Oesophageal and gastric motility

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Abstract

The oesophagus and stomach are responsible for transporting food from the oral cavity to the small intestine in a manner that does not compromise the safety of the airway, that is relevant to the composition of the meal, and that allows for optimal absorption of nutrients. To achieve this, several control mechanisms interact to ensure that the swallowed bolus traverses the oropharynx into the oesophageal body and enters the gastric reservoir. Here the bolus undergoes mixing and is transformed into a consistency suitable for emptying through the pylorus at a rate consistent with the maximal absorptive capacity of the small intestine.

In health we take this process for granted, but relatively small changes in the coordination of these motor events may induce a diverse array of symptoms. Such symptoms may have serious clinical sequelae, such as aspiration pneumonia and malnutrition, which can be life threatening. In this article, we shall aim to provide a concise overview of the anatomy, physiology and motor function of the oesophagus and stomach, paying particular attention to investigative methods that are currently used in clinical practice.

Keywords physiological measures; upper gastrointestinal motility

Introduction

In patients presenting with symptoms thought to emanate from the foregut, motility testing of the upper GI tract may be undertaken, which may facilitate diagnosis and aid in management decisions. In this brief review we will describe the motor function of the oesophagus and stomach and provide a summary of the contemporaneous diagnostic approaches.

The oesophagus

Upper oesophageal sphincter

The proximal oesophagus begins at the pharyngo-oesophageal junction with a region of high pressure known as the upper oesophageal sphincter (UOS). The cricopharyngeus and thyropharyngeus muscles are major contributors to this high-pressure zone. The UOS performs two main functions: it prevents air from entering the oesophagus during breathing; and it prevents refluxed intra-oesophageal content from entering the airway. The UOS is located at the level of cricopharyngeus muscle and this 1-cm segment usually corresponds to the region of maximal UOS pressure.[1] The UOS is 2–4 cm in length and is closed at rest. Excitatory neural input maintains closure but is suppressed during deglutition, which, in conjunction with anterior and superior movement of laryngeal structures, leads to UOS relaxation and opening. This process is coordinated with pharyngeal peristalsis to allow entry of the bolus into the oesophageal body. The UOS will also relax in response to rapid distension of the proximal oesophagus, in order to allow venting of gas during belching or solid matter during vomiting. Slow distension or acidification of the proximal oesophagus leads to an increase in UOS pressure, consistent with this region's role in the prevention of refluxed gastric contents entering the airway.[2]

Oesophageal body

The normal adult oesophageal body is approximately 20 cm long. The proximal 5% of the oesophagus is composed of striated muscle, with the following 35–40% being a mixture of striated and smooth muscle, representing a transition zone, the remaining portion being composed exclusively of smooth muscle. The muscularis propria comprises an inner circular layer and an outer longitudinal layer. As one progresses distally there is a transition zone where the musculature changes from striated to smooth muscle, which predominates in the distal two-thirds of the oesophageal body.[3] The peristaltic sequence reflects this change in musculature and three distinct contractile segments can be observed on topographic assessment; these determine three distinct regions referred to as the cervical, thoracic and abdominal oesophagus (see below).

The oesophagus has both parasympathetic and sympathetic innervation and comprises efferent and afferent pathways.[4]

Efferent pathways: the parasympathetic nervous system provides motor innervation to the oesophageal smooth musculature. In contrast, the sympathetic nerve supply emanates from T1–T10 and regulates the associated vasculature, sphincteric tone and oesophageal muscular relaxation. The striated muscle of the oesophagus is innervated by lower motor neurons, of the somatic type, arising from the nucleus ambiguous in the brainstem. Within the distal smooth muscle segment of the oesophagus, vagal innervation, arising from the dorsal motor nucleus of the medullary portion of the brainstem, is composed of two distinct types of pre-ganglionic fibres. The first of these are short latency fibres, which synapse on postganglionic nitrergic inhibitory neurons in the myenteric plexus, also known as Auerbach's plexus. The second are long latency fibres, which synapse on postganglionic excitatory neurons in the myenteric plexus.

Afferent pathways: these are vagal afferents, whose cell bodies are in the nodose ganglia, which arise from the smooth muscle layer and serosa, and are sensitive to mechanical stretch in contrast to those arising from the mucosa, which encode chemical, thermal and intraluminal distension. Sympathetic, more commonly referred to as spinal, afferents have their cell bodies in dorsal root ganglia and are predominantly nociceptors.

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There are four phases of swallowing. In the first phase, referred to as the preparation phase, the food bolus is masticated and mixed with saliva, allowing it to be shaped and sized, and moved to the dorsum of the tongue. This is followed by a second oral phase during which the tongue contracts against the soft and hard palates, thereby generating a wave of pressure that advances the bolus into the pharynx. During the third pharyngeal phase, the soft palate rises, thereby sealing the nasopharynx to prevent regurgitation into the nasal cavities, and the UOS relaxes, enabling the bolus to enter the proximal oesophagus. This stage is associated with concomitant closure of the larynx to protect the airway. In the final phase, the oesophageal phase, peristalsis within the striated muscle of the proximal oesophagus is mediated by sequential excitation of lower motor neurons arising from efferent vagal fibres. In contrast, peristalsis within the smooth muscle of the distal oesophagus is mediated by a combination of central and peripheral mechanisms.

Central mechanism: the central mechanism involves the synchronization of short- and long-latency vagal fibres that arise in the dorsal motor nucleus. On swallowing there is near-concurrent depolarization of short-latency fibres that inhibit contraction along the length of the oesophagus, a process referred to as deglutitive inhibition. In addition, there is a delayed activation of cholinergic, excitatory, long-latency vagal efferent fibres that regulate oesophageal peristalsis.

Peripheral mechanism: the peripheral mechanism involves the combination of inhibitory (non-cholinergic) and excitatory (cholinergic) neurons, in which the former become activated while the latter are suppressed, increasing the duration of deglutitive inhibition and leading to enhanced efficiency of oesophageal peristalsis.

Lower oesophageal sphincter (LOS)

The LOS is a thickened segment within the circular muscle layer of around 2–4 cm in length, which is also tonically contracted (closed) in the resting state. LOS pressure decreases within 1.5–2 seconds following initiation of a swallow and the sphincter remains relaxed until the peristalsis reaches its proximal margin. Following passage of the bolus, the LOS often enters into a hypercontractile phase for several seconds. Resting LOS tone is maintained by clasp and sling muscle fibres that project around this region from the proximal stomach, as well as via active tonic neural excitation.[5] This tone is modulated by many factors, including meal composition, body position, oesophageal acidification and hormonal factors. Relaxation of the LOS also occurs in response to distension of the proximal stomach or oesophageal body and these events may trigger what have come to be known as transient lower oesophageal sphincter contractions (TLOSRs).[6] TLOSRs are vagally mediated and thought to play an important pathophysiological role in gastro-oesophageal reflux disease.

Assessing oesophageal motility

Oesophageal motility can be evaluated most effectively by using a combination of technologies. The recent advent and widespread adoption of high-resolution oesophageal manometry,

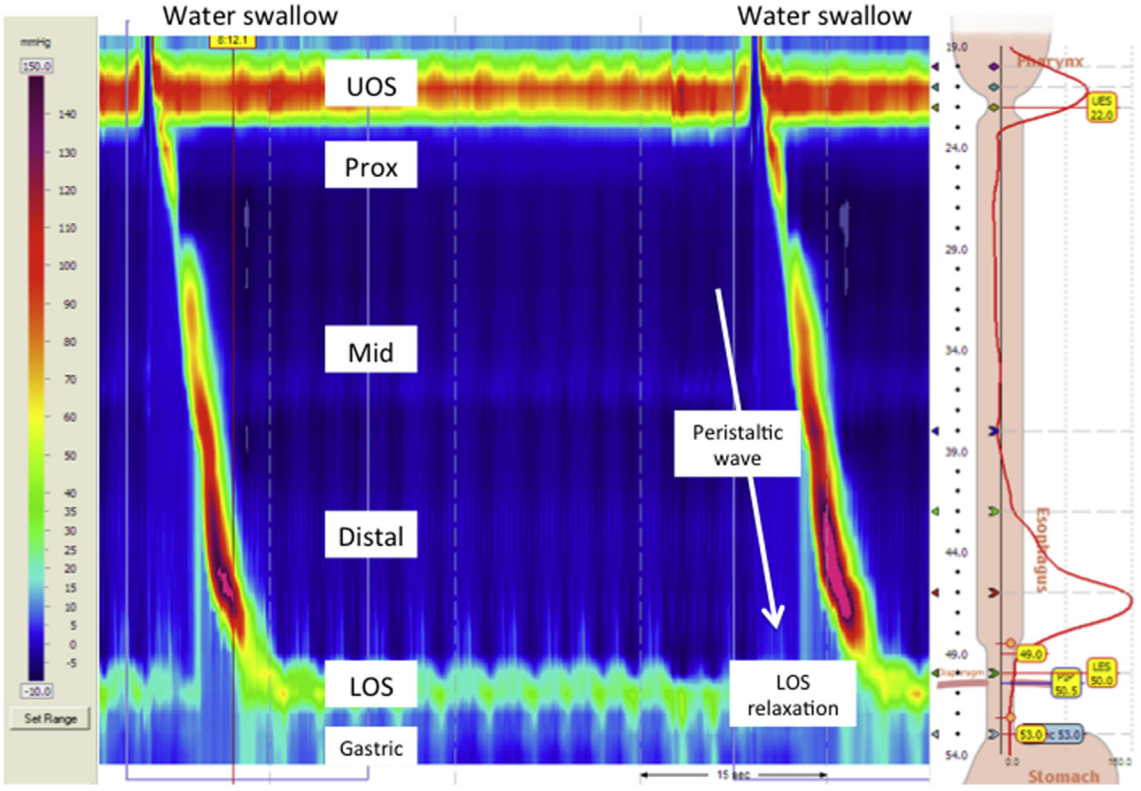

Figure 1 High-resolution oesophageal manometry with spatiotemporal plot. The spatiotemporal plot is a combination of time on the x axis, length along the oesophagus on the y axis overlaid with pressure contours on the z axis, with reds indicating high pressure and blues representing low pressures. LOS, lower oesophageal sphincter; UOS, upper oesophageal sphincter.