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The Physiology of Swallowing

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Introduction

Swallowing occurs as an orderly physiological process that transports saliva or ingested material from the mouth to the stomach. This process normally occurs so smoothly and effortlessly that it belies the complexity of the neuromuscular apparatus that executes the swallowing sequence. Generally, swallowing is considered to be "voluntary" because deglutition can be elicited by cerebral input when one thinks "swallow." Most swallows, however, particularly those between meals, occur without conscious input. Swallowing occurs surprisingly frequently, about once a minute in awake subjects, irrespective of eating [1, 2]. This high swallowing rate is elicited by saliva that is produced at a rate of about 0.5 ml/min and must be either swallowed or expectorated [3, 4]. The high natural rate of swallowing leads to about 1000 swallows daily or 3–4 million swallows per decade. During sleep, salivation and swallows virtually cease [5]. Clusters of swallows occur during arousals from sleep, but swallows seldom occur during physiological sleep [2].

Although the majority of swallows occur subconsciously in response to salivation, close clustering of voluntary swallows is evoked consciously during the processes known as "feeding" or "eating." These processes are an essential part of nutrition by which nutrients are gathered and ingested. During eating, swallowing is associated with increased salivation, which acts as a lubricant.

Phases of Swallowing

For descriptive purposes, physiology texts commonly divide swallowing into four phases called the preparatory phase, oral phase, pharyngeal phase, and esophageal phase. The preparatory phase and part or all of the oral phase are considered to be voluntary. In contrast, the pharyngeal and esophageal phases of swallowing are involuntary.

Preparatory and Oral Phases

The preparatory phase consists of taking material into the mouth and preparing it for a swallow. In the case of solid or semisolid food, the food is chewed, mixed with saliva, and usually positioned on top of the anterior tongue in anticipation of a swallow. At the onset of most normal swallows, the tongue tip is poised against the superior incisors or maxillary alveolar ridge. A semisolid or liquid bolus is cupped within a depression of the anterior one- to two-thirds of the tongue [6]. In the case of a large bolus, the posterior oral cavity is sealed off due to elevation of the tongue base against the soft palate. During the oral phase of swallowing, the tongue elevates and rolls posteriorly in a peristaltic motion, making sequential contact with the hard and soft palate, and thereby propelling the bolus into the pharynx [7]. Entry of the bolus into the pharynx occurs virtually coincident with elevation of the soft palate against the posterior pharyngeal wall, a motion that concurrently allows the bolus to leave the mouth and seals off the nasopharynx from regurgitation.

Pharyngeal Phase

The pharyngeal phase of swallowing begins as the moving wave of glossopalatal opposition traverses the fauces. The point of glossopalatal contact imparts an inverted "V" shape to the tail of a liquid or semisolid bolus [8]. Pharyngeal peristalsis continues as the posterior aspect of the tongue makes

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sequential descending contact with the posterior wall of the pharynx. Concurrently, the pharyngeal constrictors contract sequentially in a descending sequence [9]. In the hypopharynx, peristaltic obliteration of the pharyngeal lumen is achieved by opposition of the closed larynx and the inferior pharyngeal constrictor. Pharyngeal peristalsis propagates at 9–25 cm/s.

During the swallow sequence, the upper esophageal sphincter (UES), also termed the pharyngoesophageal sphincter, relaxes for about 0.5 s during which transsphincteric flow of a swallowed bolus occurs [10]. The transiently relaxed UES, formed mainly by the cricopharyngeus, is opened by anterior traction exerted by the superior–anterior excursion of the hyoid and also by pulsion forces imparted by a swallowed bolus [11, 12]. Normally, the oral phase of swallowing occurs in 0.7–1.2 s, while the pharyngeal phase lasts about 0.6 s.

Laryngeal Elevation

During the oral and pharyngeal phases of swallowing, the larynx is lifted substantially upward and forward by the combined contraction of the suprahyoid muscles, thyrohyoids, and pharyngeal elevators. This superior–anterior excursion of the larynx not only serves to open the UES by traction but also enlarges the pharynx to receive the bolus, engulfs the bolus, and acts as an ancillary mechanism to protect the larynx against aspiration. The major mechanism preventing aspiration of swallowed material into the larynx is contraction of the intrinsic laryngeal muscles that approximate the arytenoids and epiglottis, close the false cords, and adduct the vocal cords [13].

Esophageal Phase

Passage of the pharyngeal peristaltic contraction wave through the cricopharyngeus terminates UES relaxation and marks the transition between the pharyngeal and esophageal phases of swallowing. Normally, swallow-induced peristalsis propagates at about 2–4 cm/s and traverses the entire body of the esophagus, about 20 cm in length, in 6–10 s [3, 14]. Peristalsis in the striated musculature of the proximal esophagus appears phenotypically similar to that in the smooth muscle of the distal two-thirds of the esophagus. The pharynx and proximal esophagus are the only areas in the human body where striated muscle is not under voluntary neural control. Relaxation of the UES generally occurs as the transient cessation of background excitatory neural input.

Anatomy

The anatomical components of the swallowing apparatus include the bony and cartilaginous support structures, striated and smooth muscle, and neural elements.

Solid Support Structures

The alveolar arch and hard palatal plate of the maxilla form the rigid roof of the oral cavity that opposes the tongue during the oral phase of swallowing. The hinged mandible allows chewing during the preparatory phase of swallowing, but generally becomes fixed in a closed position during swallowing to provide a solid support for the tongue. The cervical spine provides a firm but flexible posterior wall for the pharynx, while the skull forms a rigid roof for the superior nasopharynx and supports the pharynx as well as the esophagus. The pharyngobasilar fascia originates from the basiocciput of the skull and interdigitates with the pharyngeal musculature, thereby anchoring the pharynx firmly to the skull base. Thus, the pharynx and connected esophagus hang from the skull base like a curtain. A tough phrenoesophageal membrane connects the distal esophagus to the diaphragm at the diaphragmatic hiatus. Membranous planes that ensheathe the pharynx and esophagus allow free axial movement of these structures during swallowing. Suspended by the suprahyoid musculature, the hyoid bone serves as an intermediate supporting structure for the larynx. The thyrohyoid membrane and paired thyrohyoid muscles connect the larynx to the hyoid. The larynx itself, comprised of cartilage and ligamentous connections, forms the anterior wall of the hypopharynx, whereas the tongue base forms the anterior wall of the oropharynx.

Muscular Components and Their Innervation

The muscles of the mandible, supplied by the mandibular division of the trigeminal nerve (V_3) and facial musculature (orbicularis and buccinators), supplied by the facial nerve (VII), play an important role during chewing and the preparatory phase of swallowing. During the oral phase of swallowing, however, these muscle groups show only a minimal to moderate contraction that serves to stabilize the jaw and, in some instances, stiffen the lips and cheeks. Tongue movement – the major action in the oral phase of swallowing and also a major component of the pharyngeal phase of swallowing – is determined by the four intrinsic and four extrinsic muscles of the tongue. The in-

Table 1. Motor nerves for swallowing

Muscles of the face	VII
Muscles of mastication	V_3
Intrinsic muscles of the tongue	XII
Palate,[a] pharynx,[b] Larynx, and esophagus	X
Intrinsic muscles tongue	XII
Extrinsic muscles tongue[c]	C_1-C_2
Muscles of the hyoid	V_3, VII, C_1-C_2

[a] Exception: Tensor velum (V_3)
[b] Exception: Stylopharyngeus (IX)
[c] Exception: Glossopalatinus (X)

Table 2. Sensory nerves of the mouth and pharynx

Nerve	Area of Innervation	Sense
V	Soft palate, entire mouth Anterior 2/3 tongue, nasopharynx	Touch
VII	Anterior 2/3 tongue	Taste
IX	Posterior 1/3 tongue Oro/hypopharynx	Touch/taste Touch
X	Root tongue, Interior larynx	Touch

trinsic musculature of the tongue is innervated by the hypoglossal nerve (XII), while the four extrinsic muscles (with the exception of the glossopalatinus [X]) are innervated from the ansa cervicalis (C_1–C_2) that run within the hypoglossal nerve. The musculature of the soft palate is under vagal nerve (X) control, except for the tensor velum, which is innervated by V_3. With the exception of the stylopharyngeus, which is innervated by the glossopharyngeal nerve (IX), all of the pharyngeal musculature (elevators and constrictors), including the cricopharyngeus, are innervated by the vagus. The vagus also innervates all eight intrinsic muscles of the larynx. Seven paired laryngeal muscles are innervated by the recurrent laryngeal nerves (RLN) while one pair (the cricothyroids) is innervated by the superior laryngeal nerves (SLN). Hyoid and laryngeal movement is produced by five muscle groups innervated by V_3, VII, and C_1–C_2.

To summarize (Table 1), the muscles of mastication are innervated by the mandibular nerve (V_3) while cranial nerve XII (hypoglossal and ansa cervicalis) controls tongue movement. With few exceptions, the vagal nerve (X) controls the muscles of the palate and pharynx as well as the intrinsic muscles of the larynx. The only exclusions are the tensor velum palatini (V_3) and stylopharyngeus (IX). Multiple cranial nerves (V_3, VII, and C_1–C_2) regulate deglutitive movement of the hyoid and larynx.

Neural Control Mechanism

The neural control of swallowing involves four major components: efferent motor fibers contained in cranial nerves and the ansa cervicalis, afferent sensory fibers contained in cranial nerves, cerebral and midbrain fibers that synapse with the brain stem swallowing centers, and the paired swallowing centers in the brainstem. Fibers from the cranial nerves and higher cerebral centers send input signals to the brainstem swallowing centers, which process this information. An appropriate code of afferent neural input activates a swallow response from the swallowing centers. Output signals pass via the cranial nerves (Table 1) that operate the muscle machinery of swallowing.

Input Signals

Sensory cranial nerve input to the brainstem swallowing centers (Table 2) is provided mainly by the glossopharyngeal (IX) and vagal (X) nerves, with some participation by the maxillary branch of the trigeminal (V_2) and facial (VII) nerves [15, 16]. The facial nerve provides taste fibers over much of the tongue, and, of course, touch sensation for the lips and face. In the mouth, sensory fibers are trigeminal (V_2) whereas at the fauces and in most of the pharynx, sensation is via the glossopharyngeal nerve. The superior laryngeal nerve (SLN) of the vagus contains sensory fibers for the posterior aspect of the larynx and root of the tongue.

The optimal stimuli that induce swallowing show regional variation. For example, the most effective stimulus is light touch at the fauces, heavy touch in the posterior pharynx, and water at the posterior larynx. Taste stimulation alone is a weak stimulus for swallowing. The major sensory stimuli needed to elicit swallowing are thought to originate from the fauces, pharynx, and posterior larynx. In experimental animals, electrical SLN stimulation at specific frequencies elicits swallowing. Authorities believe that coded sensory information, of a given pattern and intensity, from the receptive field of the pharynx serves as the major trigger for swallowing [17, 18]. Cerebral input, such as the intent to swallow, or wetting of the lips and face are thought to facilitate swallowing by augmenting the input from existing background pharyngeal stimuli. Most swallows occur subconsciously. Swallowing remains intact in decerebrate animal preparations and in some humans decorticated by disease. Sensory signals from the pharynx

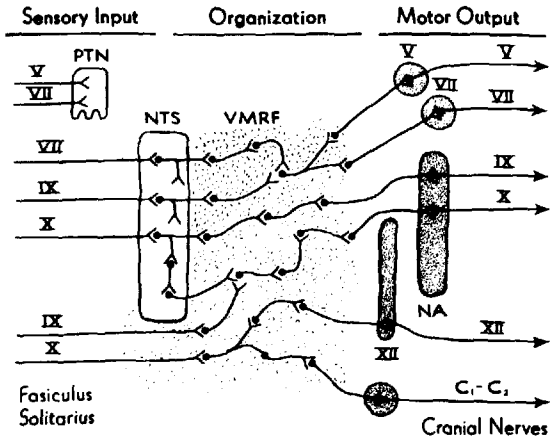

Fig. 1. Schematic representation of a brainstem swallow center. The swallow centers are paired, but only one is shown. Sensory input from the V, VII, IX, and X nerves that triggers swallowing is carried mainly to the nucleus tractus solitarius (NTS). Afferent input from V and VII also synapse in the primary trigeminal nucleus (PTN) and some fibers reach the NTS. An area comprising the NTS and ventral medial reticular formation (MRF), a neuropile with multiple interneurons and synaptic connections, constitutes the bilateral paired swallowing centers. When the swallowing center receives appropriate afferent information from the periphery or from higher cranial centers (not shown), a preprogrammed swallow response is triggered. The muscle machinery of the swallow response is driven by motor neurons that leave the swallow center and synapse in cranial nerve nuclei on the ipsilateral side. The lower motor neurons to the muscles that execute swallowing run in cranial nerves V, VII, IX, X, and XII.

that differ from the code needed to evolve swallowing may elicit a gag, cough, or other patterns of oral–pharyngeal motor responses that use the same muscle groups as swallowing.

Brainstem Swallow Centers

Paired swallowing centers reside in the hind brain. Transection studies in the dog suggest that only one intact center is needed for normal swallowing [18]. The swallow centers are not discrete focal areas, but consist of ill-defined broad zones (Fig. 1) located lateral to the midline and ventral to the caudal portion of the fourth ventricle, which incorporate the nucleus tractus solitarius (NTS) and the ventromedial reticular formation (VMRF). Input fibers from the cranial nerves and higher cerebral centers synapse within the NTS or reticular formation. Thus, each swallow center consists of an elaborate array of interneurons (the details of which are poorly understood) that process incoming information, generate a preprogrammed swallow response, and distribute signals to the appropriate cranial nerve motor nuclei and their axons, which then deliver neural signals to the many muscles involved in swallowing.

For purposes of simplification, each swallow center may be thought of as a computer. A central programming unit (CPU), comprised of an intricate, hardwired neuronal network, is capable of receiving incoming neural signals (input), processing the information, activating a menu of stock programs, and generating appropriate outgoing signals (output). Built, or programmed, into the computer is a stereotyped program for swallowing as well as other programs for gagging, coughing, and other actions. Access to and activation of the swallow program depend on a specific pattern of input. The computer output is an orchestrated sequence of signals to the cell bodies of motor neurons that operate the mechanical contractile muscular elements that allow for swallowing or other patterned physiological responses.

As an analogy, the variety of related programmed events, such as swallowing, coughing, and gagging, which use many of the same cranial motor fibers and cervical muscles, may be likened to a piano. Each key on the piano produces only a single note, similar to each cranial motor neuron, which controls only a single motor unit. The keys on the piano may be struck at different frequencies, intensities, and sequences, similar to the interplay of efferent output via cranial nerve fibers. Whereas a specific musical score determines the melody, or program, generated by the piano, specific programs determine the pattern responses, such as swallowing or coughing, which originate from the brainstem centers.

Motor Output

Swallow-related motor output to the musculature of the neck and esophagus is transmitted via axons whose cell bodies reside in the central nuclei of cranial nerves. These nuclei consist of well-defined aggregates of motor cell bodies that are located somewhat laterally or dorsally within the brainstem, like a chain of islands. The trigeminal (V) nucleus is located toward the cranial part of the brainstem, near the level of the mid pons, while the facial nucleus (VII) is at the level of the posterior margin of the pons. Motor fibers in the vagus (X) arise from two distinct sausage-shaped nuclei that extend caudal to the caudal margin of the fourth ventricle on either side of the midline. The nucleus ambiguus lies somewhat laterally and supplies vagal motor fibers that innervate the striated musculature of the palate, pharynx, larynx, and proximal esophagus. The nucleus ambiguus also

sends motor fibers to the glossopharyngeal nerve (IX) as well as to the vagus (X). The dorsal motor nucleus lies dorsomedially and supplies vagal motor fibers that innervate the smooth muscle of the distal two-thirds of the esophagus as well as other structures of the upper gastrointestinal tract. The elongate hypoglossal nucleus (XII) lies dorsally at the same level of the dorsal motor nucleus and supplies the intrinsic muscles of the tongue. Ventral horn fibers from the C_1–C_2 level of the cord form a bucket-handle loop (ansa cervicalis) and then join the hypoglossal nerve to innervate the genioglossus, anterior digastric, and infrahyoid muscles.

All the cranial nerve motor fibers to striated muscle structures pass directly to the muscle cells to form a discrete motor end plate on individual muscle cells. These pathways do not have any intervening ganglionic synapses [19]. Nerve stimulation releases acetylcholine (ACh) from the nerve terminal at the motor end plate. The ACh acts on myogenic nicotinic receptors to contract the muscle cell. Each axon supplies a discrete population of smooth muscle cells (e.g., 5–50), which form a motor unit. Graded increases in the force of muscle contraction are elicited by an increased firing rate of the nerve (temporal summation) or the recruitment of additional motor units (spatial summation).

In contrast to the nerves that innervate striated muscle cells, the nerve fibers to esophageal smooth muscle originate in the dorsal motor nucleus rather than in the nucleus ambiguus and synapse in the esophageal intramural neural plexus, known as Auerbach's plexus [14]. This neural plexus is located between the longitudinal and circular smooth muscle layers of the muscularis externa. Short postganglionic nerves, whose cell bodies lie within the ganglia of Auerbach's plexus, pass to both the longitudinal and circular esophageal smooth muscle. These nerves form neural membrane clefts with the smooth muscle cells, but are not nearly as closely applied to the muscle as those of a motor end plate on striated muscle. Therefore, motor function of smooth muscle can never be as finely tuned and discriminatory as that of striated muscle. The preganglionic fibers, enteric plexus, and postganglionic fibers of the esophagus make up part of the parasympathetic nervous system.

Unlike esophageal striated muscle, which is innervated by only one type of nerve, esophageal smooth muscle seems to be innervated by at least two different types of nerves [19, 20]. Cholinergic postganglionic fibers excite esophageal smooth muscle by causing membrane depolarization. Cryptic inhibitory nerves are noncholinergic, nonadrenergic, and release an inhibitory transmitter that is thought to hyperpolarize smooth muscle membranes. Some workers claim that this neurotransmitter is vasoactive intestinal peptide (VIP). Whatever its identity, the released transmitter causes receptive relaxation of the esophageal body and lower esophageal sphincter ahead of a circular peristaltic contraction wave, a process known as descending inhibition: and it may also result in a rebound contraction, or "off response," immediately following the cessation of cell hyperpolarization. The relative contribution of cholinergic and noncholinergic nerves to the genesis of peristalsis in esophageal smooth muscle [20] is the subject of controversy.

Theories

The precise details of the control systems that execute the oral – pharyngeal and esophageal phases of swallowing have not been determined.

Oral and Pharyngeal Phases

Two major theories have been proposed to describe the neural control mechanism that executes the oral and pharyngeal phases of swallowing: the reflex chain hypothesis and the central pattern generator hypothesis [17]. According to the reflex chain hypothesis, a bolus moving through the mouth and pharynx stimulates sensory receptors that sequentially trigger the next step in the swallow sequence. Even in the absence of a bolus, the posterior excursion of the tongue is believed to stimulate faucial or oropharyngeal receptors that trigger or augment the next increment of the swallow sequence. Thus, the theory proposes that swallowing proceeds as a chain of linked reflexes, each step stimulating the next step. The reflex chain hypothesis leads to the notion that the late oral phase of swallowing "triggers" the pharyngeal phase of swallowing. A delay between these phases is explained by a delay in "pharyngeal triggering" or the "pharyngeal response." In practical terms, the concept of pharyngeal triggering is widely used as an operational definition by health professionals who treat patients with abnormal swallowing.

The second hypothesis suggests that once swallowing is initiated, the ensuing swallowing sequence is programmed solely by a network of neurons in the medullary swallowing centers that function independently of sensory feedback. Thus, this hypothesis proposes that a central pattern genera-

tor for swallowing is not influenced by or dependent on peripheral sensation.

Some evidence exists to support both the reflex chain and central pattern generator hypotheses. Data from the dog indicate that the pharyngeal phase of peristalsis remains fixed and stereotyped, irrespective of the method used to initiate swallowing or the volume of a swallowed bolus [18]. In contrast, studies supporting the reflex chain hypothesis have shown that anesthetizing the oral and pharyngeal mucosa reduces the number of dry swallows per unit of time and alters the contractility of individual muscles involved in swallowing. Recent data from our laboratory show that variations in the volume of swallowed bolus cause quantitative alteration in many variables of swallowing. For example, graded increases in the volume of a swallowed liquid bolus speed up the velocity of oral peristalsis, increase the magnitude of hyoid and laryngeal movement, lengthen the duration of upper esophageal sphincter relaxation and opening, elicit earlier movement of the larynx in the swallow sequence, and cause earlier opening of the upper esophageal sphincter [10, 21]. Not all the variables of the oral and pharyngeal phases of swallowing, however, are altered by bolus volume. Variables independent of bolus volume include the force of oral as well as pharyngeal peristaltic contractions and peristaltic velocity in the pharynx.

Although some evidence exists to support both the reflex chain and pattern generator hypotheses of swallowing, these two are not necessarily mutually exclusive. Swallowing probably depends on a central patterned program that is modulated or reinforced by, but not dependent on, feedback sensory input. Feedback sensory modification of the oral–pharyngeal phases of swallowing might occur as a preprogram modification governed by lingual proprioreceptors that sense the bolus size before the swallow. Alternatively, sensory feedback modification might occur on-line during the swallowing sequence. Both types of mechanism may be operative.

Esophageal Phase

Esophageal peristalsis occurs normally as a smooth, uninterrupted contraction wave that traverses the entire esophagus. The control mechanism that generates peristalsis in esophageal smooth muscle differs substantially from that for esophageal striated muscle. All workers agree that peristalsis in esophageal striated muscle is determined by a descending sequence of efferent neural discharges, generated by the central swallowing program [22]. The sequenced motor output might be likened to the sequencing from an automobile distributor that sparks piston movements in a designated, predetermined sequence.

As peristalsis reaches the distal part of the striated muscle esophageal segment, the central swallowing program stimulates, at the appropriate instant, the onset of peristalsis in the proximal part of the smooth muscle segment. The precise control mechanism(s) that generate peristalsis in esophageal smooth muscle, however, are controversal. The classic notion, widely held until about 25 years ago, suggested that sequential central stimulation of esophageal cholinergic nerves generated peristalsis in the smooth muscle part of esophagus [14]. Subsequent evidence from experimental animal preparations, however, demonstrated that the organization for esophageal smooth muscle peristalsis persisted after vagal transection or when the esophagus was placed in an organ bath. Such evidence indicated that an intramural control mechanism exists within the esophagus itself and that this is capable of organizing persistalsis in esophageal smooth muscle independent of central input. Considerable debate exists at present as to whether the organization of esophageal smooth muscle is an inherent property of esophageal circular smooth muscle itself or of the intramural plexus of nerves. Moreover, debate exists about whether a neural control mechanism of esophageal peristalsis occurs as an "on response" elicited by cholinergic nerves or an "off response" mediated by nonadrenergic, noncholinergic (NANC) nerves.

Regardless of the identity of the specific underlying neural transmitters, general agreement exists that peristalsis in the esophagus as well as in the pharynx occurs as a rapid wave of relaxation followed by a slower wave of contraction. The rapid descending wave of inhibition relaxes the pharynx, upper esophageal sphincter, esophageal body, and lower esophageal sphincter in an aboral sequence so that these structures may accommodate an oncoming bolus advanced by the peristaltic contraction wave. Bolus transport is determined exclusively by pharyngoesophageal peristalsis in recumbent subjects, while gravity assists peristalsis in upright subjects.

As with the different proposed mechanisms for the oral and pharyngeal phases of swallowing, the different proposals for the control mechanisms of peristalsis in esophageal smooth muscle are not necessarily mutually exclusive. For example, some central output may stimulate the smooth muscle esophagus in a descending sequence that might overlie a control mechanism intrinsic to the esophagus. It is clear, however, that esophageal peristal-

sis must be initiated by neural excitation. This neural excitation originates centrally in primary peristalsis and in many species, such as the cat, is also central in secondary peristalsis. In other species, such as the opossum, secondary peristalsis may be initiated as a local stretch reflex.

Peripheral sensory feedback may modulate esophageal peristalsis in some species. For example, in the dog, primary peristalsis seldom traverses the entire esophagus unless a bolus is present [23]. In the dog, therefore, sensory feedback reinforcement by a vagovagal central feedback mechanism is essential to sustain peristalsis through the distal esophagus. The canine esophageal body, however, is comprised solely of striated muscle. In the human and opossum esophagus, primary peristalsis generally traverses the entire esophagus, even in the absence of a bolus [3, 20]. In these two species the distal two-thirds of the esophagus is formed of smooth muscle. In all species the presence or absence of a bolus, bolus consistency, and body position may affect the velocity and magnitude of esophageal peristalsis, particularly in the distal half of the esophagus. These phenomena further support the notion that esophageal peristalsis is modulated by sensory feedback mechanisms.

Resting tone, relaxation, and opening of the lower esophageal sphincter (LES) and UES occur by entirely different mechanisms. Resting UES tone is generated exclusively by background central neural input, while deglutitive UES relaxation represents a transient switching off of neural input. In contrast, resting LES tone is, at least in part, a property of sphincteric smooth muscle, augmented by input from cholinergic nerves. Transient LES relaxation is caused by active stimulation of inhibitory nerves that release a hyperpolarizing neurotransmitter. During sleep, active UES tone disappears [24] whereas LES tone either remains unchanged or increases [2]. The UES is opened by traction assisted by pulsion when a bolus is present, whereas LES opening is determined mainly by the pulsive forces transmitted via a swallowed bolus. With a dry swallow, the UES opens somewhat whereas the LES generally remains closed, even though it relaxes. The UES relaxes for about 0.5 s while LES relaxation lasts 6–10 s. Relaxation in each sphincter is terminated by the arrival of the peristaltic contraction wave that passes into the sphincter.

Diagnostic Evaluation of Swallowing

Diagnostic laboratory evaluation of swallowing includes radiography, intraluminal manometry, scintigraphy, and ultrasound. Ultrasound examination is confined to the oral phase of swallowing [25]. Scintigraphy provides good evaluation of bolus transport, but does not demonstrate small amounts of laryngeal aspiration and is not widely used. The two main methods for evaluating pharyngoesophageal function are radiography and intraluminal manometry. For the esophagus, I believe that radiography and manometry are comparable for evaluating motor function, but radiography also evaluates bolus transport and morphologic abnormalities whereas manometry does not. For the evaluation of the oral and pharyngeal phases of swallowing, radiology is substantially superior to manometry or any other method. Manometry is best suited to evaluating UES relaxation [26]. In my opinion, videoradiography is preferable to cineradiography because it requires substantially less radiation and is generally accessible at low cost. High speed filming with slow motion play back capability is essential to evaluate oral–pharyngeal deglutitive function. Optimal assessment includes evaluation of timing [27] as well as magnitude of structural movement [10, 21]. In addition to diagnostic evaluation, the radiographic method also allows assessment of therapeutic manipulations of bolus characteristics and head position in the search for a "safe swallow" that avoids or minimizes aspiration [13]. In studies that focus on assessment of oral–pharyngeal function, it is important to examine the esophagus as well.

Summary

Swallowing is a physiological phenomenon that occurs up to 100 times daily. Although swallowing may be initiated as a conscious volitional act during eating, the substantial majority of swallows occur subconsciously between meals, without apparent cerebral participation. Such nonprandial swallowing occurs about once a minute in awake individuals and is driven by salivation, which elicits and stimulates sensory receptors in the mouth and pharynx. Salivation and swallowing stop during sleep.

The primary stimulus for swallowing is provided by sensory stimuli originating from receptors located within the sensory fields of the mouth and pharynx. Input from these sources activates the central swallowing centers in a coded manner. Such stimuli may exceed a threshold to activate spontaneous swallowing and provide the necessary background excitation to allow voluntary cerebral input to elicit swallowing by facilitation.

After deglutition is initiated, the oral and pharyngeal phases of swallowing appear to be controlled mainly by programmed pattern generators located within the paired brainstem swallowing centers. Nevertheless, feedback sensory input, determined by bolus volume and consistency, may alter some, but not all, of the variables associated with the oral and pharyngeal phases of swallowing.

The pharyngeal and esophageal phases of swallowing are entirely involuntary. Peristaltic contractions in the striated muscle of the pharynx and esophagus are orchestrated exclusively by the swallow center program, which sends out vagal efferent signals in a caudally oriented sequence. Minimal feedback modulation of peristalsis occurs in the pharynx and proximal esophagus. Primary peristalsis in esophageal smooth muscle is initiated at the appropriate instant by the central swallowing program but is generated, at least in part, by mechanisms intrinsic to the esophagus itself. Peristalsis through both the pharynx and esophagus occurs as a rapid wave of inhibition that causes an accommodative, receptive relaxation of pharyngoesophageal tube and its sphincters, followed by a slower peristaltic contraction wave responsible for bolus transport. In the distal two-thirds of the esophagus, bolus volume and consistency modulate peristaltic variables, such as the occurrence, velocity, and force of contraction.

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