Acute dysphagia with severe epigastric or chest pain** is ominous and suggests intermittent volvulus and the danger of strangulation.
- Unexplained **anemia** is sometimes seen with these large hernias.

(2) RADIOLOGY

- Four types of hiatal hernia have been defined. The radiologic examination also aims at documenting the mucosal alterations and esophageal wall complications present with reflux disease.

Type	Description
H0	No hiatal hernia
H1	Sliding hernia. GEJ above diaphragm
H2	Normal position of GEJ. Protrusion of the stomach alongside the esophagus
H3	Components of sliding and paraesophageal hernias GEJ is in the chest. Stomach rolls through the hiatus in a paraesophageal position
H4	Large hiatal defect with components of the sliding and/or paraesophageal hernia accompanied by another abdominal organ (colon, spleen, pancreas, small bowel)

(3) ESOPHAGOSCOPY

- The **MUSE** classification encompasses all aspects of mucosal damage, describing the changes of metaplasia (M), ulcers (U), strictures (S), and erosions (E). No place is given to minimal degrees of mucosal alteration as those suggested by hyperemia. Each category of mucosal damage can be scored on its own for a more objective quantification of lesions.

Equivocal Evidence of Esophagitis	E0 = normal
	E1 = erythema
Unequivocal Evidence of Esophagitis	E2 = erosions and ulceration
	E3 = stricture or columnar-lined esophagus

(4) ESOPHAGEAL BIOPSIES (HISTOLOGY)

- The initial events leading to mucosal damage involve an increase of the basal layer to form > 15% of the epithelial thickness.
- The papillae from the lamina propria penetrated > than two thirds of the epithelial thickness and come to lie in close proximity to the esophageal lumen.
- Repeated insults, especially by a refluxate containing acid, bile, and pancreatoduodenal enzymes, may result in ulcers becoming covered by a columnar mucosa growing in a cephalad direction. This epithelium is abnormal in function and may show three types of columnar cells: gastric, junctional, or intestinal (specialized).

Equivocal Evidence of Esophagitis	H0 = normal
	H1 = basal cell hyperplasia
Unequivocal Evidence of Esophagitis	H2 = acute epithelial or subepithelial inflammation and/or ulceration
	H3 = fibrosis or columnar-lined esophagus

(5) MOTILITY STUDIES

- Studies quantifying motor function of the esophagus provide the best evidence for the presence or absence of the physiologic abnormalities associated with gastroesophageal reflux disease

Equivocal Evidence of Functional Abnormality Associated with Reflux	M0 = normal esophageal motility, LES >10 mm. Hg
	M1 = normal esophageal motility, LES 6–10 mm. Hg
Unequivocal Evidence of Functional Abnormality Associated with Reflux	M2 = normal esophageal motility, LES <6 mm. Hg
	M3 = aperistaltic esophagus, LES <6 mm. Hg

(5) MOTILITY STUDIES (CONT.)

- Esophageal body peristalsis is usually normal when reflux episodes are associated with minimal wall damage
- Esophagitis alters function in a manner proportional to damage severity
- Patients with severe esophagitis have an increased incidence of **failed peristalsis** and **weak peristaltic contractions**
- Patients with a Barrett's esophagus tend to show the worst functional changes
- Motility studies offer prognostic information when physiologic damage is present.
- Normal function in the esophageal body with an LES pressure gradient above 6 mm. Hg remains equivocal evidence of the dysfunction leading to reflux damage
- A sphincter gradient when absent or < 6 mm. Hg identifies patients at significant risk for reflux and with a poorer prognosis for long-term medical therapy.
- Absent peristalsis and absent LES tone usually accompany the worst categories of reflux disease as seen in the patient with scleroderma and the patient with end-stage esophagitis.

(6) MONITORING OF ACID AND BILE REFLUX

- 24-hour pH monitoring is the most precise measure of the presence of acid in the esophageal lumen

- The amount, frequency, and time of acid exposure are documented by a glass or antimony electrode placed 5 cm. above the LES. Multiple probes can be placed at various levels in the esophagus

- These objective measurements, when technically adequate, measure and correlate acid reflux episodes with typical esophageal symptoms. Atypical chest pain, asthma episodes, and oropharyngeal symptoms must show close correlation with reflux episodes and mucosal damage before being attributed to reflux disease.

(6) MONITORING OF ACID AND BILE REFLUX

- When direct measurement of bile reflux is needed, the information is obtained by a new fiberoptic probe, which recognizes bilirubin and allows quantification of bile exposure on the gastric and on the esophageal mucosa
- Normal acid exposure in the esophagus is suggested to vary between 4.2% and 7%
- The staging of severity in regard to acid pH parameters is noted in Table 7

Equivocal Evidence of Reflux	**R0 = no reflux or <4%**
	R1 = reflux 4–7%
Unequivocal Evidence of Reflux	**R2 = reflux 7–12%**
	R3 = reflux >12%

(6) MONITORING OF ACID AND BILE REFLUX

- Although currently regarded as the most objective methods for diagnosing reflux, both endoscopy and 24-hour pH monitoring fall far short of being gold standards

- 10% to 20% of patients with endoscopic esophagitis have negative 24-hour pH monitoring studies, and probably even a larger proportion of patients showing pH studies above 7% acid exposure have normal endoscopic findings.

(7) FINAL STAGING PROCESS

The four objective methods that help quantify gastroesophageal reflux disease are:

1. Endoscopic documentation of mucosal damage
2. Histologic proof of pathologic changes in the mucosa and submucosa
3. Recording of an abnormal exposure to an acid and/or alkaline refluxate for the esophageal mucosa
4. Recording of the functional abnormalities leading to reflux disease.

Staging of Severity in Gastroesophageal Reflux

	Endoscopy				Histology				pH Monitoring				Manometry			
Stage	0	1	2	3	0	1	2	3	0	1	2	3	0	1	2	3
0	X				X				X				X			
1		X				X				X				X		
2		X				X					X	X			X	X
3			X	X			X	X			X	X			X	X
4	Stricture formation and/or columnar-lined esophagus															

(7) FINAL STAGING PROCESS

- **Stage 0** = When all four tests are negative, so reflux disease has not been demonstrated.

- **Stage I** = When the recording methods all show equivocal documentation of reflux, again clinically significant reflux has not been demonstrated.
- In both these groups, although patients may complain of being severely symptomatic, causes for their symptoms should be sought elsewhere.
- **Stage 2** = When physiologic abnormalities and pH documentation of reflux disease are present, but without the mucosal damage.

- **Stage 3** = When all objective methods of investigation document reflux damage.

- **Stage 4** = The worst category of functional and histologic damage is seen

MEDICAL TREATMENT

- The great majority of patients seen with small hiatal hernias and occasional reflux symptoms require symptomatic treatment only with advice on modifications of some life-style habits.

- For more intensive therapy, treatment should always be based on accurate staging of the disease.

- When GERD has been well documented, medical treatment is always the first approach to control both symptoms and reflux damage.

Medical Treatment of GER

Modify Life-Style	Reduce weight
	Diet: Increase proteins, Reduce fat and sugar
	Avoid alcohol, tobacco, mint, chocolate, and medications affecting function
	Elevate the head of the bed
Protect Esophageal Mucosa	Antacids, alginates, cytoprotective agents
	H2 receptor antagonists
	Proton pump inhibitors
Improve Function	
Esophageal	Cisapride Metoclopramide Bethanechol
Gastric	Cisapride Metoclopramide Domperidone

SURGICAL TREATMENT: ANTIREFLUX OPERATIONS FOR EARLY-STAGE GERD.

- Symptoms, in the absence of physiologic abnormalities and of proven endoscopic or mucosal damage (Stage 0 and Stage 1), should never be indications for an antireflux operation.

- Patients with an incompetent LES accompanied by increased GER on 24-hour pH monitoring but without mucosal damage (Stage 2 disease) should always undergo a well-supervised period of medical management of 6-12 months. At the end of this period, patients and their physicians will usually choose whether to continue medical therapy or undergo antireflux operation

- Patients with esophagitis and defective function of the LES (Stage III disease) also should undergo intensive medical therapy, with the patient trying to lose weight during this period. As in Stage II disease, a decision is made at the end of a 6- to 12-month period in regard to further therapy.

ANTIREFLUX OPERATIONS FOR EARLY-STAGE GERD

- Patients showing severe complications of reflux disease, such as stricture with esophagitis or circumferential CLE, should be treated surgically. Antireflux surgical treatment is indicated in such patients to correct the functional defects and limit progression of damage.

- The most frequently used operations for reflux disease are total or partial **fundoplication**. Their aim is to restore normal anatomy, by restoring an intra-abdominal segment of esophagus, re-creating an appropriate high-pressure zone at the EGJ, and maintaining this repair in a normal position.

- These operations are preferred for patients presenting at the first three stages of esophagitis, where there is usually no shortening or periesophageal inflammation with the reflux damage.

ANTIREFLUX OPERATIONS FOR EARLY-STAGE GERD

1. **Total Fundoplication.** (Nissen)

2. **Partial Fundoplication**. (Belsey-Mark IV operation, Hill's posterior gastropexy, The Dor operation, Watson repair , The posterior partial fundoplication of Guarner and Lind).

ANTIREFLUX OPERATIONS FOR COMPLICATIONS OF GERD

- Standard antireflux operations fail in 45% of cases when a stricture is present. In this situation, the esophagus, narrowed from long-term reflux or damaged with extensive columnar epithelium replacement, is frequently affected by periesophagitis and shortening

- This situation dictates the use of lengthening procedures such as the **Collis gastroplasty**, where the lesser curvature of the stomach is fashioned as a tube to elongate the esophagus and obtain a proper intra-abdominal length of esophagus.
- A partial or a total fundoplication is added to the gastroplasty to obtain the antireflux function, and the repair is reduced without tension under the diaphragm.
- These repairs are considered esophageal sparing operations, and they provide satisfactory results for patients with significant reflux damage to their esophagus.

ANTIREFLUX OPERATIONS FOR COMPLICATIONS OF GERD

- The **intrathoracic fundoplications** (**Thal and Maher**) may offer good reflux control, but they are not used extensively because of dangers that have been reported for supradiaphragmatic fundoplications.