

ESOPHAGUS

Made by group A1

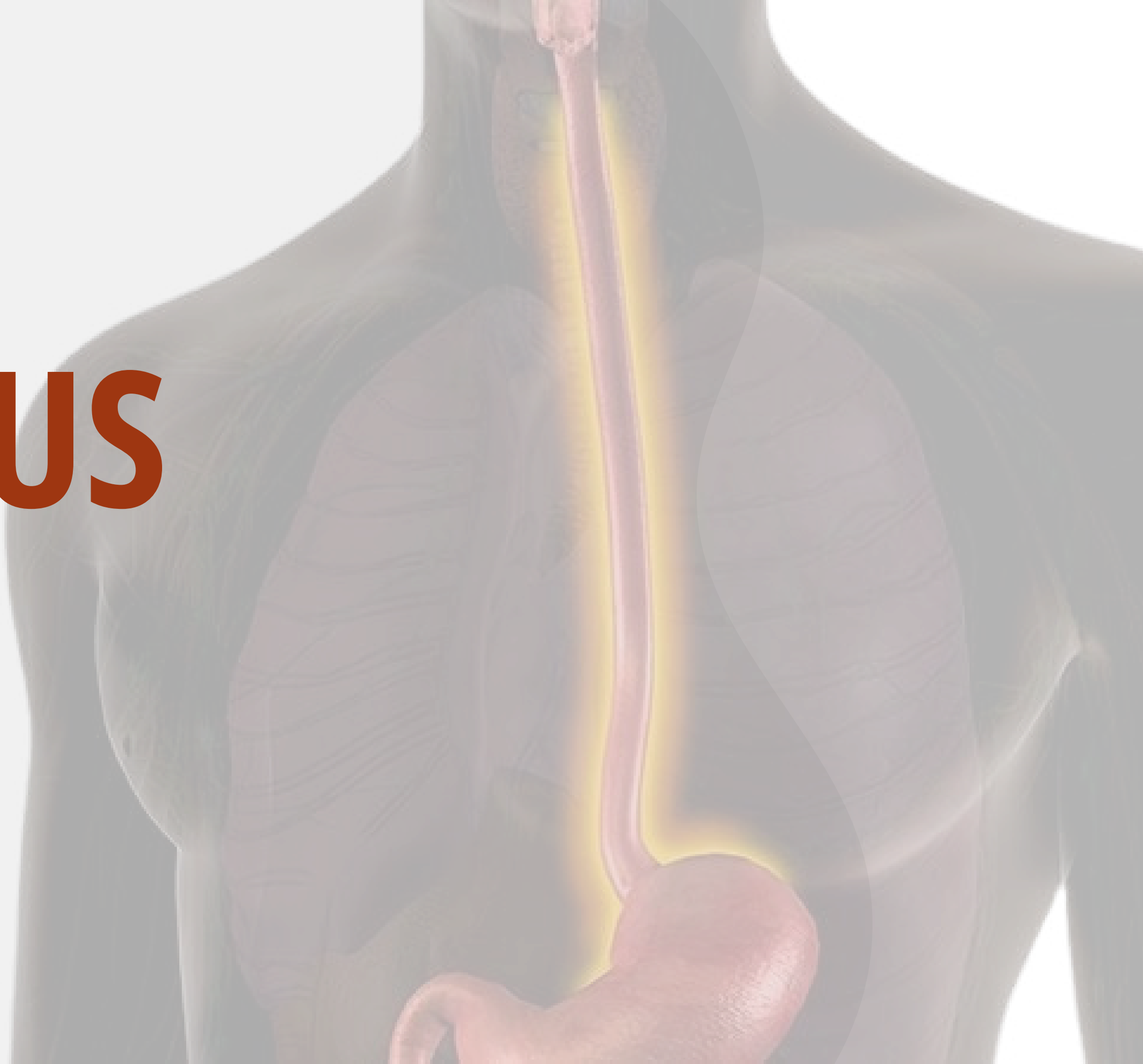
Deyaa ghanem

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ANATOMY OF ESOPHAGUS

Def: The esophagus a muscular tube, Conducts food from pharynx into stomach About (25 cm) long.

-Beginning: It begins from the lower border of the pharynx at the level of the 6th cervical vertebra.

-Termination: It ends in the cardiac orifice of the stomach at the level of T11 vertebra, (one inch) to the left of the median plane.

Curves

1-Anteroposterior curvature: The oesophagus follows the curvatures of the vertebral column.

2-Side to side: Two inclinations to the left: one at level of T1 and the other at T7.

Constrictions:

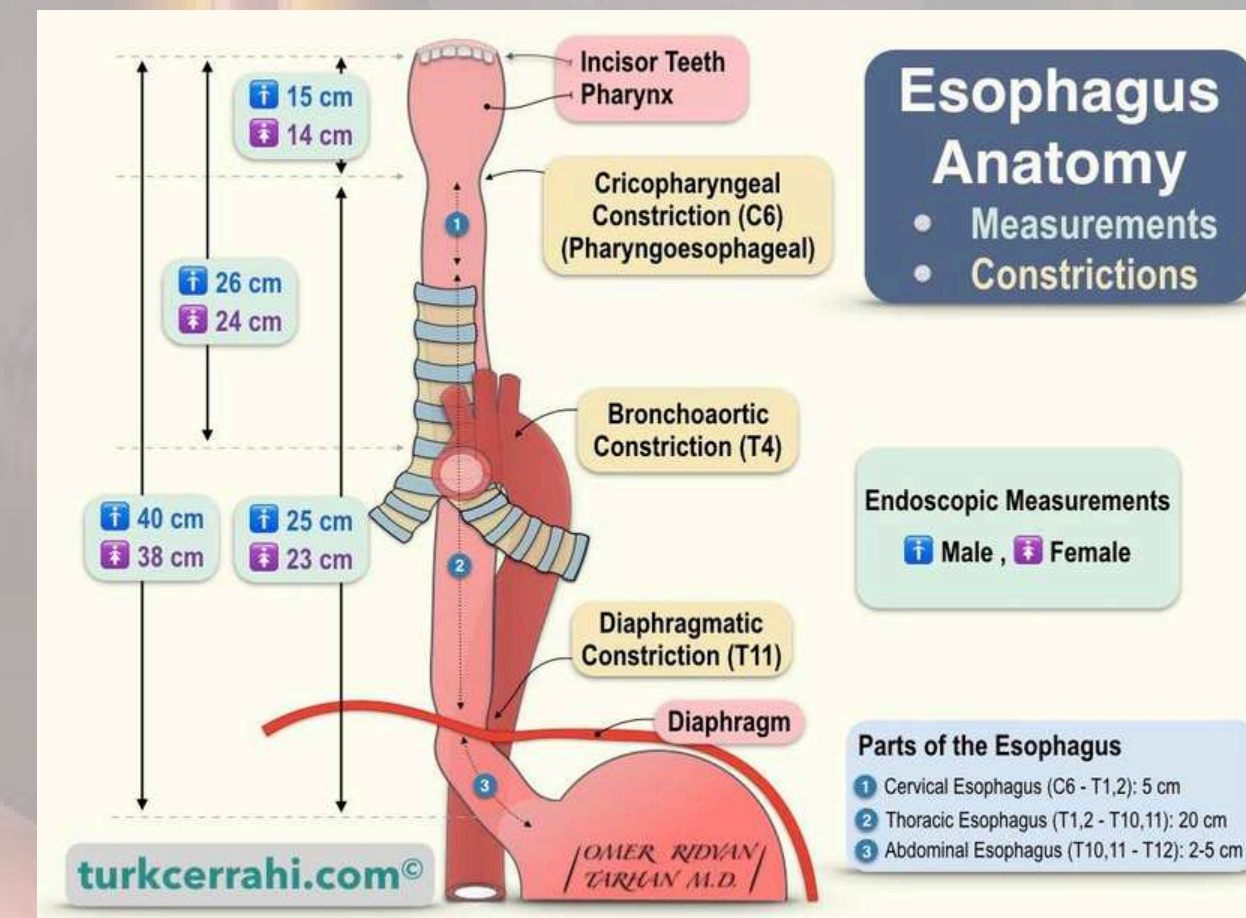
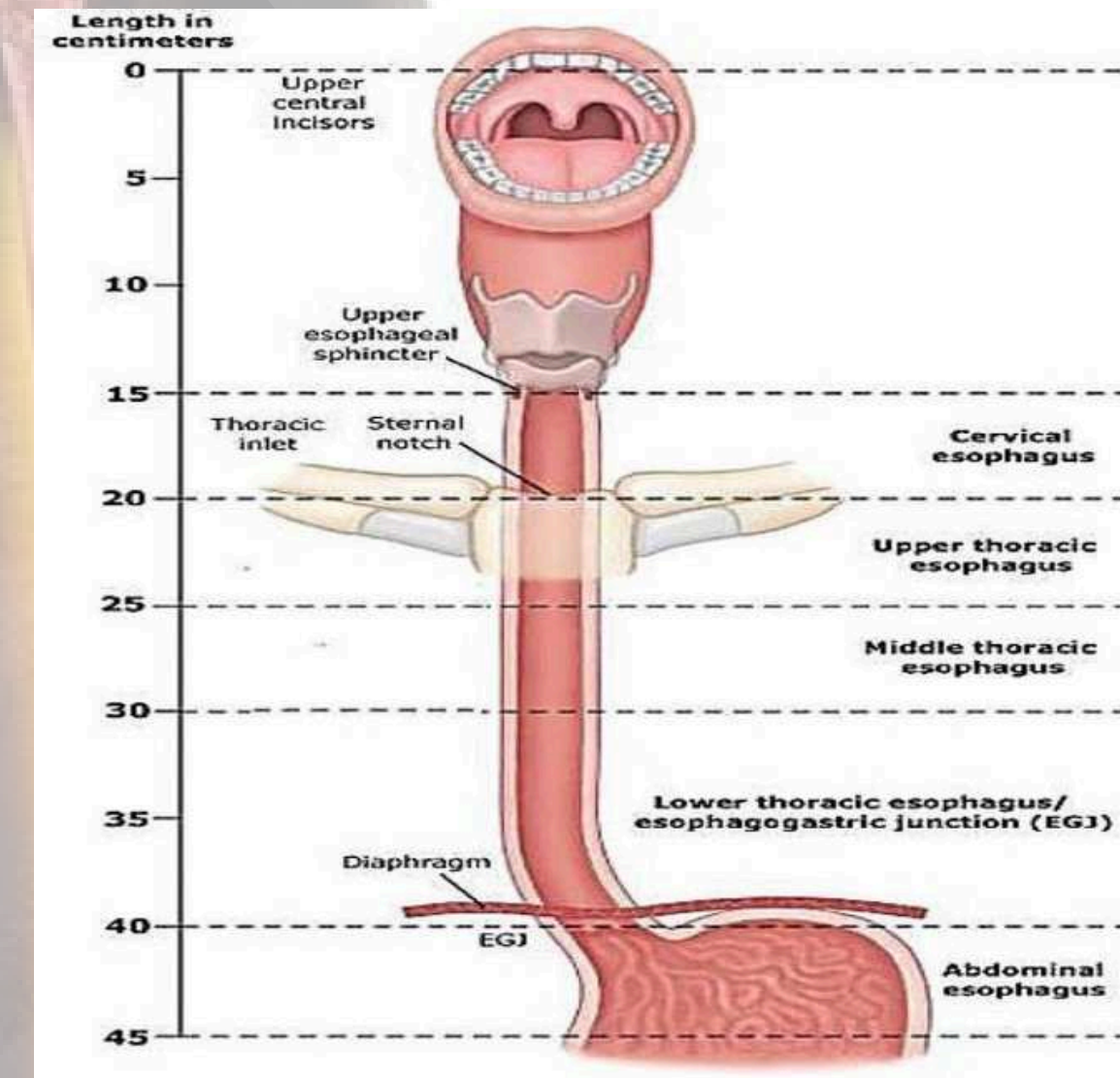
1-At its beginning 6 inches from incisors.

2-At the crossing of the arch of aorta 9 inches from incisors.

3-At the crossing of the left bronchus 12 inches from incisors.

4-At the diaphragmatic opening 15 inches from incisors.

* There is Some resistance at these constrictions at endoscopy



CERVICAL PART OF ESOPHAGUS

Anterior relations:

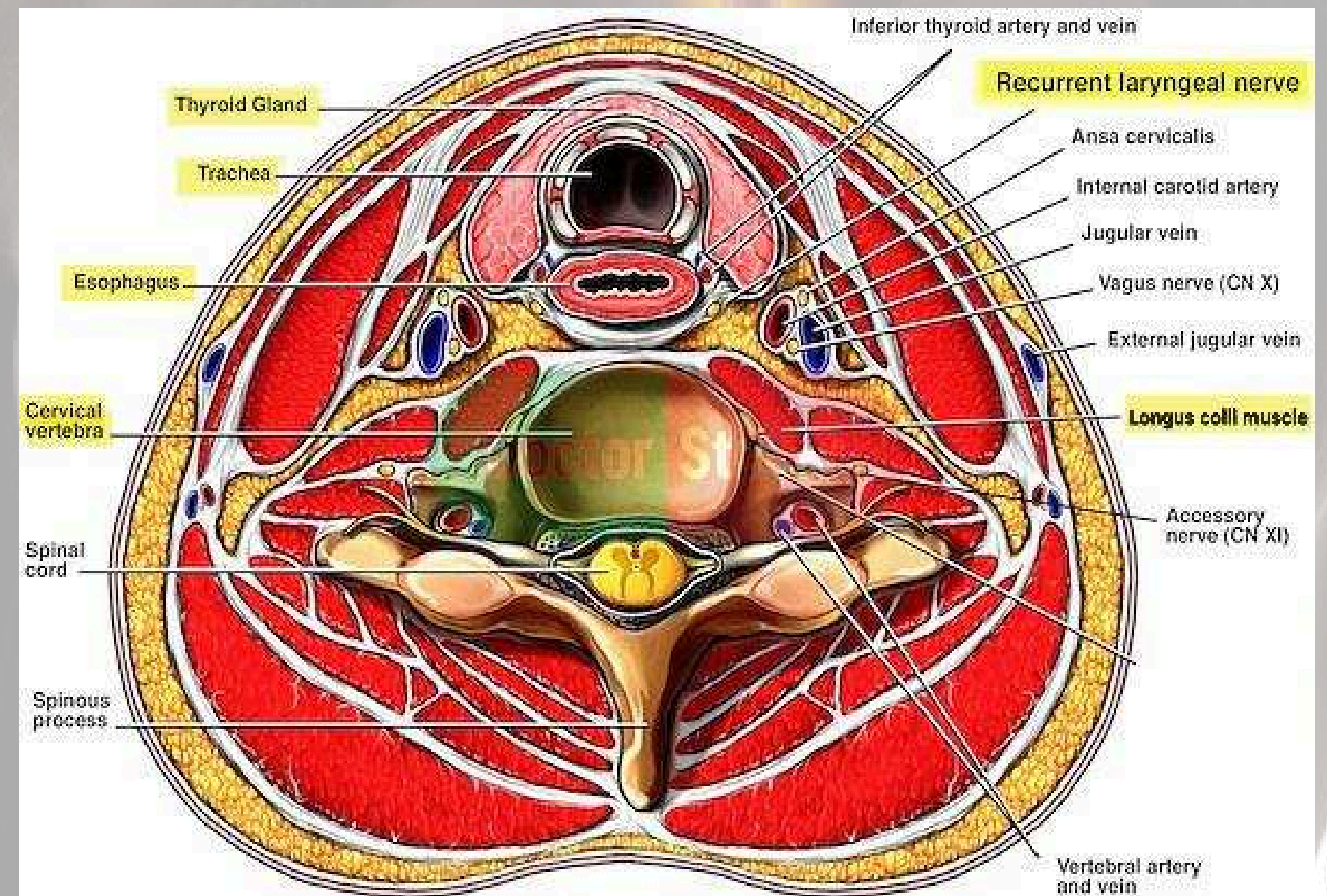
- Trachea.
- Recurrent laryngeal nerves one on each side, in the groove between trachea and esophagus.

Posterior relations:

- Longus colli muscle.
- Vertebral column.

Lateral relations:

- Common carotid arteries (on each side).
- Thyroid gland (on each side).
- Thoracic duct ascends for a short distance (along its left side)



THORACIC PART OF ESOPHAGUS

Anterior relations:

- Trachea.
- Left principal bronchus at (T5).
- Pericardium (separates esophagus from left atrium) at (T5-T8).
- Diaphragm (T8-T10).

*In cases of left-sided heart failure, the oesophagus may be compressed backwards by the enlarged left atrium.

Posterior relations:

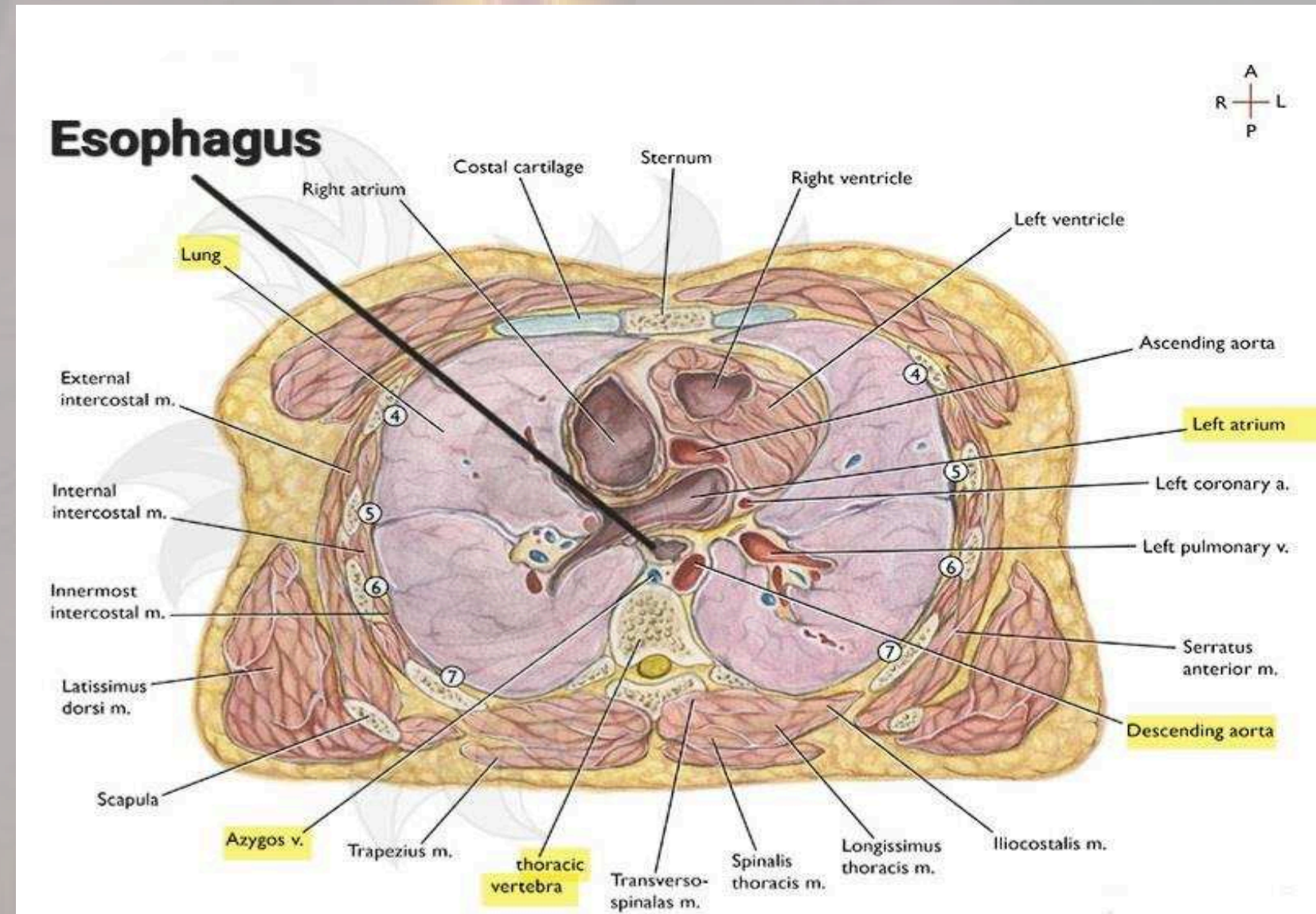
- Thoracic vertebrae.
- Thoracic duct.
- Azygos vein
- Right posterior intercostal arteries
- Descending thoracic aorta at its lower part(T7-T9).

Right relations:

- Right pleura.
- Arch of the azygosvein.

Left relations:

- Left pleura.
- Arch of aorta.
- Descending thoracic aorta.



ABDOMINAL PART OF ESOPHAGUS

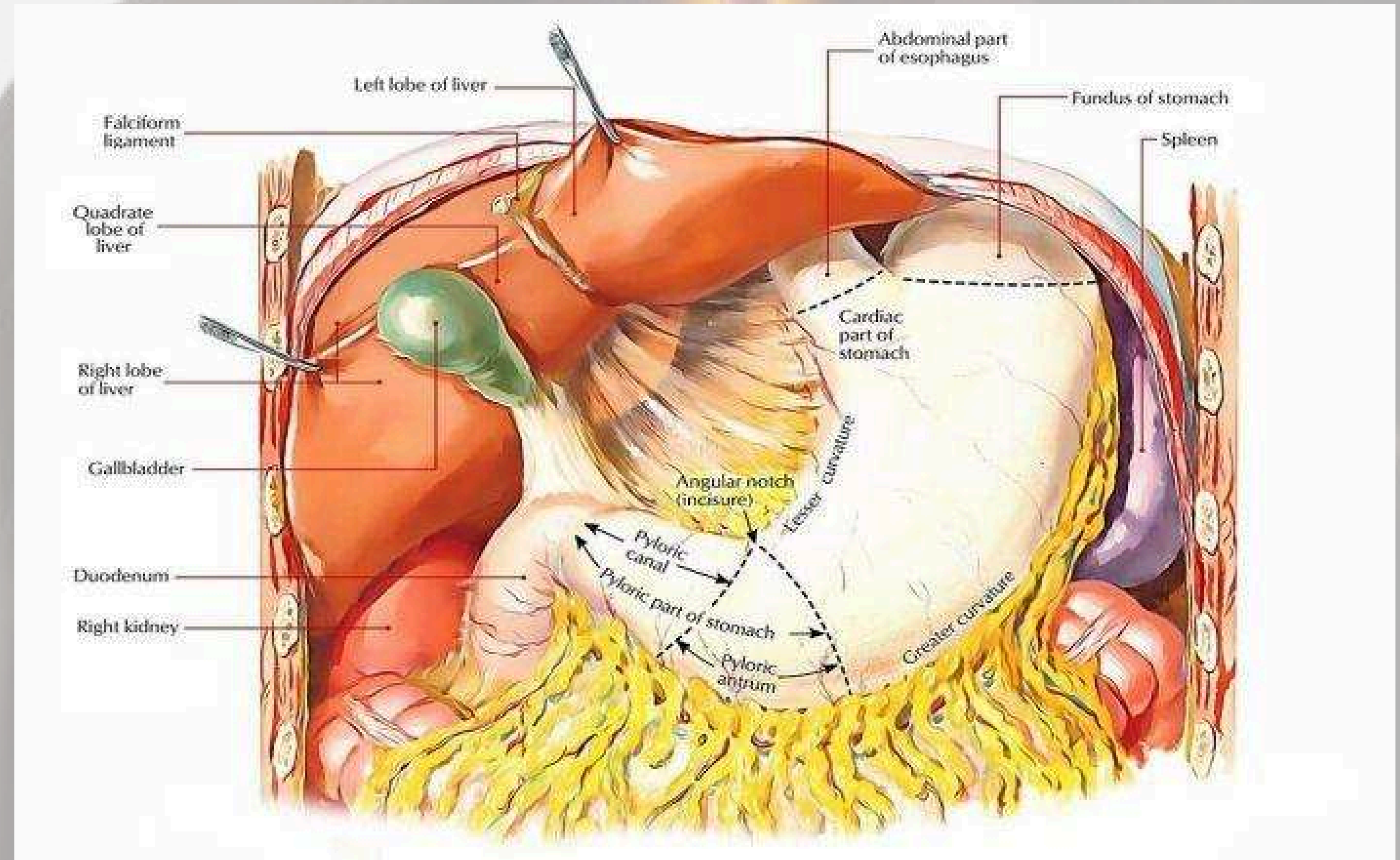
It ends in the cardiac orifice of the stomach.

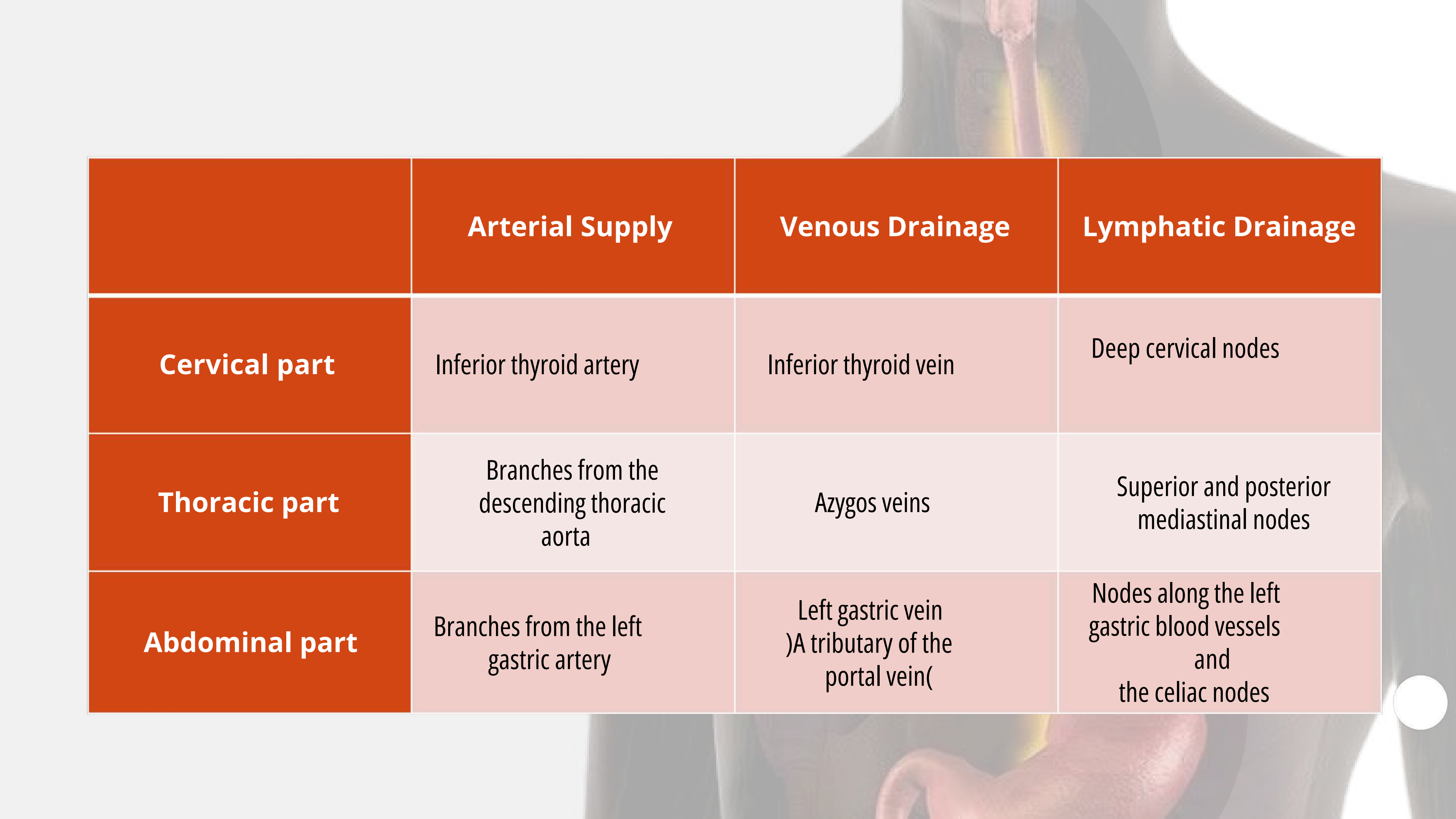
Anterior relations:

The left lobe of the liver.

Posterior relations :

The left crus of the diaphragm .





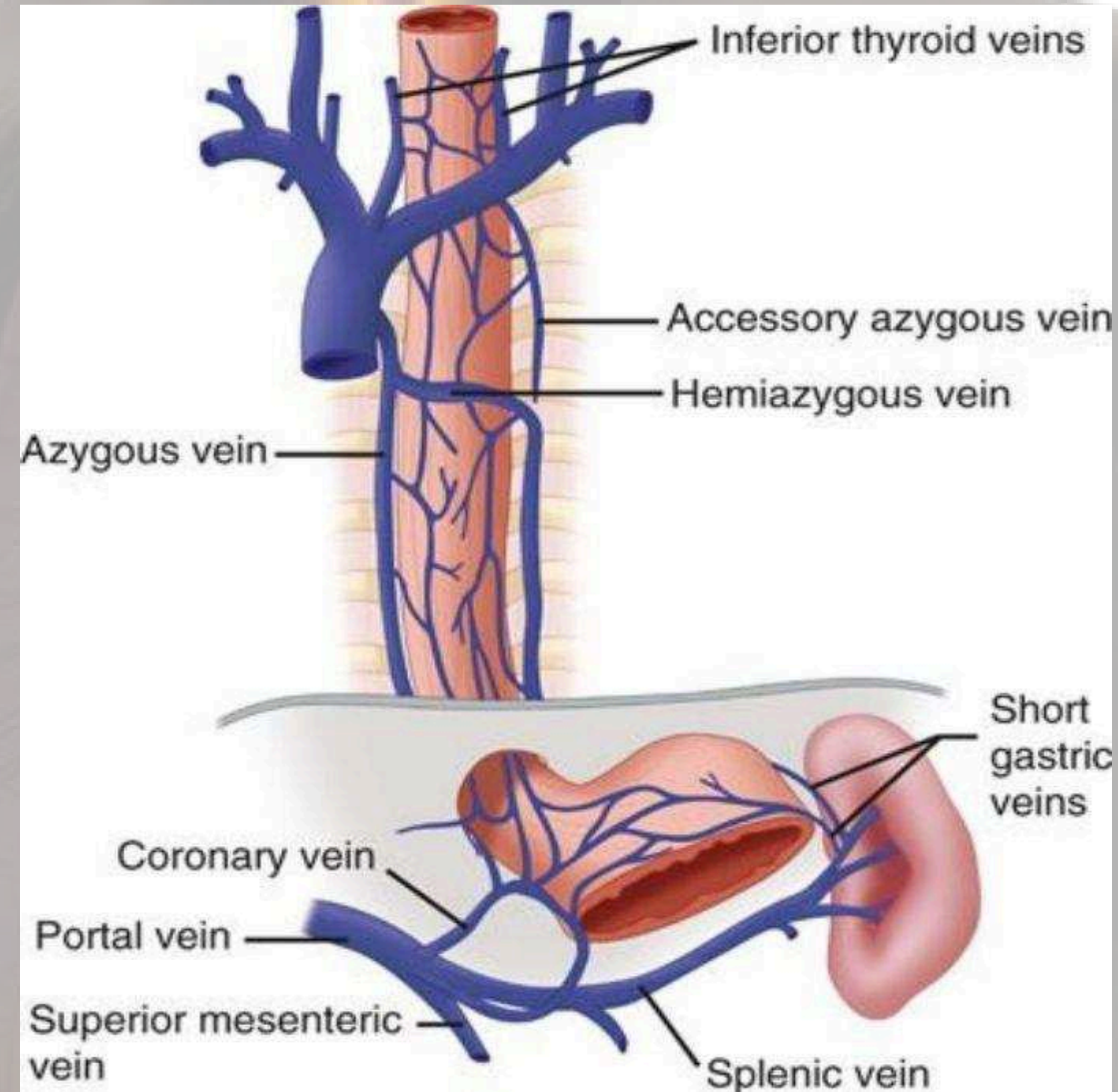
	Arterial Supply	Venous Drainage	Lymphatic Drainage
Cervical part	Inferior thyroid artery	Inferior thyroid vein	Deep cervical nodes
Thoracic part	Branches from the descending thoracic aorta	Azygos veins	Superior and posterior mediastinal nodes
Abdominal part	Branches from the left gastric artery	Left gastric vein)A tributary of the portal vein(	Nodes along the left gastric blood vessels and the celiac nodes

PORTAL SYSTEMIC ANASTOMOSES AT THE LOWER PART OF ESOPHAGUS

- Esophageal veins of azygos vein systemic circulation.
- Esophageal veins of left gastric vein portal circulation.

***That will lead to esophageal varices in case of portal hypertension.**

•Clinical symptom: Vomiting of blood



HISTOLOGY OF ESOPHAGUS

Layer of esophagus: from inner to outer :

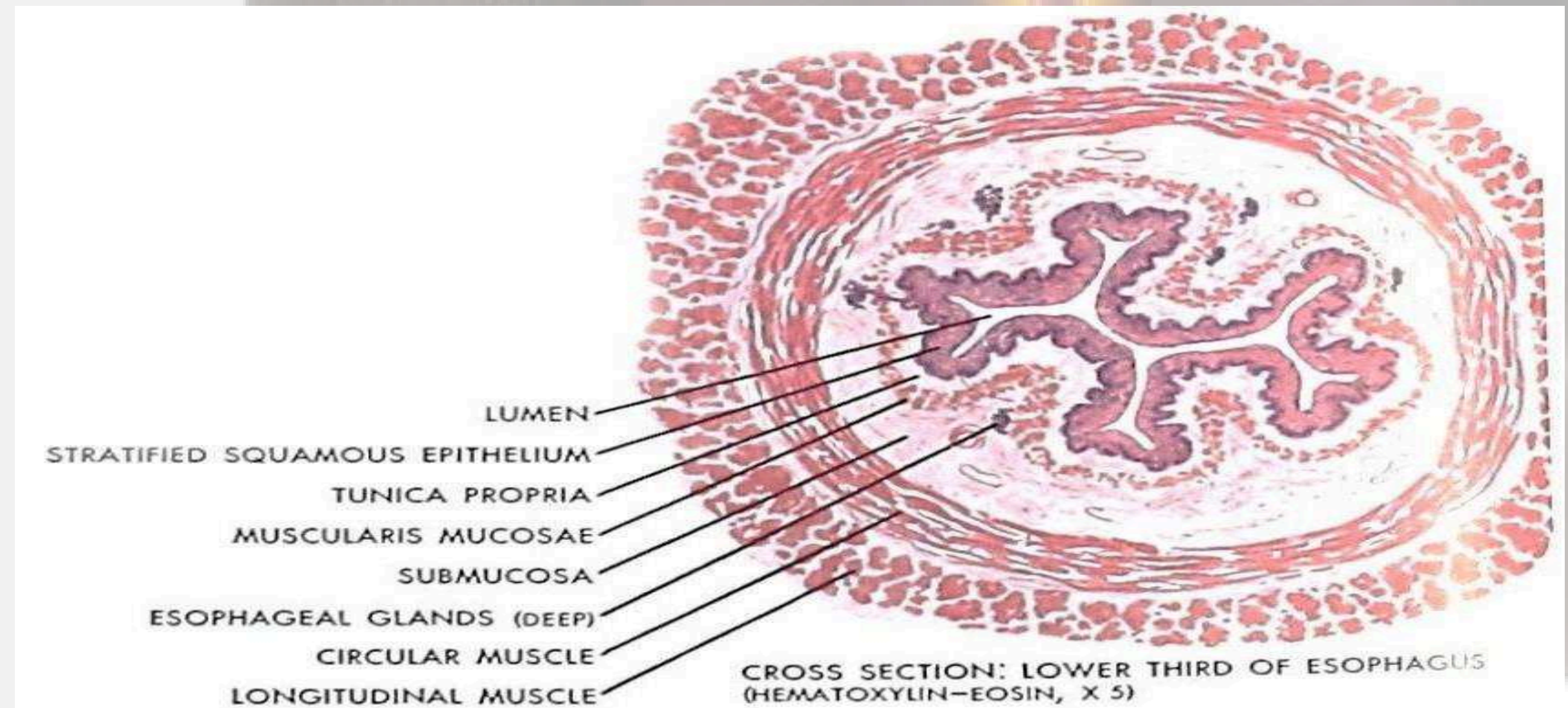
Mucosa: squamous epithelium transition to columnar before 2cm of GEJ

Submucosa

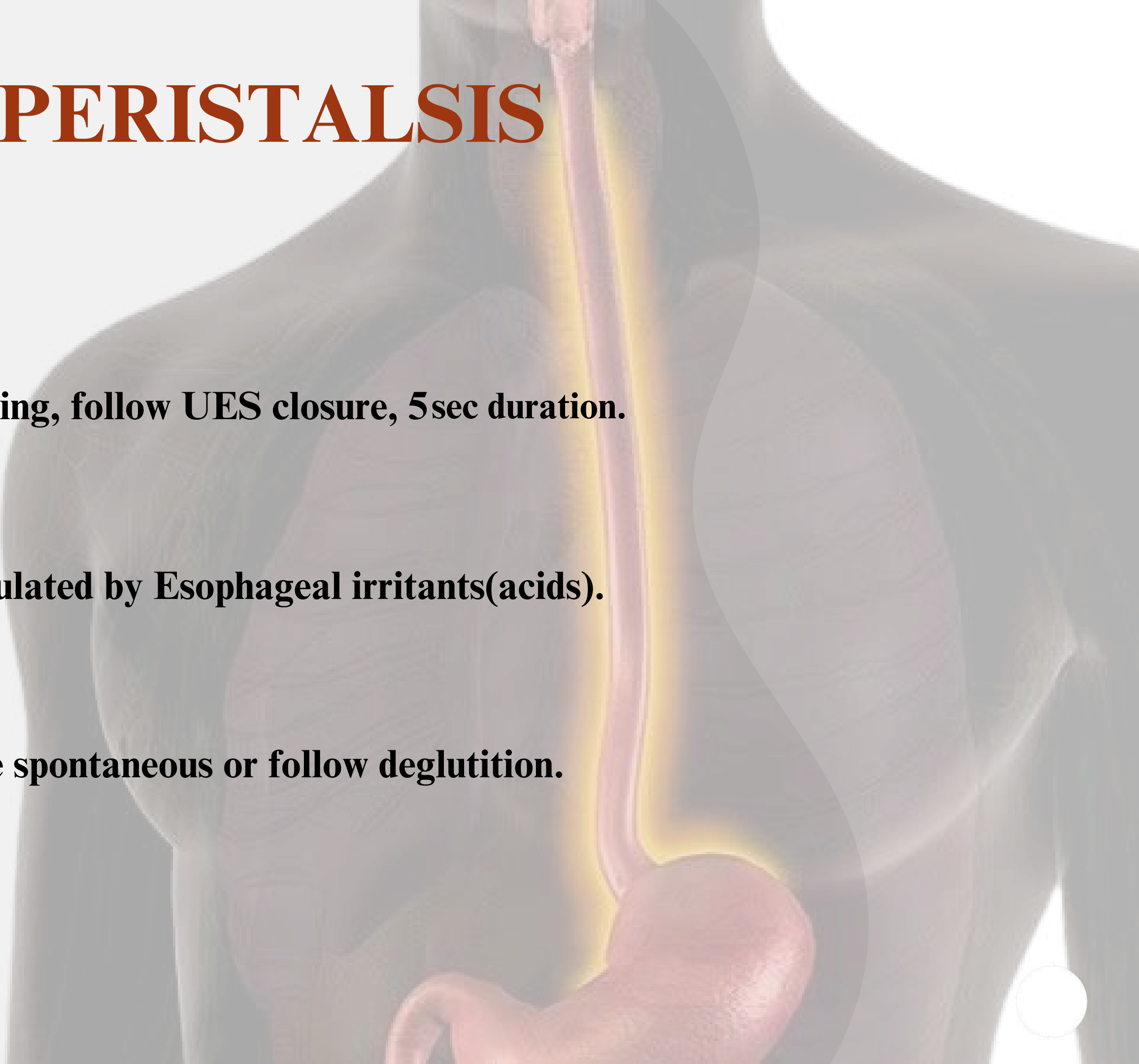
Circular muscle

Longitudinal muscle

Adventitia



ESOPHAGEAL PERISTALSIS

An anatomical illustration of the human torso, focusing on the digestive system. The esophagus is highlighted in a bright yellow color, showing its path from the throat down to the stomach. The stomach is shown in a reddish-pink color. The background is a light gray, and the overall image has a soft, semi-transparent appearance.

Primary peristalsis :

Involuntary, preceded by swallowing, follow UES closure, 5sec duration.

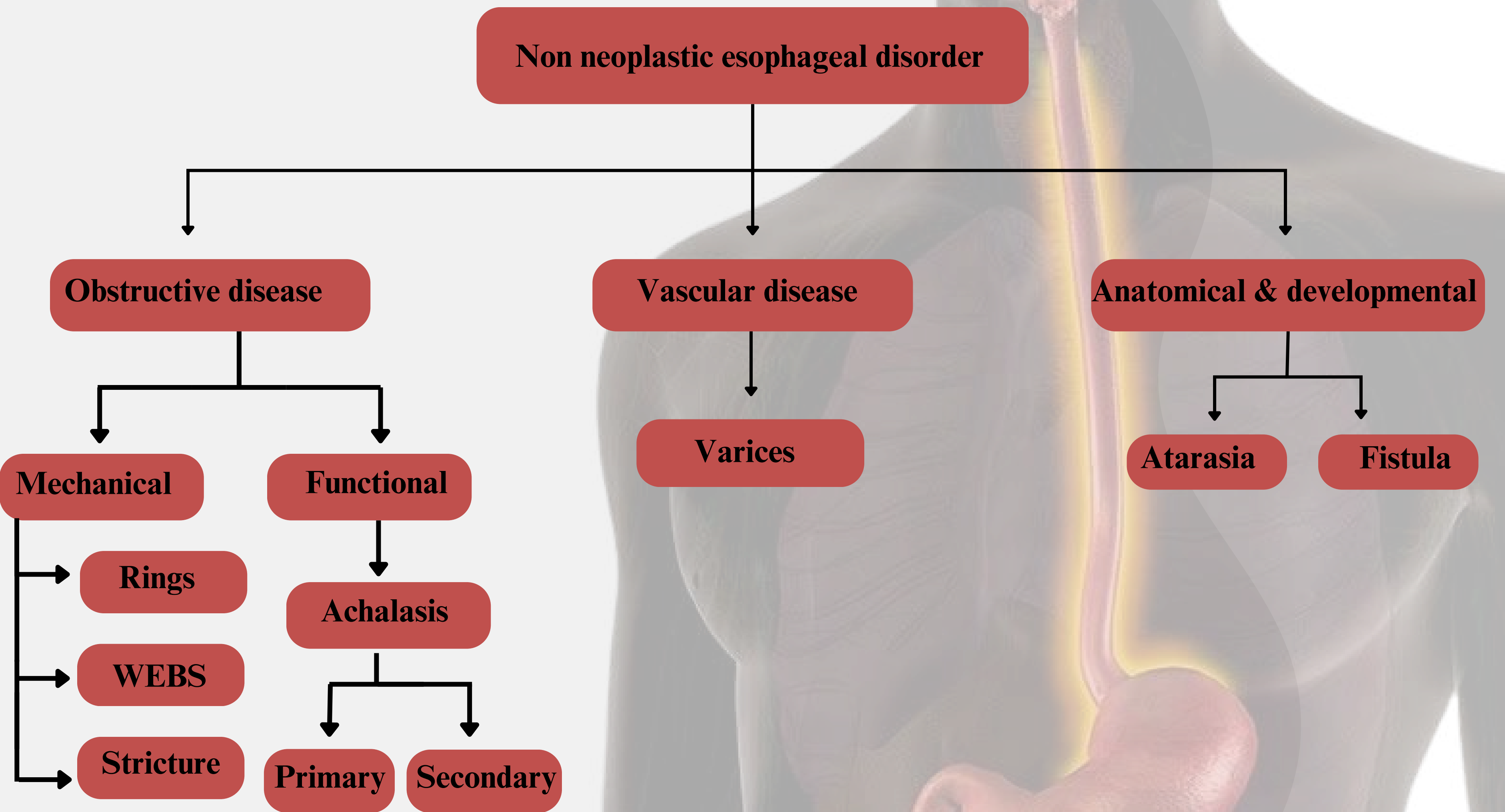
Secondary peristalsis:

Not preceded by swallowing, stimulated by Esophageal irritants(acids).

Tertiary contraction:

Non propulsive contraction, can be spontaneous or follow deglutition.

Non neoplastic esophageal disorder



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graph TD; A[Non neoplastic esophageal disorder] --> B[Obstructive disease]; A --> C[Vascular disease]; A --> D[Anatomical & developmental]; B --> E[Mechanical]; B --> F[Functional]; E --> G[Rings]; E --> H[WEBS]; E --> I[Stricture]; F --> J[Achalasia]; J --> K[Primary]; J --> L[Secondary]; C --> M[Varices]; D --> N[Atarasia]; D --> O[Fistula];
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This flowchart classifies non-neoplastic esophageal disorders into three main categories: obstructive, vascular, and anatomical/developmental. Obstructive diseases are further divided into mechanical (rings, webs, strictures) and functional (achalasia, which is primary or secondary). Vascular diseases include varices. Anatomical and developmental disorders include atarasia and fistula.

Obstructive disease

Mechanical

Rings

WEBS

Stricture

Functional

Achalasia

Primary

Secondary

Vascular disease

Varices

Anatomical & developmental

Atarasia

Fistula

Common clinical picture: (from history)

- 1- **Dysphagia:** the main symptoms of esophageal disease
- 2- **odynophagia:** painful dysphagia (usually due to esophagitis)
- 3- **Regurgitation:** passive return o f esophageal or gastric undigested Content (should be differentiated from vomiting).
- 4 - **Halitosis:** bad odor of the breath due to fermentation of retained Fluid in the esophagus
- 5- **retrosternal pain**
- 6- **complication in neglected cases**

Vomiting	Regurgitation
preceded by nausea	No
Retching is common	No
Reflex, active, anti peristalsis	passive return of esophageal or gastric content
Digested content	Undigested content
occur at any time	occur especially on lying flat or leaning foreword

Obstructive diseases: physical blockage or narrowing of the lumen this obstruction Can b e due t o problem in esophagus itself or external compression, it can be mechanical or functional.

Esophageal mucosal ring:

- **Circuler ring in the distal esophagus consist of mucosa, submucosa, sometimes hypertrophied muscles, there 3 types, type B the most common (scatzki ring), usually occur with episodic dysphagia**
- **It associated with GERD a n d hiatus hernia**

Esophageal mucosal webs:

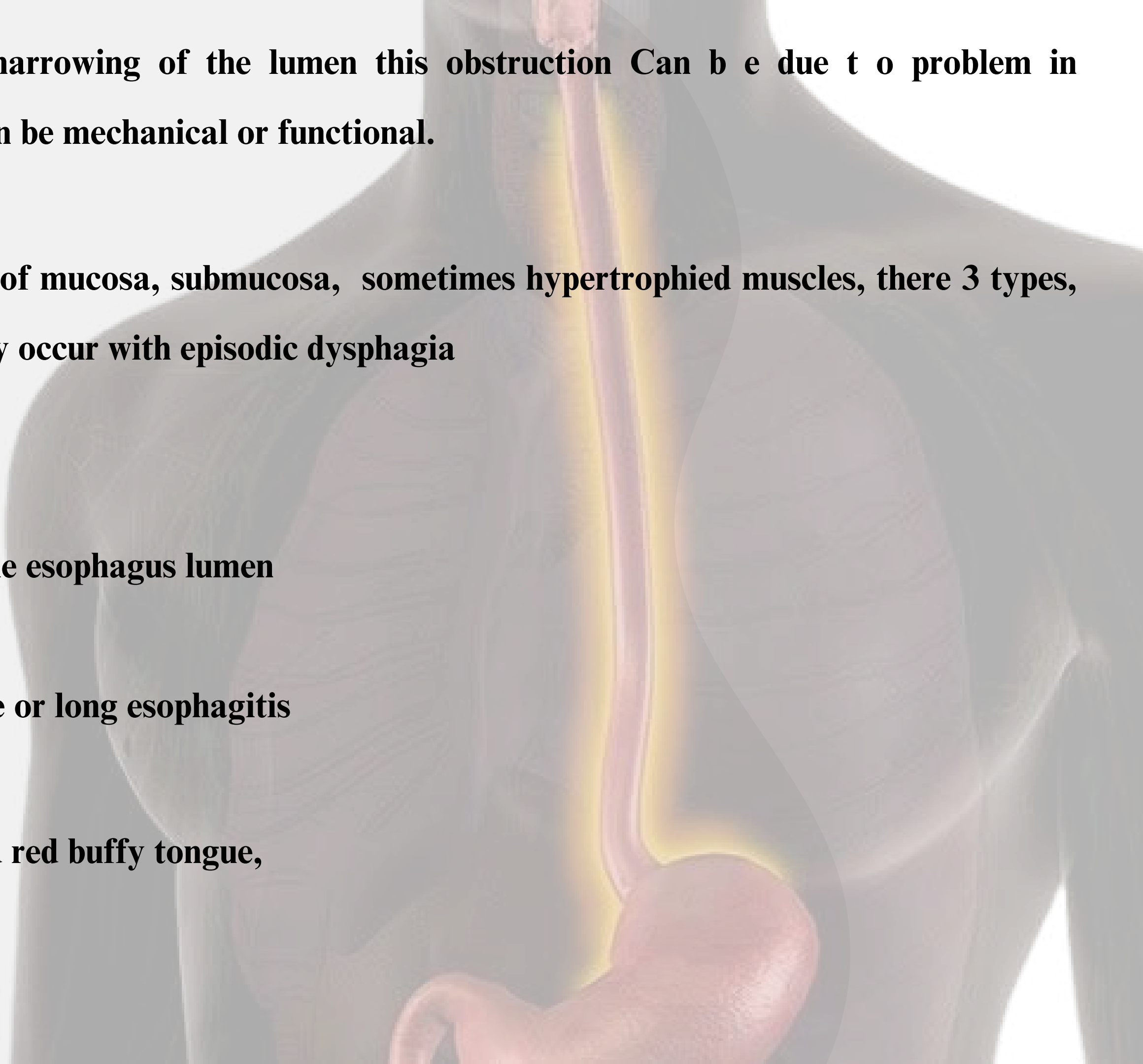
- **Protrusion of the mucosa& submucosa into the esophagus lumen**
- **common in upper esophagus,**
- **i t associated with plummer -vinson syndrome or long esophagitis**

plummer-vinson syndrome

- **triad of dysphagia, iron dificiency anemia and red buffy tongue,**

Esophageal webs

- **increase the risk for squamous cell carcinoma**

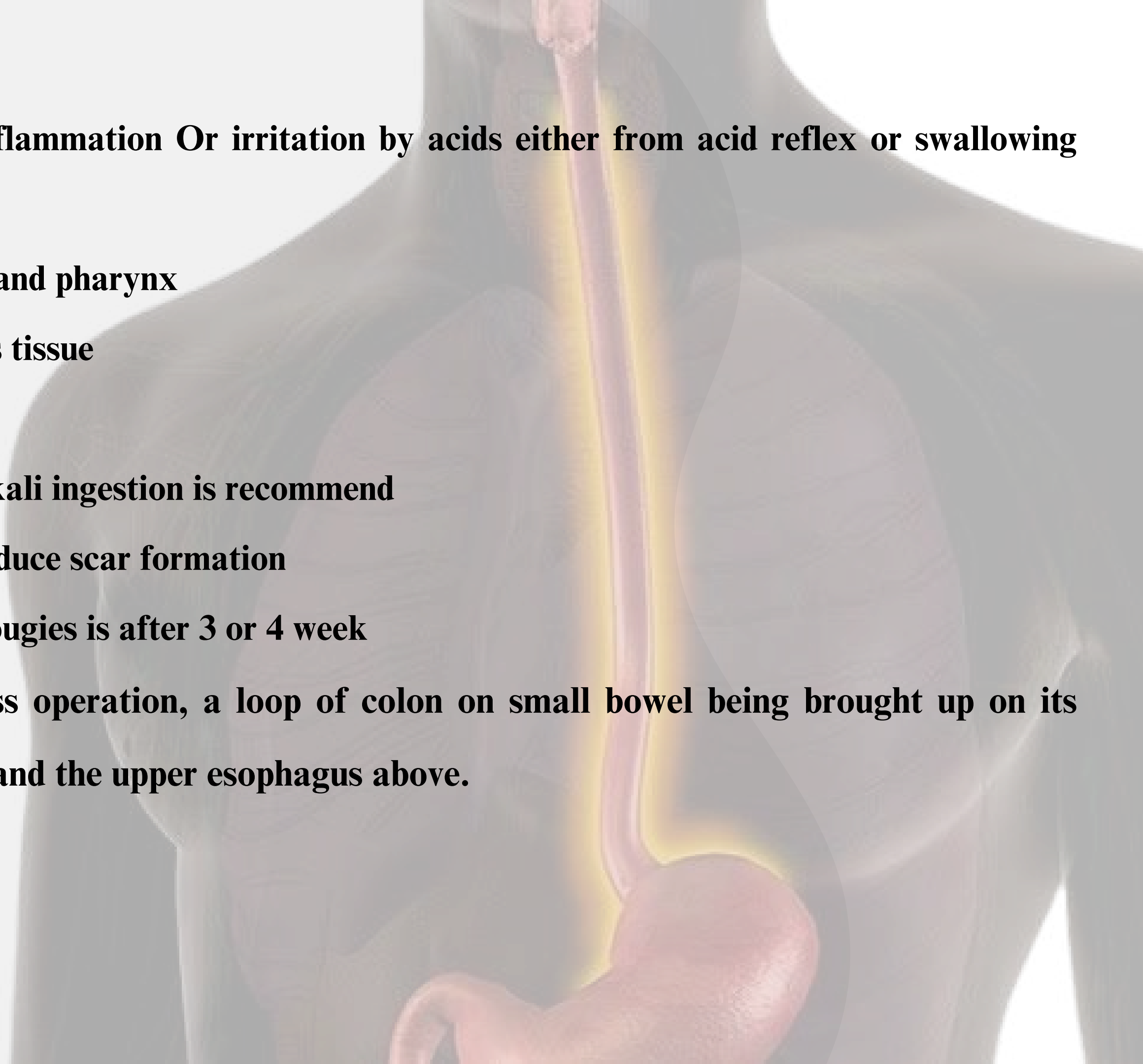


esophageal stricture:

- narrowing or constriction due to chronic inflammation Or irritation by acids either from acid reflex or swallowing corrosive substance.
- Sometimes It associated with burn to mouth and pharynx
- it cause ulcer that usually replaced by fibrous tissue

Treatment

- in the acute phase: neutralize the cause so alkali ingestion is recommend
- Before scar: systemic steroids are given to reduce scar formation
- If a stricture develops, gentle dilation with bougies is after 3 or 4 week
- impassable stricture is treated by a bypass operation, a loop of colon on small bowel being brought up on its vascular pedicle between the stomach below and the upper esophagus above.



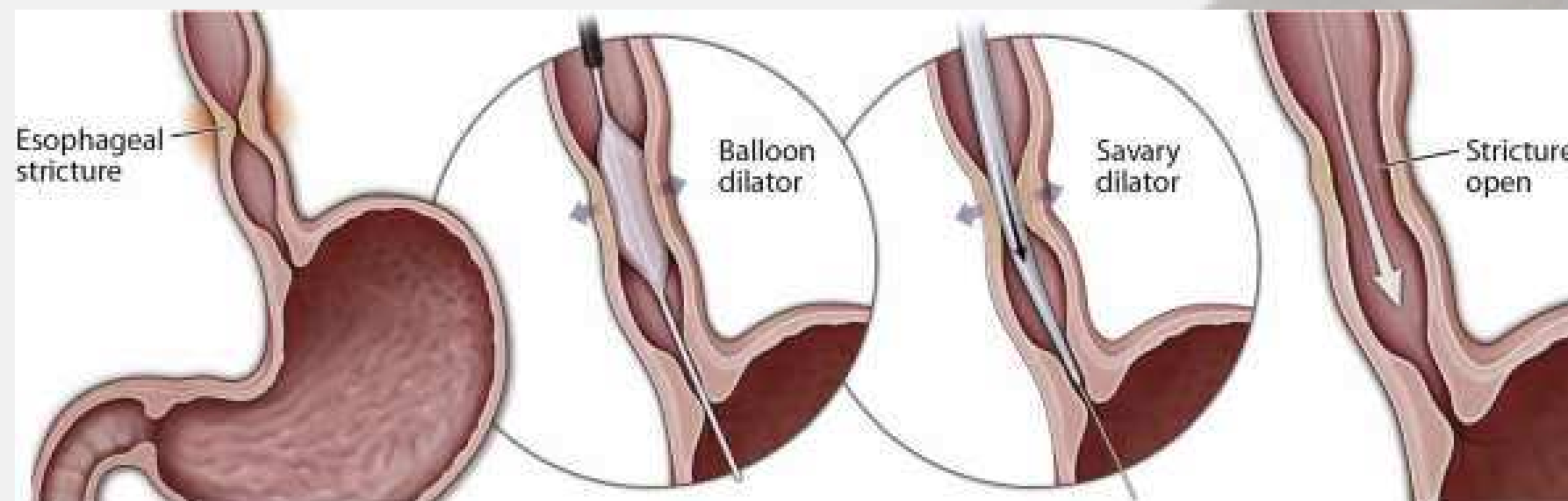
(A)



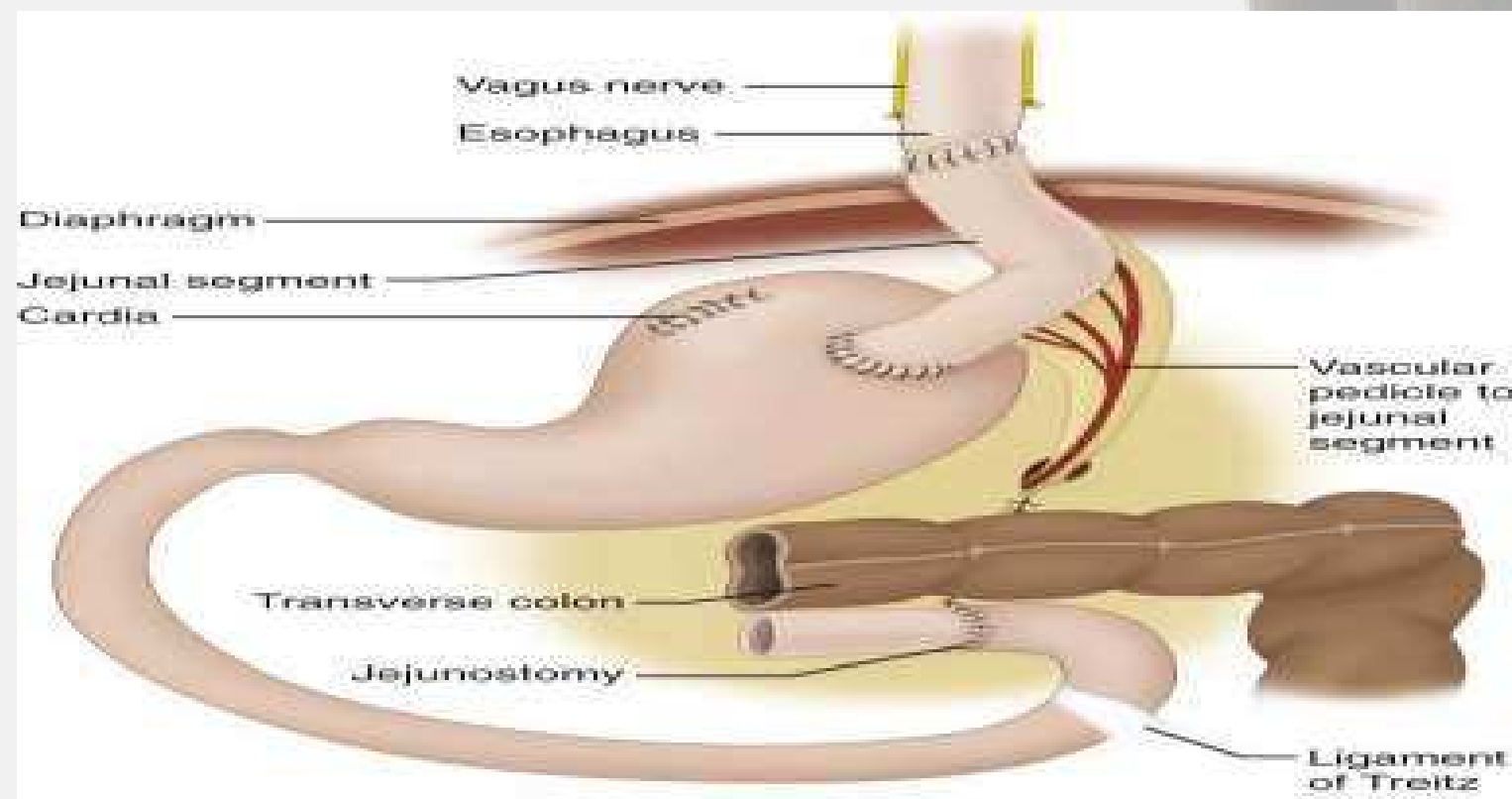
(B)



(C)



(D)



A- ESOPHAGEAL RING

B- ESOPHAGEAL WIBS

C- DILATION OF STRICTURE

D- BYPASS SURGERY

Achalasia :functional obstruction cause failure relaxation of esophagus LES due to Nuoromusculer incoordination so decrease or absent peristalsis

-Primary achalasia: idiopathic neuronal dysfunction

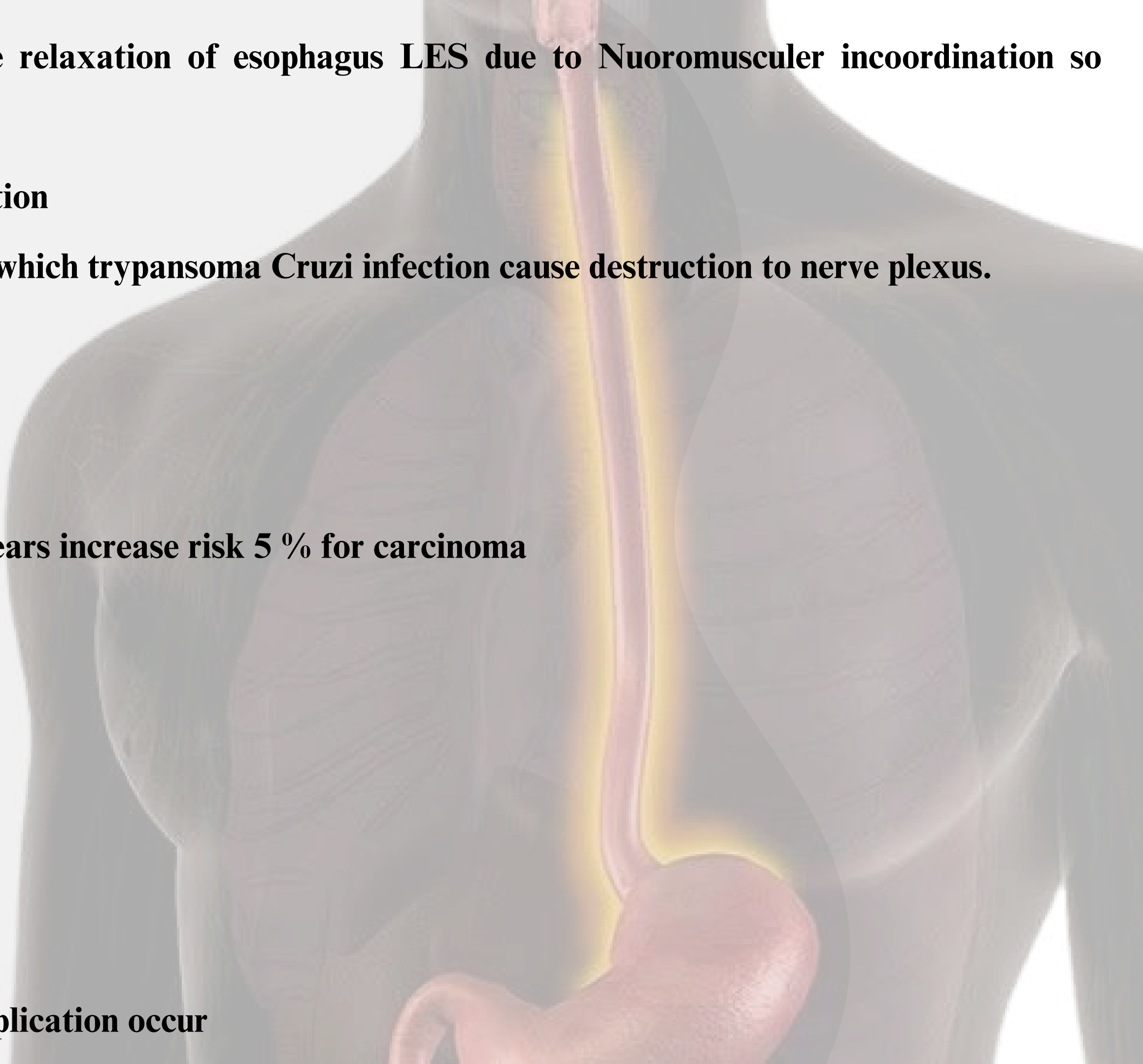
-Secondry achalasia: occur in chagas disease in which trypanosoma Cruzi infection cause destruction to nerve plexus.

***Complications:**

- Esophagitis & ulcer.
- Diverticulum.
- Pulmonary complication.
- Regurgitation
- Hematoemesis
- more than 20 years increase risk 5 % for carcinoma

***Clinical picture:**

- intermittent dysphagia to fluid then solid
- Epigstrial pain (xiphosternal junction area)
- odynophagia
- Regurgitation
- halitosis
- Ask about RS symptoms to insure that no complication occur



Investigations:

- **Chest x-ray:** may reveal dilated oesophagus as a mediastinal mass, with air fluid level .
- **Barium swallow :** show gross dilation or tortuosity of the oesophagus (bird- beak appearance)
- **Oesophgoscopy:** demonstrate a sac contain pond of stagnant Food and fluids and some abnormality in severe cases
- **Oesophageal manometry** confirm increase lower oesophageal Sphincter pressure

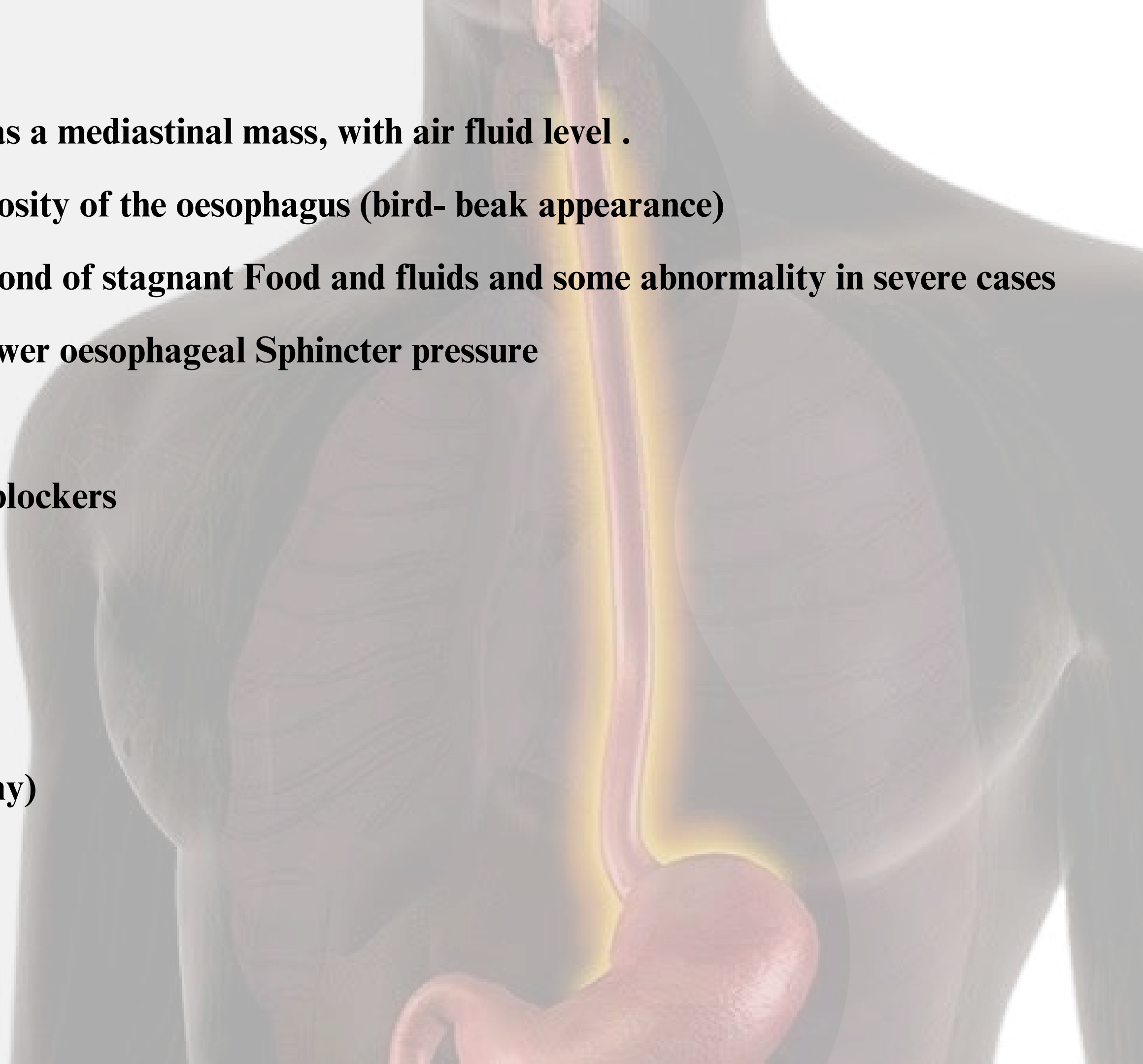
Treatment:

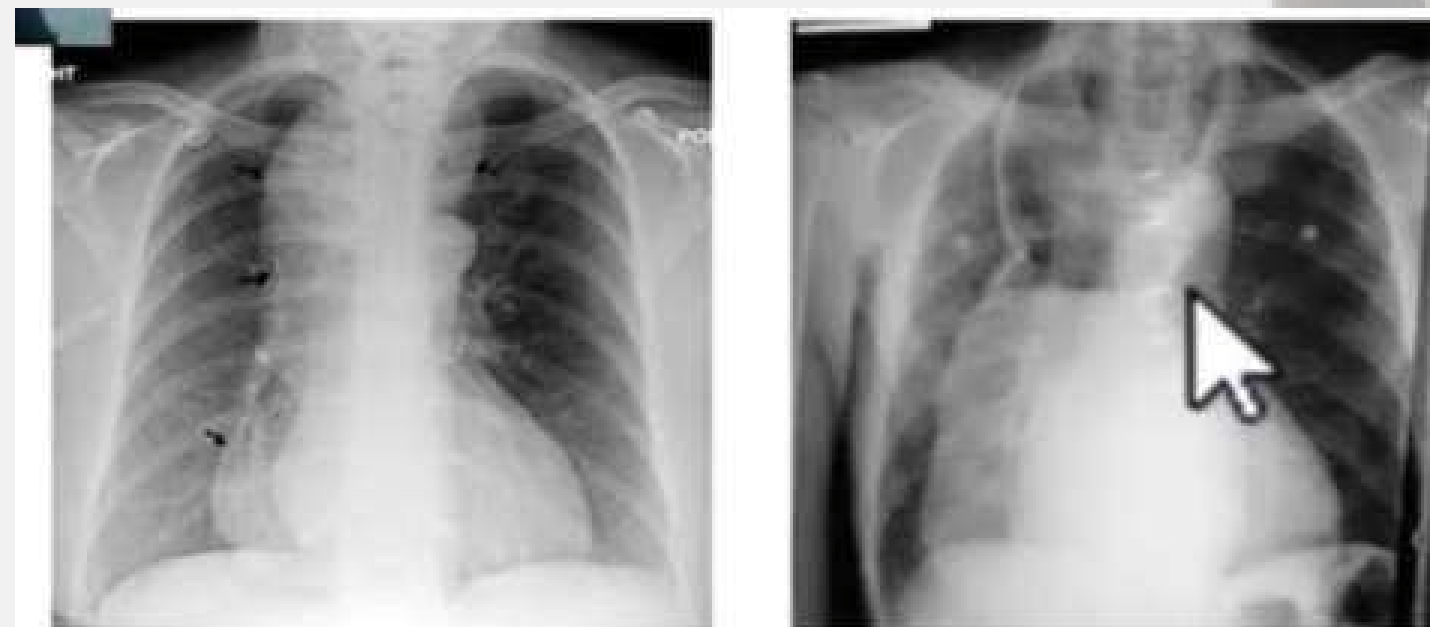
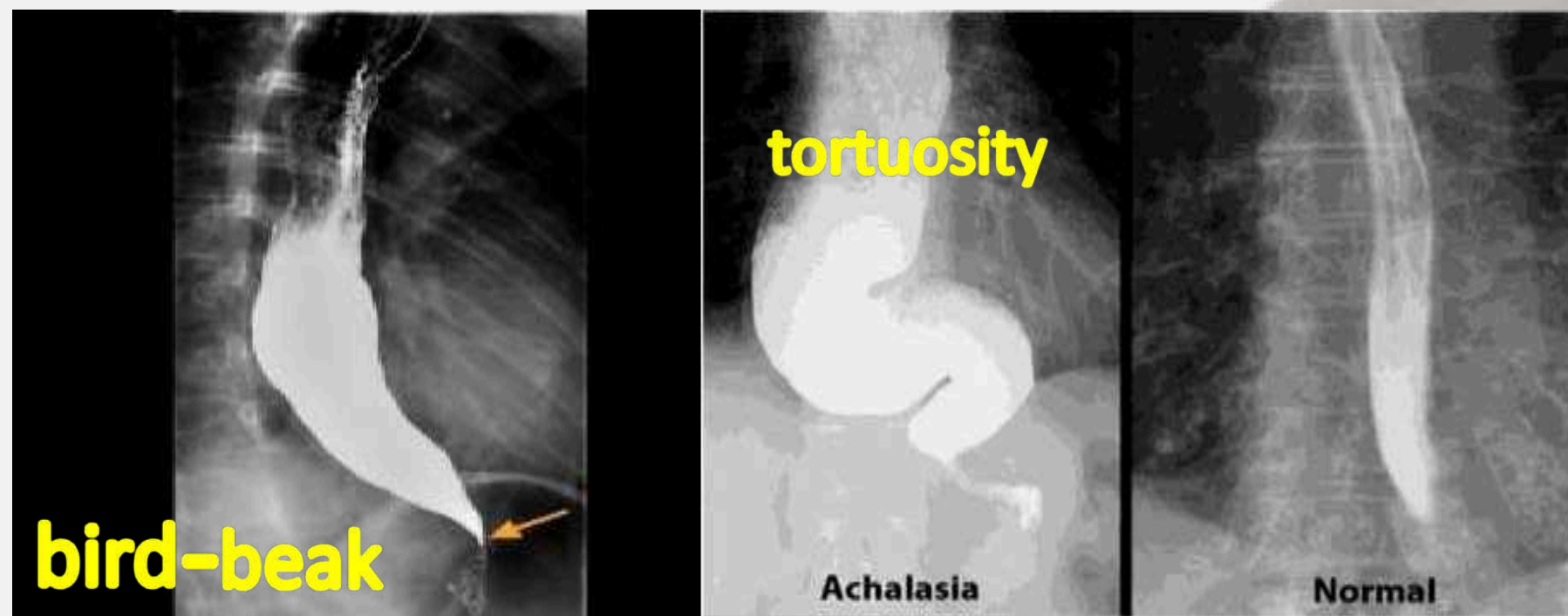
- **If mild :** medication use such as CA channel blockers
- **If severe many options available:**

endoscopic balloon dilatation

botulinum toxin injection

Hellers operation (oesophagocardiomyotomy)





A chest X-ray showing achalasia (arrows point to the outline of the massively dilated esophagus.)

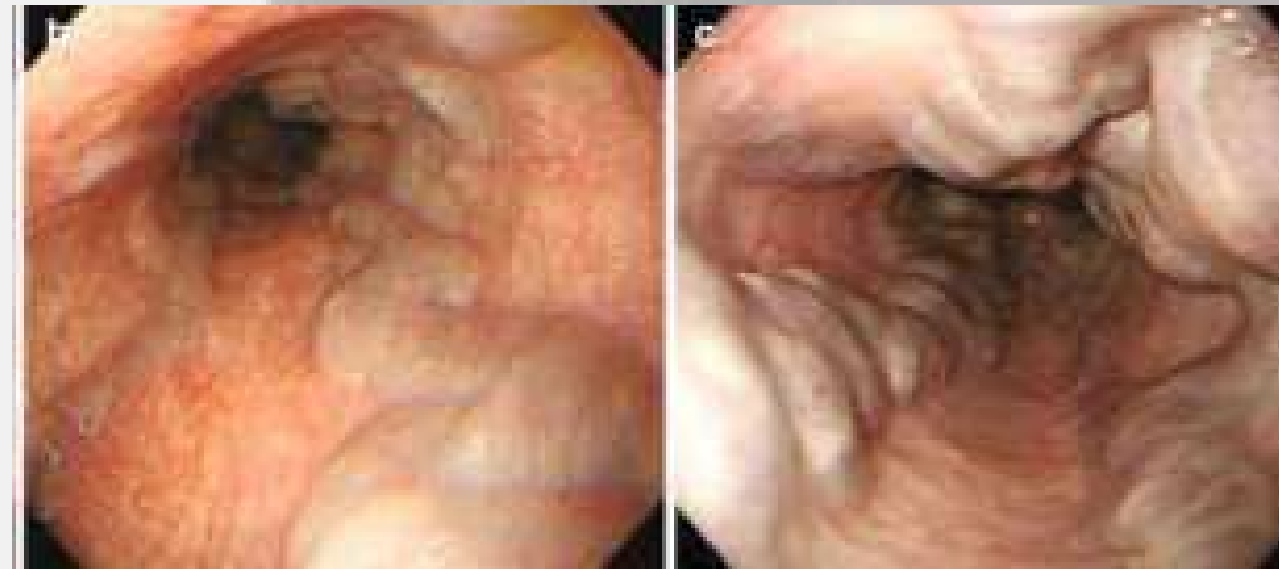
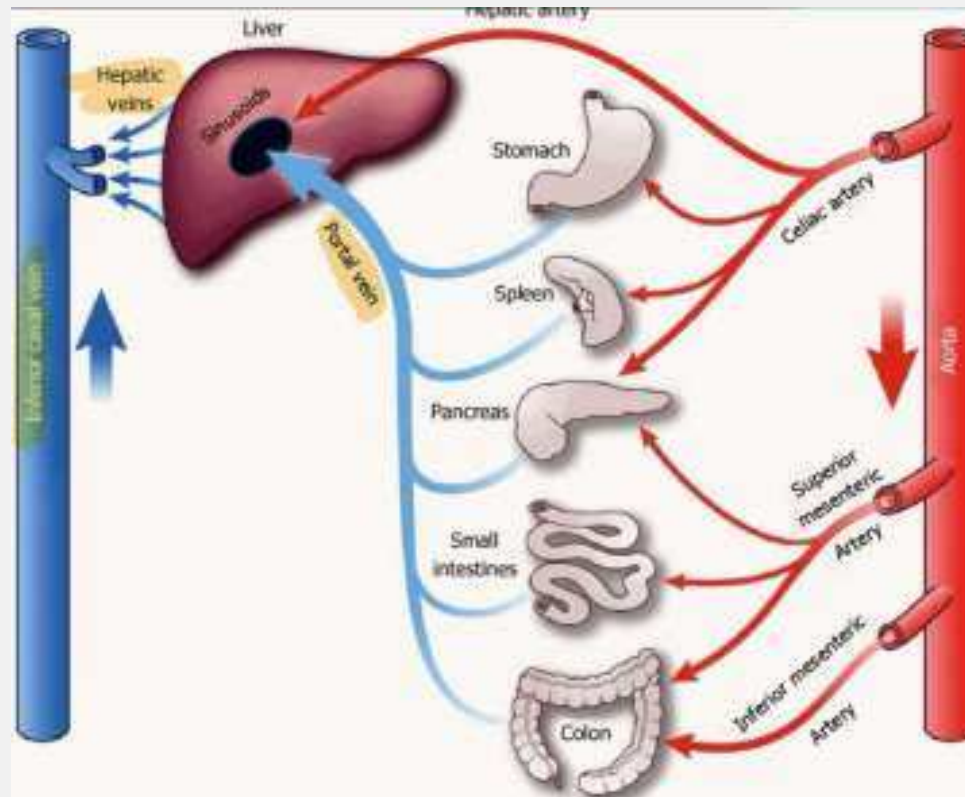
barium swallow

Chest X-ray

Esophageal varices

- venous blood from the GIT is delivered to the liver via the portal vein before reaching the IVC.
 - Diseases that impede portal flow that cause portal hypertension (mainly liver cirrhosis this hypertension lead to varices
 - By endoscopy: varices appear as tortuous dilated veins within the submucosa of the distal esophagus and proximal stomach.
- # varices often asymptomatic but their rupture can lead to massive hematoemesis and death so it emergency.

Treatment: band ligation



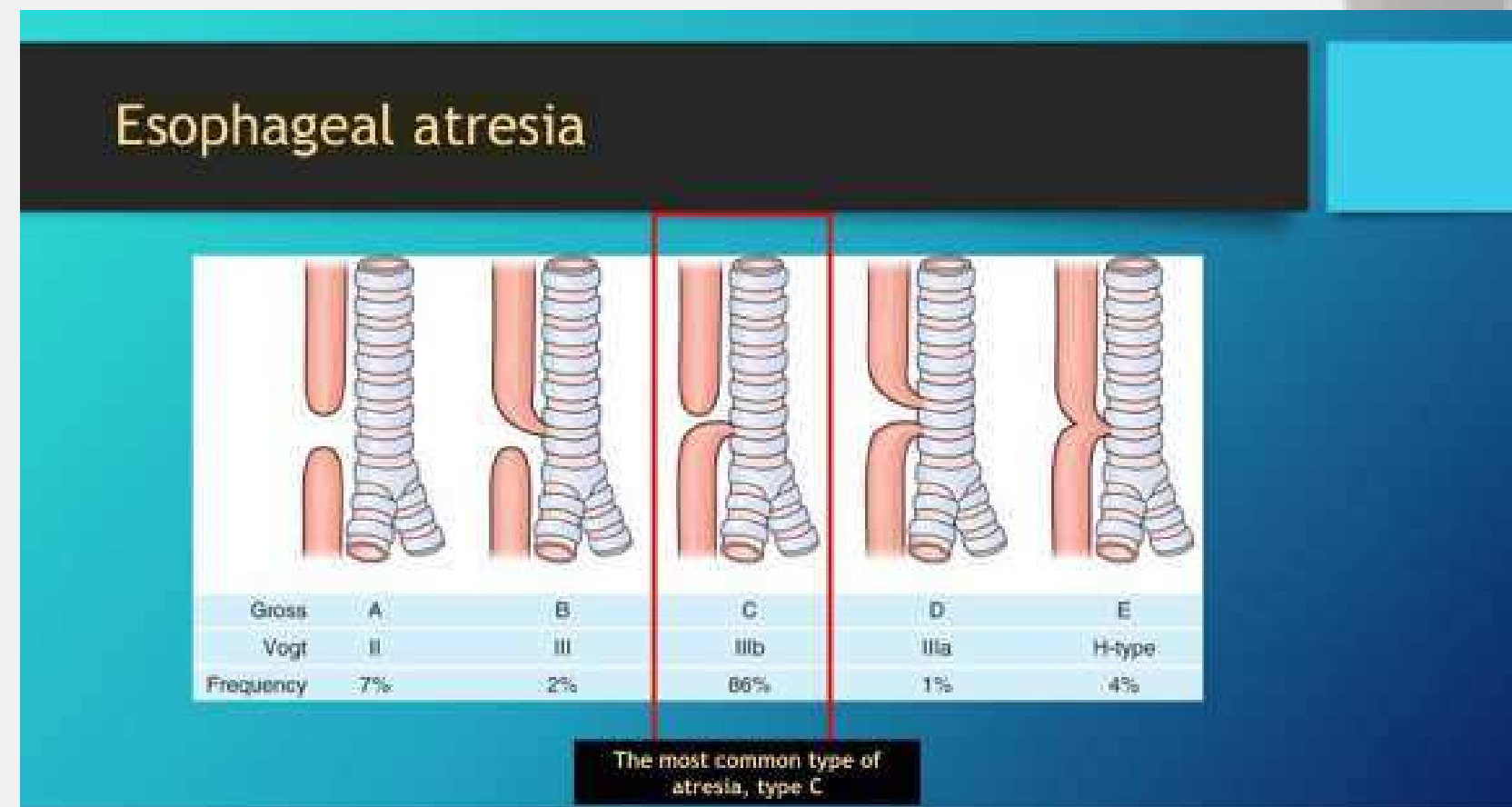
Anatomic and developmental disorder

Esophageal atresia

esophagus does not form properly. In this condition the upper part of the esophagus ends in a blind pouch , meaning it does not connect to the stomach as it should.

Esophageal fistula (specifically *tracheoesophageal fistula*, TEF)

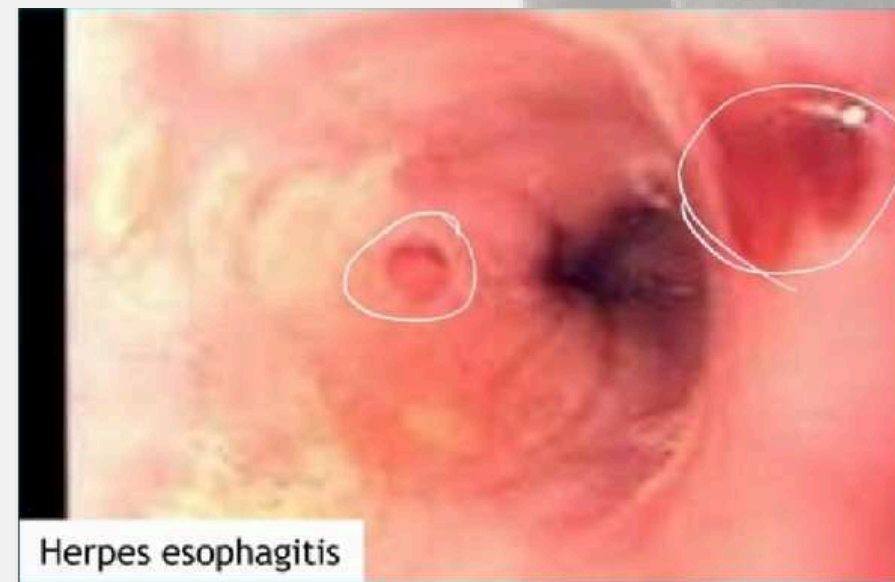
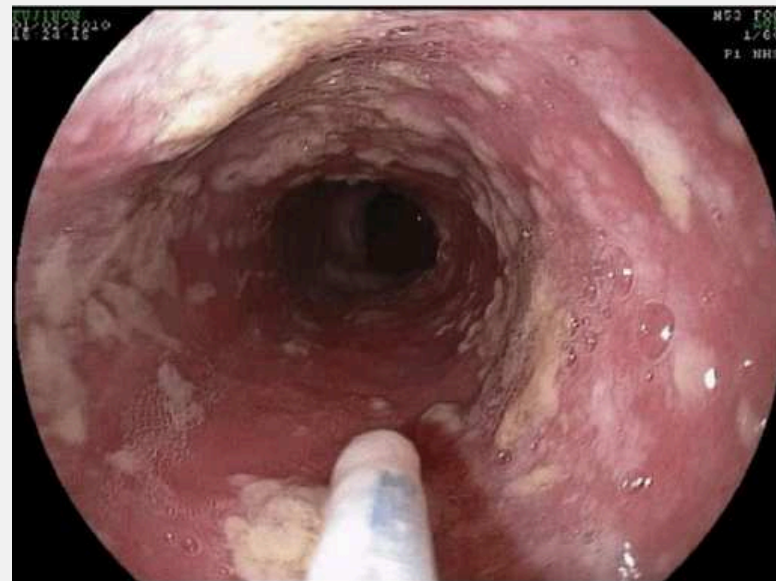
is an abnormal connection (fistula) between the esophagus and the trachea This can cause food or liquid to pass into the lungs, leading to respiratory issues



Esophagitis:

Esophagitis is an inflammatory condition of the esophagus with various causes, primarily related to acid reflux, infections, and irritants

- 1. Infectious esophagitis** : immunosuppressed, Herpes simplex viruses (HSV)-punched out ulcers, Cytomegalovirus (CMV) –linear ulcers, Fungal organisms –pseudohyphae on biopsy.
- 2. Chemical esophagitis** : irritating factors, causes odynophagia, severe cases may cause hemorrhage, stricture, or perforation
- 3. Eosinophilic Esophagitis** : chronic immunologically mediated disorder food impaction and dysphagia in adults and feeding intolerance or GERD-like symptoms in children, Endoscopically: circumferential rings



4-Reflux esophagitis

Normally:

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

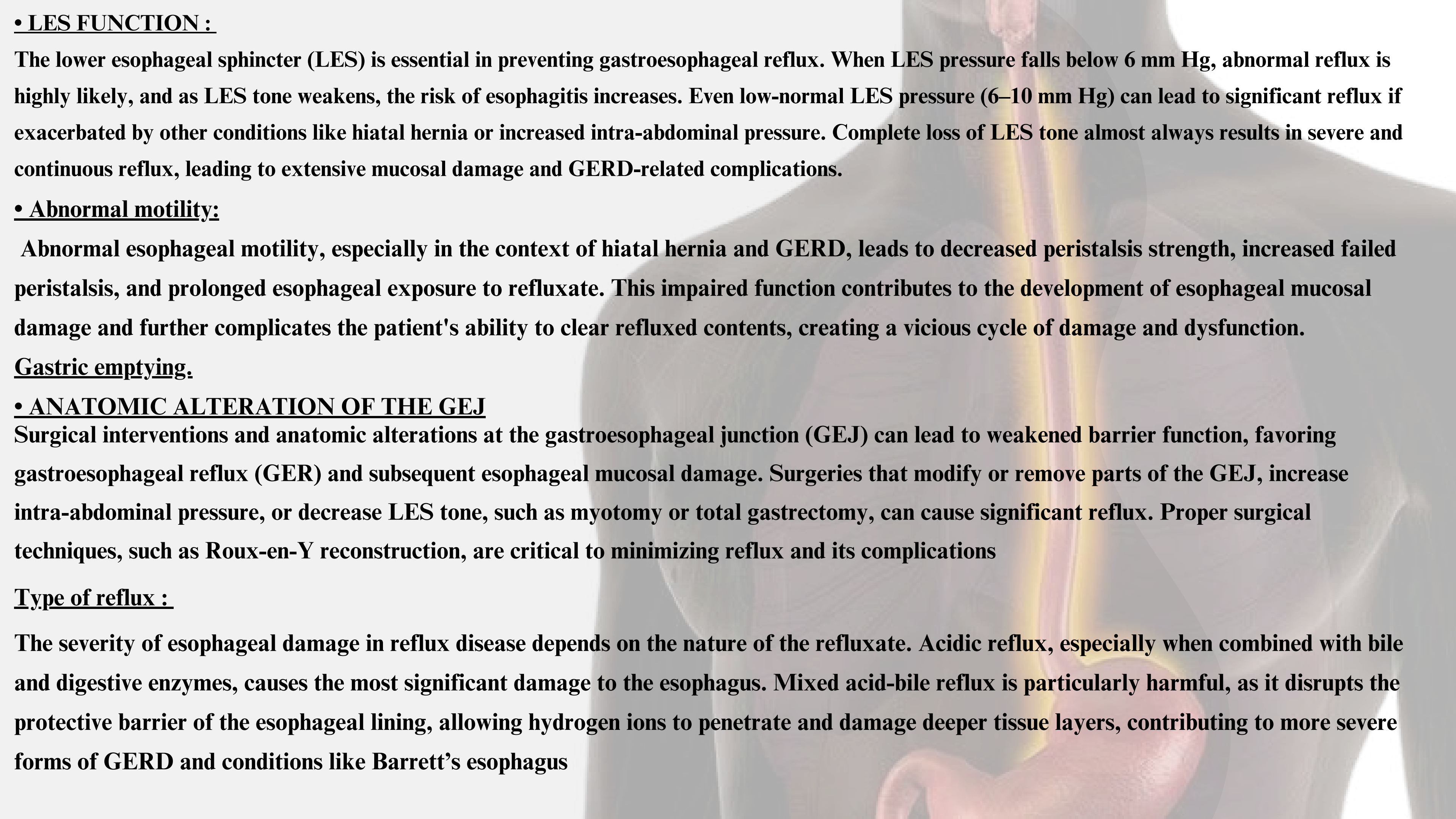
Primary and secondary peristalsis represent the most useful defense mechanism of the esophagus

- 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.

GERD :

clinical condition Reflux of gastric contents into the lower esophagus that cause heartburn, dysphagia, and, less often, noticeable regurgitation of sour-tasting gastric contents.

Factors that affect reflux:



• LES FUNCTION :

The lower esophageal sphincter (LES) is essential in preventing gastroesophageal reflux. When LES pressure falls below 6 mm Hg, abnormal reflux is highly likely, and as LES tone weakens, the risk of esophagitis increases. Even low-normal LES pressure (6–10 mm Hg) can lead to significant reflux if exacerbated by other conditions like hiatal hernia or increased intra-abdominal pressure. Complete loss of LES tone almost always results in severe and continuous reflux, leading to extensive mucosal damage and GERD-related complications.

• Abnormal motility:

Abnormal esophageal motility, especially in the context of hiatal hernia and GERD, leads to decreased peristalsis strength, increased failed peristalsis, and prolonged esophageal exposure to refluxate. This impaired function contributes to the development of esophageal mucosal damage and further complicates the patient's ability to clear refluxed contents, creating a vicious cycle of damage and dysfunction.

Gastric emptying.

• ANATOMIC ALTERATION OF THE GEJ

Surgical interventions and anatomic alterations at the gastroesophageal junction (GEJ) can lead to weakened barrier function, favoring gastroesophageal reflux (GER) and subsequent esophageal mucosal damage. Surgeries that modify or remove parts of the GEJ, increase intra-abdominal pressure, or decrease LES tone, such as myotomy or total gastrectomy, can cause significant reflux. Proper surgical techniques, such as Roux-en-Y reconstruction, are critical to minimizing reflux and its complications

Type of reflux :

The severity of esophageal damage in reflux disease depends on the nature of the refluxate. Acidic reflux, especially when combined with bile and digestive enzymes, causes the most significant damage to the esophagus. Mixed acid-bile reflux is particularly harmful, as it disrupts the protective barrier of the esophageal lining, allowing hydrogen ions to penetrate and damage deeper tissue layers, contributing to more severe forms of GERD and conditions like Barrett's esophagus

SIGNS AND SYMPTOMS

Most Common :

Regurgitations, Heartburn

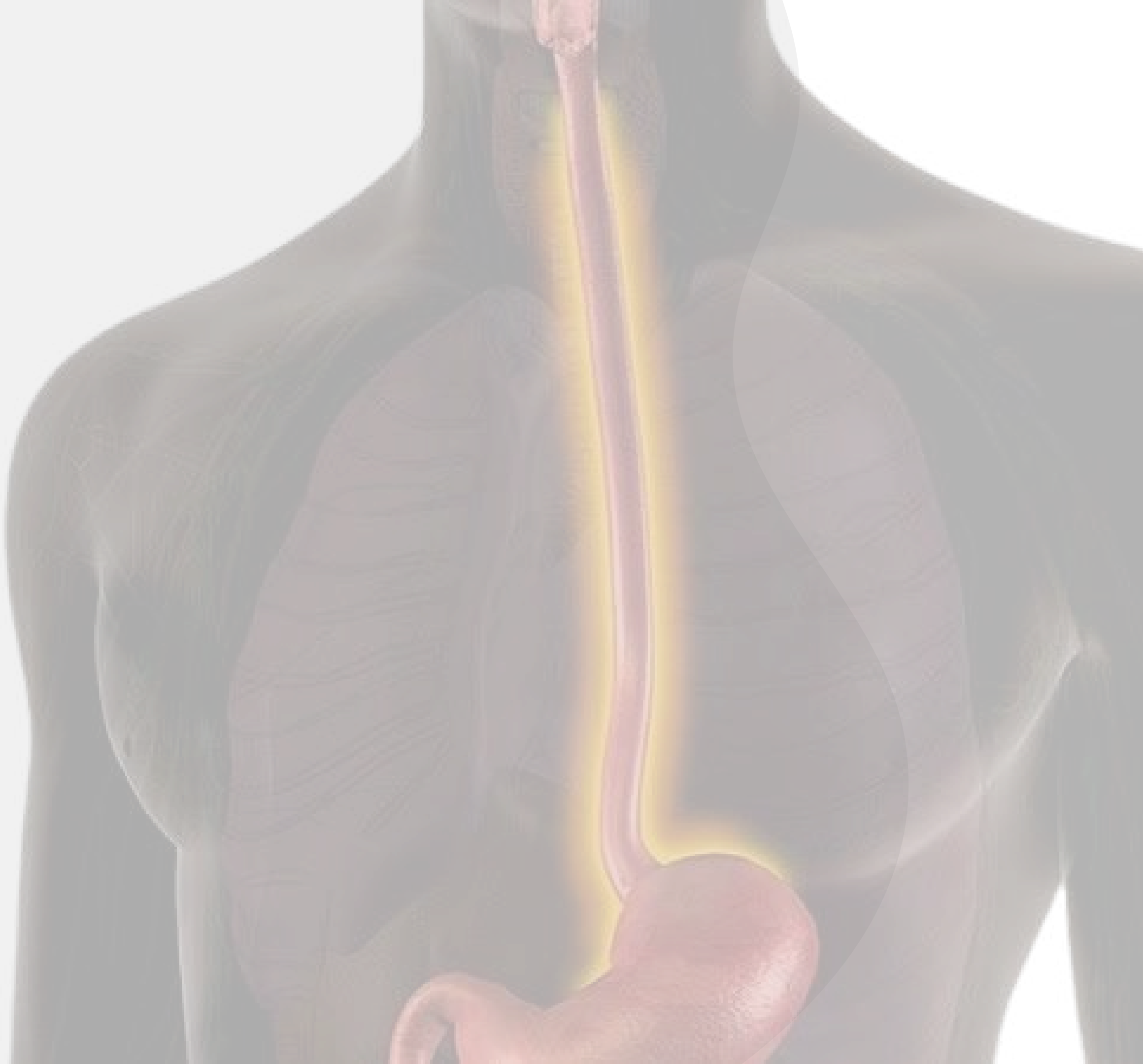
Related to Complications of Reflux Disease

Dysphagia, Odynophagia, Hematemesis, Melena

Unrelated to Esophageal Damage

Oropharyngeal dysphagia, Asthma, Chest pain,

Hoarseness, Pharyngiti



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															

Equivocal Evidence of Esophagitis

E0 = normal

E1 = erythema

Unequivocal Evidence of Esophagitis

E2 = erosions and ulceration

E3 = stricture or columnar-lined esophagus

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

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

Equivocal Evidence of Reflux

R0 = no reflux or <4%

R1 = reflux 4–7%

Unequivocal Evidence of Reflux

R2 = reflux 7–12%

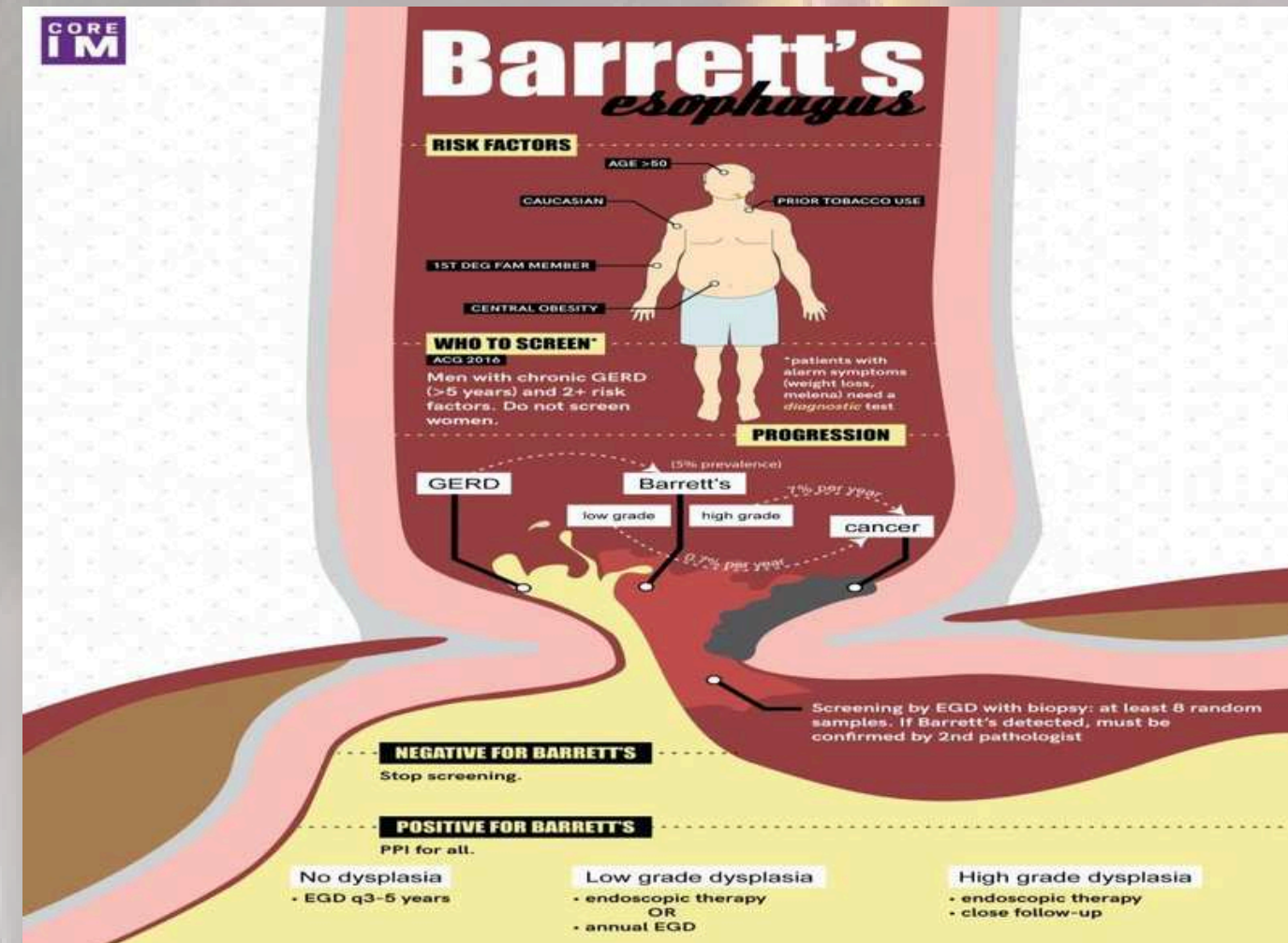
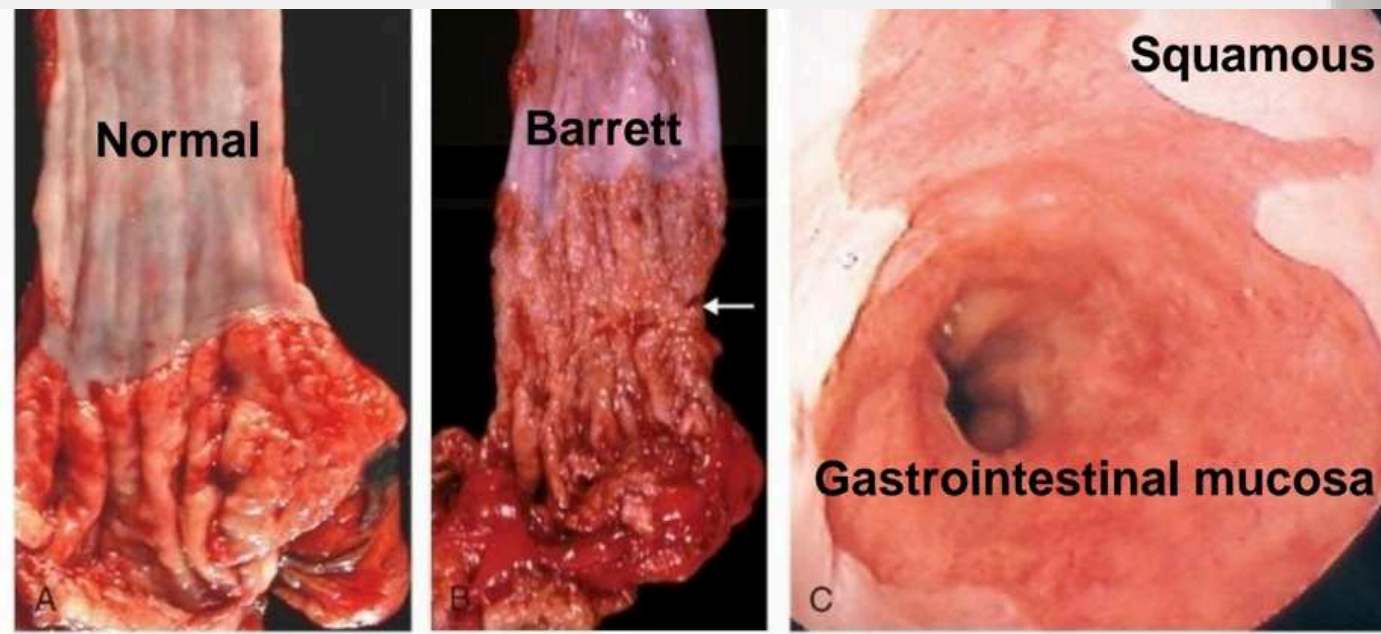
R3 = reflux >12%

Complications of GER :

destruction of mucosa > ulcer > fibrosis > obstruction of lumen > stricture, or Barrett esophagus. Other, less common complications include acute or chronic bleeding and aspiration.

Barrett esophagus

- Barrett e. is a complication of long-standing gastroesophageal reflux , it Is replacement of the normal distal esophageal stratified squamous mucosa by metaplastic columnar epithelium containing goblet cells.
- Pathogenesis: reflux > ulceration > healing > metaplasia.
- The chief complication of Barrett e. is the risk of development of adenocarcinoma. Which happen more in male than female.



Hiatal Hernia

•Dilated segment of the stomach protrude above the diaphragm.

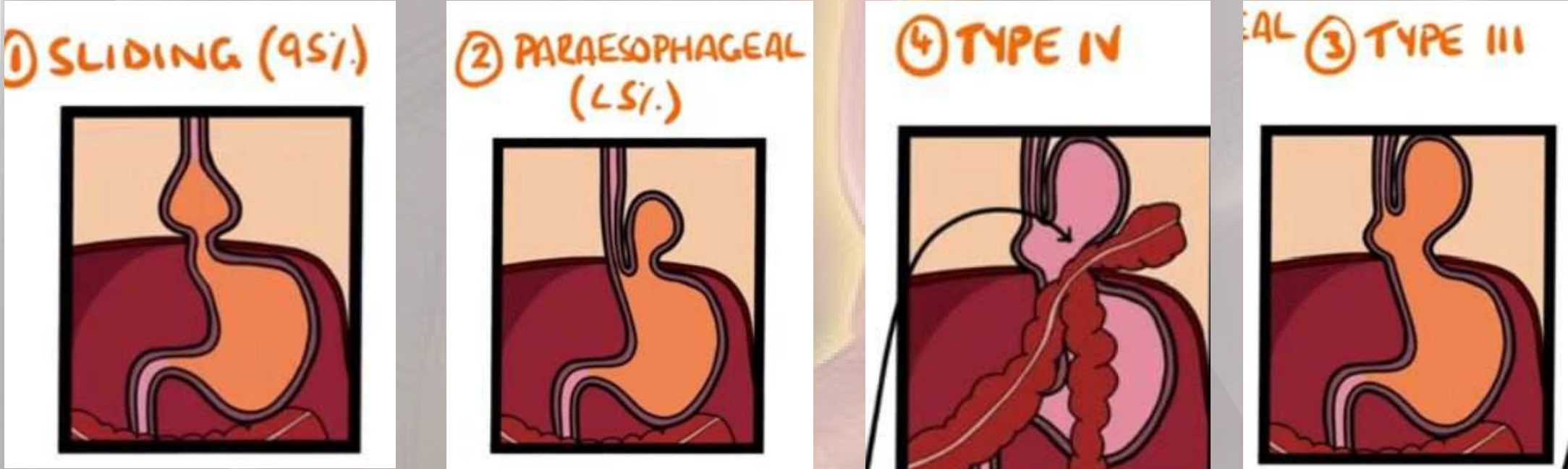
Symptoms

- 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.

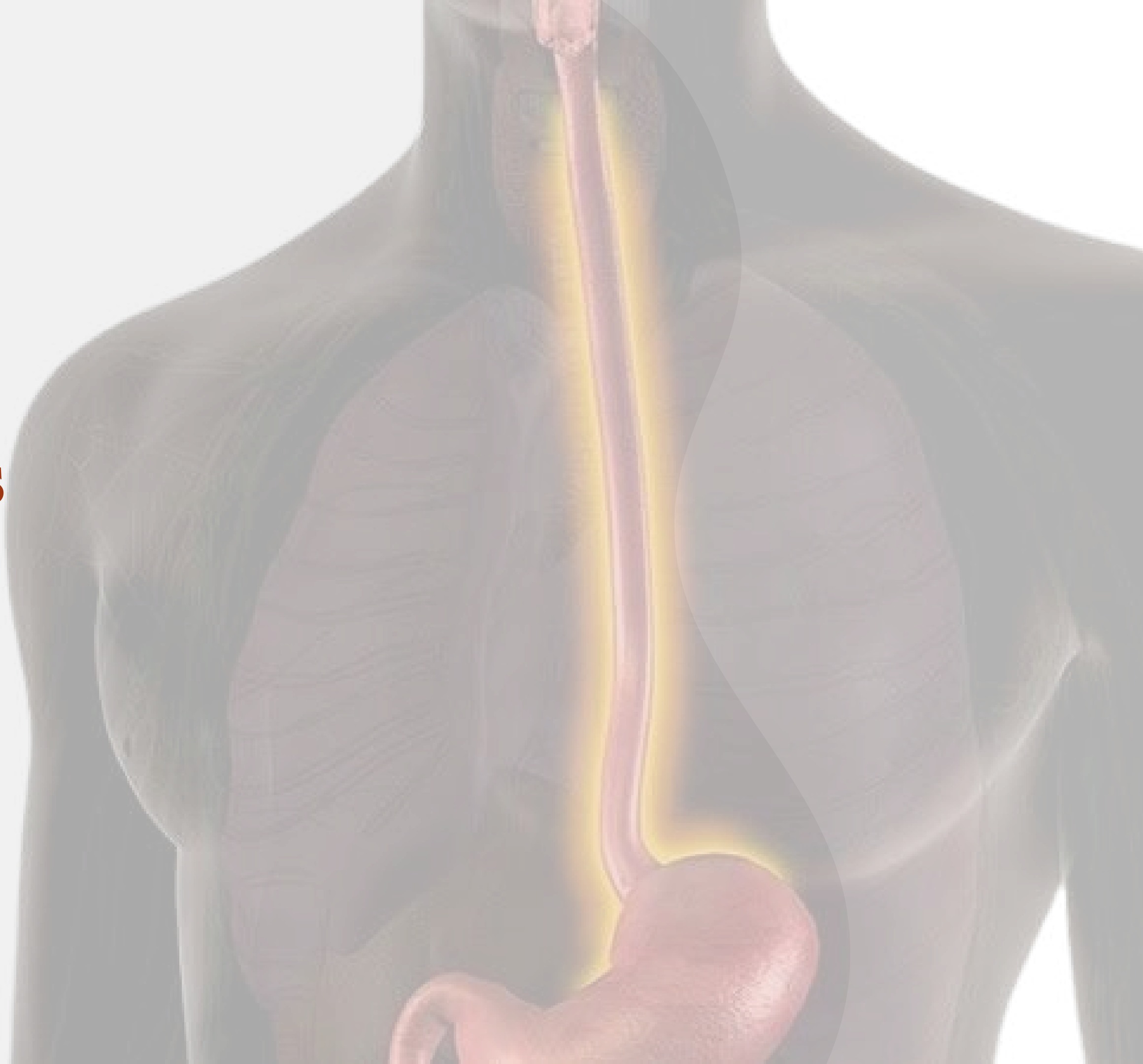
Type	Description
H0	No hiatal hernia
H1	Sliding hernia. GEJ above diaphragm
H2	Normal position of GEJ. Protrusion of the stomach alongside the esophagus
H3	Components of sliding and paraesophageal hernias GEJ is in the chest. Stomach rolls through the hiatus in a paraesophageal position
H4	Large hiatal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

- Complications:

- 1-Reflux esophagitis
- 2-Mucosal ulceration
- 3-Bleeding
- 4-Perforation



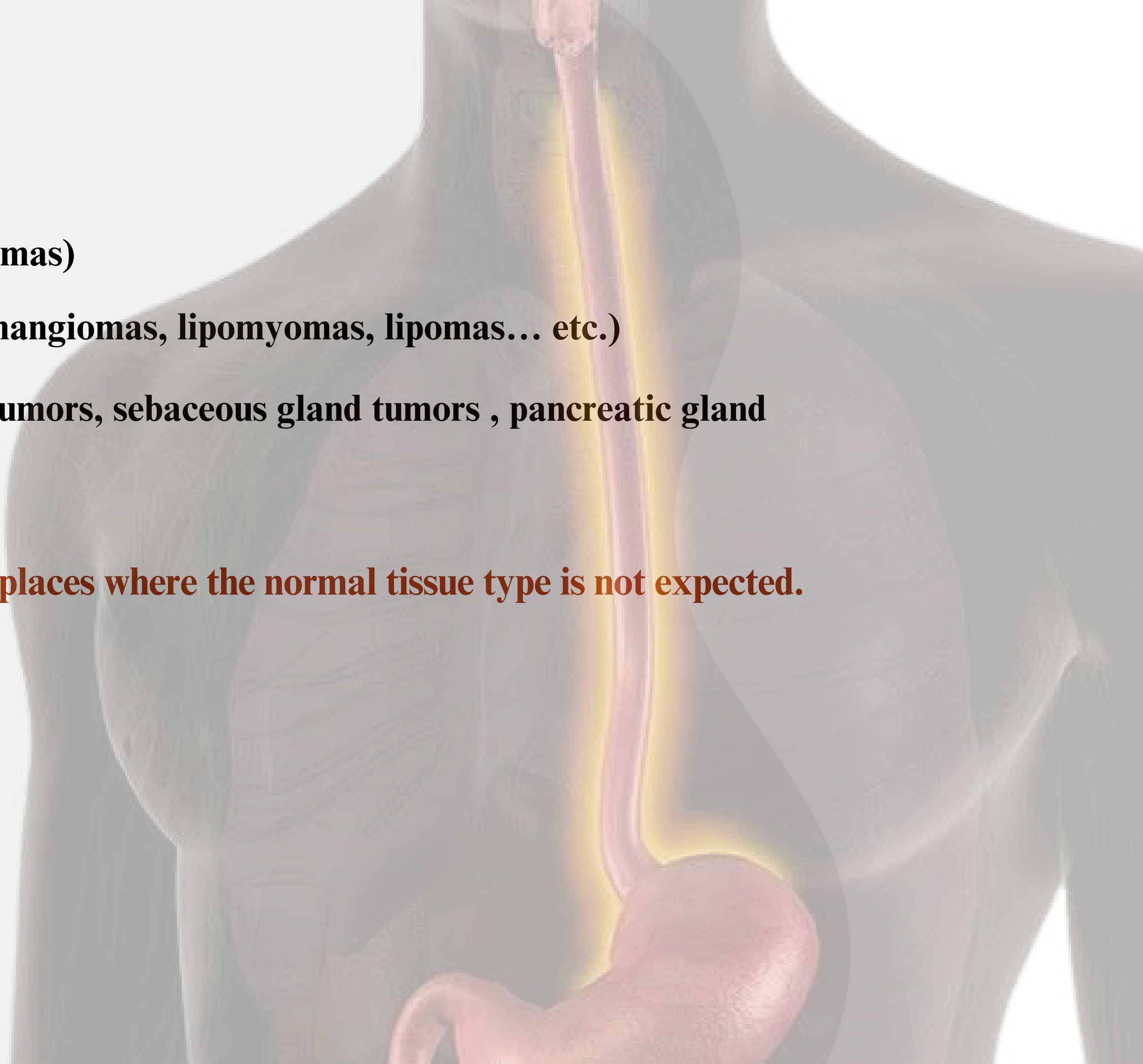
Esophageal Neoplasms



Benign Tumors

1. Epithelial tumors (e.g. polyps, cysts, adenomas)
2. Nonepithelial tumors (e.g. leiomyomas, hemangiomas, lipomyomas, lipomas... etc.)
3. Heterotopic tumors (e.g. gastric mucosal tumors, sebaceous gland tumors, pancreatic gland tumors... etc.)

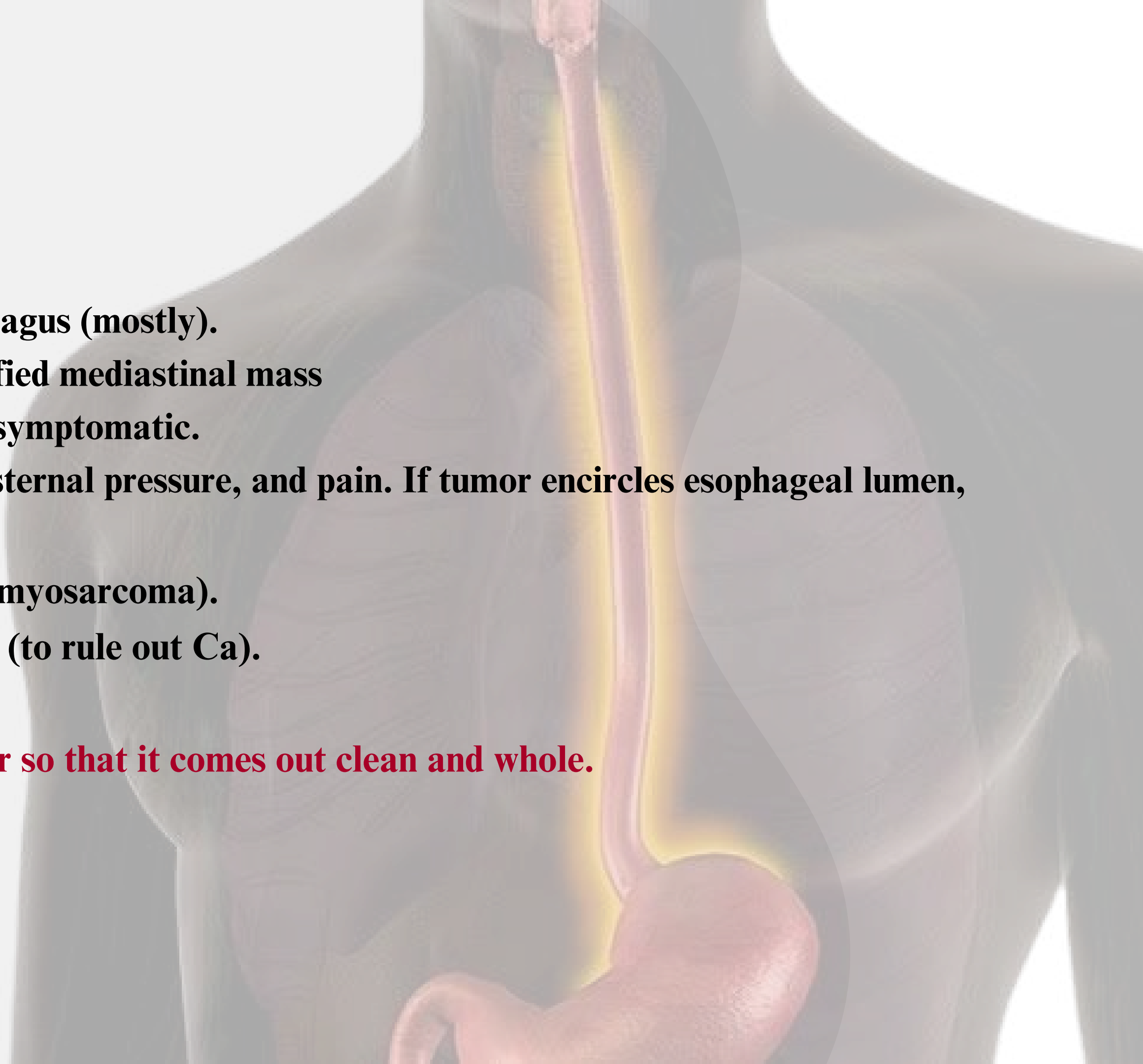
Heterotopic tumors: tumors that develop in places where the normal tissue type is not expected.



Leiomyomas

- **Type:** Benign.
- **Frequency:** Most common benign tumor.
- **Location:** Middle and lower thirds of esophagus (mostly).
- **May become calcified** (identified as a calcified mediastinal mass)
- **If size < 5 cm**, it would most probably be asymptomatic.
- **Symptoms (size > 5 cm):** Dysphagia, retrosternal pressure, and pain. If tumor encircles esophageal lumen, obstruction and regurgitation may occur.
- **Low probability of becoming malignant (leiomyosarcoma).**
- **Diagnosis:** Barium swallow, esophagoscopy (to rule out Ca).
- **Treatment:** enucleation.

Enucleation: removal of an organ or a tumor so that it comes out clean and whole.



Most Common Malignant Tumors

	Adenocarcinoma	Squamous Cell Carcinoma
Location (most common)	Lower one-third.	Upper and middle thirds.
Risk factors	Barrett esophagus, GERD, obesity, previous radiation therapy, tobacco smoking.	Hot beverages, alcohol , caustic injuries, achalasia, nitrosamine-rich foods, previous radiation therapy, tobacco smoking.
More common in (in the US)	Men. Whites	Men. African-Americans.
Lymph nodes spread	Paraoesophageal, celiac axis, and splenic hilum nodes.	Location-dependent: Upper 1/3: cervical lymph nodes. Middle 1/3: mediastinal and tracheobronchial nodes. Lower 1/3 (less common): celiac and gastric nodes.
Organ metastasis	Lung and liver.	Diaphragm, pericardium, and stomach.

Malignant Tumors Clinical Presentation

- Malignant esophageal tumors generally have a poor prognosis due to advanced disease at presentation (symptoms appear quite late in the disease).

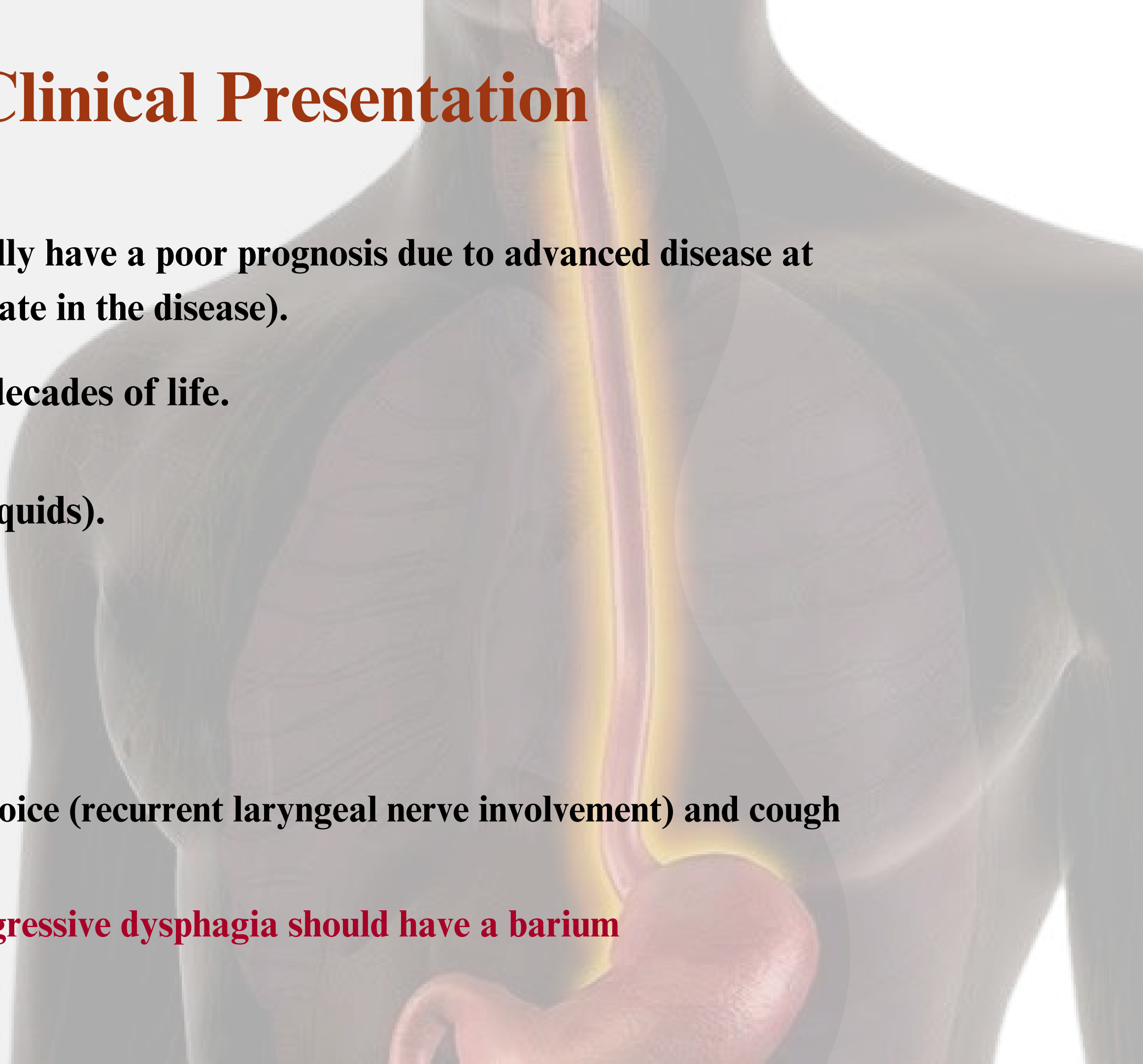
Typically occurs during the 6th-7th decades of life.

Symptoms include:

1. Progressive dysphagia (solids to liquids).
2. Odynophagia.
3. Weight loss.
4. Hematemesis.
5. Chest pain.

- SCC may present with hoarseness of voice (recurrent laryngeal nerve involvement) and cough (tracheal involvement).

Note: any adult complaining from progressive dysphagia should have a barium esophagogram and esophagoscopy.



Diagnosis

Esophagoscopy and biopsy	Mandatory in every patient with an esophageal stenosis.
Esophageal biopsy and cytology	Establishes a diagnosis in 95% of patients with malignant strictures.
Double-contrast barium swallow	Can demonstrate lesions as small as 5-15 mm.

Evaluation

Evaluation (staging of the tumor)	
Barium swallow	First study performed; provides information on the location and length of tumor.
Endoscopic ultrasound	Defines the depth of mural invasion and to assess associated paraoesophageal lymph nodes.
Chest X-ray	Abnormal in only 50% of patients (may be deceptively normal). (Findings: air-fluid level in the obstructed esophagus, dilated esophagus, abnormal mediastinal soft tissue, pleural effusion, or pulmonary metastasis.)
Abdominal and Chest CT scan	Evaluation of the esophageal wall thickness (normal: < 5 mm). Assessment of invasion and metastasis.
Bronchoscopy	To exclude the airway involvement and assess the recurrent nerve function.
MRI	Evaluate mediastinal structures.

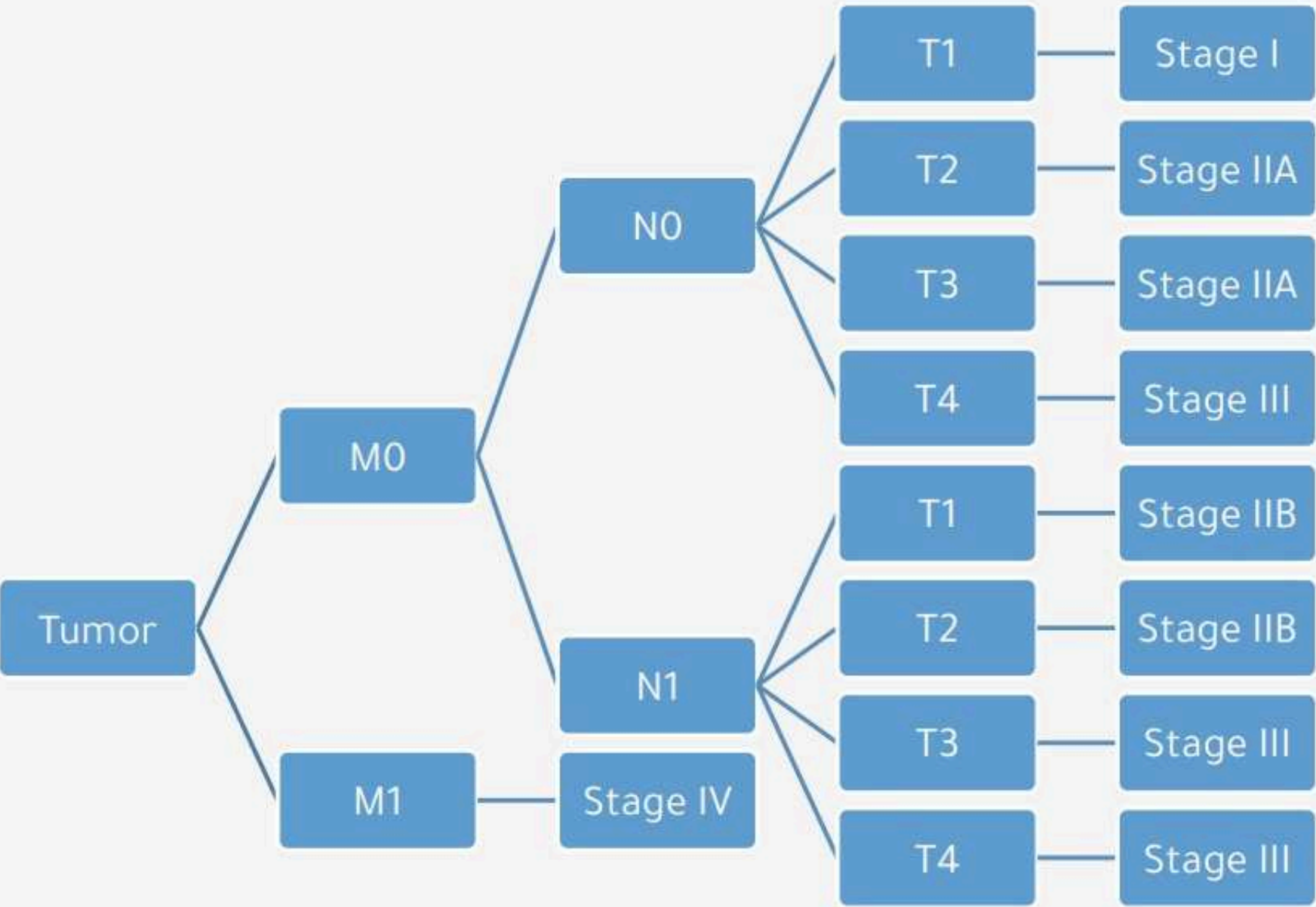
TNM Classification

T (depth of invasion of the primary tumor)	
Tx	Primary tumor cannot be assessed (cytologically positive, but not evident on endoscopy or radiology).
T0	No evidence of primary tumor (post-treatment).
Tis	Carcinoma in situ.
T1	Tumor invades up to (but not beyond) lamina propria or submucosa.
T2	Tumor invades muscularis propria.
T3	Tumor invades adventitia.
T4	Tumor invades adjacent structures.

N (regional lymph nodes involvement)	
Nx	Regional nodes cannot be assessed.
N0	No regional lymph node metastasis.
N1	Regional lymph node metastasis.

M (distant metastasis)	
M0	No metastasis.
M1	Distant metastasis.

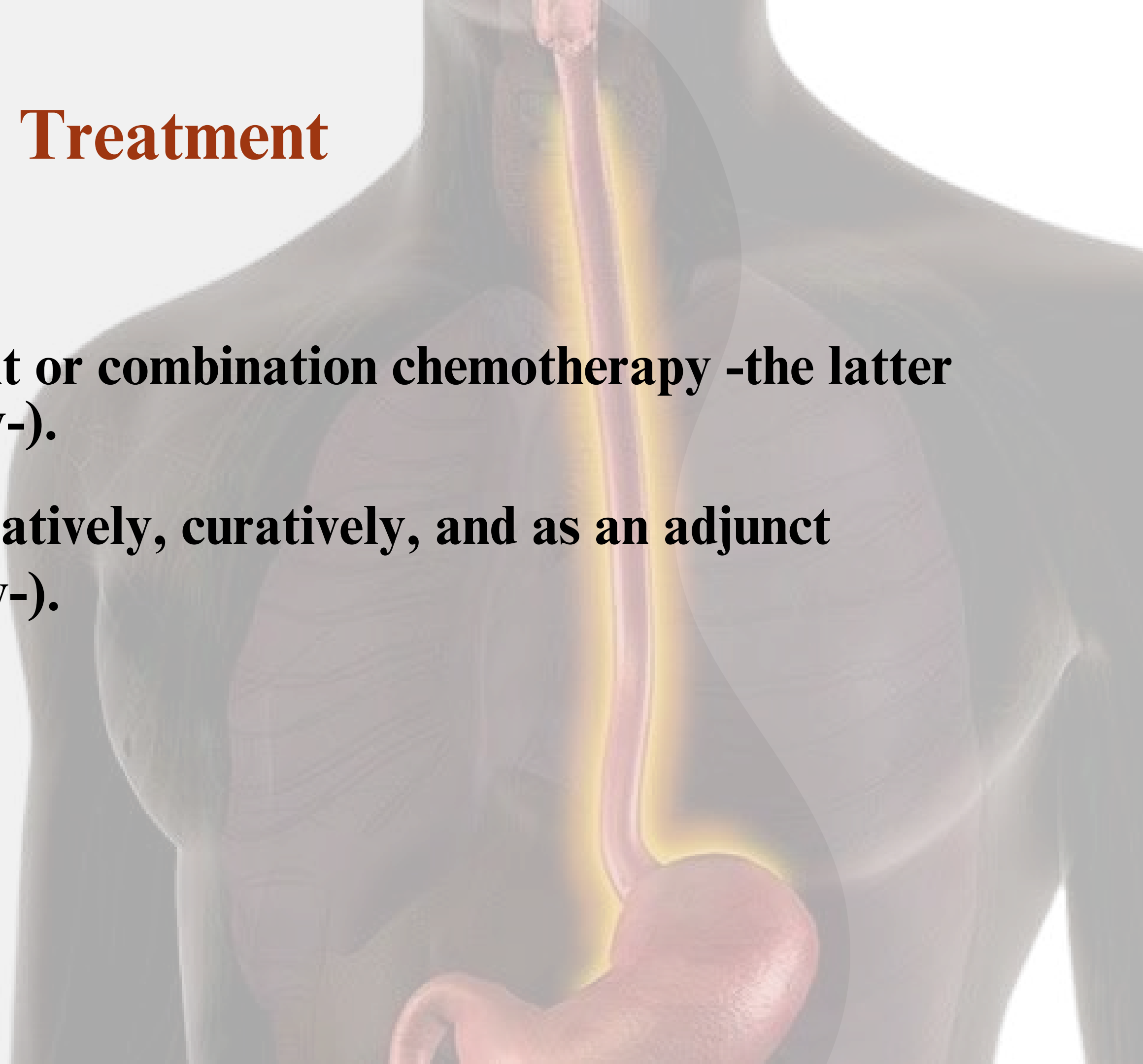
TNM Classification



Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage IIA	T2 T3	N0 N0	M0 M0
Stage IIB	T1 T2	N1 N1	M0 M0
Stage III	T3 T4	N1 Any N	M0 M0
Stage IV	Any T	Any N	M1

Treatment

- **Surgery (mentioned later).**
- **Chemotherapy (single-agent or combination chemotherapy -the latter has been used preoperatively-).**
- **Radiation therapy (used palliatively, curatively, and as an adjunct therapy -to esophagectomy-).**
- **Transoral intubation.**
- **Endoscopic laser therapy.**
- **Palliative internal bypass.**



Surgical procedure of non neoplastic esophageal disorders

Investigation:

A. Plain chest X -ray **B. Barium swallow** **C. Esophagoscopy**

D. Manometric studies (best investigations in early cases) : diagnostic tests used to assess the pressure dynamics and motility of the esophagus (peristalsis) , inserting a pressure-sensitive tube through the nose, down the esophagus, and into the stomach.

The tube has sensors that measure the muscle contractions of the esophagus as the patient swallows .

E. 24 hours esophageal pH studies: a probe in the esophagus will demonstrate reflux of gastric acid and its temporal relation to pain.

Treatment

- In mild cases

A. Medical treatment: by nitrate or calcium channel blockers are of limited temporary benefit.

B. Esophageal dilation by hydrostatic or pneumatic bag .

C. injection of botulinum toxin in the wall of the narrow segment of the esophagus

- In severe cases

D. laparoscopic Heller's operation (esophagocardiomyotomy) : The safest , most reliable and fastest treatment, Longitudinal incision is made in the muscle coat of lower 7 cm of esophagus and upper 2 cm of stomach . reflux esophagitis may occur so Laparoscopic Nissen's fundoplication and Collis gastroplasty, done to prevent the reflux.

E. Esophagotomy : surgical procedure involving an incision into the esophagus , threes diffrent types as cervical , thoracic and abdominal.

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

Stage 0,1 -> lifestyle changes

stage 2-> after medical management for 6-12 month -> decide weather to continue or anti reflux surgery

stage 3-> medication until 6-12 month with lose weight during this period ->decide if surgery needed or not

Surgical procedure of neoplastic esophageal disorders

Investigation:

I) Laboratory investigations:

Occult blood in stool : usually positive .

Blood picture : usually show anaemia.

Tumor markers : CEA , CA19-9 & CA125 evaluate response to treatment and follow up of the patient .

II) Radiological investigations :

1.Barium swallow: may show Irregular narrowing with mild proximal dilatation, Shouldering, Rat tail appearance, Irregular filling defect.

2. U/S & endoscopic U/S is very important to detect local extent of the tumor , mediastinal invasion and lymph nodes enlargement .

3. C.T. scan : chest and upper abdomen.

4. PET scan (positron emission tomography scan) : show local tumor , nodal & distal metastases for accurate staging.

III) Oesophagoscopy and biopsy are the most important investigaions.

**IV) Investigations to detect metastasis :
(Mention in any malignancy)**

Plain x-ray

U/S , CT & MRI

Radioactive isotopic scan

PET scan & PET-CT.

Aspiration of pleural effusion or ascites

Laryngoscopy , bronchoscopy ,Thoracoscopic and laparoscopic exploration with biopsy

Surgical procedure of neoplastic esophageal disorders

Treatment

1) Operable case:

- laparoscopic and thoracoscopic oesophagoectomy** with oesophago-gastric anastomosis in the neck or the thorax .
- Oesophagogastrrectomy** :in lower 1/3 carcinoma of esophagus, Through left thoraco-abdominal incision excision of the esophagus and upper part of stomach with gastric pull up to perform oesophago-gastric anastomosis in the thorax
- Ivor Lewis**: Carcinoma middle 1/3 of oesophagus .two incision , first one in upper middle line abdominal incision , to reach the mobilized stomach . Second incision in the right 5th. intercostals space the oesophagus is excised down to the cardia with oesophago- gastric anastomosis in the thorax (gastric pull up).
- Transhiatal approach** : for middle and upper carcinoma ,The most popular total oesophagectomy

2) Inoperable cases:

- oesophagoscope a rigid tube or stent is inserted into the tumor to keep the lumen patent to relieve dysphagia is the best.
- Radiotherapy and chemotherapy.
- Endoscopic laser photocoagulation cause coagulative

*THANK
YOU*

