Chapter 11: Deglutition

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Deglutition is the process whereby a bolus, liquid or solid, is transferred from the buccal cavity to the stomach. Swallowing is a complex, integrated, continuous act, involving somatic and visceral, afferent and efferent nerves together with their associated striated and smooth muscles. For simplicity of description, it is useful to divide deglutition into three phases which correspond to the three anatomical regions through which the bolus passes:

(1) oral
(2) pharyngeal
(3) oesophageal.

The initiation of the first two phases is subject to conscious control and involves striated muscles controlled by a complex of stimulatory and inhibitory signals from the brainstem. The third phase involves the smooth muscle of the oesophageal wall and depends on both central coordination and local, intramural neural arcs.

The act of swallowing has been studied from two points of view:

(1) mechanical (cineradiography and intraluminal manometry)

(2) neurophysiological, in which neural impulses are traced from afferent signals through the swallowing centre to the motor nerves in an attempt to understand the 'wiring diagram' that programmes this complex muscular response.

Oral stage

In their extensive studies of deglutition, Ardran and Kemp (1955) considered that in the first or oral stage the tongue is involved in squeezing food out of the mouth and oropharynx. When swallowing begins, the tip of the tongue is raised to the back of the incisor teeth and is then applied to the palate from before backwards, so squeezing the contents of the mouth into the pharynx. When the tail of the bolus has been expelled from the mouth, the back of the tongue arches posteriorly to meet the soft palate and pharyngeal wall. The nasopharynx is closed by the elevation of the soft palate in combination with the contraction of the superior pharyngeal constrictors.

No account of the variations in the behaviour of the tongue in swallowing can be understood without first following the movements of the hyoid bone, to which most of the extrinsic muscles of the tongue and the floor of the mouth are attached. The contraction of these muscles influences the volume and disposition of the tongue's substance relative to the mouth cavity.
Normal individuals swallow the contents of the mouth in the midline furrow in the tongue. When chewing, food may pass down the upper lateral food channels at the sides of the tongue to the valleculae, as in the rabbit.

Patients with a gap in the palate (congenital or acquired) may also divert food to the sides of the tongue and may plug the gap by raising the tongue in the midline. Patients with holes in the cheek, for example from a gunshot injury, learn to swallow on the opposite side, as do patients with unilateral weakness of the tongue. After food has been introduced into the mouth, space for it is normally made by lowering the tongue, the floor of the mouth and the hyoid bone.

During the act of swallowing, the jaws are brought together as the tongue fills the oral cavity to displace its contents and the hyoid bone raised towards the lower border of the mandible. Maximum elevation of the hyoid bone is reached as the tail of the bolus is being expelled from the mouth, and is maintained as the tongue arches backwards to displace the bolus downwards. These movements of the hyoid bone relate to the need to adjust the volume of the tongue. As the bolus is displaced from the mouth, the tongue reoccupies its position of rest in the mouth cavity. However, as the tongue must also arch backwards to displace the bolus downwards, the volume of the tongue necessary to occlude the mouth must be further reduced by raising of the floor of the mouth. In other words, maximum elevation of the hyoid bone is indicative of maximum contraction of the mylohyoid muscle, thus reducing the mouth proper to the smallest possible size. If a large bolus is swallowed, the tongue (and hyoid bone) must be pulled well forward to provide adequate accommodation for the bolus to be taken into and through the pharynx; thus maximum displacement of the hyoid forwards relates to the degree of distension of the oropharynx.

The whole of a mouthful may not always be swallowed as a single bolus; for example, when a large mouthful is taken, the subject may elect to swallow it by means of a series of boluses. To do this, the subject releases a quantity of the content into the pharynx by parting the tongue and soft palate and then raising the tongue in the back of the mouth behind the junction of the hard and soft palates, thus cutting off the bolus in the pharynx from the rest of the contents of the mouth proper. The contents of the pharynx are then cleared by apposition of the constrictors to the back of the tongue and soft palate in the usual manner. In these circumstances, there is no oral phase of swallowing until the last mouthful is to be expelled.

Accordingly, it is possible to dissociate the forepart of the tongue from swallowing and to use it for some other purpose while swallowing is continuing. The forepart of the tongue may be lowered in the front of the mouth to create suction as occurs when drinking, or as part of the mechanism of sucking, or when the mouth is opened, as in pint-swallowing, to create a large cavity into which food can be taken or poured. With the conclusion of all these acts, it is necessary to clear the mouth either by dribbling with the head inclined forwards, or by using the whole of the tongue to swallow the contents of the mouth.

The forepart of the tongue is also used for manipulation of food in preparation for mastication and in the formation of consonants in speech; manipulation may continue during pharyngeal swallowing. The anterior third of the tongue, which corresponds with the free
portion in conjunction with the lips and jaws, is mainly responsible for procedures requiring manipulation in feeding and speech. These include:

(1) taking food from a spoon or fork, for example lapping, licking etc
(2) manipulation of food in the forepart of the mouth, including cleaning the teeth and palate
(3) expressing the contents of teat or nipple, and the initial stage of swallowing
(4) forming consonants in speech, and whistling
(5) expression of emotion.

The middle third of the tongue, which corresponds to the region opposite the molar teeth, is responsible for:

(1) pressing of food on to the molars for chewing
(2) the passage of food into the upper lateral food channels at the side of the tongue
(3) the following functions when it is elevated:
   (a) cutting off the mouth cavity from the pharynx, for example, when regulating the swallowing of a large mouthful by multiple boluses
   (b) altering the shape of the oropharyngeal cavity to form vowel sounds
   (c) plugging a hole in the hard or soft palate, or cheek
   (d) compensating for a paralysed soft palate by preventing or reducing nasopharyngeal reflux in the pharyngeal phase of swallowing.

The posterior third of the tongue:

(1) acts mainly as a wedge of tissue in the pharyngeal phase of swallowing which, when opposed to the soft palate and pharyngeal constrictors, displaces the bolus downwards and helps to turn down the epiglottis
(2) in its apposition to the soft palate when the mouth is closed, normally closes off the mouth cavity at rest
(3) can be apposed to the pharyngeal wall to close off the airway to reinforce or substitute for laryngeal closure in certain circumstances; for example some normal subjects when bearing down, or in patients who can use glossopharyngeal breathing, but who cannot close the larynx.
The whole of the tongue, from before backwards, is normally involved in the coordinated peristaltic wave which squeezes food from the mouth into the lower pharynx. By a reverse response, the tongue, as it is lowered from before backwards, creates a suction mechanism, as is seen in the continuous swallowing associated with suckling or drinking.

**Pharyngeal stage**

When swallowing takes place in the erect position, the first half of the bolus is passed through the pharynx into the oesophagus by means of thrust from the tongue, assisted by gravity. The peristaltic wave usually commences when the tongue expressor wave reaches the soft palate. With a moderate-sized fluid bolus, the head of the bolus has often entered the oesophagus before the pharyngeal peristaltic wave has commenced.

This wave moves rapidly and smoothly downwards, clearing the pharynx of the hindpart of the bolus and pushing it through into the oesophagus. Normally, no hold-up occurs at the cricopharyngeal sphincter as the wave of relaxation, preceding the contraction of the pharynx, reduces the resting tone of the cricopharyngeal muscles. The peristaltic wave involves the sphincter as it descends, inducing tight closure of it. The sphincter subsequently relaxes and returns to its resting state after the peristaltic wave has passed.

Studies conducted by Ardran and Kemp (1961) showed that swallowing in the lower half of the pharynx occurs down the lateral lower food channels. The latter extend from the edge of the lateral pharyngoepiglottic folds downwards and backwards around the laryngeal air passage to the level of the cricopharyngeal sphincter where they fuse and become one channel continuous with the lumen of the oesophagus. These channels are functional entities and serve to transmit fluid or semifluid substances around the mouth of the larynx and comparatively little passes directly over the edge of the tongue or the epiglottis down the midline. If the bolus is large, the larynx is pulled further forward to accommodate it, and the two channels become one posteriorly.

When swallowing in the erect position, a bolus is usually directed down the midline of the tongue to the level of the median glossoepiglottic fold, whereupon it is deflected into the vallecula on either side. The epiglottis is tilted backwards towards the posterior pharyngeal wall and serves to arrest the head of the bolus as it descends on to the back of the tongue. As the bolus accumulates on the epiglottis, it spills over the lateral pharyngoepiglottic folds into the food channels on either side of the mouth of the larynx and passes forwards into the pyriform fossae; at this stage, a small quantity of fluid may spill directly over the edge of the epiglottis over the open entrance to the larynx. As the bulk of the bolus enters the upper pharynx, the larynx is raised towards the hyoid bone, the contents of the pyriform fossae are expressed upwards and backwards, and the lower part of the lateral channels is opened to allow the bolus to spill downwards and backwards towards the midline. The columns meet over the back of the cricoid cartilage at the level of the upper border of the cricopharyngeus muscles, which then open to allow the bolus to enter the oesophagus. During the descent of the two streams below the epiglottis, some fluid frequently passes behind the arytenoids joining the two columns: this part is used whenever one side is partly or completely obstructed below this level.
When the bulk of the bolus has entered the upper oropharynx, the tongue moves backwards towards the posterior pharyngeal wall and meets the contraction resulting from the pharyngeal constrictors. The tongue of the epiglottis is gradually displaced with the bolus and is bent downwards at the side so that it is arched like a monk’s hood over the larynx. Each half of the epiglottis serves as a chute to deflect the bulk of the bolus to either side of the midline. A certain amount of the bolus is always displaced down the midline, but this proportion of the total bolus is small unless the amount swallowed is comparatively large, when space is made to accommodate it by taking the larynx further forwards. As the main mass of the bolus is expressed from the oropharynx, the soft palate is lowered and applied to the tongue, and the posterior pillars of the fauces are also brought forwards. The tongue of the epiglottis is pressed forward over the mouth of the larynx until its tip is over the back of the cricoid cartilage. As the tail of the bolus is expressed from the pharynx, the larynx is lowered and the lateral food channels are cleared from above downwards. Finally, the cricopharyngeus muscles contract and express the last of the bolus into the oesophagus. Only a small residue normally remains in the pharynx: this is situated over the base of the tongue on the down-turned epiglottis in what are now backward-turned valleculae. When the airway is re-established, as the larynx returns to the position of rest, the tongue of the epiglottis springs upwards; the small residue which remains on the epiglottis is either retained in the valleculae or split into the lower lateral food channels and held in the pyriform fossae.

If a person swallows with the head turned fully to one side, the bolus is deflected down the lower lateral food channel on the side opposite to which the head is turned: the whole of an average-size bolus may be swallowed down the one lateral channel. Old people with rigidity of the spine or other lesions may not be able to turn the head enough to obstruct completely the side to which the head is turned. Many instruments, such as a test meal tube, the gastroscope and oesophagoscope, may be passed entirely down one side.

When a normal person swallows in a supine position, the bulk of the bolus is still directed to the side of the larynx, but it takes a relatively posterolateral course; the laryngeal opening, which is situated anteriorly, is not necessarily surrounded by the bolus unless the amount swallowed is large. The course of the bolus is likewise influenced by gravity when the patient swallows lying prone, or on one side. The effect of gravity is important in consideration of the problems of management of patients with pharyngeal palsy.

**The epiglottis and closure of the larynx during swallowing**

Ardran and Kemp (1967) have stated that there are several components associated with the above action. In the first component, when the bolus of food begins to spill down the back of the tongue, the lumen of the laryngeal vestibule is invariably narrowed. This is in consequence of the rocking movement of the arytenoids, downwards, forwards and forwards, with resulting narrowing of the glottis, bulging inward of the vestibular folds, forward movement of the cartilages of Wrisberg and obliteration of the interarytenoid space.

The second component produces backward tilting of the leaf of the epiglottis towards the posterior pharyngeal wall. The manner in which this is brought about depends on the position of the larynx and hyoid relative to the cervical spine and lower jaw at the moment swallowing is initiated.
In normal individuals at rest in the erect position, the leaf of the epiglottis is closely approximated to the tongue. With the passage of the bolus from the mouth into the pharynx, its descent is checked by a ledge constituted by the backward-tilted epiglottis: this is the phase of vallecular arrest. At this stage, the lumen of the laryngeal vestibule is usually reduced in size, but not closed.

As the mass of the bolus accumulates on the epiglottis, the larynx and hyoid are usually elevated towards the lower jaw; the hyoid rotates so that its greater cornua become horizontal (rather than their usual oblique position), thereby producing further backward tilting of the epiglottis towards the pharyngeal wall. This may result in complete closure of any remaining gap between the epiglottis and the pharyngeal wall, thus completely checking any spill of the bolus.

With further displacement of the bolus from the mouth, the larynx and hyoid are moved forwards and further upwards. Contact between the epiglottis and the posterior wall is no longer maintained and the bolus spills over the edge of the epiglottis into the lateral food channels on either side of the laryngeal entrance. The laryngeal lumen, though narrow, is usually not completely closed and a little of the bolus may enter the vestibule at this time and pass down as far as the laryngeal ventricle. During this swallowing stage, the leaf of the epiglottis projects backwards above the entrance of the larynx into the stream of the descending bolus, and the epiglottis is raised in the middle and bent down at the side, thereby serving to deflect the bulk of the bolus to one or both sides of the larynx (see above).

With further descend of the bolus, the whole of the hyoid bone is approximated more closely to the thyroid cartilage. The cricothyroid visor is opened which allows the arytenoid masses to be tilted bodily forward, with the various components reducing the lumen of the vestibule to about one-third of the anteroposterior diameter in quiet breathing.

At any stage during the descent of the bolus, but always when it has been expressed past the larynx, there is backward bulging of the lower part of the epiglottis into the vestibular lumen, resulting from the apposition of the thyroid cartilage to the hyoid. This, in turn, leads to further bulging of the vestibular folds and obliteration of the ventricles. The total effect of all these movements is to oppose the dorsal surface of the epiglottis to the cartilages of Wrisberg and obliterate the laryngeal lumen from the level of the vocal cord to the superior laryngeal aperture.

The final component of laryngeal closure is the sealing of the laryngeal aperture by the sudden downward turning of the leaf of the epiglottis within the column of the bolus. This action results from a number of factors, which are the sustained approximation of the thyroid to the hyoid - both structures being elevated to the lower jaw - force is transmitted to the bolus as the tongue moves backwards to the palate and posterior pharyngeal wall, the leaf of the epiglottis is pressed down by the descending peristaltic wave against the arytenoid masses and any residue of the bolus which lies beneath it is displaced from the entrance to the larynx.

In all normal individuals, the pharynx is not squeezed entirely clear of the bolus: there is always a small residue left on the dorsal surface of the down-turned leaf of the epiglottis, that is its ventral surface when erect. If the leaf of the epiglottis were not present, this residue
would lie over the laryngeal entrance. When the airway is re-established, the residue is swept upwards into the valleculae as the leaf of the epiglottis rises to the erect position.

There are, of course, modifications of the foregoing sequence of events, for example when an individual swallows into a large, air-filled pharyngeal cavity, when an individual is lying supine, and so on.

**Oesophageal stage**

Before discussion of the final part of deglutition, some mention should be made of the cricopharyngeal sphincter which forms the junctional zone between the pharynx above and the oesophagus below. However, functionally it is probably more accurate to use the term 'oesophageal sphincter'. This sphincter is formed by the cricopharyngeus (horizontal fibres) above which are the lower oblique fibres of the inferior constrictor, and this zone is continuous below with a variable length of the circular muscle of the oesophagus.

Lund (1965) stated that the cricopharyngeal sphincter is normally closed at rest and remains so even when subjected to raised pressure from above or below. The cricopharyngeus muscles can relax, however, to allow air etc to pass either up or down. The sphincter provides a zone of elevated pressure which is approximately 1.6 kPa (12 mmHg) above the pressure in the oesophagus.

When swallowing occurs, the sphincter initially relaxes but does not open. The bolus then passes through the relaxed sphincter by means of thrust from the tongue, assisted by gravity. The cricoid is displaced forwards by the bolus, the sphincter opening at this stage. At about the time the bolus enters the oesophagus, the peristaltic wave commences at the top of the pharynx. This moves rapidly and smoothly downwards, clearing the pharynx of the hind part of the bolus and pushing it through into the oesophagus. The peristaltic wave involves the sphincter as it descends, producing the tight closure characteristic of the second stage of the sphincter changes. After the peristaltic wave has passed, the sphincter relaxes and returns to its resting state.

However, as Goyal (1984) pointed out, part of the opening of the sphincter, with abolition of the resting pressure, is brought about by the anterior displacement of the larynx by the suprahyoid muscles. This forward and upward laryngeal movement is an early event in swallowing, with Doty, Richmond and Storey (1967) stating that mylohyoid activity is the first to appear in response to swallowing. In a sense, the suprahyoid muscle can be considered as the dilator fibres of the upper oesophageal sphincter.

**Oesophagus**

The oesophagus functions essentially as a conducting tube for conveying substances to, and occasionally from, the stomach. During discussion of deglutition, Slome (1971) wrote that in the intrathoracic oesophagus, the intraluminal pressure corresponds to the intrapleural pressure. However, the pressure varies considerably with inspiration and with expiration, particularly with the latter if the glottis is closed, as in coughing, straining etc. During swallowing, peristaltic waves pass down the oesophagus with waves of positive pressure reaching 6.67-13.3 kPa (50-100 mmHg). The form of the wave varies somewhat with the
nature of the swallowed substance, but with liquids and semisolids there is an initial negative wave resulting from the elevation of the larynx drawing on the cervical oesophagus. This is followed by an abrupt positive wave, coinciding with the entry of the bolus into the oesophagus. Next comes a slow rise of pressure succeeded by a final, large positive pressure wave which rises and falls rapidly, this being the peristaltic stripping wave.

Secondary peristaltic waves arise locally in the oesophagus in response to distension, and they complete the transportation of bolus portions which have been left after the primary peristaltic wave. Tertiary oesophageal contractions are irregular, non-propulsive contractions involving long segments of the oesophagus, which frequently occur during emotional stress. The velocity of oesophageal transport is more rapid in the upper oesophagus than in the distal half, on account of the differences in muscle type and neural mechanism of the propagation of the peristalsis.

At the lower end of the oesophagus there is a zone of raised pressure about 3 cm in length, extending above and below the diaphragm, with a mean pressure of approximately 1 kPa higher (8 mmHg) than the intragastric pressure which can be regarded as the location of the 'physiological sphincter' of the oesophagogastric region. As with the cricopharyngeal sphincter, the oesophagogastric sphincter, which is normally in tonic contraction, undergoes relaxation before the peristalsis reaches it, with a reflex preventing the occurrence of contraction immediately the bolus has passed into the stomach.

The physical consistency of the swallowed material determines to some extent the mechanism involved in its passage through the oesophagus. When fluid is swallowed, it may be projected from the pharynx to the oesophagogastric junction in about 1 s (with the subject in a standing position), and is well ahead of the peristaltic wave. In consequence of this rapid passage, the swallowing of corrosive fluids causes burns which are often localized to the distal end of the oesophagus. When the bolus is solid or semisolid, it is passed down the oesophagus by a peristaltic contraction of the oesophageal musculature, with gravity playing little part in the process.

In the upper part of the oesophagus, peristalsis progresses rapidly; in the lower one-third, the contraction wave is more sluggish. The differences in motor activity are related to the muscular coat’s being striated in the former situation and unstriated in the latter.

Oesophageal sphincter (the lower oesophageal sphincter)

Jewell and Selby (1982) stated that the lower oesophageal sphincter forms the major barrier to gastro-oesophageal reflux; when the muscle of the sphincter is destroyed by diseases, such as systemic sclerosis, or by cardiomyotomy for achalasia, reflux commonly occurs. The resting sphincter pressure in patients with gastro-oesophageal reflux is often subnormal but there is a wide overlap with the normal range. Many variables govern the actual pressure recorded. Radial asymmetry of pressure is present, with higher pressures being recorded towards the patient’s left (Luckman and Welch, 1977). Recording technique varies and, for these and other reasons, no generally accepted normal range of lower oesophageal sphincter pressure has yet been agreed. However, it seems probable that in the protection against reflux, the capacity of the sphincter to respond to stress is more important than the resting pressure.
Circular muscle fibres from the oesophago-gastric junction behave differently from those in the body of the oesophagus, in that when the former are stretched, the length-tension curve is steeper, that is they have a greater resistance to being stretched. The cause of the tone of the lower oesophageal sphincter in humans is poorly understood. It has been variously suggested either that neural influences are responsible as in the upper oesophageal sphincter, or that tone is a result of hormonal factors, or that it is an intrinsic property of the muscle fibres themselves. In the opposum, the isolated circular muscle of the sphincter retains its tone after treatment with tetrodotoxin, which blocks all nerve conduction, implying that, in this animal, tone is myogenic in origin (Goyal and Rattan, 1976). However, the tone of the lower oesophageal sphincter may certainly be influenced by neural and possibly by hormonal factors.

**Neural regulation of the lower oesophageal sphincter**

The vagus nerve is concerned with the regulation of lower oesophageal sphincter function. It is predominantly an afferent nerve which carries impulses from the whole alimentary tract, except for the distal large intestine, but efferent fibres influence motor and secretory activities. Vaso-vagal reflexes are poorly understood but they are known to exert both excitatory and inhibitory actions on gut motility. Section of the vagal nerve appears to have a variable effect upon the lower oesophageal sphincter in different species. In the dog, high bilateral vagotomy results in oesophageal dilatation and aperistalsis, as might be expected with denervation of striated muscle, and the lower oesophageal sphincter pressure also falls (Khan, 1981). The opossum has much more smooth muscle in the oesophagus and here a transient increase in sphincter pressure follows bilateral vagotomy, while stimulation of the peripheral end of the severed nerve causes the sphincter to relax. Stimulation of the central end causes sphincteric contraction, even when vagotomy is bilateral, which indicates that the efferent pathway for this centrally mediated mechanism lies outside the vagi (Rattan and Goyal, 1974).

In humans, gastro-oesophageal reflux is a common consequence of surgical truncal vagotomy. Resting pressure in the lower oesophageal sphincter falls to the low or low normal range. However, surgical truncal vagotomy does impair the sphincteric response to stress, and the increase in sphincter pressure normally seen after an increase in intra-abdominal pressure is thus inhibited (Angorn et al, 1977). As the vagal nerve supply to the oesophagus and the lower oesophageal sphincter comes off the vagi above the level of section at truncal vagotomy, it seems probable that the operation will have severed the afferent fibres of the reflex concerned in the sphincteric response to the increased abdominal pressure. Patients with gastro-oesophageal reflux who have not undergone previous surgery, show a similar lack of sphincteric contraction in response to a rise in intra-abdominal pressure, and it appears that disruption of this reflex may well be of aetiological importance in the causation of reflux.

Much less is known about the role of the sympathetic nerve supply to the oesophagus. No gross disturbance in oesophageal function followed bilateral thoracolumbar sympathectomy when this was employed in the treatment of hypertension. This suggests that the sympathetic nerve supply to the oesophagus is not of vital importance in the regulation of motor activity.
Hormones and the lower oesophageal sphincter

Gastric causes an increase in lower oesophageal sphincter pressure (Giles et al, 1969), and this action is mediated through cholinergic mechanisms which can be blocked by atropine. There can be no doubt that this and other alimentary hormones do exert a pharmacological effect upon the sphincter. However, the evidence that these hormones are of physiological importance in the regulation of sphincter tone is much less certain. Changes in serum gastrin levels in health and disease do not correlate closely with changes in sphincteric pressure. In pernicious anaemia, the lower oesophageal sphincter pressure tends to be low, yet the serum gastrin level may be high; and in patients with the Zollinger-Ellison syndrome, the lower oesophageal sphincter pressure is certainly not increased. Although meals will influence the lower oesophageal sphincter pressure, and the rise in pressure roughly coincides with increased secretion of gastrin by the antral G cells, it seems more likely that the fluctuations in sphincteric tone are mediated by way of nervous rather than hormonal pathways.

Many other gut hormones have been shown to increase or decrease the tone of the lower oesophageal sphincter. As in the case of gastrin, it is difficult, in the present state of knowledge, to conclude that any of these play a significant part in the physiological regulation of oesophageal motility, or in the prevention of gastro-oesophageal reflux. Other agents exert a pharmacological effect on the lower oesophageal sphincter, and a number of drugs with anticholinergic actions, notably the tricyclic antidepressants, may in clinical use aggravate gastro-oesophageal reflux.

Nervous regulation of deglutition

As Mountcastle (1980) has outlined, 'the oropharyngeal phase of swallowing, completed in less than 1 second, is an intricate, stereotyped, bilaterally symmetrical sequence of inhibition and excitation, involving more than 25 muscle groups and controlled by the swallowing centre of the brainstem. This complex neuroreaction pattern can be initiated by stimulation of a single efferent nerve, the superior laryngeal nerve. Although the initiation of swallowing is under voluntary control, in the same way as other voluntary movements, it is often effected without conscious effort. It appears in the fetus as early as 12 weeks of gestation, long before suckling and respiratory movements occur. On average, normal adults swallow 600 times in 24 hours, with 50 swallows occurring during sleep and only 200 during eating. During the act of drinking, swallows can be repeated as frequently as 1 second but in the absence of a bolus, the frequency is greatly reduced, which is an indication that swallowing depends upon peripheral stimuli from the oropharynx as well as messages from higher centres. In fact, it would appear that swallowing can be initiated voluntarily if afferents from the pharynx are blocked by administration of a local anaesthetic. In this situation, swallowing can still be initiated by direct electrical stimulation of the superior laryngeal nerve.

In addition to the superior laryngeal nerves, other afferents which stimulate swallowing converge on the nucleus solitarius by way of the maxillary branch of the trigeminal nerve. Afferent stimuli over these same nerves also initiate other patterned responses, such as gagging, coughing and chewing. The temporal and spatial patterns of the afferent stimuli determine the patterned response that is evoked. Appropriate afferent stimuli are relayed to the swallowing centre in the reticular formation of the rostral medulla. The centre is actually
two paired half centres that continue to initiate swallowing responses from the ipsilateral muscles when the connections between them are severed. Impulses from the swallowing centre then activate the appropriate motor neurons in the nucleus ambiguus and the fifth, seventh and twelfth cranial nerve nuclei. The resultant motor activity can be analysed from electromyographic tracings. Once the swallowing centre has been activated there is little evidence that afferent impulses play any role in modifying the oropharyngeal patterned muscular response. Activation of the swallowing centre influences the activities of other centres, most notably the respiratory centre, as evidence by an apnoeic pause of 0.5-3.5 seconds that accompanies every swallow. The continuous train of action potentials that maintains the resting tone of the upper oesophageal sphincter is interrupted, with resultant sphincter relaxation before the onset of pharyngeal peristalsis. Closure is effected as the neural discharge propagating the peristaltic wave passes through the sphincter and the resting tone is re-established.

It is frequently considered that the motor nerve supply to the cricopharyngeal sphincter is derived from either the recurrent laryngeal nerve or the autonomic nervous system, but there is no clinical or experimental evidence to support this view. Much more likely is that the former is supplied by the pharyngeal branch of the vagus.

Lund (1965) has shown that there are experimental findings to imply that the sphincter changes are probably attributable to a reflex arc, stimulated by the peristaltic wave when it reaches the level just above the cricopharyngeal sphincter. It is likely that this 'sphincter reflex' is merely part of a series of coordinated reflexes which give rise to the peristaltic wave itself. It is suggested that the nerve supply to the cricopharyngeus muscles (a definite entity in the dog) constitutes the efferent pathway of this reflex arc. The fibres passing in these nerves are either excitatory or inhibitory, producing contraction or relaxation of the sphincter respectively. The latter change is probably a result of a reduction of the nerve impulses which normally pass continuously to the sphincter, thereby maintaining its resting tone. The arrangement of the afferent pathways is uncertain, but it may be formed by nerve fibres in the pharyngeal wall which are derived from the pharyngeal branch of the vagus.

When a swallow is produced by stimulation of the upper pharynx, an initial wave of relaxation passes downwards. When it reaches a point just above the sphincter, the reflex arc is stimulated and the sphincter relaxes. This is followed by a contraction wave which also passes down the pharynx but, in this case, stimulates the reflex to produce contraction of the sphincter. Thus the sphincter initially relaxes and subsequently contracts, both changes being coordinated with the peristaltic wave.

When the pharynx in the dog is experimentally divided above the sphincter, the peristaltic wave is unable to pass downwards in the normal way. As a result, the afferent pathway is not stimulated and the normal sphincter changes do not occur, even though the nerve supply to the cricopharyngeus muscles is intact. Similarly, the sphincter again fails to function normally if the nerve supply to the cricopharyngeus muscles is divided, but the pharynx is continuous with the sphincter. In this case, it is the efferent pathway which has been destroyed.
Sphincter actions, therefore, depend upon the integrity of the nerve supply to the cricopharyngeus muscles and also on continuity being maintained between the pharynx above and the sphincter below.

A distinction has been made between primary and secondary peristalsis. Mountcastle (1980) pointed out that it was originally believed that primary peristalsis, which is initiated by swallowing, was mediated by impulses originating from the swallowing centre, whereas secondary peristalsis, which is initiated by a bolus in the oesophagus, was mediated by local intramural neural pathways. Secondary peristalsis serves to return material, following its reflux into the oesophagus, to the stomach, or to carry material to the stomach that has been left behind by an ineffectual peristaltic wave. Secondary peristalsis can be produced experimentally by inflation and deflation of a balloon in the oesophagus. It now appears that the designations 'primary' and 'secondary' peristalsis should be used only to indicate how peristalsis is initiated as both central and local neural pathways appear to be involved, regardless of the means of initiation. The initial even in oesophageal peristalsis is stimulation of the longitudinal muscle of the oesophagus followed by segmental activation of circular muscle and inhibition of the distal oesophagus.

The stimulation of the longitudinal muscle is cholinergic (atropine sensitive). The circular muscle displays a different response in that it does not contract until after the end of the electrical stimulus (off-response) and is not inhibited by antagonists to any of the known autonomic transmitters. It has been suggested that vagal non-cholinergic non-adrenergic inhibitory nerves hyperpolarize the circular muscle by an as yet unidentified neurotransmitter. When the source of stimulation is turned off, rebound depolarization leads to contraction. Oesophageal peristalsis will not be understood until the relation between the longitudinal and circular muscle activity is clarified and the nature of the mediator of the vagal inhibitory nerve is identified. The ability of anticholinergic drugs to block peristalsis in the oesophagus may be through their action on the cholinergically innervated longitudinal muscle.

With increasing age, the frequency of 'misfires' and abnormal segmental, non-progressive contractions in the smooth muscle segment increases (tertiary contractions). If, instead of a single swallow, the subject takes a series of swallows - as in drinking a glass of water - each swallow initiates the complete oropharyngeal sequence, but peristalsis in the oesophagus is inhibited until the last swallow of the series. The cricopharyngeal sphincter relaxes and closes with each swallow, whereas the cardiac sphincter opens with the first swallow and closes only when the peristaltic wave enters the sphincter. Secondary peristalsis, initiated by balloon distension of the body of the oesophagus, can be inhibited by a swallow. These observations are best interpreted as an indication of the inhibition of distal neuromuscular activity during that of the proximal segments, presumably mediated through the swallowing centre. In addition, local afferent impulses from the oesophagus alter oesophageal peristalsis, for example the course of the peristaltic contraction is proportional to the size of the bolus. On the other hand, swallows of hot liquid increase the speed and amplitude of oesophageal peristalsis, whereas cold swallows decrease speed and amplitude and finally abolish peristalsis altogether. Acid swallows cause disorder and delay of oesophageal peristalsis. Recent studies suggest that afferent impulses of local origin may not only modify the pattern of oesophageal peristalsis, but may indeed be essential for its normal propagation. In conscious dogs, with a cervical oesophageal cannula in place, it was found that with the cannula closed oesophageal peristalsis occurred after 90% of water swallows,
while with the cannula open and the water swallows diverted to the outside, no peristalsis was recorded in the lower oesophagus. These findings may be relevant to oesophageal function in humans for transection of the smooth muscle portion of the oesophagus in the monkey interferes with primary peristalsis. It appears that primary and secondary peristalsis both depend on descending neural activity originating in the swallowing centre, as well as on sequential afferent input from the oesophagus.

**Innervation of the oesophagus**

Although knowledge of the afferent innervation of the oesophagus is of great importance to an understanding of its motor function, very little is known at present. Neither the location and characteristics of the sensory receptors nor the functional significance of the afferent impulses that are found in the vagus and sympathetic nerves have been defined. Electrical records from the nodose ganglion in the cat show two kinds of afferent signals arising from the oesophagus in response to balloon distension. One is a continuous discharge for the duration of the distension, and the other is an ‘on-off’ signal firing one burst after inflation and another after deflation of the balloon.

Although the striated portion of the oesophagus appears to have the same innervation as that of other striated muscles, it contracts slowly, remains contracted for 1-2 seconds, and then relaxes slowly.

The motor innervation of the smooth muscle portion of the oesophagus is believed to be from the vagal oesophageal plexus. The preganglionic parasympathetic fibres synapse in the myenteric plexus with postganglionic neurons that innervate the oesophageal smooth muscle. Exactly how the peristaltic wave is propagated is not understood, but it is unlikely that it is mediated by serial excitation of cells in the motor nuclei of the vagus. Distension of isolated segments of the opossum oesophagus - which, like the human oesophagus, has smooth muscle in its distal half - produces peristalsis. This peristalsis is neural rather than myogenic in origin because it is blocked by tetrodotoxin, a pharmacological agent that selectively depresses transmission along nerve fibres. Studies of this preparation suggest that there are motor nerves in the oesophagus that are neither cholinergic nor adrenergic, the so-called non-adrenergic inhibitory nerves.

The contribution of the adrenergic nerves to oesophageal motor function is still undefined. It seems clear, however, that the classic view that cholinergic impulses are excitatory and adrenergic impulses are inhibitory is an over-simplification.

In summary, deglutition, or swallowing, is a complex neuromuscular operation initiated consciously but carried to completion by an integration of afferent impulses and central nervous system efferent impulses, organized both in the swallowing centre and in local intramural arcs, the latter act on a muscular tube made up of both striated and smooth muscle. The oesophagus is separated from the pharynx above and the stomach below by a pair of sphincters, the upper and lower oesophageal sphincters, which maintain a resting intraluminal pressure greater than the adjacent segments. Swallowing may be viewed as a relaxation of the swallowing tube to receive the swallowed bolus, followed by caudally progressing muscular contracture, the peristaltic wave, sweeping the bolus before it. Relaxation is most obvious in those segments with a high resting tone, namely the sphincters, whereas the progressive nature
of the peristaltic wave is most obvious in the remaining segments that exhibit a low resting tone. It is possible that the sphincters are differentiated from adjacent segments, not so much by special neural arrangements, but rather by an increased sensitivity of the neuromuscular elements of the sphincters to the neurohumoral determinants of motor function.

**Applied physiology of deglutition**

**Pharyngeal paralysis**

The cranial nuclei, containing the cell bodies of the neurons which supply the muscles in swallowing, lie close together in the medulla. Lesions in this region, such as those of bulbar poliomyelitis, motor neurone disease, or thrombosis of the posterior inferior cerebellar artery, may cause dysphagia. The palatal and pharyngeal muscles may be paralysed, but, interestingly, the cricopharyngeal sphincter is usually unaffected and will relax normally. A complete hold-up of the bolus at the level of the sphincter may occur very rarely in a total pharyngeal palsy, but after an interval of time, the bolus normally passes through successfully by virtue of thrust by the tongue and gravity in the erect position. Nasal regurgitation and laryngeal spill with coughing can occur on attempted swallowing. In these cases, a cricopharyngeal myotomy may be indicated in progressive neurosurgical disorders; however, it should be remembered that there is a risk of a reflux of the oesophageal contents upwards through the divided sphincter, particularly if the oesophagus is also paralysed.

As already mentioned, the opening of the oesophageal sphincter results from two separate mechanisms, that is the central inhibition of the ongoing activity in the cricopharyngeal constrictors, together with active contraction of the suprahyoid muscles. Abnormalities in the opening of the upper sphincter may be due, therefore, to involvement of either the cricopharyngeus or the suprahyoid muscles, or both, as suggested by Goyal and Cobb (1981).

**A lump in the throat**

If local and distant (gastro-oesophageal junction) causes have been excluded, the lump in the throat symptom will usually be a consequence of spasm or failure of the cricopharyngeal sphincter to relax.

Although cineradiography is of enormous help in assessing these patients, in a review of 100 consecutive cine films, Lund (1965) found no radiological abnormality in 85 patients. In the remaining 15 patients, the commonest irregularity was gastro-oesophageal reflux.

Watson and Sullivan (1974) would seem to confirm this aetiology as they found high resting upper oesophageal sphincter pressures in patients with 'globus sensation' to the order of 140-220 mmHg, as compared with pressures of 70-140 mmHg in their control subjects.

Lane (1980) also believed that incompetence of the lower oesophageal sphincter is relevant in that it reduces the pH in the oesophagus; this, in turn, initiates incoordinated peristaltic movements, with reflux production of alkaline saliva which the patient continually swallows. It is the latter which exacerbates the feeling of a lump in the throat.
The posterior pharyngeal or Zenker’s diverticulum

This condition occurs posteriorly between the upper and lower halves of the inferior constrictor muscles, that is the thyro- and cricopharyngeal muscles respectively. The pouch arises as a result of a combination of early closure of the cricopharyngeal sphincter and weakness or incoordination of the pharyngeal peristaltic wave.

If the peristaltic wave in a normal swallow is likened to the smooth, progressive squeezing of a toothpaste tube (held upside down) from the bottom to the top, then a similar analogy can be drawn to describe the formation of these early pouches. If the toothpaste is first squeezed down from the bottom of the tube (the peristaltic wave sweeping down the pharynx) and then suddenly, with the other hand, pressure is applied in front (the cricopharyngeal muscles contracting), a bulge will appear in the tube between the two flattened portions (this is the equivalent of the pouch). Thus the pouch is formed when the sphincter is closing and not when it is relaxing, with relaxation generally being adequate. This explanation is supported by the manometric findings of Ellis et al (1969) in which all patients showed an abnormal temporal relationship between pharyngeal contraction and termination of sphincter relaxation, as the sphincter contraction occurred before completion of contraction in the pharynx.

Oesophageal motility

Bouchier et al (1984) maintained that the three indices of normal oesophageal motility (peristalsis, sphincter tone and sphincter relaxation) may be disturbed in several ways in oesophageal motility disorders. The peristaltic nature of the contraction wave may become lost. This often results in pressure waves, which develop simultaneously or in a non-sequential way at different levels of the oesophagus and, as such, are deprived of much of their propulsive force. The contraction may also be too strong and last too long, resulting in painful spasm or dysphagia. Contractions that are too weak will have little or no propulsive force. Other manifestations of disordered motility are 'spontaneous' motor activity (not elicited by swallowing) and repetitive contractions in response to a single swallow. Sphincters may be hypotensive and relax inappropriately, thus allowing reflux. They may be hypertensive and produce pain, or they may fail to relax sufficiently so that dysphagia ensues and stasis develops proximally. In most cases of disordered motility, several of these abnormalities are combined. However, the combination is not always sufficiently typical and specific to be pathognomonic.

Oesophageal motility disorders have been classified as primary and secondary. In primary motor disorders, the oesophagus is the site of major involvement; this group includes achalasia, diffuse oesophageal spasm and related motor disorders, as well as the conditions termed 'presbyoesophagus' and 'symptomatic (or hypertensive) peristalsis' (nutcracker oesophagus). In secondary motor disorders, oesophageal abnormalities are caused by more generalized nervous, muscular, or systemic diseases, metabolic disturbances, or to inflammatory or new growth lesions of the oesophageal wall. Examples in this secondary group are systemic sclerosis, various muscle diseases such as myotonia dystrophica, and lesions of the central nervous system involving the brainstem.