Review Article

Radiologic Assessment of Abnormal Oral and Pharyngeal Phases of Swallowing

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American Journal of Roentgenology 1990;154:965-974.

In our companion report [1], we reviewed the normal physiology and radiologic appearance of the oral and pharyngeal phases of swallowing. Our purpose here is to review abnormalities of the first two phases of swallowing.

The most frequent symptoms of abnormal oral or pharyngeal swallowing include difficulty initiating swallowing, cervical dysphagia, nasal regurgitation, coughing, and choking. Generally, abnormal oral or pharyngeal swallowing function is associated with high cervical dysphagia in the upper neck, whereas the abnormal esophageal motor function or obstructive morphology may be in the retrosternal area or lower neck. The symptoms of persistent sore throat or pain with swallowing are suggestive of neoplasm or infection.

On clinical examination, abnormalities of liquid bolus preparation for swallowing include drooling from the lips due to facial muscle weakness and failure to gather the bolus normally within the mouth. Disorders in the pharyngeal phase may not be obvious by observing the patient swallow. Contrary to a widely held opinion, the efficacy of the pharyngeal swallow does not correlate with an intact gag reflex. Additionally, "silent aspiration" may go undetected on clinical examination. For these reasons, swallow function is optimally evaluated by radiologic examination.

Preparation for Swallowing

Functional abnormalities in the preparatory and oral phases of swallowing are listed in Table 1. The majority of these abnormalities are caused by abnormal tongue movement and, occasionally, by a soft-tissue defect of the tongue or oral cavity. Problems in motor function or reduced bulk of the anterior two-thirds of the tongue cause abnormalities in lifting, shaping, and propelling oral boluses; paresis, restricted movement, or loss of bulk of the posterior tongue impairs function of the glossopalatal sphincter and bolus pulsion.

Abnormalities of the preparatory phase for the oral swallow demonstrated radiologically include an inability to shape the bolus or hold it above the tongue. The latter circumstance must be distinguished from a normal dipper-type swallow in which the bolus is initially positioned in the anterior lingual sulcus [2]. Inability to segregate the bolus from the oropharynx by elevation of the posterior tongue and approximation to the soft palate may lead to premature spilling of the bolus into the oropharynx (Fig. 1).

Oral Phase of Swallowing

In some instances, an impairment of the oral phase of swallowing is seen as a repetitive attempt at initiating swallowing, piecemeal swallows, or rolling movements of the tongue (Table 1). Occasionally, some patients who are capable of normal pharyngeal swallowing may have difficulty initiating oral swallowing because of problems in cooperation, cognition, or oral sensation (Fig. 2). Abnormalities in the oral phase of swallowing generally lead to delayed oral transit and clearance of the oral bolus with retained residual in the mouth.

Received October 30, 1989; accepted after revision December 14, 1989.

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AJR 154:965-974, May 1990 0361-803X/90/1545-0965

TABLE 1: Abnormalities in the Preparatory and Oral Phases of Swallowing

Radiologic Findings	Type of Impairment
Preparatory Phase	
Cannot hold barium in mouth anteriorly	Reduced lip closure
Cannot form bolus	Reduced range or coordination of tongue movement
Cannot hold bolus posteriorly (premature spill)	Reduced tongue shaping
Cannot chew	Reduced tongue lateralization
Oral Phase	
Hesitancy initiating swallow	Impaired cognitive function, neural function, or oral sensation
Tongue moves forward at onset of swallow	Forward tongue thrust
Stasis in sulci	Reduced labial or buccal tension
Stasis in floor mouth	Reduced tongue shaping
Stasis in midtongue depression	Tongue scarring
Abnormal lingual peristalsis	Impaired tongue motion
Poor tongue to palate contact	Reduced tongue elevation
Repetitive tongue rolling	Parkinson disease
Premature spill of bolus	Reduced tongue or palatal control
Piecemeal deglutition	Abnormal neural control
Slow oral transit	Impaired tongue movement

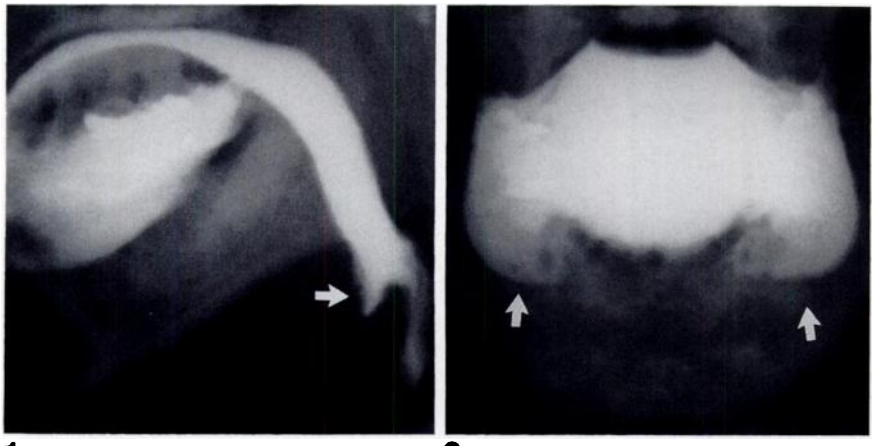

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Fig. 1.—Premature spill of barium from oral cavity into pharynx of elderly patient after a stroke. Barium fills valleculae (*arrow*) and spills over free margin of epiglottis. Also noted is dipper-type bolus preparation near tongue with barium in lateral and anterior sublingual sulci.

Fig. 2.—Posteroanterior view in elderly patient who had difficulty initiating swallowing. On initial efforts to swallow, patient stored liquid barium in buccal pouches (*arrows*) and did not initiate swallowing. Patient was then given applesauce with barium, which he swallowed normally. Patient liked taste of applesauce but not regular barium. He then swallowed liquid barium normally with only slight storage of barium in buccal pouches, which is a normal variant in some subjects.

Impaired lingual movement or conditions such as a soft-tissue defect lead to barium stasis in the mouth. Premature spilling of barium into the pharynx may be accompanied by failed transition into normal pharyngeal peristalsis or aspiration or both. Impaired oral function is commonly associated with abnormal pharyngeal swallowing, but one may be abnormal while the other is normal. Normally, the oral phase of swallowing undergoes a smooth transition to a brisk pharyngeal phase, with vigorous transport of the swallowed bolus into and through the pharynx. In some patients, however, the pharyngeal phase is delayed, but once initiated is normal. In the presence of severe oral impairment, the pharyngeal phase may be triggered by injecting a small barium bolus directly into the pharynx through a soft tube passed into the pharynx through either the mouth or nose. Such techniques, however, are used only for examination and not for feeding.

Radiation may cause an impaired oral phase as well as a pharyngeal phase of swallowing for several reasons: (1) decreased salivation that helps initiate swallowing, (2) fibrosis with restrictive tongue or hyoid movement, (3) damage to lingual nerves, and (4) damage to tongue muscles with accompanying atrophy. Additionally, the pharyngeal phase is delayed or impaired in some radiated patients. Such delays may arrest or retard pharyngeal bolus transport or lead to aspiration.

Pharyngeal Phase of Swallowing

Abnormalities of the pharyngeal phase of swallowing are listed in Table 2. Nasal regurgitation of fluid and nasal speech may occur when the soft palate does not make a good seal against the posterior pharyngeal wall or the superior pharyngeal constrictors do not contract normally. The generic causes include impaired neurologic function, disease of striated muscle, or a structural abnormality, such as a palatal defect. In some instances, the palatal sealing appears normal when the patient is upright and regurgitation is not seen until the patient is positioned horizontally or in a slightly head-down position (Fig. 3). In some patients, nasal regurgitation can be caused

TABLE 2: Abnormalities in the Pharyngeal Phase of Swallowing

Radiologic Findings	Generic Impairment
Absent or decreased hyoid movement	Feeble contraction, suprahyoid muscle
Absent or decreased laryngeal movement	Abnormal contraction, thyrohyoid
Poor palatal movement	Impaired function of velopharngeal closure mechanism
Delayed or absent pharyngeal phase	Impaired sensation; damage to neural programming in the brainstem
Increased unilateral residual valleculae and/or piriform sinus	Unilateral impairment of pharyngeal peristalsis
Abnormal movement, epiglottis	Impaired function of thyrohyoids, cervical spur, mass
Laryngeal penetration/aspiration	Early: premature oral spill; during: impaired laryngeal closure; late: pharyngeal residue
Abnormal opening of upper esophageal sphincter (UES)	Impaired UES relaxation; decreased UES compliance; feeble hyoid movement; feeble pharyngeal transport

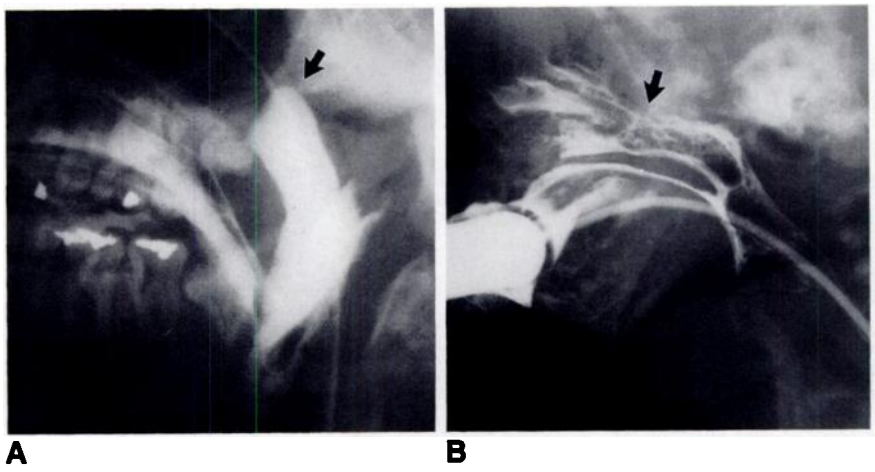

Fig. 3.—Two examples of nasal regurgitation due to feeble palatal contraction during swallowing. Regurgitation occurred only while patients were recumbent.

***A*, Elderly man several weeks after a stroke. Nasogastric tube is present. Substantial amount of barium (*arrow*) has regurgitated into nasopharynx.**

***B*, 6-month-old infant with neuromuscular dysfunction and respiratory symptoms. Oral esophageal tube is present to search for tracheoesophageal fistula. Regurgitated barium is visible in nasopharynx and nasal cavity (*arrow*).**

by disorders of pharyngeal transport and accompanying residual when swallowed material refluxes into the nasopharynx after palatal closure and reopening.

A number of factors may contribute to impaired pharyngeal bolus transport. A feeble or disorganized posterior tongue thrust will weaken the tongue driving force that is so important in initiating bolus transport into the oropharynx. Such impaired tongue movement may be caused by neurologic disease, muscle disease, fibrosis, or a surgical defect. In the hypopharynx, bolus transport is achieved largely by peristalsis of the pharyngeal constrictors. In some instances, impaired constrictor function, often neurologic in origin, may be limited to a single constrictor, for example, the middle constrictor. The bolus will lose its sharp tail and move slowly through the area of involvement. In other instances, constrictor weakness is unilateral and seen only on frontal imaging. The abnormal side bulges, compared with the normal contralateral side (Fig. 4), and retains residual swallowed material. The cardinal feature of impaired pharyngeal transport is increased residual volume, generally in the piriform sinuses but also in the valleculae (Fig. 5). With unilateral pharyngeal paresis the residual in the hypopharynx is on the ipsilateral side.

Another abnormality that may be observed during the oral and pharyngeal phases of swallowing is a diminished superoanterior excursion of the hyoid and larynx, which normally becomes maximal during the pharyngeal swallow. Diminished movement of these structures is generally related to impairment of the neural program that drives the muscles that move the structures or to tissue fibrosis (e.g., from surgery or radiation). Decreased hyoid and laryngeal movement may contribute to impaired oropharyngeal filling, impaired bolus transport, aspiration, or abnormal upper esophageal sphincter (UES) opening.

Yet another abnormality that may be observed during the pharyngeal swallowing phase is impaired movement of the epiglottis. As discussed earlier, normal epiglottic movement is a major factor in closure of the laryngeal vestibule. On lateral videofluoroscopy, bilateral impairment of thyrohyoid contraction is seen as a lack of laryngeal-hyoid approximation accompanied by absent or only partial vestibular closure. Such impairments are generally neurologic in origin. On posterolateral projection, unilateral thyrohyoid impairment causes upward epiglottic tilting (Fig. 6) to the ipsilateral side [3]. Other causes of epiglottic tilting include cervical spurs or a mass on

the ipsilateral side, such as a mass of the aryepiglottic fold that mechanically prevents normal epiglottic closure.

Perhaps the most significant abnormality sought during radiologic examination of pharyngeal function is aspiration [4]. We define aspiration as barium penetration into the laryngeal vestibule with passage below the vocal cords. The term penetration is applied to barium entry into the laryngeal vestibule without passage below the cords. Penetration alone occurs more commonly than penetration with aspiration. Often the penetrated barium is cleared during the course of the same swallow as the laryngeal elevation increases (Fig. 7) or during a subsequent cough. Penetration occurs most commonly with swallows of thin barium and may be seen as a "finger" of barium extending into the vestibule. On posteroanterior views, penetration is seen as a thin vertical line of barium in the midline (Fig. 4). Severe penetrations are often

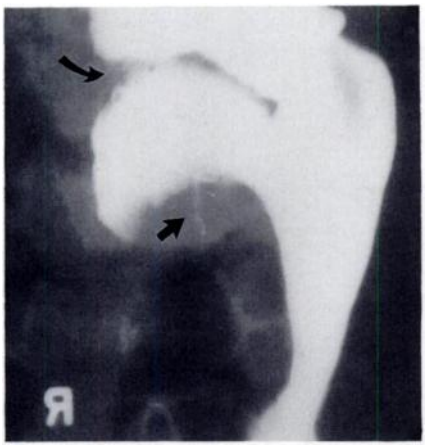

Fig. 4.—Posteroanterior view. Bulging of left pharynx in patient with left-sided paralysis of pharyngeal peristalsis. All of swallowed barium flowed down left pharynx and none through right pharynx. Trace amount of penetration into laryngeal vestibule is seen in midline (*straight arrow*). Slightly oblique defect is caused by epiglottis (*curved arrow*). When patient's head was turned to right, swallowed barium flowed mainly down right side of pharynx and vestibular penetration did not occur.

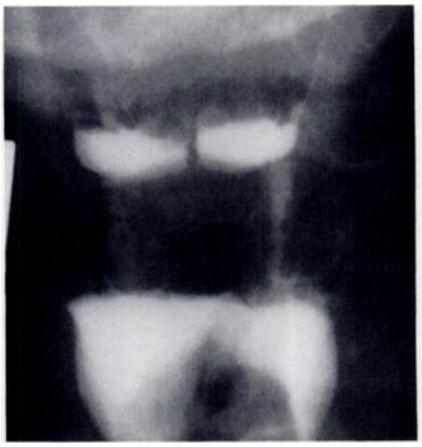

Fig. 5.—Posteroanterior view. Substantial barium residue in valleculae and piriform sinuses after 10-ml barium swallow in a patient in whom pharyngeal peristalsis was absent after a stroke.

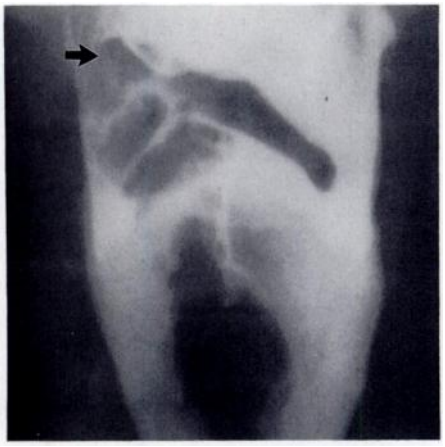

Fig. 6.—Example of epiglottic tilt during pharyngeal phase of 10-ml barium swallow in 45-year-old man with amyotonia dystrophy. Free margin of epiglottis is tilted upward on right (*arrow*). Gas bubble is trapped beneath right side of epiglottis. Barium is seen to stream down side channels of pharynx. Epiglottic tilt in this patient is attributed to impaired contraction of right thyrohyoid muscle. There was no evidence of a cervical spur or pharyngeal mass.

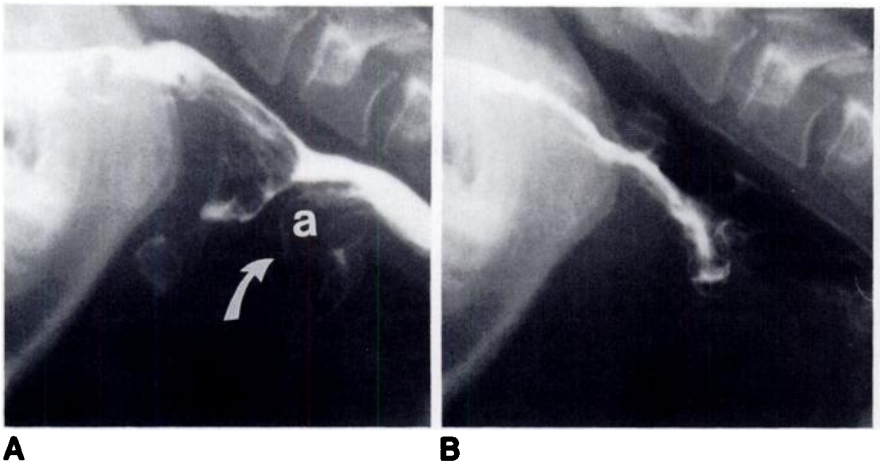

Fig. 7.—Laryngeal penetration during swallowing of 5-ml barium bolus by patient who had undergone cranial surgery and radiation for craniopharyngioma.

***A*, During pharyngeal phase of swallowing, thin finger of barium (*arrow*) has penetrated laryngeal vestibule. Epiglottis is bowed over arytenoids (a); its free margin has flipped caudad.**

***B*, By completion of swallowing sequence, most of penetrated barium has been "kicked out" of laryngeal vestibule, which is now open, and only a thin coating of barium remains to be seen.**

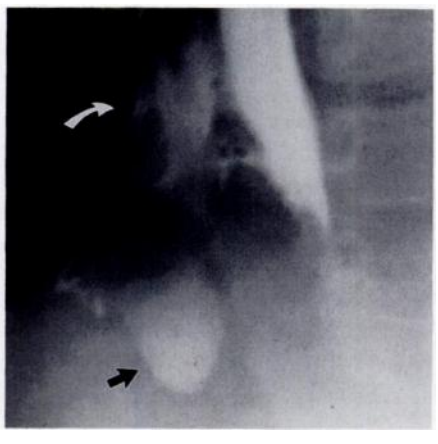

Fig. 8.—Tracheal aspiration in patient with dermatomyositis. Barium is seen in laryngeal vestibule (*curved arrow*) and also in trachea (*straight arrow*). Pharyngeal bolus transport was impaired because of weakness of pharyngeal constrictors. High residuals of barium remained in pharynx and piriform sinuses. Aspiration occurred both during and after a swallow.

accompanied by aspiration and are seen as a substantial extension of barium on the lateral view (Fig. 8) and as a thick vertical line, or wedge, on the posteroanterior view (Fig. 9B). On the posteroanterior view, the inner margin of the aryepiglottic folds often becomes well coated with barium (Fig. 9A). On the lateral view the arytenoids may remain vertical and split the barium column (Fig. 10), with some barium going into the larynx and the remainder filling the hypopharynx or crossing the UES [5]. In such cases, there is severe impairment of vocal cord closure. We believe that mild penetration is generally caused by a feeble or dyssynergic contraction of one or both of the thyrohyoid muscles, thereby showing laryngeal elevation. Aspiration has several general causes and may occur (1) early, before the pharyngeal swallow; (2) during the pharyngeal swallow; or (3) late, after the pharyngeal swallow. Early aspiration is caused by premature spill of fluid from the mouth and is related to impaired lingual or lingual-palatal function. Aspiration during a swallow is usually related to impaired function of the intrinsic laryngeal muscles, generally from a neural cause or surgical ablation. Late aspiration is generally related to increased pharyngeal residual of any cause, for example, impaired bolus transport; abnormal UES opening; or, occasionally, esophagopharyngeal regurgitation. Other types of conditions in the differential diagnosis of aspiration include a cleft epiglottis or tracheoesophageal fistula. This last condition may simulate aspiration when barium abruptly appears in the upper trachea and larynx and is coughed through the vestibule into the pharynx. The key point with a tracheoesophageal fistula is that the barium does not enter the trachea via the larynx, but rather enters the upper trachea from below. The therapy for aspiration depends on its generic type. Some patients exhibit more than one type and cause of aspiration.

Incomplete UES opening during the pharyngeal phase of

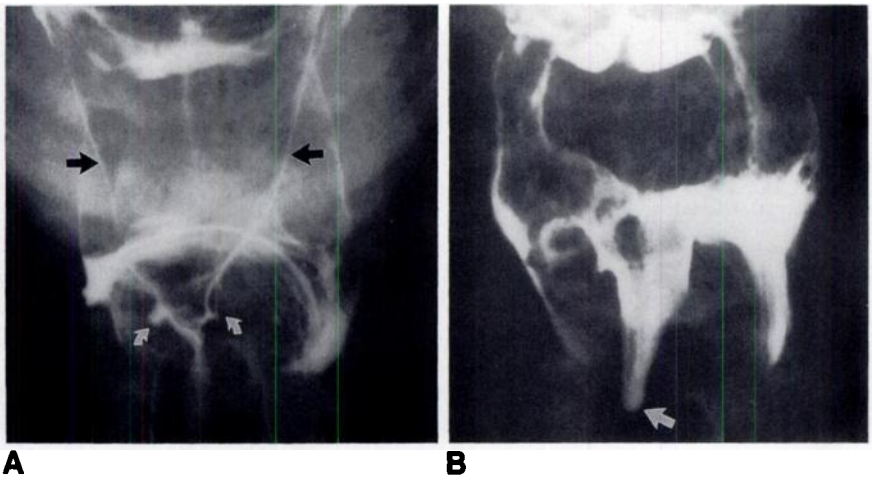

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Fig. 9.—Posteroanterior views. Two examples of severe aspiration.

A, Elderly woman after a stroke. Laryngeal vestibule and glottis are widely patent. Barium outlines aryepiglottic folds (*straight arrows*) and laryngeal ventricles (*curved arrows*). Barium is also seen in trachea.

B, Elderly man with nodular carcinoma in right piriform sinus that involves base of right aryepiglottic fold. Large vertical wedge of barium fills laryngeal ventricle and extends into trachea (*arrow*).

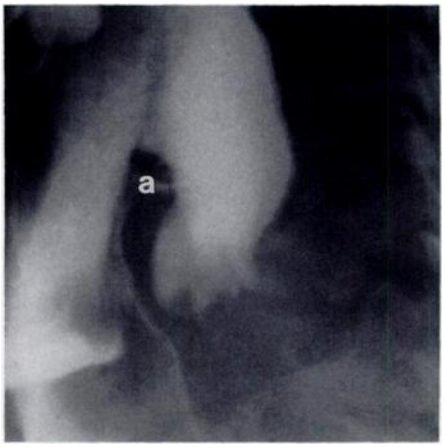
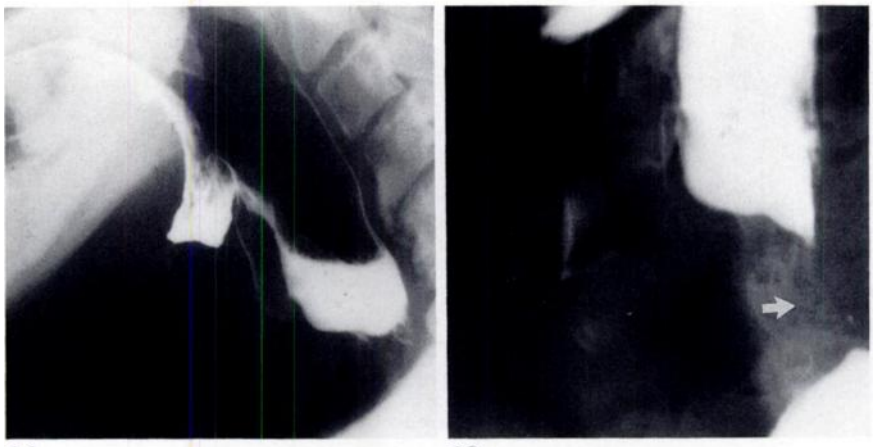


Fig. 10.—Severe aspiration. Elderly senile man who exhibited absent pharyngeal peristalsis, aspiration, and a large amount of residual barium in pharynx. Upper esophageal sphincter did not open normally. Arytenoid (a) cartilages remained vertical and split barium column.

Fig. 11.—Failed opening of upper esophageal sphincter (UES) in 51-year-old man with abrupt onset of difficulty in swallowing. Patient was hypertensive but without CT or clinical evidence of stroke. Radiologic examination showed absent pharyngeal peristalsis and failed UES opening. Oral phase of swallowing was normal. Patient did not aspirate but held his breath and coughed residual barium out of his pharynx. A substantial residue of barium is present in valleculae and piriform sinuses. On manometry, UES did not relax with swallowing. During a 2-month period, pharyngeal swallows, peristalsis, and UES relaxation returned to normal. This patient's condition was attributed to lacunar infarcts of brainstem.

Fig. 12.—Abnormal opening of upper esophageal sphincter (UES) in elderly man with oculopharyngeal dysphagia. Radiologic examination demonstrated normal oral phase of swallowing with feeble pharyngeal contractions, high residual in pharynx, and aspiration. On some swallows, UES opened partially or, as in this example, not at all (*arrow*); however, UES relaxed normally on manometry.



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swallowing may impair pharyngeal bolus transport, lead to excessive pharyngeal residual, and cause late aspiration. Impaired UES opening is caused by an abnormality in one or more of the four factors associated with normal sphincter opening.

Failure of UES relaxation is most commonly caused by neurologic disease, such as stroke or head trauma, and is generally accompanied by abnormalities in pharyngeal bolus transport (Fig. 11). Thus, impaired UES (cricopharyngeus) relaxation rarely occurs as an isolated abnormality. Intraluminal manometry is the only satisfactory clinical laboratory method for validating impaired UES relaxation [6, 7]. Simply observing absent or incomplete UES (cricopharyngeal) opening radiologically does not establish impaired sphincter relaxation (Fig. 12). Some patients exhibit a pronounced cricopharyngeal indentation (Fig. 13) that has often been judged, simply on the basis of its radiologic appearance, to represent isolated impaired UES relaxation; this has been termed cricopharyngeal achalasia [8, 9]. In our experience, such patients generally exhibit normal UES relaxation on manometry and the underlying abnormality is decreased compliance of the relaxed UES. Decreased stretchability of the relaxed cricopharyngeus is probably caused by fibrosis. A subset of patients with Zenker diverticulum exhibit the same abnormality. Feeble anterior movement of the hyoid may contribute to incomplete UES opening but seldom occurs as an isolated finding. The most common cause of incomplete UES opening seems to be decreased intrabolus pressure related to impaired bolus transport through the pharynx (Fig. 12). The underlying cause may be neurologic or muscular.

A useful concept has been suggested for evaluating and understanding abnormalities of the oral and pharyngeal phases of swallowing. This concept stresses the relationship between specific functional impairments, adaptive compensation, and decompensation [10]. For example, glossopalatal closure and tongue thrust control fluid flow from the mouth to the pharynx. In some patients, weakness of the posterior tongue may be compensated for by increased downward movement of the soft palate. Decompensation consists of premature escape of fluid from the mouth (Fig. 1), often associated with aspiration. Weakness of the soft palate may be associated with prominence of Passavant ridge as a compensation to seal off the nasopharynx. This compensation seems especially common in children. Decompensation consists of nasal regurgitation during swallowing (Fig. 3). A third example is weakness of the intrinsic laryngeal muscles. Compensation against aspiration may be achieved by flexion of the head, which tucks the larynx beneath the epiglottis. Decompensation consists of aspiration.

Radiation therapy is believed to contribute to abnormalities in the pharyngeal phase of swallowing. Such functional abnormalities include pharyngeal paresis, delayed pharyngeal clearance, epiglottic dysfunction, and aspiration [11].

As stressed earlier, the examination for cervical dysphagia is incomplete without a careful examination of esophageal function and motility. Findings of significance may include esophageal motor dysfunction, esophageal stricture, esophageal neoplasm, reflux esophagitis, and reproducible gastroesophageal reflux. Morphologic abnormalities in the pharynx or proximal esophagus include tumor, circular webs (Fig. 14), or Zenker diverticulum (Fig. 15). Generally, small incomplete webs or transient pharyngeal pouches are of no clinical significance. Recently, however, Curtis et al. [12] reported that pharyngeal pouches that appear late and persist late into the swallowing sequence are associated with cervical dysphagia (Fig. 16), whereas pharyngeal pouches in asymptomatic subjects appear and disappear earlier in the sequence. Persistent pharyngeal diverticula are commonly associated with clinical symptoms.

During the past decade numerous claims have emerged that the symptoms of globus or cervical dysphagia are associated with gastroesophageal reflux disease. Many of these claims have been based on the notion that gastroesophageal acid may cause spasm or dysfunction of the UES. In our experience, however, we have been unable to document that intraesophageal infusion of acid or spontaneous episodes of acid gastroesophageal reflux cause any alteration of normal UES function [13]. Nevertheless, in occasional patients with reflux disease, regurgitated acid may cause pharyngitis or aspiration laryngitis. To our knowledge, however, acid regur-

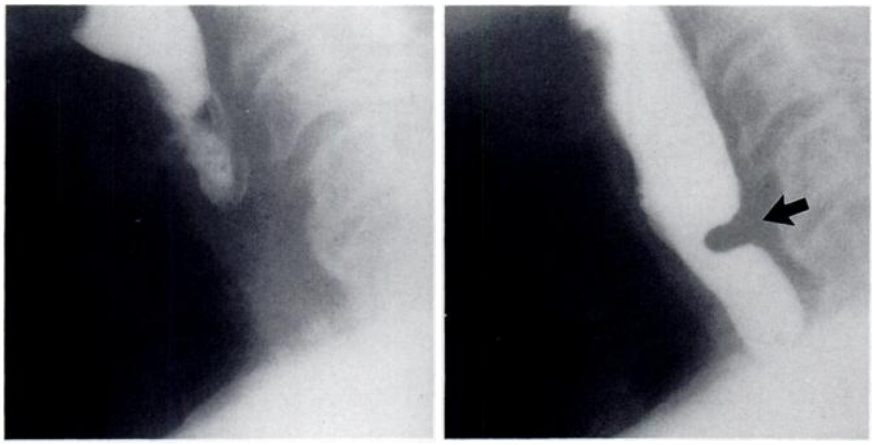

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Fig. 13.—Prominent cricopharyngeal (CP) bar in elderly patient with mild cervical dysphagia. Swallows of 10-ml barium bolus.

***A*, Head of barium bolus is just reaching upper margin of upper esophageal sphincter (UES).**

***B*, Prominent restrictive CP bar (*arrow*) is seen at level of UES. This bar persisted throughout entire interval of barium flow into cervical esophagus and narrowed aperture of open UES did not change in dimension. On pharyngeal manometry, pharyngeal peristalsis and UES relaxation were normal. We attribute prominent CP bar to reduced compliance, or stiffness, of CP muscle, even when relaxed [7]. Reduction in CP compliance is probably related mainly to idiopathic fibrosis or muscle contracture.**

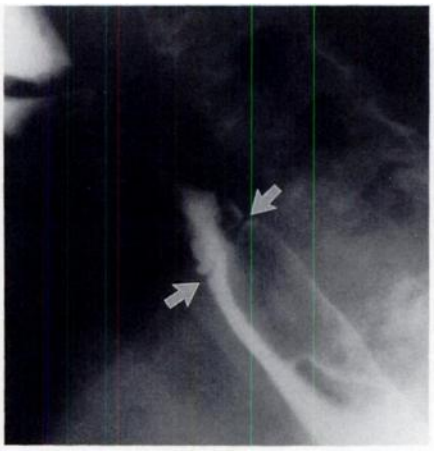


Fig. 14.—Circumferential web (*arrows*) in cervical esophagus in a patient with dysphagia for solids. Half of a marshmallow was caught at web and produced clinical symptoms.

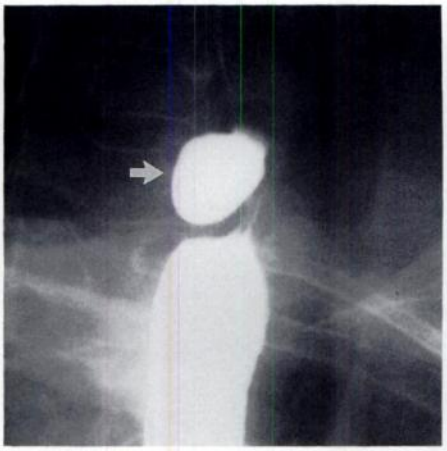


Fig. 15.—Lateral oblique view of Zenker diverticulum (*arrow*) in elderly man with cervical dysphagia. Pharyngeal peristalsis and clearance were normal.

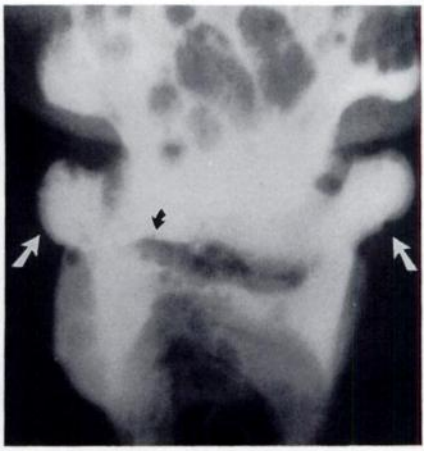


Fig. 16.—Bilateral pharyngeal pouches (*straight arrows*) in patient with unexplained cervical dysphagia. Pouches appeared during middle of pharyngeal swallow and lasted until late in pharyngeal swallow. Curtis et al. [12] suggest this timing is abnormal. Also noted is slight tilting of epiglottis (*curved arrow*), caused by cervical osteophyte. All other components of swallowing appeared normal.

gitation is rarely associated with any overt change in the oral-pharyngeal function detectable on videofluoroscopy, or pharyngeal manometry. For this reason, we believe that although a relationship may exist between acid gastroesophageal reflux disease and cervical symptoms in some patients, this association is overstated and poorly substantiated. In fact, only a minority of patients with acid reflux disease have acid reflux into the proximal esophagus and regurgitation into the pharynx. Therefore, the finding of a hiatal hernia or isolated episodes of gastroesophageal reflux on radiologic examination do not establish a reflux cause for globus or cervical dysphagia.

Value of Radiologic Examination

Thorough radiologic evaluation of each component of the oral and pharyngeal phases of swallowing leads to two beneficial circumstances: (1) the judgment that swallowing function is normal is a highly accurate one, rather than signifying that abnormal function simply has not been noticed and (2) an accurate complete description is obtained of the normal and abnormal features of oral or pharyngeal swallowing. Such descriptions are essential even when a specific cause is not apparent. For example, a given patient may exhibit impaired tongue movement with difficulty forming a bolus and a delay in initiating swallowing while pharyngeal function is entirely normal. Another patient may exhibit normal oral and pharyngeal function in the presence of weak palatal function and nasal regurgitation. Virtually unlimited combinations of impaired and normal function of the different components of swallowing may be observed in individual patients. This phenomenon is especially true in patients who have suffered a sudden neurologic event such as a stroke or head injury.

Accurate description of swallow function forms the basis for patient management and the design of therapy, where appropriate. A partial list of possible causes of impaired oral or pharyngeal swallowing is given in Table 3. It must be emphasized, however, that an accurate description of the constellation of impairments during oral and pharyngeal swallowing often does not lead to a specific diagnosis. Nevertheless, the radiologic diagnosis provides critical information relative to the patient's swallowing function and management. A specific diagnosis is generally provided mainly by the clinical history alone, or the clinical history in combination with the radiologic findings.

The most common cause of neurologic dysfunction is cerebral vascular disease. Commonly, abnormalities of swallowing related to stroke are reversible over weeks to months. Primary muscle disorders tend to affect the pharyngeal phase of swallowing more than the oral phase, but this pattern is variable and nonspecific. Deteriorating swallow function with repetitive swallows suggests a diagnosis of myasthenia gravis. Such changes may be reversed with IV administration of edrophonium (Tensilon), which should be given with caution.

In some instances, pharmacologic therapeutic agents may cause abnormalities in swallowing. For example, many drugs, such as thorazine, have an anticholinergic effect that impairs salivation and thereby may lead to difficulty in initiating swallows. Other drugs, such as metoclopramide, may cause rhythmic protrusion of the tongue. For these reasons, examination of swallowing should include a comprehensive clinical history of drug therapy.

Neoplasms in the mouth or pharynx may cause abnormalities in swallowing function. For example, a neoplasm in the anterior or middle tongue may impair bolus preparation or oral clearance, and a neoplasm of the tongue base may compromise delivery of the oral bolus into the pharynx or

cause premature spill into the oropharynx (Fig. 17). A lesion of the pharyngeal wall may impair pharyngeal bolus transport and lead to an increased residual in the pharynx. As discussed earlier, a tumor of the aryepiglottic fold or medial wall of the piriform sinus may cause tilting of the epiglottis during the pharyngeal swallow [3]. Such lesions may also lead to aspiration (Fig. 18).

The effect of aging on swallowing is controversial. Although aging per se probably causes subtle quantitative changes in the oral and pharyngeal phases of swallowing, such changes remain to be fully elucidated and measured.

TABLE 3: Causes of Abnormal Oral or Pharyngeal Swallowing

Neurologic disease
Cerebrovascular
Posttraumatic
Degenerative (e.g., Alzheimer)
Parkinsonism
Amyotrophic lateral sclerosis/motor neuron disease
CNS neoplasm
Bulbar poliomyelitis
Pseudobulbar palsy
Friedreich spastic ataxia
Familial dysautonomia
Postoperative CNS tumor
Muscle disease
Muscular dystrophy
Oculopharyngeal dystrophy
Myotonic dystrophy
Dermatomyositis
Myasthenia gravis
Radiation injury
Gastroesophageal reflux disease?
Pharmacologic agents (e.g., atropine, thorazine)
Malignancy
Tongue
Pharynx
Larynx
Postoperative
Tongue
Pharynx
Larynx

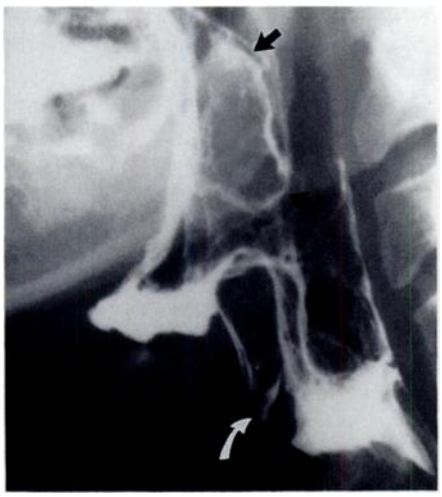
Postoperative Studies

As with the general examination for patients with a suspected abnormality of oral or pharyngeal swallowing, the radiologic examination in patients who have undergone surgery for oral, pharyngeal, or laryngeal malignancies involves a careful evaluation of morphology [14] as well as function [15]. Such surgeries include resection for primary malignancies of the tongue, pharynx, and larynx. In many cases, the patient may also undergo a radical lateral neck dissection and/or subsequent irradiation.

The aim of surgical resection is to include a 1-cm margin free of tumor. Therefore, even a small tongue lesion of 2 cm would require resection of a 4-cm block of tissue, probably with a primary closure. Such surgery might not be expected by the surgeon to cause a severe abnormality with swallowing. However, difficulty may be caused by loss of tongue mass or restrictive tongue movement because of fibrosis [16]. Loss of normal tongue movement often leads to difficulty shaping a bolus, difficulty initiating swallowing, and increased residual in the mouth following the oral swallow. The pharyngeal phase of swallowing is generally normal.

Tumors involving the alveolar ridge, tongue base, tonsil, lateral pharyngeal wall, or palate require wider resection than simple tumors confined to the tongue. The large defect may be dealt with by primary closure or a skin flap. These patients may have a substantial tissue defect, abnormal pharyngeal bolus transport, nasal regurgitation, or aspiration [17].

Carcinoma of the larynx may be focal, for example, limited to a vocal cord or aryepiglottic fold. However, such carcinomas may extend to multiple sites, such as both supraglottic and glottic areas or the subglottic region or even beyond the glottis into the piriform sinus. The type of treatment is determined by the extent of the tumor. Lesions limited to one vocal fold are commonly treated by radiation alone. Types of surgical procedure include a hemilaryngectomy, supraglottic laryngectomy, and total laryngectomy with tracheostomy. Hemilaryngectomies are seldom performed. With a supraglottic laryngectomy, the hyoid and epiglottis and aryepiglottic folds are removed [17, 18]. On lateral view, the vocal cords and infraglottic larynx are pulled up to the base of the tongue.



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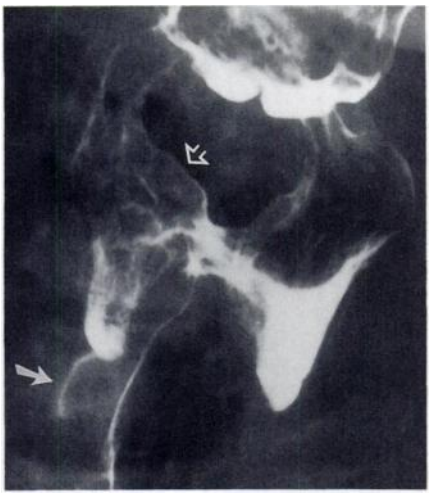

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Fig. 17.—Bulky carcinoma (*straight arrows*) involving right posterior tongue and right pharyngeal wall. Tumor caused premature spill of barium from mouth, impaired posterior tongue thrust, impaired pharyngeal bolus transport, and increased residual barium in the valleculae and piriform sinuses and barium penetration into laryngeal vestibule (*curved arrow*).

Fig. 18.—Posteroanterior view of nodular carcinoma originating in right piriform sinus. Carcinoma causes constriction of right piriform sinus (*solid arrow*), invades right side of glottis, and causes flat mass on aryepiglottic fold (*open arrow*). During swallowing, barium was aspirated into trachea.

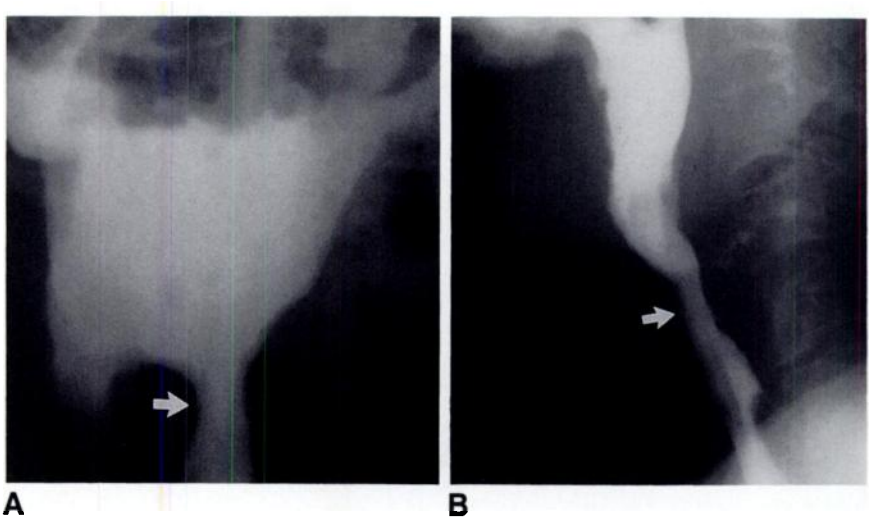

Fig. 19.—Patient after partial pharyngectomy and total laryngectomy for carcinoma of larynx and left piriform sinus. Hyoid and larynx are absent.

***A*, Anteroposterior view. Left piriform sinus is absent. Stricturelike narrowing (*arrow*) exists in region of upper esophageal sphincter (UES).**

***B*, Lateral view. Stricture seen at level of UES (*arrow*). Also seen is substantial regurgitation of barium into nasopharynx.**

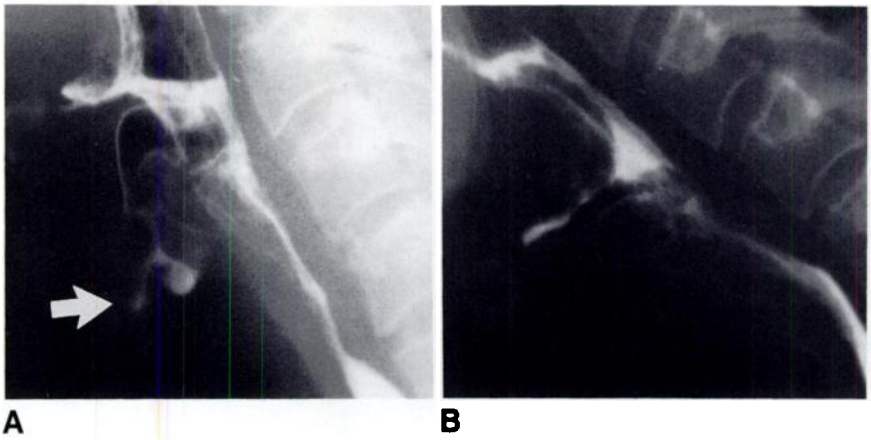

Fig. 20.—Value of head flexion in prevention of aspiration.

***A*, Head in neutral position. With barium swallow, barium is seen to have been aspirated below glottis (*arrow*). Aspiration also occurred with thick barium and barium paste.**

***B*, Head in flexed position. During this swallow, barium penetrated laryngeal vestibule, but aspiration did not occur. On other swallows with head flexed, there was no vestibular penetration. Vestibular penetration did not occur with thick barium or barium paste.**

Although closure of the cords alone is generally sufficient to prevent aspiration during the swallow, some aspiration of pharyngeal residue may occur after the swallow when the vocal folds open for respiration. Oral swallowing and pharyngeal bolus transport are generally normal. With total laryngectomy, an effort is made to reconstruct an upper esophageal sphincter by attaching the free ends of the cricopharyngeus or filling in the gap of the resected cricoid with soft tissue. Therefore, some narrowing or stricture of the reconstructed UES may be present (Fig. 19) with increased residual in the pharynx, or the patient may have a pseudoepiglottis at the tongue base [17]. The oral phase of swallowing is usually normal. In some patients, laryngectomy is accompanied by a cricopharyngeal myotomy.

Evaluation of Therapeutic Maneuvers

Videofluorography is not only a useful method for the diagnostic evaluation of oral-pharyngeal swallowing disorders but can be used to search for a "safe swallow" without aspiration accompanied by satisfactory pharyngeal bolus transport [4]. Therapeutic maneuvers include (1) manipulations of bolus variables such as volume and consistency, (2) variation of head position, and (3) use of certain specific therapeutic maneuvers such as supraglottic swallow [19]. Some patients exhibit aspiration with a small barium bolus (e.g., 2 ml), while others require a larger bolus (e.g., 10 ml). For this reason, at least several volumes should be used during the diagnostic examination. Examination with several bolus consistencies is critical. In some patients, aspiration occurs most commonly with thin barium (e.g., 30% wt/vol), but may not occur at all with thick barium or paste material (e.g., barium paste, applesauce, or pudding). Head position can be very important. Head flexion often prevents aspiration by reducing premature spill from the mouth and also by tucking the larynx anteriorly beneath the epiglottis (Fig. 20). Tilting the head backward can enhance oral transit. Turning of the head directly to the contralateral side may compensate for unilateral pharyngeal weakness (Fig. 4). Bending the head laterally directs swallowed liquid to the ipsilateral side and may also be useful therapeutically to enhance pharyngeal bolus transport and prevent aspiration.

Two special maneuvers that may be evaluated during videofluoroscopy include the supraglottic swallow and the Mendelsohn maneuver. The supraglottic swallow is done for patients with aspiration before or during a pharyngeal swallow.

The subject is instructed to take a deep breath and then swallow during breath-holding. A cough immediately following the swallow clears the larynx of aspirated material. The Mendelsohn maneuver is done to facilitate UES opening and, therefore, enhance pharyngeal emptying and reduce aspiration. For the Mendelsohn maneuver, the subject is instructed to produce a sustained hyoid-laryngeal elevation during a swallow. Placing a finger on the subject's hyoid or having the patient look at the video monitor may help in the completion of this maneuver. This maneuver puts sustained traction on the UES and prolongs its opening, thereby facilitating sphincter opening and pharyngeal emptying.

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