Oral and Pharyngeal Function and Dysfunction

Olle Ekberg

Contents

O. Ekberg
Diagnostic Centre of Imaging and Functional Medicine, Skåne University Hospital,
205 02 Malmö, Sweden
e-mail: olle.ekberg@med.lu.se

Abstract

Normal pharyngeal swallow is coordinated in a precise and exact manner. It is controlled from a swallowing center in the brain stem. Normal swallowing is adjusted to bolus volume temperature and viscosity. Abnormal pharyngeal swallow may lead to misdirected swallowing that challenges the airways. Inefficient transportation into the esophagus, stomach, and bowel may lead to dehydration and malnutrition.

1 Introduction

Normal pharyngeal swallow is precisely scheduled and symmetric. It is finely tuned and coordinated in a precise and exact manner to establish a safe swallow (Miller 1986; Dodds 1989). The swallowing process is regulated by a command center in the brain stem, a central program generator which receives input from the cerebral cortex and peripheral muscle and directs the sequence of swallowing. This process is both voluntary and involuntary and incorporates motor activity from the oral cavity, pharynx, and esophagus. It involves both motor and sensory activity. There is an evolving amount of knowledge concerning normal and abnormal swallowing (Jones and Donner 1991; Ekberg and Wahlgren 1985; Hannig and Hannig 1987; Brühlmann 1985; Pokieser et al. 1995; Dodds 1989).

Med Radiol Diagn Imaging (2017)
DOI 10.1007/174_2017_60, © Springer International Publishing AG
Published Online: 09 August 2017

2 The Normal Swallow

2.1 The Oral Stage

The oral, pharyngeal, pharyngoesophageal segment (PES), and esophageal stages of swallowing are readily appreciated radiographically (Fig. 1). The oral stage of swallowing is bolus-specific, i.e., the patient handles different boluses differently, i.e., a strawberry is handled differently from a cup of tea. Therefore, the oral stage is notoriously more difficult to evaluate radiologically than the rest of the swallowing apparatus. However, the oral stage should be included in the radiologic evaluation. The recording should start with the ingestion. During oral processing there is superior and inferior and some anterior–posterior movement of the hyoid bone. However, liquid barium should not be processed or modified in the oral cavity. Therefore, oral preparation for swallowing is tested with a solid or semisolid bolus. When the ingested material is ready to be swallowed, the material is brought onto the back of the tongue, which obtains the shape of a groove (Hamlet et al. 1988). This is the preparatory position for swallowing.

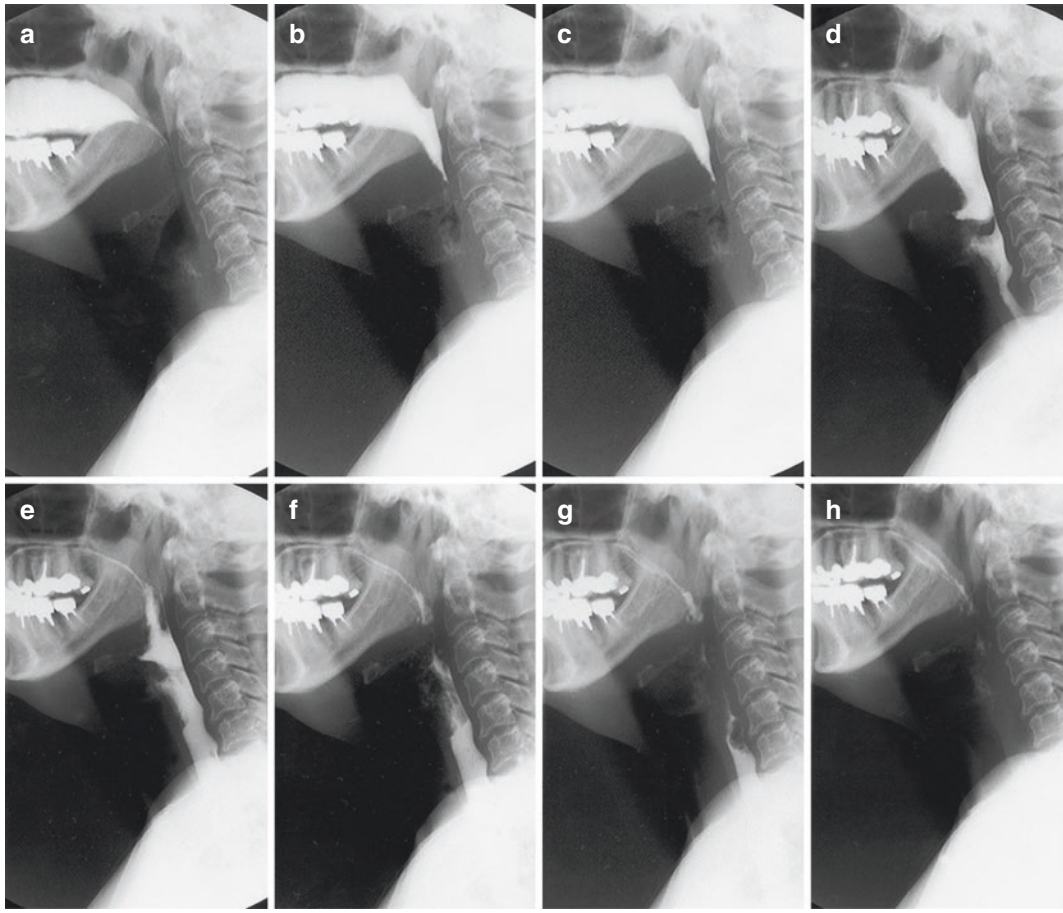

Fig. 1 The oral, pharyngeal, and pharyngoesophageal segment (PES) stages are readily appreciated radiographically. This is a sequence of a barium swallow (**a**–**h**) in lateral projection. The bolus is gathered in the oral cavity (**a**–**c**) and is propelled into the pharynx by an upward and backward movement of the back of the tongue (**d**). The tilting down to a horizontal position of the epiglottis is seen in (**d**). The airways are closed and none of the barium reaches into the laryngeal vestibule or trachea. The PES opens. The upward and forward movements of the larynx including the hyoid superiorly and the PES inferiorly are extremely important for the normal execution of pharyngeal transit. There are no, or only minimal, remnants of barium in the pharynx after swallowing

No part of the bolus is allowed to leak anteriorly from the mouth through the lips. Even more important radiologically is to observe if posterior leak occurs. The patient should be able to control the sealing of the tongue-base to the soft palate and posterior pharyngeal wall.

2.2 The Pharyngeal Stage

The pharyngeal swallow is initiated voluntarily and the material to be swallowed is usually called a bolus from this point. Initiation of the pharyngeal swallow coincides with the beginning of the anterior movement of the hyoid bone from an elevated position. Pharyngeal constriction is probably cued by the bolus interfering with sensory innervation at the faucial isthmus. Radiologically it is convenient to use the beginning of the anterior hyoid movement as the starting point of pharyngeal swallow. The tongue then propels the bolus posteriorly into the pharynx and further down into the PES and cervical esophagus. If the pharyngeal constrictor wall has normal compliance, only minor dilatation of the pharynx occurs (Fig. 2).

The palatopharyngeal isthmus is closed by elevation of the muscular palate and constrictor convergence, which is most medial of the lateral walls. Normally, no regurgitation of barium into the nasopharynx occurs.

In a patient with severe oral impairment, the pharyngeal phase may be elicited by injecting a small barium bolus directly into the pharynx through a soft tube. This may be placed into the pharynx via either the mouth or the nose. Such techniques, however, are used only for examination and not for feeding. Patients with uncoordinated, weak, or jerky tongue movements commonly cannot correctly position the bolus on the tongue. Accordingly, the tongue cannot displace the bolus posteriorly. There is a strong correlation between an abnormal anterior movement of the hyoid bone and overall abnormal oral and pharyngeal function, as well as defective opening of the PES.

Protection of the airways occurs at four separable anatomically and functionally different sites, i.e., the vocal folds, the supraglottic portion of the laryngeal vestibule, the subepiglottic portion of the laryngeal vestibule, and the epiglottis (Curtis and Hudson 1983; Curtis and Sepulveda 1983; Ekberg 1982). The most crucial of these levels is the supraglottic portion of the laryngeal vestibule. Barium in the vestibule commonly extends into the trachea, as the vocal folds offer poor protection of the lower airway (Ekberg and Hilderfors 1985). Radiographic observation of

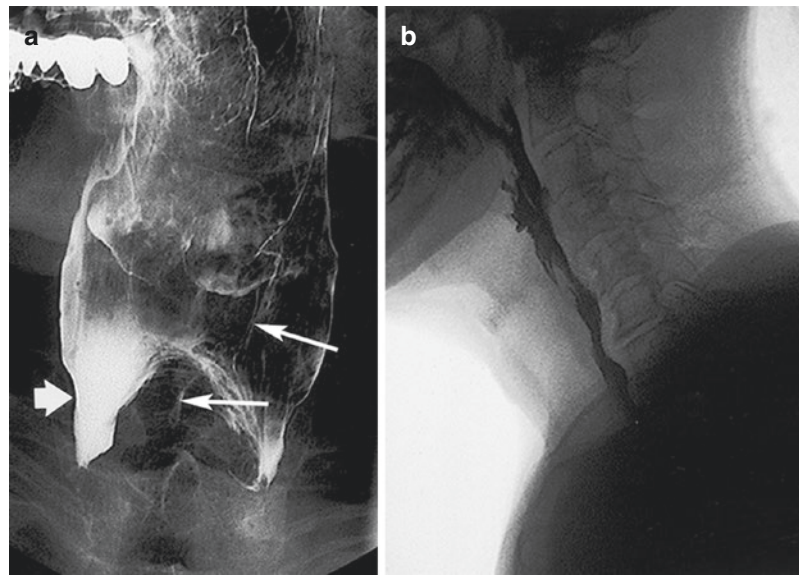

Fig. 2 An 80-year-old man with cerebrovascular disease. There is paresis in the right side of the pharynx. This is not seen in lateral projection (**b**) and is only seen in frontal projection (**a**). There is pooling of contrast medium in the right piriform sinus (*broad arrow*). Small amounts of barium are also seen coating the inside of the laryngeal vestibule down to the false vocal cords

barium penetration into the larynx and trachea is strategic in dysphagia evaluation. Bedside evaluation for aspiration has a low sensitivity. This is partly because many of these patients have sensory impairment in the larynx and/or trachea and fail to cough (Splaingard et al. 1988).

Of even more fundamental importance, and basically a prerequisite for airway closure and constrictor activity, is the elevation of the pharynx and larynx. The airways are also protected by a movement of the thyroid cartilage toward the hyoid and by closure of the laryngeal vestibule. Additional protection is offered by the epiglottis and the vocal folds. Closure of the larynx starts at the vocal folds and progresses in a superior direction in a peristaltic-like manner.

The constrictors have a minor role in the conveyance of a bolus through the pharynx. The tongue-base pressure does not differ significantly between those individuals with and those individuals without retention (Olsson et al. 1997). This finding is important because many research groups advocate normal tongue-base and constrictor activity as a prerequisite for a successful outcome of cricopharyngeal myotomy (Buchholz 1995). Tongue-base pressure has been appreciated as the driving force of the bolus, and the tongue-base has been associated with a compensatory function, namely, overcoming weak pharyngeal constrictors by increasing tongue-base activity. This tongue driving force propels the bolus and the pharyngeal constrictors stabilize the pharyngeal tube; the tongue then closes the lumen behind the bolus to prevent retrograde escape (McConnel et al. 1988; Cerenko et al. 1989; McConnel 1988).

In patients with pharyngeal retention, the PES does not open as much as in patients without such retention. This reduced opening of the PES is not associated with an increased intrabolus pressure, but is seen together with decreased laryngeal elevation. This is in agreement with a study in which traction on the PES was found to be the main force leading to the PES opening (Hsieh et al. 1995). The suprahyoid muscles are therefore much more important than other muscles in the pharynx. Also, tongue-base pressure, which is reflected by the intrabolus pressure, was the same in patients with and in patients without retention. Thus, pharyngeal shortening could be the most important mechanism in pharyngeal bolus transport (Ergun et al. 1993a, b).

Such intrabolus pressure was shown to be the same for the two groups in this study.

The pharyngeal phase of swallowing is complex because of the intricate anatomic relationships and the close temporal activation of the more than two dozen muscles that are required to function together to effectively transport the bolus from the mouth to the esophagus. In a classic electromyographic study by Doty and Bosma (1956), temporal activation of the muscles of deglutition was shown. Activation begins in the mylohyoid muscle, and the muscle action is then propelled inferiorly. Early events during the pharyngeal phase of swallowing include activation and sealing of the nasopharynx and contraction of the mylohyoid, hyoglossus, and geniohyoid muscles (i.e., the suprahyoid pharyngeal shorteners). A number of other muscles also contract early, namely, those that effect airway protection, such as the intrinsic and extrinsic muscles of the larynx. Contraction of the superior pharyngeal constrictor, styloglossus, palatoglossus, pterygopharyngeal, palatopharyngeal, stylopharyngeal, salpingopharyngeal, stylohyoid, and posterior digastric muscles then occurs. In terms of bolus transportation, this early stage represents the conveyance of the bolus from the oral cavity into the pharynx. This activity is often described as being achieved by the tongue thrust, although a multitude of muscles are involved (McConnel et al. 1988; Cerenko et al. 1989; McConnel 1988). The pharyngeal constrictors give only stability to the gullet—they do not contribute to bolus transportation in other ways (Dodds 1989). In contrast, the late part of the pharyngeal stage consists of contraction of the thyrohyoid, sternohyoid, sternothyroid, and omohyoid muscles (i.e., the strap muscles), and also the middle and inferior pharyngeal constrictors. This latter activity is thought to clear the bolus from the pharynx. The late pharyngeal constrictor activity for clearance of the pharynx is seen as a contracting wave traversing inferiorly from the superior pharyngeal constrictor level. This wave is usually best appreciated on

the anteroposterior view as an inverted V-shape to the tail of liquid or semisolid bolus (Dodds 1989). It is impairment of such clearance that is recognized as retention in the valleculae, piriform sinus, or both.

The exact reason why laryngeal elevation leads to retention in patients with manometrically normal pharyngeal constrictor activity is not clear. It is possible that reduced elevation is caused by impaired function of the suprahyoid muscles and other elevators. Such impaired function might lead to abnormal compliance of the wall, against which the constrictors may then act. As retention was not seen in a location immediately cranial to the cricopharyngeal muscle, it is more likely that a defective opening reflects a more profound and widespread dysfunction in the pharynx. Defective opening may be the sole result of impaired elevation of the pharynx.

2.3 The Pharyngoesophageal Segment

The PES is the transition between the pharynx and the esophagus. Between swallows it is kept closed as a sphincter of circular striated muscles. Anatomically it consists of the cricopharyngeal muscle and the inferior portion of the pharyngeal constrictors and the superior portion of the cervical esophagus. The length of the segment is about 3–4 cm, where the cricopharyngeal muscle makes up 1–1.5 cm. Closure is the effect of muscle tonicity and the pressure from surrounding tissues (Kahrilas et al. 1988). Normal opening, which is crucial for bolus transport, is achieved by relaxation of muscle tone. However, this probably only accounts for 10–20% of the total sphincter tone. More important is the movement of the PES superiorly and anteriorly together with the larynx and the hyoid bone. In addition, intrabolus pressure created mainly by the tongue-base and constrictor activity also helps to open the PES.

2.4 The Esophageal Stage

This is comprehensively presented in Sect. 5.1.

3 The Abnormal Swallow

In terms of what abnormalities can be expected on the four different anatomic levels, a rule of thumb is that dysfunction is by far the principal abnormality in the oral cavity and pharynx. In the PES, dysfunction and structural abnormalities may coexist. In the esophagus, structural abnormalities predominate.

3.1 The Oral Stage

Leaking of barium anteriorly through the lips, laterally into the buccal pouches, or posteriorly into the pharynx is abnormal. An overly large ingestion in a patient with impaired pharyngeal function and misdirected swallowing is also abnormal. This may indicate impairment of bolus sizing. Impaired lingual movement or jerky uncoordinated movements of the tongue during the preparatory phase of swallowing are also abnormal.

In patients with neurologic diseases, oral dysfunction regularly predominates over pharyngeal dysfunction. Radiographically, this can be appreciated as defective containment, i.e., leakage of barium anteriorly through the lips, laterally into the buccal pouches, or posteriorly into the pharynx, where it potentially may reach the airways if the laryngeal vestibule is not closed. If the patient swallows an abnormally large bolus, this may indicate impairment of bolus sizing. Abnormalities in the oral phase of swallowing, i.e., impaired lingual movement or a soft tissue defect, generally lead to delayed oral transit and clearance of the oral bolus with retention of barium. Premature spill of barium into the pharynx may be accompanied by failed initiation of swallowing, aspiration, or both. Impaired oral function is often associated with abnormal pharyngeal swallow (Fig. 3). Normally, the oral phase of swallowing undergoes a smooth transition into the pharyngeal phase with vigorous transport of the swallowed bolus into and through the pharynx. In some patients, however, the pharyngeal phase is delayed but otherwise normal.

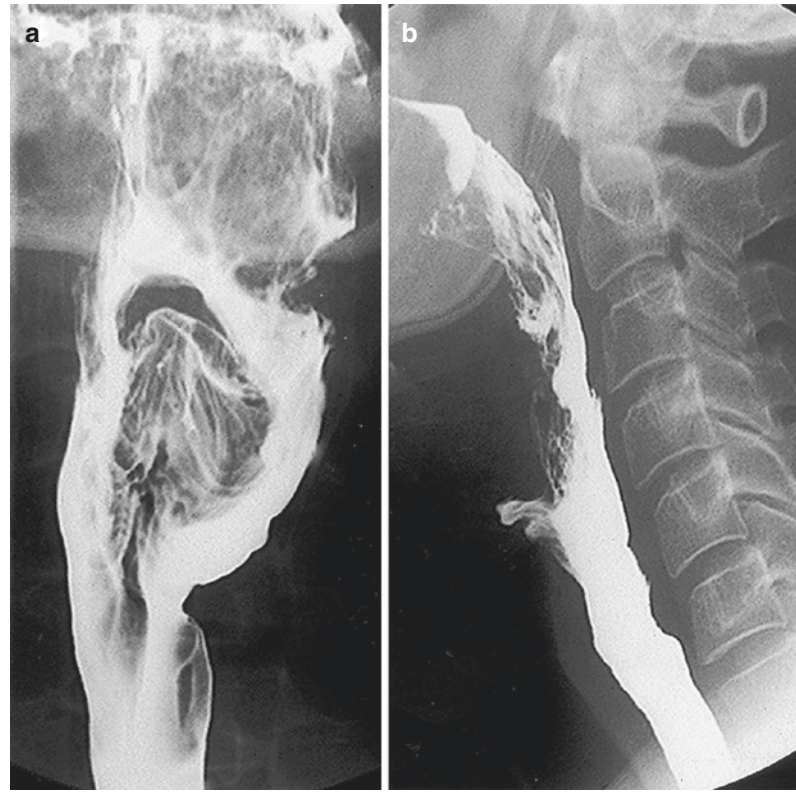

Fig. 3 A 49-year-old man with sudden onset of dysphagia. There is a left-sided weakness of the pharynx. The left side of the pharynx bulges laterally. There is also abnormal opening of the PES, probably due to impaired constrictor strength cranially. The pharyngeal constrictor abnormality is not appreciated in the lateral projection (**a**) and is only appreciated in the frontal projection (**b**)

3.2 The Pharyngeal Stage

Abnormal initiation of the pharyngeal stage of swallowing is easily appreciated when the bolus is conveyed into the pharynx without the pharynx being elevated and without occurrence of constrictor activity. Again, lack of anterior displacement of the hyoid bone is a conspicuous indicator of a serious abnormality. Demonstration of dissociation between the oral and pharyngeal stages depends upon observation of structural displacement. Except for this failure of voluntary elicitation of pharyngeal swallow, the oral and the pharyngeal stages of swallowing basically appear radiologically normal. In borderline cases, in which the bolus is retained in the valleculae or in the piriform sinuses for 2–5 s, the assessment of normalcy is based upon observation of the bolus.

The ultimate consequences of this dissociation are that the bolus reaches the pharynx when the larynx is still open. Even if the barium does not penetrate into the larynx, the delayed initiation is a potential threat to safe swallowing. Even the slightest amount of barium in the vestibule indicates an abnormality (Fig. 4). The wide range of normalcy can be observed during chewing and swallowing of a mixture of solids and liquid, especially during talking, etc., when the barium and/or solids are brought into the pharynx while chewing continues without tongue propulsion being elicited. Again, what happens to the bolus is more important than observing wall displacement in this circumstance. It is even more important to recognize that if pharyngeal swallow is not triggered at the faucial isthmus either by the bolus or by the tongue, swallowing may be elicited from the vallecula or from the posterior pharyngeal wall.

Defective closure of the velopharynx is due to either soft palate dysfunction or defective function in the superior pharyngeal constrictor. Medial movement of the lateral wall is more extensive than the anterior movement of the posterior constrictor wall. Compensation may be in the form of a Passavant's ridge, a protrusion

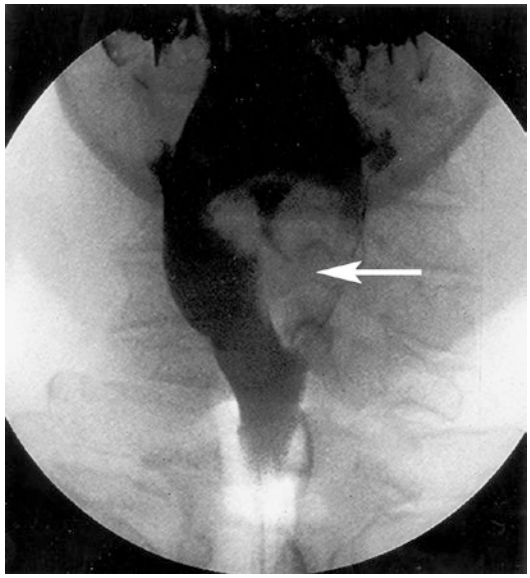

Fig. 4 Another example of a unilateral pharyngeal constrictor paresis, this time on the right side. The deviation of contrast medium into the right side may erroneously give the impression of a tumor on the normal left side (*arrow*). This pseudotumor appearance has led to numerous unnecessary endoscopic examinations

similar to that seen as a compensatory maneuver in speech in individuals with a cleft palate.

The pharyngeal constrictors play a crucial role in swallowing (Ardran and Kemp 1956). If the constrictor muscles are paretic, the pharynx is flaccid and allows an abnormal expansion of the chamber during the compression phase of swallowing (Figs. 3 and 4). Such lack of distensibility may result in impaired transit of bolus from the oral cavity into the esophagus even if the tongue acts normally (Thulin and Welin 1954; Ekberg et al. 1986). This can be seen as a dilated and wide flaccid pharynx, but this is a nonspecific finding. Action of the pharyngeal constrictors is also crucial for clearing barium from the pharynx. However, there are pitfalls (Fig. 5). This constrictor activity is best evaluated in the posteroanterior view (Fig. 2). The consequence of defective constrictor activity is residual barium in the pharynx after swallowing. As the middle pharyngeal constrictor is the pharyngeal constrictor most commonly involved, retention occurs at the level of the laryngeal inlet and may lead to aspiration after swallowing.

In the pharynx, retention of contrast medium may occur and indicate abnormalities in the tongue-base and/or in the pharyngeal constrictors. The finding of any contrast medium that reaches into the laryngeal vestibule and trachea is abnormal (Figs. 1, 2, and 3). This is especially true if the patient does not cough during this event. This indicates desensitization and is seen in patients with impairment lasting for more than about 2 months. Defective tilting down of the epiglottis is also abnormal and is generally seen together with impaired movement of the hyoid bone and larynx. More fundamental abnormalities are impaired elevation of the larynx and pharynx together with the hyoid bone at the initiation of pharyngeal swallow. This is seen in patients who do not elicit the pharyngeal stage of swallowing. We have used the beginning of the anterior movement of the hyoid bone as a key event. During the preparatory stage the hyoid bone and larynx are moving up and down, but at the initiation of the pharyngeal swallow the hyoid bone starts to move anteriorly (from an elevated position). If that movement is missing, the patient does not elicit pharyngeal swallow and is thereby at great risk if fed orally.

3.2.1 Misdirected Swallowing

Confusion exists regarding the terminology confined to barium reaching into the airways. "Penetration" has been used either to describe barium reaching into the airways *during* swallowing or merely to describe barium reaching only into the laryngeal vestibule and not beyond the vocal folds. "Aspiration" has been used either to describe barium reaching into the airways *after* swallowing and usually due to residue in the pharynx or to describe barium reaching beyond the vocal folds. Different use of the terminology has created unnecessary confusion. It is much more rational to describe (1) when the barium reaches into the airways, namely, *before*, *during*, or *after* elicitation of pharyngeal swallow, and (2) how far into the airways the barium reaches, namely, *into the subepiglottic*, *supraglottic portion* of the laryngeal vestibule or *beyond the vocal folds*. There is no consensus regarding the terminology and therefore it is warranted to be precise

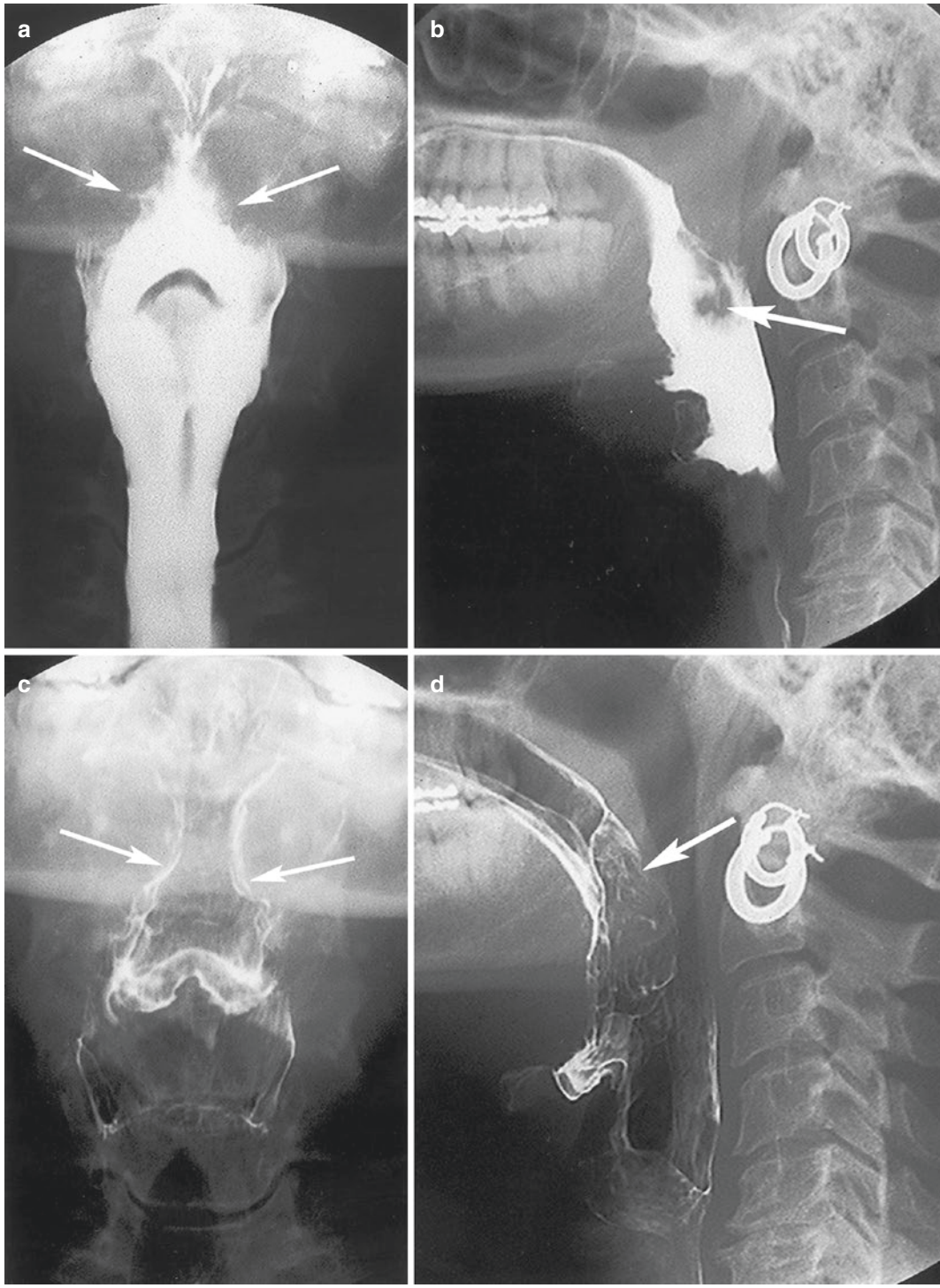

Fig. 5 (**a**, **b**) Barium swallow, single-contrast full column films. (**c**, **d**) Double-contrast films. In this 30-year-old woman there are extremely large tonsils (*arrow*). This finding was seen in both frontal and lateral projection. They may simulate normal or exaggerated function in the constrictor musculature, but they are in fact caused by a morphologic abnormality. Such hypertrophic tonsils may cause dysphagia

as the implicit meaning of "penetration" and "aspiration" is not widely accepted.

Of crucial importance is the coordination between the oral and pharyngeal stages of swallowing. The most common cause of misdirected swallowing is dissociation between the oral and pharyngeal stages of swallowing. This means that those two compartments are acting normally but the synchronization between them is impaired. The oral stage includes propulsion of the bolus into the pharynx. The pharyngeal stage of swallowing includes elevation of the hyoid bone, the larynx, and the pharynx. It is crucial to observe a conspicuous anterior movement of the hyoid bone as this is an indicator of elicitation of pharyngeal swallow. Any patient who does not show such hyoid bone movement potentially has a severe pharyngeal dysfunction. There should also be an approximation between the larynx and the hyoid bone due to contraction of the thyrohyoid muscles. In normal individuals the timing of the oral and pharyngeal stages is tightly coordinated and there is usually a time lag of not more than 0.5 s between when the apex of the bolus passes the faucial isthmus and start of the anterior movement of the hyoid bone. In patients with such a tight coordination, misdirected swallowing is seldom seen. However, the longer the delay, the more likely misdirected swallowing is to occur. Such incoordination between the oral and pharyngeal stages of swallowing is particularly common in the elderly. It is often the sole cause of misdirected swallowing (Fig. 6).

Airway closure and constrictor activity are both important elements of swallowing. Absence of elevation of the pharynx and larynx and failure of anterior movement of the hyoid bone are crucial elements of abnormal function. These are seen not only in the neurologically impaired patient but also after extensive surgery and/or radiotherapy. During barium swallow, the quality of the cough reflex is readily appreciated and can

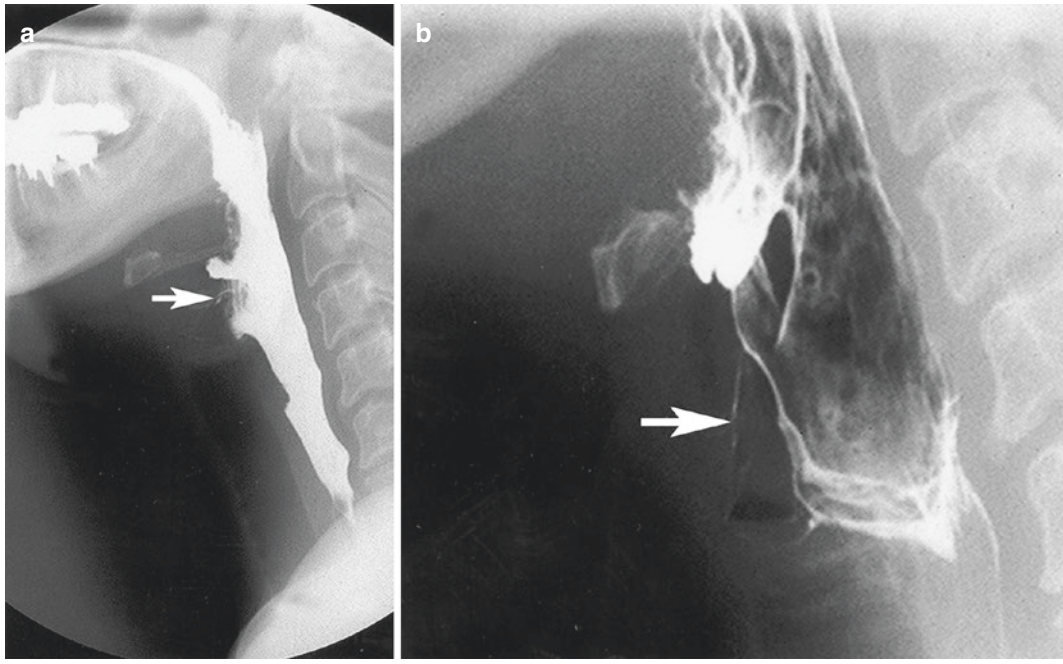

Fig. 6 This patient complained of an occasional sensation of having a foreign body in the neck. She pointed to the lateral part of her neck. Several swallows were normal. However, small amounts of barium eventually reached into the laryngeal vestibule. This was due to a slight dissociation (1.7 s) between the oral and pharyngeal stages of swallowing. This was enough to cause misdirected swallowing into the laryngeal vestibule. The patient then indicated that she had the foreign body sensation in the neck. This was seen both on single-contrast full column films (**a**) (*arrow*) and on double-contrast films (**b**) (*arrow*)

be defined as to the level where it is elicited, as well as roughly how much and for how long the barium must be in contact with the laryngeal and/or tracheal mucosa. However, for proper quantification of misdirected swallowing to the trachea, scintigraphy is more accurate than barium swallow (Muz et al. 1987).

Discovery of misdirection of the barium into the larynx and/or trachea should not lead to the interruption of the study. On the other hand, there are patients with massive penetration into the trachea in whom a very limited study is sufficient for answering the clinician's immediate questions concerning possible oral feeding. It is important to elucidate the underlying pathophysiologic processes in these patients and a few swallows should be observed in lateral projection. However, the risk of acquiring bronchopneumonia secondary to misdirected bolus is probably much less than many would expect (Ekberg and Hilderfors 1985).

According to Kun et al. (1987), elderly patients have misdirected swallowing due to dysfunction in the oral stage or a combination of oral and pharyngeal stage dysfunction. Oral stage dysfunction was due to ingestion of a large volume or rapid acquisition. However, dissociation between the oral and pharyngeal stages was the main finding. It has been shown that thermal stimulation of the faucial isthmus reduces such a dissociation (Logemann 1983; Logemann and Kahrilas 1990). For many years such tactile and thermal stimulation of the faucial isthmus has been a widely used technique for treatment of these patients.

Misdirected swallowing has been considered a major cause of aspiration pneumonia. If major aspiration occurs during barium swallow, it is common for the patient to be fed nonorally. The rationale for such treatment has been questioned (Siebens and Linden 1985). It was shown that patients who were fed nonorally showed more frequent aspiration pneumonia than those who were fed orally. Their explanation was that saliva contaminated with bacteria in the oral cavity caused the pneumonia. The oral hygiene in these patients is usually low. It was concluded that artificial feeding does not seem to be a satisfactory solution for preventing pneumonia in elderly patients with prandial aspiration.

Patients who have defective protection of the airways during swallowing are also at risk of fatal choking episodes. Prandial aspiration most often results in reflexive coughing, gagging, and forced exploration. This is often referred to by the patient as "choking." It is usually uncomfortable but brief and usually familiar in nature to the patient. This is in contrast to airway obstruction during oral intake, which may be fatal unless the occluding material is removed or displaced. Such food asphyxia is an important cause of accidental death in children (Editorial 1981; Lima 1989). It is estimated that 8000–10,000 adults choke to death each year (Donner and Jones 1985). The development of the Heimlich maneuver and its inclusion as part of basic cardiopulmonary resuscitation training has increased public awareness of the problem in the USA (Heimlich 1985). The cause of near-fatal choking episodes has been studied in 58 individuals (Feinberg and Ekberg 1990). Most of these patients who had survived a Heimlich maneuver applied because of food impaction showed abnormalities during the barium swallow. Most of them aspirated liquid bolus. This was due to bolus leak or dissociation. Some patients also showed defective closure of the laryngeal vestibule. However, a subset of patients (14 of 58) were able to vocalize during the near-fatal choking episode and they demonstrated structural abnormalities of the PES and the esophagus. Therefore, patients who have had a near-fatal choking episode should undergo an elective radiologic barium study in order to reveal an underlying cause and prevent new episodes that may otherwise prove to be fatal.

4 Dysfunction of the Pharyngoesophageal Segment

Defective opening of the PES is a common finding. This may be seen as a posterior indentation of the cricopharyngeal muscle. However, it has been shown that the indentation per se does not impinge on the lumen diameter (Olsson and

Ekberg 1995). In fact, the diameter of the PES at the level of the cricopharyngeal muscle is the same in patients with and without that posterior cricopharyngeal bar. Instead the pharynx is dilated above and the cervical esophagus is dilated below the cricopharyngeal bar, thereby giving the false impression of lumen narrowing. Manometrically, the cricopharyngeal muscle in these patients has been shown to act normally, and there are decreased peak amplitudes in the pharynx above. There is also a small subset of patients who have fibrosis of the PES and the cricopharyngeal muscle, but this is rare. Most patients who appear to have an abnormal opening of the PES do have normal morphology and function in that particular segment, and the impairment in fact is defective elevation of the PES and weakness of the pharyngeal constrictors (Olsson and Ekberg 1995). The most common cause of this, in turn, is lack of initiation of the pharyngeal stage of swallowing. Therefore, any surgical procedure on the PES is not likely to be beneficial, the main reason being that only a small fraction of the closure of the PES is due to muscle tonicity.

Failure of the PES to open may be seen in neurologic disease but is generally not accompanied by abnormal pharyngeal bolus transport (Curtis et al. 1984; Ekberg 1986a). Failure of the cricopharyngeal muscle to open or elongate may be due to (1) defective muscle relaxation, (2) defective extensibility, and (3) hypertrophy or hyperplasia. The posterior bar intruding into the barium and created by the cricopharyngeal muscle is seldom an isolated dysfunction. It is commonly associated with abnormal motor function in the segment above (i.e., the inferior pharyngeal constrictor) and/or in the segment below (i.e., the cervical esophageal muscles). Therefore, the cricopharyngeal indentation, though the most conspicuous, is only one aspect of severe motor dysfunction in the adjacent PES (Ekberg 1986a). Cervical esophageal webs are also common in dysphagic patients (Figs. 5 and 7).

Adapted and compensated swallowing basically have the same radiologic appearance and, in most patients, they are notoriously difficult to demonstrate. Therefore, a normal radiologic study does not rule out pharyngeal abnormality.

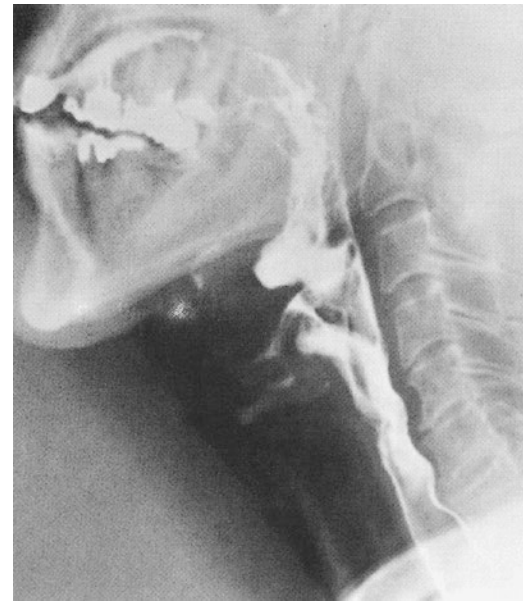

Fig. 7 A 76-year-old woman 2 week after a stroke. Lateral radiogram of the pharynx during swallowing. Contrast medium has reached into the pharynx, through the PES, and into the cervical esophagus. The epiglottis is in a horizontal position. The hyoid bone is elevated and brought slightly forward. However, contrast medium has reached into the laryngeal vestibule to the level of the sinus of Morgagni. No contrast medium is seen in the trachea. The patient did not subjectively experience this misdirected swallowing. There was no cough. This indicates chronic aspiration (see also Fig. 8)

5 Radiologic Evaluation in Specific Disease Entities

Radiologic evaluation of oral and pharyngeal function during swallowing has a high sensitivity but is nonspecific in terms of the type and extent of the underlying abnormality (Figs. 7, 8, and 9). Specific disease is seen to cause pharyngeal dysfunction and dysphagia with varying frequency and the cause of dysphagia during the disease varies. This has been studied radiologically in cerebrovascular disease (Veis and Logemann 1985; Donner and Silbiger 1966), poliomyelitis (Silbiger et al. 1967; Ardran et al. 1957), amyotrophic lateral sclerosis (Bosma and Brodie 1969a), myasthenia gravis (Murray 1962), myotonic dystrophy (Bosma and Brodie 1969b), Parkinson's disease (Calne et al. 1970; Robbins

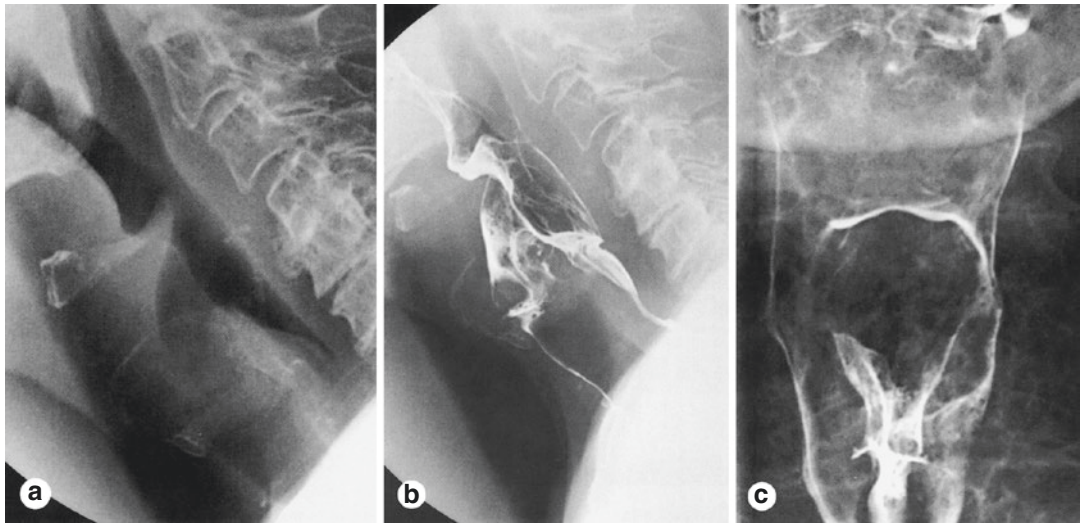

Fig. 8 A 74-year-old man who had undergone radiotherapy to the neck for laryngeal carcinoma 3 year previously. There was no sign of recurrence but the patient now had difficulty swallowing. He had sustained several choking episodes. (**a**) Lateral radiogram of the pharynx. There is thickening of the prevertebral soft tissue and of the epiglottis. (**b**) After barium swallow, there is contrast medium in the laryngeal vestibule and in the trachea, but only minor retention in the piriform sinuses. (**c**) Anteroposterior view of the pharynx. There is minor retention in the effaced valleculae and shallow piriform sinuses. Contrast medium is also seen in the laryngeal vestibule and sinus of Morgagni (see also Fig. 7)

et al. 1986), brainstem tumor (Kun et al. 1987), and multiple sclerosis (Daly et al. 1962). Even the distinction between upper and lower motor neuron disease, e.g., cortical bulbar tract dysfunction (pseudobulbar palsy), and lower motor neuron disease, i.e., pontomedullary dysfunction (bulbar palsy), is ambiguous. However, the latter patients usually have more widespread pharyngeal paresis and they also lack initiation of pharyngeal swallow. Basically, all neurologic impairments lead to the same dysfunction. This is especially so for the "end-stage" dysfunction, which tends to lead all types of disease entities down a common pathway with impaired elevation of the larynx and pharynx and impaired anterior movement of the hyoid bone.

The cause of dysphagia in terms of symptoms and functions often does not match. Deterioration or progression of dysphagia is as a rule compensated. So, if we could decompensate swallowing, this would reveal progression. The radiologist may intentionally elicit the decompensation by extension of the neck by provision of a large bolus and other stresses (Ekberg 1986b; Buchholz et al. 1985).

5.1 Cerebrovascular Diseases

Most patients with pharyngeal dysfunction are patients who suffer from stroke. The prevalence of stroke in the USA has been estimated to be approximately 1.6 million persons, with 250,000 new cases of stroke each year. Of these stroke patients, 30–50% will develop dysphagia. Strokes that are multiple (Veis and Logemann 1985; Rosenbek et al. 1991; Horner et al. 1991; Splainard et al. 1988), bilateral (Logemann 1983), or localized in the brain stem (Veis and Logemann 1985; Horner et al. 1991; Logemann and Kahrilas 1990; Linden and Siebens 1983) are considered to cause severe swallowing impairment. However, even unilateral cortical or subcortical strokes (Veis and Logemann 1985; Robbins and Levine 1988; Robbins et al. 1993; Gordon et al. 1987; Meadows 1973) can cause swallowing problems. Dysphagia in the stroke population may last for weeks or months, but occasionally much longer. Although dysphagia is a subjective symptom, dysfunction of the pharyngeal stage of swallowing can be objectively registered using barium swallow and video recording.

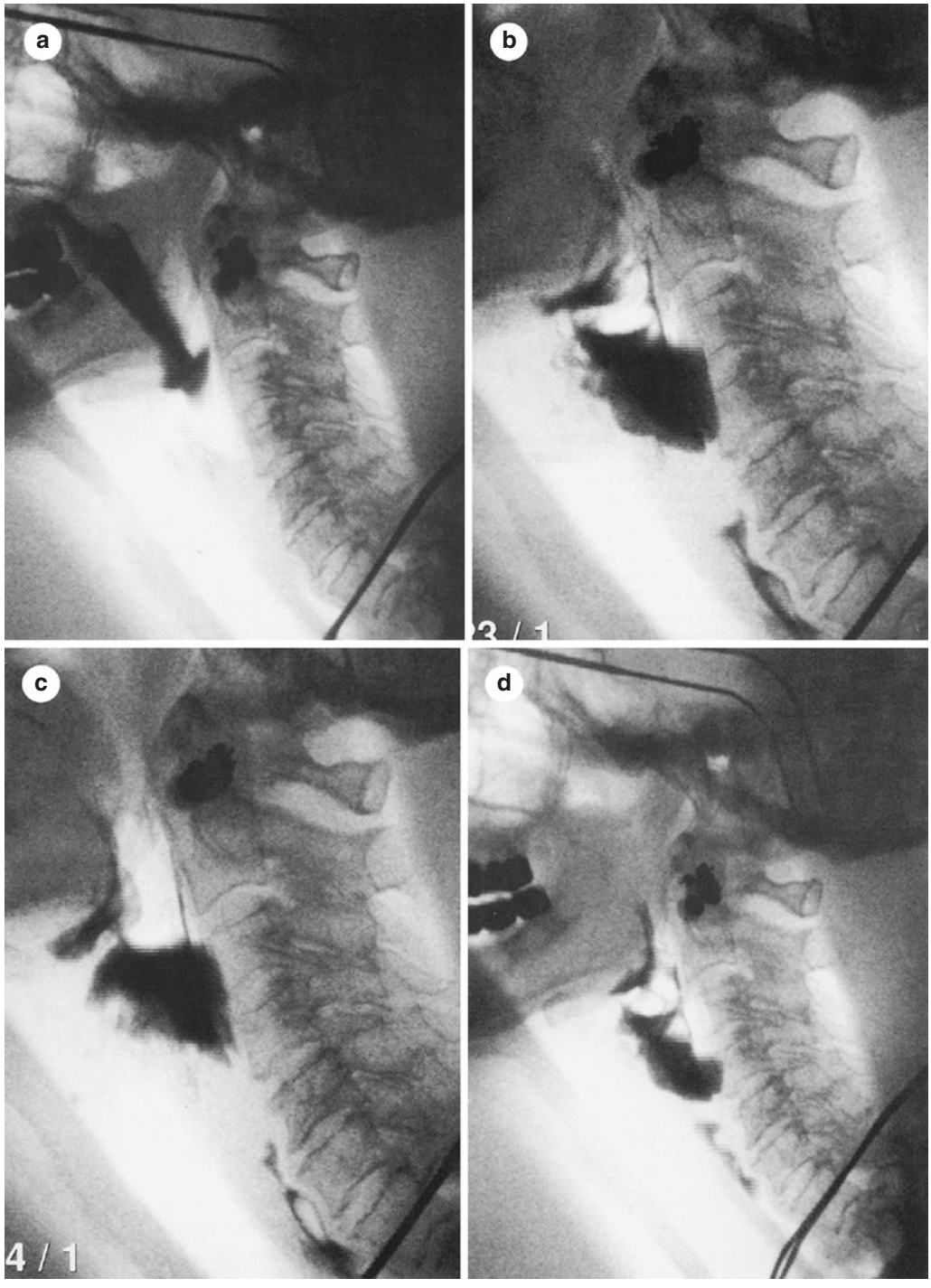

Fig. 9 A 65-year-old woman with no prior history of stroke. She had developed dysphagia 2 year previously. The dysphagia progressed over a couple of weeks. She had difficulty with all kinds of textures and had a very prolonged eating time. (**a**) The contrast medium is brought into the pharynx. (**b**) Pronounced dilatation of the pharynx indicating high compliance secondary to weakness of pharyngeal constrictor musculature. (**c**) Eventually contrast medium reached into the laryngeal vestibule but not further down into the airways. (**d**) Only very tiny streaks of barium reach through the PES during each swallowing attempt. There was minor anterior movement of the hyoid bone that could indicate that pharyngeal swallow had been elicited. However, there was no constrictor activity. The patient spontaneously learned to compensate for the absence of contractility in the constrictors (from Ekberg and Olsson 1997)

This technique is now the gold standard for evaluation of normal and abnormal swallowing.

Oropharyngeal impairment is recognized as a frequent cause of morbidity, disability, and costly dependence in stroke patients (Veis and Logemann 1985). Bolus misdirection into the larynx and trachea is perhaps the most significant abnormality that we routinely observe during barium swallow. Especially in the elderly, swallowing dysfunction is prevalent, particularly in those who are hospitalized, institutionalized, or well advanced in years. These patients represent some of the most challenging cases that clinicians must deal with on a routine basis. The diagnosis is difficult and so is management. However, this is not because of unique abnormalities in swallowing behavior or morphodynamics, but is because of the patients themselves (Veis and Logemann 1985). In fact, swallowing problems may go undetected in these patients because the signs and symptoms are vague, subtle, or unreported by the patients' caregivers. Critical management decisions regarding dietary alterations, degree of oral intake, and institution of artificial feeding often depend on the radiologic assessment of such misdirected swallowing.

Radiologists are important members of multidisciplinary teams that address swallowing disorders. Diagnosis and treatment of dysphagia depend on videofluorographic deglutition examinations during barium swallow. Patients with cerebrovascular disease, Parkinson's disease, and other neurologic conditions, including elderly patients with dementia, need to undergo videofluorographic examination in order to correctly describe functional status and thereby provide a platform for rational therapy. Treatment strategies must be founded on objective grounds (see Sect. 5).

6 The Role of the Radiologist in the Design of Therapy

Treatment of oral and pharyngeal dysfunction strives to correct or compensate for damage to specific neuromuscular components of the oral and pharyngeal swallow, such as reduced tongue movement, delay in triggering the pharyngeal swallow, pharyngeal constrictor paralysis, and defective closure of the laryngeal vestibule. For evaluation of such therapy, successive radiologic evaluations are necessary (see Sect. 5). The radiologic examination must (1) reveal the anatomic and/or physiologic abnormalities causing the patient's dysphagia, and (2) identify those compensatory strategies or therapy techniques which are most effective in improving the efficiency of the patient's oral and pharyngeal swallow. The radiologic examination should start with ingestion and include also the passage of the bolus into the stomach. Following liquid swallows, the patient should be given other food consistencies, particularly pudding consistency materials and food requiring chewing, such as cookies. A variety of foods mixed with barium can be introduced in the radiologic study. The volume of each food or liquid should be measured because the dynamics of normal pharyngeal swallow vary as the volume of the bolus increases.

Two types of therapy techniques are used for reeducation for oral and pharyngeal dysphagia: (1) "direct" swallowing therapy procedures which passively facilitate swallowing by use of particular foods and liquids; and (2) "indirect" therapy techniques designed to compensate for dysfunction by increasing muscle strength, range of motion, or coordination independent of swallowing. The effectiveness of either of these techniques is assessed by observing the transport of the swallowed bolus during radiography.

References

Ardran GM, Kemp FM (1956) Radiologic investigation of pharyngeal and laryngeal palsy. Acta Radiol Diagn 46:446–457

Ardran GM, Kemp FM, Wegelius C (1957) Swallowing defects after poliomyelitis. Br J Radiol 30:169–189

Bosma JF, Brodie DR (1969a) Disabilities of the pharynx in amyotrophic lateral sclerosis as demonstrated by cineradiography. Radiology 92:97–103

Bosma JF, Brodie DR (1969b) Cineradiographic demonstration of pharyngeal area myotonia in myotonic dystrophy patients. Radiology 92:104–109

Brühlmann WF (1985) Die röntgenkinematographische Untersuchung von Störungen des Schluckaktes. Huber, Bern

Buchholz DW (1995) Cricopharyngeal myotomy may be effective treatment for selected patients with neurogenic oropharyngeal dysphagia. Dysphagia 10:255–258

Buchholz DW, Bosma JF, Donner MW (1985) Adaptation, compensation, and decompensation of the pharyngeal swallow. Gastrointest Radiol 10:235–239

Calne DB, Shaw DG, Spiers AS, Stern GM (1970) Swallowing in parkinsonism. Br J Radiol 43:456–457

Cerenko D, McConnel FMS, Jackson RT (1989) Quantitative assessment of pharyngeal bolus driving forces. Otolaryngol Head Neck Surg 100:57–63

Curtis DJ, Cruess DF, Berg T (1984) The cricopharyngeal muscle. A video-recording. Am J Roentgenol 146:497–500

Curtis DJ, Hudson T (1983) Laryngotracheal aspiration: analysis of specific neurmuscular factors. Radiology 149:517–522

Curtis DJ, Sepulveda GV (1983) Epiglottic motion: video recording of muscular dysfunction. Radiology 148:473–477

Daly DD, Code CF, Andersson HA (1962) Disturbances of swallowing and esophageal motility in patients with multiple sclerosis. Neurology 59:250–256

Dodds WJ (1989) The physiology of swallowing. Dysphagia 3:171–178

Donner MW, Jones B (1985) Editorial. Gastrointest Radiol 10:194–195

Donner MW, Silbiger ML (1966) Cinerafluorographic analysis of pharyngeal swallowing in neuromuscular disorders. Am J Med Sci 251:600–616

Doty RW, Bosma JB (1956) An electromyographic analysis of reflux deglutition. J Neurophysiol 19:44–60

Editorial (1981) Inhaled foreign bodies. Br Med J 282:1649–1650

Ekberg O (1982) Defective closure of the laryngeal vestibule during deglutition. Acta Otolaryngol 93:309–317

Ekberg O (1986a) The cricopharyngeus revisited. Br J Radiol 59:875–879

Ekberg O (1986b) Posture of the head and pharyngeal swallow. Acta Radiol Diagn 27:691–696

Ekberg O, Hilderfors H (1985) Defective closure of the laryngeal vestibule: frequency of pulmonary complications. Am J Roentgenol 145:1159–1164

Ekberg O, Lindgren S, Schultz T (1986) Pharyngeal swallowing in patients with paresis of the recurrent nerve. Acta Radiol Diagn 27:697–700

Ekberg O, Olsson R (1997) Oper Tech Otolaryngol Head Neck Surg 8:153–162

Ekberg O, Wahlgren L (1985) Dysfunction of pharyngeal swallowing: a cineradiographic investigation in 854 dysphagial patients. Acta Radiol Diagn 26:389–395

Ergun GA, Kahrilas PJ, Lin S, Logemann JA, Harig JM (1993a) Shape, volume, and content of the deglutitive pharyngeal chamber imaged by ultrafast computerized tomography. Gastroenterology 105:1396–1403

Ergun GA, Kahrilas PJ, Logemann JA (1993b) Interpretation of pharyngeal manometric recordings: limitations and variability. Dis Esophagus 6:11–16

Feinberg MJ, Ekberg O (1990) Deglutition in near fatal choking episodes: radiologic evaluation. Radiology 176:637–640

Gordon C, Hewer RL, Wade DT (1987) Dysphagia in acute stroke. Br Med J 295:411–414

Hamlet SL, Stone M, Shawker TH (1988) Posterior tongue grooving in deglutition and speech: preliminary observations. Dysphagia 3:65–68

Hannig C, Hannig A (1987) Stellenwert der Hochfrequenzröntgenkinematographie in der Diagnostik des Pharynx und Ösophagus. Rontgenpraxis 40:358–377

Heimlich JH (1985) A life-saving maneuver to prevent food choking. JAMA 234:398–401

Horner J, Bouyer FG, Alberts MJ, Helms MJ (1991) Dysphagia following brain-stem stroke: clinical correlates and outcome. Arch Neurol 48:1170–1173

Hsieh PY, Brasseur JG, Shaker R, Kern MK, Kahrilas PJ, Ren J (1995) Modeling and timing of UES opening events. Paper presented at the Dysphagia Research Society meeting, Tysons Corner, 26–28 October 1995

Jones B, Donner MW (1991) Normal and abnormal swallowing, imaging in diagnosis and therapy. Springer, Berlin

Kahrilas PJ, Dodds WJ, Dent J, Logemann JA, Shaker R (1988) Upper esophageal sphincter function during deglutition. Gastroenterology 95:52–62

Kun WS, Buchholz D, Kuman AJ, Donner MW, Rosenbaum AE (1987) Magnetic resonance imaging for evaluating neurogenic dysphagia. Dysphagia 2:40–45

Lima JH (1989) Laryngeal foreign bodies in children: a persistent, life-threatening problem. Laryngoscope 99:415–420

Linden P, Siebens A (1983) Dysphagia: predicting laryngeal penetration. Arch Phys Med Rehabil 69:637–640

Logemann JA (1983) Evaluation and treatment of swallowing disorders. College-Hill Press, San Diego

Logemann JA, Kahrilas PJ (1990) Relearning to swallow after stroke-application of maneuvers and indirect biofeedback: a case study. Neurology 40:1136–1138

McConnel FMC (1988) Analysis of pressure generation and bolus transit during pharyngeal swallowing. Laryngoscope 98:71–78

McConnel FMS, Cerenko D, Jackson RT, Guffin TN Jr (1988) Timing of major events of pharyngeal swallowing. Arch Otolaryngol Head Neck Surg 114:1413–1418

Meadows JC (1973) Dysphagia in unilateral cerebral lesions. J Neurol Neurosurg Phychiatry 36:853–860

Miller AJ (1986) Neurophysiological basis of swallowing. Dysphagia 1:91–100

Murray JF (1962) Deglutition in myasthenia gravis. Br J Radiol 35:43–52

Muz J, Mathog RM, Miller PR, Rosen R, Borrero G (1987) Detection and quantification of laryngotracheopulmonary aspiration with scintigraphy. Laryngoscope 97:1180–1185

Olsson R, Castell J, Johnston B, Ekberg O, Castell DO (1997) Combined videomanometric identification of abnormalities related to pharyngeal retention. Acad Radiol 4:349–354

Olsson R, Ekberg O (1995) Videomanometry of the pharynx in dysphagic patients with a posterior cricopharyngeal indentation. Acad Radiol 2:597–601

Pokieser P, Schober W, Schima W (1995) Videokinematographie des Schluckaktes—Indikation, Methodik und Befundung. Radiologe 35:703–711

Robbins J, Levine RL (1988) Swallowing after unilateral stroke of the cerebral cortex: preliminary experience. Dysphagia 3:11–17

Robbins J, Levine RL, Maser A, Rosenbek JC, Kempster GB (1993) Swallowing after unilateral stroke of the cerebral cortex. Arch Phys Med Rehabil 74:1295–1300

Robbins JA, Logemann JA, Kirshner HS (1986) Swallowing and speech production in Parkinson's disease. Ann Neurol 19:283–287

Rosenbek JC, Robbins J, Fishback B, Levine RL (1991) The effects of thermal application on dysphagia after stroke. J Speech Hear Res 34:1257–1268

Siebens AA, Linden P (1985) Dynamic imaging for swallowing re-education. Gastrointest Radiol 10:251–253

Silbiger M, Pikielney R, Donner MW (1967) Neuromuscular disorders affecting the pharynx. Investig Radiol 2:442–448

Splainard ML, Hutchins B, Sulton LD, Chaudhuri G (1988) Aspiration in rehabilitation patients: videofluoroscopic versus bedside clinical assessment. Arch Phys Med Rehabil 69:637–640

Thulin A, Welin S (1954) Radiographic findings in unilateral hypopharyngeal paralysis. Acta Otolaryngol Suppl 116:288–293

Veis SL, Logemann JA (1985) Swallowing disorders in persons with cerebrovascular accident. Arch Phys Med Rehabil 66:372–375