#### Foreword:

What follows is the work of 4 colleagues bewildered by too many sources and with no desire to jump through books. It includes:

- Re-written and revised 2021-2022 slips
- Unigaster when necessary
- Something about the old blocks

It can be considered a one-stop source for taking the gastroenterology and endocrinology exam, but, as always, no handout replaces textbooks.

Your choice.

Good luck.

# Gastroenterology

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## **Esophageal Symptoms**

**Heartburn (heartburn)** = retrosternal burning sensation that occurs as a result of gastroesophageal reflux, occasionally reaching the neck and/or back. It takes the form of an intermittent pain often exacerbated by meals (rich in fat, chocolate, mint), alcohol intake, supine position (gravity problems, that's why we tend to raise the head of the bed) and abrupt increases in abdominal p. The pathogenesis would seem to be related to dilation of intercellular spaces with H+ ions entering the deep layers of the mucosa where chemoreceptive nerve endings are located.

**Regurgitation** = perception of rising stomach contents in esophagus and oropharynx, more frequent in supine or anti-flexion position. This is a symptom that usually represents more major reflux disease. It may represent a symptom during acid-suppressive therapy because of weakly acidic refluxes (pH is controlled but if there is still reflux, regurgitation is felt).

**Dysphagia** = a sensation of halting, difficulty and/or slowed transit of the bolus on its way from the mouth to the stomach ("the pc reports that he cannot get the mouthful down"). It differs, however, from *bolopharyngeal*, a continuous sensation of a foreign body referred to the throat, independent of swallowing that is often present in patients with an anxious psychological attitude (the pc cannot swallow, but the pathway is pervious). It is distinguished:

<u>Oropharyngeal (or high or globe)</u> = striated muscle involvement, pc reports that the fact that he cannot progress the bolus from mouth/hypopharynx forward. It indicates neuromuscular dysfunction, which is more common in the elderly and thus the most relevant causes are:

- Cerebrovascular problems
- Amyotrophic lateral sclerosis
- Parkinsonisms (frequent, for an elderly person with globe should always be considered)
- Myasthenia gravis (more frequent in women)
- Tardive dyskinesia

Another possible cause is **Zenker's diverticulum**, an extroversion at the level of the esophageal tract upper, which is investigated with radiography.

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<u>Esophageal (or proper)</u> = inability to progress the bolus from the esophagus to the stomach, usually reported at the level of the epigastrium with and undigested, nonacidic regurgitation (food has not yet reached the stomach), is the most common (the bolus after being swallowed gets stuck at the passage between the esophagus and stomach). Causes:

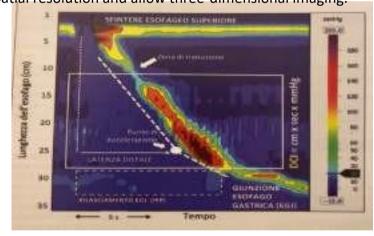
- Motility disorders with involvement of the smooth muscle of the esophageal body or LES (achalasia and related non-acalasia disorders)
- Mechanical (e.g., neoplasm)
- Autoimmune diseases (SLE, scleroderma, polymyositis)
- GERD (not a major manifestation, but sometimes there are some atypical ones)
- Esophageal diverticula (more frequent in the elderly)
- Foreign bodies
- Pill-induced damage, caustic injury
- Infectious esophagitis (e.g., candida, which may raise suspicion of immunodeficiency)

Achalasia is the most frequent cause of dysphagia, and is characterized by inability to release the lower esophageal sphincter, with increased pressure, and lack of peristaltic contraction. The etiology is unknown, but genetic, degenerative, autoimmune, and infectious (VZV) causes are possible hypotheses; in any case, there is functional loss of the neuronal component of the myenteric plexus, which should convey the post-deglutition release signal: normally, neurons innervating the LES release NO; when they decrease, cholinergic neuron-mediated constriction prevails, which counterbalances the nitrergics. The consequence is the impact of the bolus against the sphincter and, in the long run, the esophageal musculature becomes exhausted and dilated (pcs often report having to drink a lot during meals). In addition to dysphagia for solids and liquids (but obviously the probability of a liquid passing is higher), symptomatology manifests with nonacid regurgitation (60-70%) and chest pain. Complications: malnutrition, weight loss, and ab-

ingestis pneumonia.

**Diagnosis** is made by manometry, the requirement for which is secondary to either an <u>x-ray</u> with <u>mdc</u> (barium) showing esophageal dilatation with 'bird's beak' appearance of the SES (filiform appearance of contrast) or an <u>endoscopy</u> always indicated to rule out stenosis, foreign bodies or neoplastic lesions at the gastro-oesophageal junction, which could result in pseudoacalasia. The gold standard, however, is <u>esophageal manometry</u>, which is instead performed with a tube inserted through the oral cavity (hydraulic pressure tube) that physiologically, at the level of the SES, should record a p of 80 mmHg, then a negative intraesophageal p of -5 mmHg, and then a p of 20 mmHg at the level of the LES: the latter in cases of achalasia can be as high as >40 mmHg. However, to date, it has been replaced by a newer technique which is <u>high-resolution manometry</u> (HRM) that performs recording with air-hydraulic pumps, software and hardware that assess motor activity continuously withbetter spatial resolution and allow three-dimensional imaging.





### Chicago classification of achalasia:

- Type 1: there is peristalsis, but the esophageal sphincter does not relax
- Type 2: There is still residual, but not peristaltic, activity of the esophageal musculature
- Type 3: premature and spastic contractions of the muscles of the esophagus

This classification is useful in guiding the surgeon and patient to the best treatment approach (e.g., those of type 3 will be referred to a first-choice treatment approach, i.e., POEM).

### Achalasia therapy

-Pharmacological: calcium channel blockers, anticholinergics or nitroderivatives (to relax muscles) that are poorly effective in the long term. They are generally used only as a gateway therapy.