Esophageal Motility in Health and Disease*

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The development of small sensitive pressure-detecting devices for the accurate and continuous registration of esophageal pressures has revived interest in the mechanism of deglutition in the normal man and in his disease states. As a result of the application of these new techniques to the study of esophageal motility and the correlation of physiologic, pharmacologic, and anatomic data, new interpretations of dysphagia in certain disease states are now available. It is the purpose of this paper to review these newer developments as they pertain to esophageal motility in health and disease.

Methods for Studying Esophageal Motility

Until recently, the two methods most frequently used to study esophageal motility and the effect of pharmacological agents were roentgenologic techniques and methods employing balloons to obtain kymographic records of esophageal motor activity. The advantages and limitations of these methods have been discussed previously.1

Roentgenologic methods must, of necessity, be employed during brief periods of observation, and continuous records of motor activity are difficult to obtain. For visual interpretation of the dynamics of deglutition, however, roentgenologic techniques are unsurpassed.

Balloon methods for studying esophageal motor activity offer the advantage of prolonged periods of observation, and they are easily adapted to the continuous recording of motor activity. The disadvantage of this method is that the balloon must be expanded if changes in its volume are to be recorded. The filling of a balloon with air or water under positive pressure results in distention of the esophagus where intraluminal pressure is normally negative. The resulting distention thus provides a local stimulus for motor activity. Although modifications of the balloon method have been used to excellent advantage in studies of the effects of various pharmacologic agents on the esophageal musculature,2, 3 definite limitations in recording motor activity resulting from deglutition exist. The balloon, which is anchored at one site, acts as an obstruction to the swallowed liquid or bolus. The almost continuous activity which the balloon stimulates interferes with the recording of sequential motor activity resulting from deglutition.

By new techniques,4, 5, 6 it is now possible with miniature pressure transducers or small open tipped tubes to record, accurately and continuously, changes in esophageal intraluminal pressure resulting from motor activity. These methods do not obstruct or stimulate the esophagus.

From the Department of Internal Medicine of the Scott and White Clinic,
Following deglutition, pressure changes occurring in different levels of the esophagus can be recorded with ease.

**Stages of Deglutition**

As stated by Starling, "It has been customary since the time of Magendie to divide the act of swallowing into three stages." The first stage is a voluntary act which initiates the other two stages, which are reflex in origin. In the first stage, liquid or a bolus of food is passed from the mouth into the pharynx. This is accomplished principally by the mylohyoid muscles which retract and elevate the tongue against the hard palate, thus forcing the liquid or bolus into the pharynx.

The second stage is concerned with the passage of the swallowed material through the pharynx. This stage is short in duration, and is a highly complicated and integrated mechanism. In order for the swallowed material to continue on its intended course into the esophagus, it is necessary that other pathways into the pharynx be closed. This is accomplished by a number of almost simultaneous actions.

The swallowed material is prevented from re-entering the mouth by the retracted and elevated tongue against the hard palate and also by the contraction of the posterior pillars of the fauces which tends to close off the cavity of the mouth. Above, the nasopharynx is sealed off by the elevation of the soft palate which comes in contact with the posterior pharyngeal wall. The entrance into the larynx and trachea is effectively closed off by the elevation and forward movement of the larynx, due mainly to the action of the mylohyoid muscles. This results in the larynx coming to rest under the base of the tongue. Both the true and false vocal cords are approximated and this further aids in sealing off the respiratory pathway.

At the completion of the above described events, which requires only a fraction of a second, the pharyngeal constrictors contract to produce a rapid peristaltic wave. This wave arises at about the level of the laryngeal opening and propels the bolus toward the opening of the esophagus. The cricopharyngeus muscle, which has kept the esophagus closed until now, relaxes as the oncoming bolus approaches and allows it to enter the upper esophagus. The peristaltic wave continues into the esophagus to become the primary esophageal peristaltic wave.

The third stage of deglutition involves the passage of liquids and solids through the esophagus. This stage of deglutition varies somewhat with the handling of liquids and a solid bolus. The mechanisms of each will be discussed in detail under esophageal transport.

**Types of Esophageal Contractions**

In the normal individual, three types of esophageal contractions have been described.

The primary peristaltic contraction, according to Templeton, actually originates in the pharynx during the second stage of deglutition. From the pharynx, this peristaltic contraction travels down into the esophagus in an unbroken manner to the level of the diaphragm. The rate of propa-
gation of the peristaltic wave becomes progressively decreased as it descends the esophagus. The faster rate of travel in the upper esophagus is thought to be due to the striated muscle which is found there. By actual measurement of the time required for the primary peristaltic wave to reach different levels of the esophagus, it has been found that it takes 3.2 seconds for the peak pressure produced by the wave to occur in the upper, 6.3 seconds in the middle, and 9.7 seconds in the lower esophagus. From data such as these, it has been computed that the velocity of a primary peristaltic wave varies from 2 to 3 cm. per second.

The mechanism of production of the primary peristaltic contractions is entirely reflex. The afferent limb of the reflex arc is principally the pharyngeal branches of the glossopharyngeal nerve. The efferent limbs of the reflex arc are the vagi. Central connections of the arc are thought to be in the medulla in the neighborhood of the vagus nucleus. Certain observations indicate that there is considerable central integration responsible for the orderly manner in which the primary peristaltic wave descends down the esophagus. For example, when a primary peristaltic wave approaches a local pathological process, such as an annular carcinoma of the mid-esophagus, interrupting the local muscular and neural elements, the peristaltic wave ceases upon reaching the lesion, only to be observed a moment later appearing below the lesion and continuing on towards the stomach. Local neural connections, the Auerbach plexuses, also may be of importance in the propagation of the primary peristaltic wave.

The secondary peristaltic contraction occurs in the normal esophagus only in response to distention. The distention may be localized such as that produced either by a solid bolus of food or experimentally in response to the distention of a small balloon placed in the esophagus. Kramer and Ingelfinger have determined the average rate of occurrence of secondary contractions induced by a distended balloon, and found it to be 9.5, 6.5, and 8.1 waves per minute in the upper, middle, and lower parts of the esophagus, respectively.

Distention over a considerable portion of the esophagus, as occurs with liquids, also may result in the production of secondary peristaltic waves. When such contractions are observed roentgenoscopically, they usually arise in the region of the arch of the aorta. A segment of the esophagus is seen to undergo spontaneous contraction which forces barium both toward the mouth and stomach. The contraction wave then proceeds to move down the esophagus in a manner similar to the primary peristaltic wave. That the secondary peristaltic wave is definitely propulsive in nature also has been demonstrated by balloon studies.

Tertiary esophageal contractions have been observed and described in apparently normal individuals past middle age. These contractions occur irregularly and locally, and are observed in the lower esophagus. They are not peristaltic. Their function is not known. Tertiary contractions occur rapidly and are of brief duration. Roentgenologically, they may produce a varied picture such as a serated or beaded appearance, multiple
diverticula-like pockets, and sometimes a picture which is referred to as "curling" and the "corkscrew esophagus." It has been suggested that contractions of the spiral muscular coat of the lower esophagus account for this phenomenon.\textsuperscript{12} Although Carlson\textsuperscript{13} may have observed the tertiary type of contraction using a balloon method to study esophageal motility, tertiary contractions apparently have not been observed using pressure devices. Roentgenologically, they are observed to occur spontaneously. Also, at times, they are seen to appear in the lower esophagus when a primary peristaltic wave has reached about the level of the aortic arch. The peristaltic wave may stop at this level or continue down the esophagus to obliterate the tertiary contractions.

**Esophageal Transport**

Transport within the esophagus, as in other parts of the alimentary canal, is dependent upon a gradient of pressure to move materials within the lumen. In the esophagus, the principal factor producing this pressure gradient is the primary peristaltic contraction. It has been stated that the peristaltic contraction is preceded by a wave of relaxation.\textsuperscript{8,14} This statement apparently has not been based on experimental evidence but upon the "law of the intestine" proposed many years ago by Bayliss and Starling.\textsuperscript{15} They found that mechanical stimulation of the intestine caused contraction above and relaxation below the point stimulated. There is no evidence that the increased pressure produced by the esophageal peristaltic contraction is preceded by a reduction in intraluminal pressure, indicating that relaxation occurs ahead of the peristaltic wave. There are also doubts of the validity of the "law of the intestine" as applied to the intestinal tract. Later investigators\textsuperscript{16,17} have been unable to confirm the observations of Bayliss and Starling. It would seem that the important factor concerned with transport in the esophagus is that some propelling force exists, not whether the canal ahead is relaxed or offers resistance.

In transport of a solid bolus, the primary peristaltic contraction is of particular importance. It sweeps the bolus ahead of it as it progresses from the upper to the lower esophagus. If the primary peristaltic wave is ineffective due to the size, consistency or dryness of the bolus, secondary peristaltic contractions occur which complete the transport of the bolus through the esophagus. These latter contractions, produced by bolus distention of the esophagus, are initiated locally.

The manner in which liquids are transported through the esophagus is dependent upon the position of the subject since gravity apparently plays an important role. In the upright position, liquids may travel through the esophagus at a speed that greatly exceeds the rate or propagation of the primary peristaltic wave. This rapid transfer of liquid to the lower end of the esophagus is considered to be due to the propelling force created by the contraction of the pharyngeal constrictors during the second stage of deglutition, as well as by the action of gravity upon the swallowed liquid. Thus, from an area of positive pressure, the liquid is more or less squirted into the lumen of the esophagus, in which there is a negative pressure. In the upright position, the rapid passage of liquids through
the esophagus requires only a few tenths of a second, and occurs while the esophagus is relaxed and before the primary peristaltic contraction in the esophagus has begun. Fluoroscopy has shown that swallowed liquids are usually arrested at the lower end of the esophagus and await the arrival of the peristaltic contraction before they are admitted into the stomach. At times, however, when a series of swallows occurs rapidly, as when drinking a glass of water, the liquid may pass on through the cardia with little pause and enter the stomach within a second or two after drinking has begun. Almost everyone has experienced this when drinking a very cold or very hot substance by becoming aware of an almost immediate feeling of cold or warmth in the epigastrium. When a quick succession of swallows occurs, the primary peristaltic wave is inhibited until the last swallow is finished. The peristaltic wave then passes down the esophagus emptying any remaining contents.

In either the supine or head down position, the force of gravity is eliminated and the liquid bolus is handled in a manner similar to a solid bolus; i.e., transport is dependent upon the primary peristaltic wave.

The exact mechanism of esophageal evacuation into the stomach has been a matter of much dispute and still has not been established. Lerche has postulated a mechanism of esophageal evacuation which is based on detailed anatomic studies and roentgenographic correlations. His anatomical findings indicated the presence of two dilatations of the lower end of the esophagus as demonstrated in Figure 1.

The upper dilatation is called the esophageal ampulla, and the lower one the gastroesophageal vestibule. The ampulla is simply a bulbar dilatation

![ESOPHAGEAL AMPULLA, LOWER ESOPHAGUS, INF. ESOPH. SPHINCTER, GASTRO-ESOPHAGEAL VESTIBULE, CONSTRUCTOR CARDIAE, STOMACH](image)

**FIGURE 1**: Schematic diagram of anatomy of the distal esophagus, according to Lerche.
of the lower end of the esophagus. It is separated from the gastroesophageal vestibule by the "inferior esophageal sphincter." The gastroesophageal vestibule is separated from the stomach by the "constrictor cardiae" which also is said to have a sphincteric action. The intradiaphragmatic portion of the esophagus is the gastroesophageal vestibule, the diaphragm encircling the vestibule at the level of about one centimeter below the inferior esophageal sphincter.

Lerche believes the inferior esophageal sphincter normally remains tonically closed and opens mechanically or reflexly to the peristaltic pressure above the ampulla. After the inferior esophageal sphincter has opened, the ampulla contracts forcing its contents into the vestibule. When the vestibule is filled, being closed below by the constrictor cardiae, the inferior esophageal sphincter contracts. The constrictor cardiae now relaxes and the vestibule contracts and shortens itself, emptying its contents into the stomach. As the vestibule relaxes, it elongates and extends through the open constrictor cardiae, evertting a portion of the vestibular mucosa into the stomach.

**Esophageal Pressures**

With the development of techniques for determining esophageal intraluminal pressures directly, a clearer conception of the mechanical factors involved has been possible. It is desirable to discuss these pressures on the basis of their origin. In the normal esophagus, three types of pressures have been observed. These include: (1) basal pressure, which is the intraluminal pressure of the resting esophagus; (2) respiratory and cardiovascular pressures, which are transmitted to the lumen of the esophagus and are superimposed on basal pressure; and (3) pressures resulting from deglutition or spontaneous esophageal motor activity.

**Basal Pressure:** Basal intraluminal pressure is defined as that pressure existing within the lumen of the esophagus when there is an absence of

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motor activity. Various factors influence the basal pressure such as the tonus of the muscular wall of the esophagus and intrathoracic and intra-abdominal pressures. Variations in pressure due to respiratory movements and cardiovascular pulsations, and the presence of certain adjacent viscera which may exert a compression or traction upon the esophagus also may influence intraluminal pressure. The most important factor which influences basal pressure is that the esophagus is subjected to the negative pressure that exists within the thoracic cavity.

Basal pressure determined by a direct measuring method, in a group of normal adults, was found to be —5.5 cm. of water when recorded from the middle third of the esophagus. (Table I).

Respiratory and Cardiovascular Pressures: Superimposed upon the negative basal intraluminal pressure are pressure changes due to respiration and cardiovascular pulsations. Inspiration produces a further increase in the negative intraluminal pressure while expiration decreases the negative intraluminal pressure. (Figure 2). Respiratory pressure changes usually vary from 4 to 12 cm. of water pressure. In normal adults, eupnic respiration was found to produce a mean pressure change of 6.5 cm. of water when recorded from the lower esophagus. (Table I).

Changes in esophageal intraluminal pressure due to cardiovascular pulsations are usually quite constant in any one individual, but have been found to vary from 1.2 to 3.6 cm. of water in different individuals. When recorded from the lower esophagus, the mean pressure change which cardiovascular pulsations produced was 2.4 cm. of water. Both respiration and cardiovascular pulsations produce a greater change in intra-

![FIGURE 2: Example of deglutition pressure pattern recorded from mid esophagus illustrating the initial negative wave and subsequent positive waves of pressure. Note the entire complex occurred while respirations were voluntarily inhibited. (From Hightower, N. C., Jr.)](image-url)
luminal pressure in the esophagus than in any other site within the alimentary canal.

Deglutition Pressures: Characteristic pressure changes occur within the esophagus during the act of swallowing. Butin and associates,\textsuperscript{10} using a method of recording esophageal intraluminal pressures directly,\textsuperscript{4} have made an extensive study of pressure changes occurring within the esophagus of normal individuals following deglutition. They had their subjects swallow liquid as well as a solid bolus; and recorded the pressure changes in the upper, middle, and lower esophagus. In these studies, they found a characteristic pattern of pressure change regardless of whether the subject swallowed liquid or a solid bolus. They termed this pattern, which consisted of an initial negative wave followed by three positive waves, the “normal swallowing complex.”

The initial negative wave in the “normal swallowing complex” described by Butin and associates was observed 42 years ago by Schreiber,\textsuperscript{18} using a balloon technique to study esophageal motility. Schreiber and later Lerche attributed the initial negative wave as being due to the stretching of the esophagus resulting from the elevation of the larynx. During the second stage of deglutition when elevation of the larynx occurs, the esophagus is closed off at both the pharyngeal and cardiac ends. With the elevation of the larynx, the esophagus is pulled up in front by the tendon of its longitudinal muscle layer and in back by the stylo-pharyngo-palatino-esophageal muscle apparatus.\textsuperscript{12} As both ends of the esophagus are closed, this elongation results in the negative intraluminal pressure already present becoming more negative. In the studies of Butin, et al., the initial negative wave was observed in about a third of the complexes recorded from normal individuals. It occurred more frequently in the upper and lower third of the esophagus than in the middle third. If the mechanism of production of this negative wave is as postulated by Schreiber and Lerche, one might expect to observe it more frequently in the upper and lower esophagus as contrasted to the middle portion. It is at the ends that the esophagus is anchored, and a pull or stretch of the organ might produce a greater negative pressure in these areas than in the mid-portion. Butin et al. found the initial negative wave to occur immediately after the onset of swallowing, that is, usually within 0.1 second, and that it was approximately 0.4 seconds in duration, and the mean negative pressure produced was 8.4 cm. of water.

Sanchez, Kramer, and Ingelfinger,\textsuperscript{6} using open tipped catheters to record esophageal deglutition pressures, stated they rarely observed the initial negative pressure wave described above. They attributed minor significance to the wave as a component of the normal pressure complex. Ingelfinger\textsuperscript{19} questions whether the initial negative wave is an essential feature of the swallowing complex or whether or not it is related to the respiratory phase and fixation of the diaphragm at the onset of swallowing. That the initial negative wave is an integral part of the esophageal deglutition pressure pattern and not related to respiration is evident from Figure 2. This illustration clearly shows the initial negative wave in a deglutition
pressure pattern recorded while the subject held his breath. Butin et al. have adopted the explanation offered by Schreiber and Lerche for the mechanism of production of the initial negative wave and the available data indicate that such an explanation is correct.

The second component of the “normal swallowing complex” is an abrupt and almost instantaneous positive pressure change. This sudden increase in pressure is shown in Figure 2 where it is seen to occur immediately after the negative pressure wave. Sanchez et al. attribute this component of the pressure complex to the sudden injection of liquid into the esophagus as they observed it to occur simultaneously in the upper seven-eighths of the esophagus after swallowing liquids. It was seldom observed after a “dry” swallow.

Butin and associates favor the explanation that the origin of the sudden pressure change is due to transmission of buccopharyngeal pressures into the esophagus. The exact origin of this component of the pressure pattern has not been determined. It was observed in 87 per cent of the records studied by Butin, et al. They found that the time of onset of the first positive wave varied with the depth of the transducer in the esophagus, beginning at 0.5, 0.8, and 1.5 seconds in the upper, middle, and lower parts of the esophagus, respectively. The magnitude of the first positive pressure wave is usually about 12 cm. of water.

Following the abrupt positive pressure wave just described, the third component of the pressure complex occurs. This consists of a slowly increasing positive pressure change, or else a plateau of positive pressure is maintained. Rarely, after the first positive pressure wave does there occur a decrease in pressure. The origin of this component of the pressure pattern is thought to be related to the approaching peristaltic wave and to represent a slowly increasing pressure head. The time of onset and duration of this second positive pressure wave increases as the recording device is placed deeper into the esophagus. These time relationships strongly suggest that this component of the pressure pattern is related to the primary peristaltic contraction. Butin, et al. give the time of onset as 1.5, 2.7, and 4.4 seconds in the upper, middle, and lower parts of the esophagus. The greatest pressure attained, found in the lower esophagus, was 21.9 cm. of water. Sanchez, et al. state the duration of this component of the pressure pattern varies from two to 19 seconds.

The fourth and most prominent component of the deglutition pressure pattern is a large, simple, monophasic, positive pressure wave which rises rapidly to a peak pressure and declines with equal speed. It immediately follows the gradual rise or the plateau of pressure described above. This wave, which has been described in detail,\(^8\)\(^,\)\(^10\) represents the pressure produced by the primary peristaltic wave. Butin and associates found that mean values for the time elapsed from swallowing to the beginning of the wave were 2.1 seconds in the upper, 5.1 seconds in the middle, and 7.9 seconds in the lower esophagus. They noted that the peak pressure occurred at 3.2, 6.3, and 9.7 seconds in the upper, middle, and lower esophagus, respectively. When recorded from the middle esophagus with the
subject in the supine position, it was found that a 15 ml. swallow of water produced a mean peak pressure of 100 cm. of water while a solid bolus produced a pressure of 74 cm. of water. (Table I). A later study, using the same method of recording pressures, gave similar results.20 In this study, subjects were given 10 ml. of water to swallow and the pressure transducer was located at the junction of the middle and lower third of the esophagus. The liquid produced a peak pressure of 88 cm. of water and with a solid bolus (2 by 1 cm. circular piece of white bread) a pressure of 72 cm. of water was recorded (Table I). In still another study using open-tipped catheters filled with water and connected to Sanborn electromanometers, Sanchez, Kramer, and Ingelfinger6 found the peak pressure from swallowing liquids to vary from 40 to 110 mm. Hg. in the upper seven-eighths of the esophagus (Table I). The duration of the pressure wave produced by the primary peristaltic contractions varies from 2.3 seconds in the upper to 3.0 seconds in the lower esophagus.10

Secondary Peristaltic Pressures: With the methods employed to register deglutition pressures, as given above, distention of the esophagus is not produced, and secondary peristaltic waves and their accompanying pressure changes are rarely observed. By distending the esophagus with a small (35 ml. capacity) water-filled balloon under 15 cm. of water pressure and recording simultaneously with a pressure transducer, secondary peristaltic contractions can be induced and the pressure changes resulting from such contractions have been recorded.5 The secondary peristaltic contraction produces a pressure wave similar to the final pressure wave of the deglutition pressure pattern. It is a simple, monophasic, positive pressure wave which arises rapidly to a peak then returns promptly to the base line. An analysis of 54 secondary peristaltic pressure waves recorded from the mid esophagus revealed the mean peak pressure waves attained to be 32 cm. of water (Table I). The mean duration of the waves was 7.6 seconds. The secondary peristaltic contractions were observed to occur spontaneously at a rate of 4 to 6 per minute.

Pressure Changes in the Distal Esophagus: A recent investigation of pressure changes in the distal esophagus during deglutition has been carried out by Sanchez, Kramer, and Ingelfinger.6 The authors have adopted the anatomical terminology proposed by Lerche in reporting their findings. In the esophageal ampulla, they recorded a pressure pattern following deglutition which was similar to that observed in the lower esophagus but different in some important respects. The initial positive pressure wave was present and this was followed by a plateau of pressure. The positive pressure wave due to the primary peristaltic contraction was observed to rise rapidly to a peak; but, instead of dropping off rapidly, it returned to the base line in a gradual slope with respiratory pressure changes superimposed upon the slowly declining wave. The peristaltic pressure wave in the ampullary complex had a duration of 15 to 22 seconds and traveled at a rate of 0.7 to 1.2 cm. per second. The average height of the wave is not given, but one published record indicates that it is approximately 27 mm. Hg. (Table I). The authors point out that the
characteristics of the ampullary deglutition pressure pattern are compatible with fluoroscopic observations which show that the peristaltic wave slows down as it reaches the lower esophagus. As characteristic ampullary complexes were recorded from a patient with a large diaphragmatic hernia and also one with paralysis of the left diaphragm, it would seem that the diaphragm is unimportant in the production of the pressure pattern. There is little doubt that the pressure pattern represents esophageal peristalsis.

From the vestibule, a decidedly different pressure pattern was observed. Records from this area showed only a gradual rise and fall of pressure lasting from 20 to 40 seconds. There was an absence of the first positive pressure wave. The maximum pressure attained was only 5 to 15 mm. Hg. (Table I). These observations indicate that the vestibule is not in communication with the lower esophagus but is closed off, possibly as suggested by Lerche by the inferior esophageal sphincter. This is in keeping with the fluoroscopic observation that liquids frequently collect in the lower esophagus above the diaphragm and await the arrival of a peristaltic wave before being emptied into the stomach. If the gastroesophageal vestibule exists as a separate anatomical entity, one must postulate a separate motor activity—one that is highly integrated with the motor activity of the lower esophagus. These pressure observations in the distal esophagus seem to support the mechanism of esophageal evacuation proposed by Lerche. They would not support the mechanism of esophageal emptying proposed by Mosher in which the diaphragm supposedly takes an active part.21

**Disturbances of Deglutition by Disease**

The term dysphagia means difficulty in swallowing. As it applies to the entire act, a disturbance in one or more of the stages of deglutition may give rise to dysphagia. Dysphagia is only a symptom, and it is not pathognomonic of any one specific disease. Pain may or may not be asso-

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**FIGURE 3:** Effects of Mecholyl on esophageal intraluminal pressure in cardiopasm. Note the marked rise in intraluminal pressure at 9 and 17 minutes after subcutaneous administration of 10 mg. of Mecholyl. (From Hightower, N. C., Jr., Olsen, A. M. and Moersch, H. J. Reproduced with kind permission of the editor of Gastroenterology.)
associated. The occurrence of dysphagia may indicate serious disease, therefore, its presence usually warrants thorough clinical investigation, including roentgenoscopic and endoscopic examination of the esophagus.

Dysphagia due to emotional and psychic disturbances, as well as the psychosomatic aspects of cardiospasm, are purposely omitted.

**Dysphagia of the First and Second Stages of Deglutition:** There are many causes of dysphagia resulting from disturbances of the first and second stages of deglutition. Only a few will be mentioned.

Any painful inflammatory process involving the mouth, tongue, or pharynx may make swallowing difficult. Neoplasms of the tongue or posterior pharyngeal wall can interfere with the passage of a bolus into and through the pharynx. Pharyngeal paralysis is a common cause of dysphagia involving the second stage of deglutition. In such instances, it may be impossible for the soft palate to be elevated, thus, allowing liquids to be regurgitated through the nose. The pharyngeal constrictors may be unable to initiate the peristaltic contraction which empties the contents of the pharynx into the esophagus. Pharyngeal paralysis usually results from a bulbar lesion as seen in syringomyelia, poliomyelitis, cerebrovascular hemorrhage, and multiple sclerosis. Cranial nerve (glossopharyngeal) neuritis also may result in pharyngeal paralysis. Myasthenia gravis can produce dysphagia by interfering with the second stage of deglutition. Diphtheria is sometimes followed by palatine and pharyngeal paralysis which produces dysphagia.

Mobility of the larynx is essential to the second stage of deglutition, and any disease process which prevents the larynx from ascending to lie beneath the base of the tongue causes marked dysphagia. Neoplasms, such as carcinoma of the larynx and thyroid, and infectious processes, such as tuberculosis and syphilis, may result in fixation of the larynx; thus, making swallowing extremely difficult. A few congenital anomalies, such as cleft palate, may also produce dysphagia.

**Dysphagia of the Third Stage of Deglutition:** Any interference with transport through the esophagus may cause dysphagia. The process may be inflammatory, as in esophagitis; obstructive, as occurs with carcinoma or benign stricture; neurogenic, as in cardiospasm or possibly diffuse spasm; or due to some systemic disease such as a generalized scleroderma.

**Cardiospasm:** The etiology and mechanism of lower esophageal obstruction in achalasia or cardiospasm has been a matter of much dispute. The consensus of opinion today holds that cardiospasm is not a localized process which produces "spasm of the cardia" but one in which most, if not all, of the esophagus is involved.\(^1\)\(^2\)\(^3\)\(^10\)\(^22\) This idea was suggested by Hurst\(^23\) when he recommended the term achalasia instead of cardiospasm. It was Hurst's opinion that achalasia was due to a degeneration of the myenteric plexus of the esophagus and not to hypertrophy and spasm of the cardiac sphincter. More recent investigations into the pathologic changes of cardiospasm have revealed a marked degeneration of Auerbach's plexus, especially in the lower parts of the esophagus.\(^24\)\(^25\)\(^26\)\(^27\) The unusual sensitivity of the cardiospastic esophagus to Mecholyl (Figure 3)
supports the pathologic findings that achalasia is due to a disorder of the intrinsic nerve plexuses. The effect of Mecholyl is in keeping with Cannon's law of denervation. This drug produces a marked contraction of the dilated atonic esophagus. (Figure 4).

The disturbance of motility which occurs in cardiospasm also indicates that the condition is not one of simple obstruction at the cardia. Motor activity of the esophagus in cardiospasm has been studied by roentgenologic, balloon-kymographic, and pressure detecting techniques.

In a study of 39 patients with cardiospasm, Templeton found at fluoroscopy that in all but three the primary peristaltic wave traveled only to about the level of the suprasternal notch or the superior margin of the aortic arch. In one of the patients, the primary wave did not go beyond the level of the cricopharyngeal muscle. In two patients, the primary wave continued on down the esophagus, but its depth was insufficient to influence the passage of the bolus. No secondary peristaltic waves were observed. In all but two of these patients, Templeton noted "purposeless, shallow, segmented contractions constantly appeared and disappeared at different levels of the esophagus." This type of motor activity was most prominent in patients with only slight dilatation. These contractions were similar to the tertiary contractions he observed in normal individuals. In addition to these undulating waves, generalized tonic contractions were observed which would force barium into the stomach when they produced a diffuse narrowing of the esophageal lumen. Wolf and Almy, in a fluoroscopic study of 14 patients with cardiospasm, also noted the undulating contractions of the lower esophagus described by Templeton. In addition, they found that in nine of the 14 patients a
sustained contraction of the diaphragm would intensify the degree of esophageal obstruction.

Abnormal motor activity in cardiospasm also has been demonstrated by balloon methods. Kramer and Ingelfinger\(^2\) have found decreased tone, lack of propulsion, and irregular phasic activity in the lower two-thirds of the esophagus. These findings have been confirmed by Sleisenger, Steinberg, and Almy.\(^3\)

Butin, Olsen, Moersch, and Code\(^4\) have compared esophageal intraluminal pressures in normal individuals and in patients with cardiospasm, and have found some important differences. Spontaneous motility was observed frequently (18.5 per cent) in the records obtained from patients with cardiospasm. In one, the incidence of spontaneous motility was reduced from 44 to 18.5 per cent by therapeutic dilatation which relieved most of his dysphagia. In another study,\(^5\) using the same technique of recording pressure, spontaneous activity was rarely observed following careful aspiration of the esophagus. This suggests that spontaneous activity is related to the degree of obstruction and retained secretions or swallowed material.

From an analysis of records, obtained, after deglutition, from patients with cardiospasm, Butin and associates found there was no fixed pattern of waves of pressure. Most of the pressure responses, however, were of three types: complexes that resembled the normal swallowing complex; absence of pressure waves related to deglutition; and multiple phasic contractions in response to a single swallow. An example of the abnormal

![Diagram](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21266/)

**FIGURE 5:** Deglutition pressure pattern recorded from mid esophagus of patient with cardiospasm. Note complex was recorded during breath holding and consists of only slight, irregular positive pressure wave. (Published through courtesy of Dr. C. F. Code, Section on Physiology, Mayo Foundation.)
pressure pattern observed in cardiospasm following deglutition is seen in Figure 5.

In a comparison of deglutition pressures in normal persons and patients with cardiospasm, it was found that a 10 ml. swallow of water produced a mean peak pressure of 87.9 cm. of water in normals and only 24.8 cm. of water in patients with cardiospasm. A solid bolus resulted in a peak pressure of 71.6 cm. of water in the normals and 17.3 cm. of water in those with cardiospasm.20

Diffuse Spasm: Moersch and Camp,3 in 1934, first described diffuse spasm of the lower part of the esophagus as a clinical entity. Dysphagia due to diffuse spasm usually is intermittent and painful. Roentgenoscopically, three types of disturbed motor activity have been described: (1) diffuse, irregular spasm of the lower half or third of the esophagus; (2) diffuse narrowing of the lower half or third of the esophagus, suggesting tetanic spasm; and (3) multiple spastic segments of concentric narrowing. Characteristic of diffuse spasm is an absence of marked dilatation above

FIGURE 6: Roentgenographic picture of diffuse spasm of lower esophagus. Note irregularity of contraction producing pseudodiverticula and minimal dilatation proximal to point of obstruction.
the area of constriction. (Figure 6). The etiology of diffuse spasm has not been established, but it is thought that the localized intermittent spasm is reflex in origin as it is frequently associated with upper gastrointestinal disease.

Few observations of esophageal motility, other than roentgenoscopic descriptions, have been made in diffuse spasm. Recently, however, esophageal pressures in two patients with diffuse spasm were studied. It was found that a 10 ml. swallow of water produced a mean peak pressure of 52.3 cm. of water, while a solid bolus resulted in a peak pressure of 47.7 cm. of water. Unlike cardiospasm, Mecholyyl does not produce a significant increase in intraluminal pressure in patients with diffuse spasm.

*Mechanical Obstructions*: Dysphagia produced by mechanical obstruction of the esophagus may be caused by intrinsic or extrinsic lesions. Intrinsic lesions include congenital anomalies as stenosis and bands; inflammatory processes, as esophagitis due to peptic ulceration, or swallowed caustics which may result in stricture; neoplasms, as carcinoma (Figure 7) and benign tumor; and foreign bodies. Examples of extrinsic lesions which produce mechanical obstructions are large inflammatory peri-esophageal lymph nodes, lymphomas, and aneurysms.

In general, most conditions causing mechanical obstruction of the esophagus produce similar disturbances in motility. When a small incomplete obstructive lesion involves the upper or middle esophagus, the primary peristaltic contraction may be observed fluoroscopically to “skip over” the lesion. When the obstruction exists in the lower esophagus, the primary peristaltic wave usually progresses to the site of the lesion with-

*FIGURE 7*: Illustrations of carcinoma of the lower esophagus: A. Constricting carcinoma of junction of middle and lower third of esophagus. Note moderate dilatation proximal to obstruction. History of dysphagia for three months. B. Obstructing carcinoma of distal esophagus with extension into cardia of stomach. Marked dilatation proximal to obstruction. Dysphagia with pain had been present for one year.
out interruption. In some instances, the waves will be shallow and not strong enough to force the barium past the stenosed portion. As most obstructive lesions occur in the lower esophagus, distention of the proximal esophagus with retention of secretions and swallowed material is not unusual. The distention acts as a stimulus for secondary peristaltic contractions, and these waves frequently are observed at the time of roentgenologic examination.

Using a balloon-kymographic method, investigations of motility in two patients with mechanical obstruction of the lower esophagus revealed decreased esophageal tone and a wave pattern consisting of strong, rhythmical contractions occurring at a rate of 4 to 6 per minute. In each patient, the inflated balloon was carried down to the point of obstruction before its progress was arrested. There is little doubt that the wave pattern described represented secondary peristaltic contractions.

Deglutition pressures have been determined in one patient with a benign stricture of the distal esophagus. Swallows of 10 ml. of water produced

![FIGURE 8: Scleroderma involving the esophagus. Cutaneous scleroderma present 4 to 5 years, the dysphagia for 16 months. Note constriction of distal esophagus just above "phrenic ampulla" with atony and dilatation of the lower esophagus. Minimal obstruction to barium flowing into stomach in upright position.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21266/)
a mean peak pressure of 66.7 cm. of water at the junction of the middle and lower third of the esophagus. A solid bolus resulted in a mean peak pressure of 34.2 cm. of water. Although these pressures are not as great as those recorded from normal individuals, they exceed the deglutition pressures observed in cardiospasm, diffuse spasm, and scleroderma. In mechanical obstruction of the esophagus, Mecholyl does not produce the marked increase in intraluminal pressure as observed in cardiospasm.20

Scleroderma: Like cardiospasm, scleroderma apparently involves most, if not all, of the esophagus. Lindsay, Templeton, and Rothman22 studied five patients with scleroderma roentgenoscopically, and found that the primary peristaltic contraction in four progressed only to the level of the suprasternal notch and ceased entirely. In one of the patients the contraction continued on down the esophagus, but was too shallow to push the bolus along. The contraction differed from a normal peristaltic wave in that the esophagus behind the advancing wave did not remain contracted, but distended immediately. Small, ineffective, secondary peristaltic contractions were observed in only one of the patients. In three, a localized narrowing of the esophagus was observed immediately above the "phrenic ampulla." Proximal to this area, mild dilatation was present. An example of the roentgenographic changes seen in scleroderma is illustrated in Figure 8.

In the upright position, swallowed barium passed from the pharynx to the stomach, with some delay at the site of stricture but not at the cardia. In either the supine or Trendelenburg position there was an absence of esophageal transport, the esophagus becoming progressively distended by successive swallows. When the esophagus filled, each swallow then forced small amounts of barium into the stomach, apparently, by transmitting pressure through the column of barium. The esophagus was unable to empty itself in the horizontal position, but if the patient was raised to a sitting or standing position, most of the barium passed into the stomach.

In balloon-kymographic studies of the disturbed motility in four patients with scleroderma, Kramer and Ingelfinger11 have reported a marked decrease in esophageal tone. They noted that propulsion of the inflated balloon down the esophagus was slow or absent. The wave pattern recorded by the balloon from the upper, middle, and lower esophagus was markedly diminished or absent.

Deglutition pressures have been measured in only one patient with scleroderma. In this one it was observed that few primary peristaltic contractions reached the lower esophagus, and those that did were of low amplitude. After swallows of 10 ml. of water, a mean peak pressure of only 10.6 cm. of water was produced, while swallows of a solid bolus resulted in a mean maximum pressure of 11.2 cm. of water.20

Mecholyl does not increase esophageal intraluminal pressure markedly in patients with scleroderma.2, 20

All of these investigations demonstrate that the primary disorder of esophageal motility in scleroderma is the defective propagation of the
primary peristaltic contraction. As there is little gradient of pressure, transport is definitely retarded. The apparent relaxation demonstrated probably represents myogenic failure.

CONCLUSION

Recent investigations of esophageal motility in health and disease have been reviewed. Although the methods of investigation have varied widely, there is a striking similarity in the results obtained. The data complement one another and their correlation affords a better understanding of the anatomy and physiology of the esophagus in the normal and diseased state. It is not to be implied that all problems have been solved with finality; on the contrary, many avenues of research are now opening. It is to be expected that further investigations along these lines and, in particular, studies of intraluminal pressures will continue to add to our knowledge.

RESUMEN

Se revisaron las investigaciones recientes sobre la motilidad del esófago normal y patológico. Aunque los métodos empleados son muy diferentes los resultados obtenidos son notablemente similares.

Los datos suministrados se complementan entre sí y su correlación permite una mejor comprensión de la anatomía y de la fisiología del esófago normal y patológico. No por eso debe pensarse que todos los problemas han llegado a su solución final. Por el contrario se están abriendo nuevos caminos a la investigación. Es de esperarse que las investigaciones ulteriores en estos lineas y en especial el estudio de la presión intraluminal esofágica agregue más a nuestros conocimientos.

RESUME

L’auteur a passé en revue les investigations récentes concernant la motilité oesophagienne, normale et pathologique. Bien que les procédés d’investigations aient largement varié, il y a une similitude frappante entre les résultats obtenus. Les constatations se complètent l’une l’autre, et leur corrélation permet une meilleure compréhension de l’anatomie et de la physiologie de l’oesophage normal et pathologique. On ne doit pas en déduire que tous les problèmes ont été résolus. Au contraire, beaucoup de voies sont maintenant ouvertes à la recherche. On doit s’attendre à ce que des investigations ultérieures dans cette direction, en particulier l’étude des pressions intra-oesophagiennes, continuent à enrichir nos connaissances.

REFERENCES

19 Ingelfinger, P. J., in discussion on Butin, J. W., et al.