

---

## Introduction

The development of feeding skills is an extremely complex process influenced by multiple anatomic, neurophysiologic, environmental, social, and cultural factors. Oral feeding in infants should be efficient to preserve energy for growing. Moreover, it should be safe so as to avoid aspiration, and it should not compromise respiratory status. This can only be achieved if sucking, swallowing, and breathing are properly coordinated [1]. This entire process is dynamic because of ongoing growth and development. Functional feeding skills, which depend on the integrity of anatomic structures, undergo changes based on neurologic maturation and experimental learning. Eating/feeding requires active effort by infants who must have exquisite timing and coordination of sucking, swallowing, and breathing to be efficient [2].

A variety of neurological, neuromuscular conditions in children and infants can impair the physiological phases of sucking/swallowing and cause disorders of feeding and dysphagia. The causes of feeding and swallowing problems include combinations of structural deficits, neurologic conditions, respiratory compromise, feeder–child interaction dysfunction, and numerous medical conditions such as genetic, metabolic, and degenerative disease [3].

In the recent years, there has been an increase in infant swallowing disorders as a result of improved survival rates for infants born prematurely or with life-threatening medical disorders. Negative experiences related to feeding, such as intubation, tube feeding, or airway suctioning may further disturb sucking and swallowing development [4]. Disorders of feeding and swallowing in children are serious and potentially fatal problems. Aspiration due to dysphagia

may lead to severe pulmonary disease, and impaired oral and pharyngeal function may rapidly result in failure to thrive. Prompt evaluation of swallowing disorders is therefore critical.

The differential diagnosis of dysphagia in children is wide. The diagnostic work-up can be extremely difficult and exhaustive in many cases. Because of this complexity, multidisciplinary team evaluations should be conducted.

Successful rehabilitation of children with swallowing disorders requires knowledge of the parameters of normal and abnormal swallowing plus skill in the integration of a variety of essential therapeutics techniques.

---

## Epidemiology

Data on the incidence of swallowing disorders are lacking, because in clinical practice, disorders of swallowing are often considered in the general context of a feeding disorder. Feeding (or eating) is different from swallowing. Eating is primarily an oral phase function that includes oral preparation and oral transit of a bolus [5]. Feeding is a complex process that involves a number of phases in addition to the act of swallowing, including the recognition of hunger (appetite), the acquisition of the food, and the ability to bring the food to the mouth [6]. The estimated prevalence of feeding problems in the pediatric population ranges from 25–35% in normally developing children to 40–93% in children with developmental delay [7, 8]. Early sucking and swallowing problems were reported to be present in 35–48% of infants with different types of neonatal brain injury [9]. However, knowledge of the true epidemiology of pediatric dysphagia remains largely unavailable because of the lack of a standardized reporting system assessing dysphagia in all of the possible contexts that may occur in infants and children [10].

---

A. Staiano (✉) · F. P. Giugliano · E. Miele  
Section of Pediatrics, Department of Transitional Medical  
Science, University of Naples “Federico II”, Via S. Pansini 5,  
Naples 80131, Italy  
e-mail: staiano@unina.it

**Table 20.1** Differential diagnosis of dysphagia. (Adapted from Ref. [12], Copyright Elsevier, 1983, and reprinted with permission from Ref. [13], Table 15.1, p. 234)

Prematurity		
Upper airway obstruction	Nasal and nasopharyngeal	Cohanal atresia, stenosis, septal deflections and abscess, infections, tumors, sinusitis
	Oropharynx	Defects of lips and alveolar processes, cleft lips or palate, hypopharyngeal stenosis, craniofacial syndromes or sequences (e.g., Cruzon, Treacher Collins syndrome, Pierre Robin sequence)
	Laryngeal	Laryngeal cleft and cyst, laryngomalacia, subglottic stenosis, and paralysis
Congenital defects of the larynx, trachea, and esophagus	Laryngotracheoesophageal cleft	–
	Tracheoesophageal fistula with associated esophageal atresia	–
	Esophageal anomalies (e.g., strictures, webs)	–
	Vascular anomalies	Aberrant right subclavian artery Double aortic arch Right aortic arch with left ligamentum
Acquired anatomic defects	Trauma	External trauma, intubation, endoscopic, and foreign body
	Chemical ingestion	–
Neurologic disorders	Central nervous system	Trauma
		Hypoxia and anoxia
		Cortical atrophy, hypoplasia, agenesis
		Infections (meningitis, brain abscess)
Peripheral nervous system disease	Trauma	–
	Congenital defects	
Neuromuscular	Guillan-Barre syndrome	
	Poliomyelitis (bulbar paralysis)	
	Myasthenia gravis	
	Myotonic muscular dystrophy	
Anatomic and functional defects	Cricopharyngeal dysfunction	
	Esophageal achalasia	
	Esophageal spasm	
	Paralysis of the esophagus	
	Associated atresia-tracheoesophageal fistula, nerve defect	
	Peptic and eosinophilic esophagitis	
	Riley-Day syndrome (dysautonomia)	
	Brain stem compression (e.g., Chiari malformation, tumor)	

## Etiology

Disorders of sucking/swallowing may be caused by multiple etiologic factors that may interfere with the child's ability to coordinate swallowing and breathing maneuvers and may be manifested as a unique set of symptoms. Potential causes responsible for three broad categories include: immaturity, delay, or a defect in neuromuscular control; an anatomic abnormality of the aerodigestive tract; and/or systemic illness. The magnitude of the dysfunction depends on the balance between the extent of the structural or functional abnormality and the child's compensatory adaptations [11]. Disorders

associated with sucking/swallowing difficulties are reported in Table 20.1 [12, 13].

## Pathophysiology

The fetus is capable of swallowing amniotic fluid in utero, indicating that the motor program for swallowing is functioning before gestation is complete.

However, oral feeding is not initiated in preterm infants before 32 weeks of postconceptional age, partly because the coordination of sucking, swallowing, and respiration is

not established [14]. Even at 34 weeks, minute ventilation during sucking decreases more than that of infants at 36–38 weeks. Therefore, the coordination between swallowing and breathing is not yet fully organized at 34 weeks of postconceptional age [15, 16].

Anatomic structures, which are essential to competent feeding skills, undergo growth that changes their physical relationship to one other and consequently affects their function. The swallowing mechanism, by which food is transmitted to the stomach and digestive organs, is a complex action involving 26 muscles and 5 cranial nerves. The neurophysiologic control involves sensory afferent nerve fibers, motor efferent fibers, paired brainstem swallowing centers, and suprabulbar neural input. Structural integrity is essential to the development of normal feeding and swallowing skills [17]. The central patterning of aeroingestive behavior is based on volitional and reflexive control mechanisms and benefit from sensory feedback to modify the spatiotemporal organization of the feed sequence to allow safe swallow [18]. Central pattern generators (CPGs) are primarily composed of adaptive networks of interneurons that activate groups of motor neurons to generate task-specific motor patterns [19]. The essential components of the masticatory CPG are found between the rostral poles of the fifth and seventh motor nuclei, although they are normally synchronized by commissural axons each hemisection side can generate a rhythm [20]. Mastication patterns differ greatly between foods and change systematically during a chewing sequence based on sensory feedback. Functional imaging has revealed that swallowing is controlled through a network of cortical areas which shares loci with other ororhythmic movements including speech [21].

Deglutition is generally divided into phases of swallowing based on anatomic and functional characteristics: pre-oral, pharyngeal, and esophageal [22, 23].

---

## Anatomic Considerations

An understanding of the anatomy of the pharynx is essential to a thorough understanding of the swallowing process. The anatomy changes during development. The tongue, the soft palate, and the arytenoids mass (arytenoids cartilage, false vocal cords, and true vocal cords) are larger relative to their surrounding chambers when compared with the adult [24]. In the infant, the tongue lies entirely within the oral cavity, resulting in a small oropharynx [25, 26]. In addition, a sucking pad, composed of densely compacted fatty tissue that further reduces the size of the oral cavity, stabilizes the lateral walls of the oral cavity. The larynx lies high in the infant, and the tip of epiglottis extends to and may overlap the soft palate. These anatomic relationships are ideal for the normal infant feeding pattern of suck or suckle feeding from a breast or a

bottle in a recumbent position [26]. In the infant, the larynx sits high in the neck at the level of vertebrae C1 to C3, allowing for the velum, tongue, and epiglottis to approximate, thereby functionally separating the respiratory and digestive tracts. This separation allows the infant to breathe and feed safely. By age 2–3 years, the larynx descends, decreasing the separation of the swallowing and digestive tracts [7].

---

## Development and Normal Swallowing Function

Swallowing skills develop progressively during fetal and neonatal maturation [27]. At approximately 26 days' fetal age, the developmental trajectories of the respiratory and swallowing systems diverge and start to develop independently. Swallowing in fetuses has been described as early as 12–14 weeks' gestational age. A sucking response can be provoked at 13 weeks' postconceptional age by touching the lips [28]. Real sucking, defined by a posterior–anterior movement of the tongue, in which the posterior movement is dominant, begins at 18–24 weeks' postconceptional age [29]. Between 26 and 29 weeks' there is probably no significant further maturation of sucking [30]. By week 34, most healthy fetuses can suck and swallow well enough to sustain nutritional needs via the oral route, if born at this early age. Sucking movements increase in frequency during the final weeks of fetal life due to an increase in amniotic fluid swallowed by a fetus during pregnancy from initially 2–7 ml a day to 450 ml a day. This is approximately half of the total volume of amniotic fluid at term [31]. The normal maturation of sucking and swallowing during the first months of life after full-term birth can be summarized by increased sucking and swallowing rates, longer sucking bursts and larger volume per suck [16]. The skill of safe and efficient oral feeding is based on oral-motor competence, neurobehavioral organization, and gastrointestinal maturity [32]. Two forms of sucking are distinguished: nutritive sucking (NS) and nonnutritive sucking (NNS). NS is an infant's primary means of receiving nutrition while NNS is regarded as an initial method for exploring the environment. The rate of NNS is approximately twice as fast as that of NS [33]. In NS, however, the ability to integrate breathing with sucking and swallowing is essential for coordinated feeding [1] and it becomes consistent by 37 weeks' gestation [34]. By increasing the intraoral space, the infant begins to suppress reflexive suckle patterns and starts to use voluntary suck patterns. In contrast to suckling, true sucking involves a raising and lowering of the body of the tongue with increased use of intrinsic musculature. Most of the infants complete the gradual transition from suckling to true suck by 9 months of age. This is considered a critical step in the development of oral skills that will permit handling of thicker textures and spoon-feeding [35].

**Table 20.2** Phases of normal deglutition. (Reprinted with permission from Ref. [13], Table 15.2, p. 236)

Phase	Activities	Time
Pre-oral (voluntary)	Food introduced into oral cavity	Varies; depends on substance
Oral phase (voluntary/involuntary)	Bolus formation and passage to pharynx	Less than 1 s
Pharyngeal phase (involuntary)	Respiration ceases. Pharyngeal peristalsis. Epiglottis closes. Larynx closes, elevates, and draws forward. UES relaxes	1 s or less
Esophageal phase (involuntary)	Esophageal peristalsis. Opening of lower esophageal sphincter	8–20 s

As with sucking, chewing patterns emerge gradually during infancy. Between birth and 5 months of age, a phasic bite-release pattern develops. At this series of jaw openings and closing begins as a reflex and evolves into volitionally controlled bite. True chewing develops as the activity of the tongue, cheeks, and jaws coordinates to participate in the breakdown of solid food. The eruption of the deciduous teeth between ages of 6 and 24 months provides a chewing surface and increased sensory input to facilitate the development of chewing [35].

The concept of a “critical period” is relevant to feeding development. A critical period refers to a fairly well-delineated period of the time during which a specific stimulus must be applied to produce a particular action. After such a critical period, a particular behavior pattern can no longer be learned. Critical periods have been described for chewing and for taste. The critical period for chewing is that time following the disappearance of the tongue protrusion reflex that should occur around 6 months of age [36]. Critical periods have also been reported for introduction of tastes. Newborn infants detect sweet solutions, reject sour flavors, and are indifferent to the taste of salt [37]. McFarland and Tremblay emphasized that sensory experience is crucial to optimize pattern formation and brain development during the critical period for attainment of swallowing proficiency [38].

Current knowledge of the swallowing mechanism is derived mainly from radiographic studies, which have been in use since the early 1900s. Plain films of the pharynx were replaced in the 1930s by cineradiography, which was subsequently in the 1970s replaced by videofluoroscopy. Videofluoroscopy permits instant analysis of bolus transport, aspiration, and pharyngeal function [39]. Using this descriptive method, deglutition can be divided into four phases: oral preparatory phase, oral voluntary phase, pharyngeal phase, and esophageal phase (Table 20.2) [13, 40].

The oral preparatory phase occurs after food is placed into the mouth. The food is prepared for pharyngeal delivery by mastication and mixing with saliva. This is a highly coordinated activity that is rhythmic and controlled to prevent injury to the tongue. The tongue is elevated toward the palate by the combined actions of the digastric, genioglossus, geniohyoid, and mylohyoid muscles. Intrinsic tongue mus-

cles produce both the initial depression in the dorsum that receives the food and the spreading action that distributes the food throughout the oral cavity. The buccinator muscles hold food between the teeth in dentulous infants and help to generate suction in neonates. In this phase, the soft palate is against the tongue base, secondary to contraction of the palatoglossus muscles, which allows nasal breathing to continue [7, 41].

During the oral propulsive phase, the bolus is propelled into the oropharynx. The oral phase is characterized by elevation of the tongue and a posterior sweeping or stripping action produced mainly by the action of styloglossus muscles. This propels the bolus into the pharynx and triggers the “reflex swallow.” The receptors for this reflex are thought to be at the base of the anterior pillars, but there is evidence that others exist in the tongue base, epiglottis, and pyriform fossae. Sensory impulses for the reflex are conducted through the afferent limbs of cranial nerves V, IX, and X to the swallowing center. Oral transit time is less than 1 s [7, 42].

The pharyngeal phase of deglutition is the most complex and critical. The major component of the pharyngeal phase is the reflex swallow. This results from motor activity stimulated by cranial nerves IX and X. Swallowing is elicited involuntarily by afferent feedback from the oral cavity and has a duration of approximately 530 ms [1]. The reflex swallow may be triggered by a voluntary oral phase component or any stimulation of the afferent receptor in and around the anterior pillar [7]. Bolus passage through the pharynx is accompanied by soft palate elevation, lingual thrust, laryngeal elevation, and descent upper esophageal sphincter (UES) relaxation and pharyngeal constrictor peristalsis. The pharyngeal phase commences as the bolus head is propelled past the tongue pillars and finishes as the bolus tail passes into the esophagus [42]. Once it begins, the pharyngeal phase is very quick, 1 s or less [7]. It is characterized biomechanically by the operation of three valves and several propulsive mechanisms. During pharyngeal swallowing, respiration is inhibited centrally [43]. The larynx closes and the palate elevates to disconnect the respiratory tract. The UES opens to expose the esophagus. At the completion of the pharyngeal phase, the airway valves (larynx, palate) open and the UES closes so that respiration can resume [42].

Pharyngeal bolus transit occurs in two phases: an initial thrust phase and a mucosal clearance phase [44]. Bolus thrust, which propels most of the bolus into the esophagus, is provided by lingual propulsion, laryngeal elevation, and gravity. The tongue has been linked to a piston, pumping the bolus through the pharynx [45]. Patients with tongue impairment cannot generate large bolus driving forces despite an intact pharyngeal constrictor mechanism [46]. Laryngeal elevation creates a negative postcricoid pressure to suck the oncoming bolus toward the esophagus, and the elevated larynx holds the pharyngeal lumen open to minimize pharyngeal resistance [45].

As the bolus enters the pharynx and is stripped inferiorly by the combined effects of gravity, the negative pressure mentioned above, and the sequential contractions of the pharyngeal constrictors, the soft palate moves against the posterior pharyngeal wall to close off the nasopharyngeal port. The bolus divides around the epiglottis, combines and passes through the cricopharyngeal muscle, or UES [7].

UES refers to the high-pressure zone located in between the pharynx and the cervical esophagus. The physiological role of this sphincter is to protect against reflux of food into the airways as well as prevent entry of air into the digestive tract [47]. Posteriorly and laterally the cricopharyngeus muscle is a definitive component of the UES. Cricopharyngeus has many unique characteristics: it is tonically active, has a high degree of elasticity, does not develop maximal tension at basal length, and is composed of a mixture of slow- and fast-twitch fibers, with the former predominating. These features enable the cricopharyngeus to maintain a resting tone and yet be able to stretch open by distracting forces, such as a swallowed bolus and hyoid and laryngeal excursion. Cricopharyngeal, however, constitutes only the lower one-third of the entire high-pressure zone. The thyropharyngeus muscle accounts for the remaining upper two-thirds of the UES. The UES function is controlled by a variety of reflexes that involve afferent inputs to the motorneurons innervating the sphincter [47]. Based on functional studies, it is believed that the major motor nerve of the cricopharyngeal muscle is the pharyngoesophageal nerve. Vagal efferents probably reach the muscle by the pharyngeal plexus, using the pharyngeal branch of the vagi [48]. The superior laryngeal nerve may also contribute to motor control of the cricopharyngeus muscle [6]. Sensory information from the UES is probably provided by the glossopharyngeal nerve and the sympathetic nervous system. There is probably little or no contribution by the sympathetic nervous system to cricopharyngeal control [48].

The relaxation phase begins as the genioglossus and suspensory muscle pulls the larynx anteriorly and superiorly. The bolus is carried into the esophagus by a series of

contraction waves that are a continuation of the pharyngeal stripping action [7]. Proposed functions of the UES include prevention of esophageal distention during normal breathing and protection of the airway against aspiration following an episode of acid reflux [6, 48]. Qualitative abnormalities of UES have been documented in infants with reflux disease [49].

The esophageal phase occurs as the bolus is pushed through the esophagus to the stomach by esophageal peristalsis. Esophageal transit time varies from 8 to 20 s [26].

---

## Dysphagia

Dysphagia is an impairment of swallowing involving any structures of the upper gastrointestinal tract from the lips to the lower esophageal sphincter [50]. Dysphagia in children is generally classified as either oral dysphagia (abnormal preparatory or oral phase) or pharyngeal dysphagia (abnormal pharyngeal phase).

Oral dysphagia is seen most commonly in children with neurodevelopmental disorders. Infants with oral dysphagia often have an impaired oral preparatory phase. These children typically demonstrate poor lingual and labial coordination, resulting in anterior substance loss and poor labial seal for sucking or removing food from a spoon. Other abnormal patterns include jaws thrust and tongue thrust on presentation of food. Oral dysphagia also may involve the oral phase of swallowing. Children with impaired oral phase function often have difficulty in coordinating the “suck, swallow, breathe” pattern of early oral intake, resulting in diminished endurance during oral feeds. Apraxia of oral swallow as well as reduction of oral sensation also is common. Other deficits include reduced bolus formation and transport, abnormal hold patterns, incomplete tongue to palate contact, and repetitive lingual pumping [26].

Oropharyngeal dysphagia results from either oropharyngeal swallowing dysfunction or perceived difficulty in the process of swallowing. Major categories of dysfunction are: (1) an inability or excessive delay in initiation of pharyngeal swallowing, (2) aspiration of ingestate, (3) nasopharyngeal regurgitation, and (4) residue of ingestate within the pharyngeal cavity after swallowing. Each of these categories of dysfunction can be subcategorized using fluoroscopic and/or manometric data [29].

---

## Clinical Signs/Symptoms

Clinical signs and symptoms of sucking/swallowing disorders in infants and children are listed in Table 20.3 [13].



**Table 20.3** Clinical signs and symptoms of dysfunctional sucking/swallowing. (Reprinted with permission from Ref. [13], Table 15.3, p. 239)

<i>Clinical signs</i>
Failure to thrive
Meal-time distress
Refusing food
Nasal regurgitation
Wet or hoarse voice
Drooling
Spitting
Vomiting
Gastroesophageal or pharyngeal reflux
<i>Symptoms</i>
Oral-tactile hypersensitivity
Feeling of obstruction
Odynophagia
Atypical chest pain
<i>Respiratory manifestations</i>
Coughing
Choking
Stridor
Change in respiration pattern after swallowing
Apnea and bradycardia (predominantly in infants)
Noisy breathing after feeding
Chronic recurrent wheezing
Chronic recurrent bronchitis, pneumonia, and atelectasis

## Complications

### Malnutrition

In the severely affected child with impaired swallowing, poor oral and/or pharyngeal function may lead to decreased energy intake as a consequence of prolonged feeding time and the inability to ingest adequate volumes, and malnutrition may result [6]. Malnutrition has many adverse effects. The most significant effects are on behavior and immune status. Malnutrition negatively influences immune status. This leads to recurrent infections that increase caloric requirements but decrease intake, leading to worsening nutritional status. In addition, malnutrition causes behavioral apathy, weakness, and anorexia, which can all profoundly affect feeding, and secondarily, nutritional status. Thus, although malnutrition is often a direct result of poor feeding skills, it can also have compounding, and even perpetuating, effect on feeding problems in children [26].

### Sialorrea

Sialorrea, or excessive drooling, is defined as the unintentional loss of saliva and other oral contents from the mouth.

Drooling usually occurs in patients with neurologic disease complicated by abnormalities of the oral phase of deglutition. Clinical complications of drooling include soaking of clothes, offensive odors, macerated skin, and if “posterior” drooling occurs, aspiration [51].

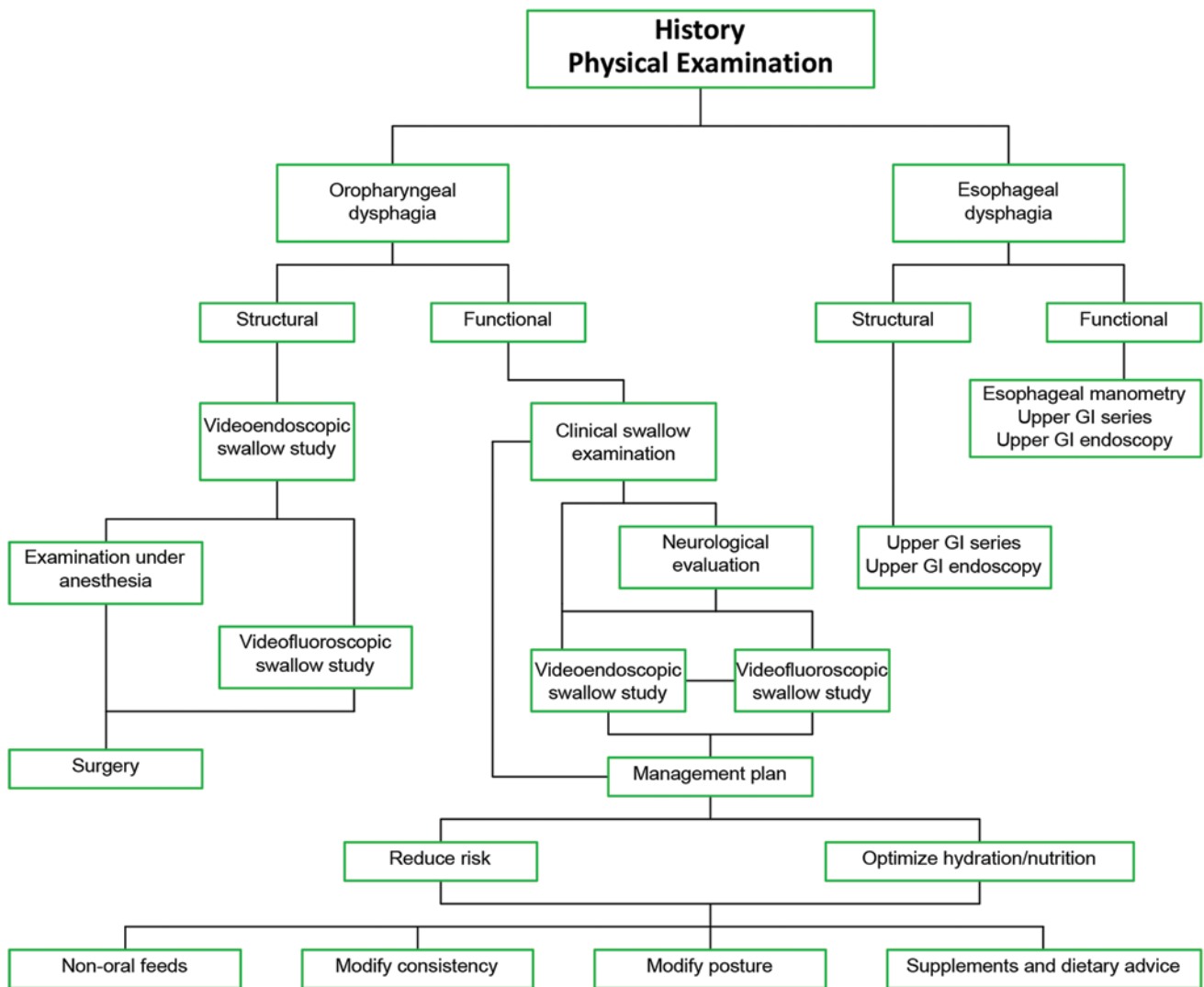
## Respiratory Complications

Respiratory complications of swallowing disorders include apnea and bradycardia, choking episodes, chronic or recurrent pneumonia, bronchitis, and atelectasis [52].

Apnea and bradycardia may result from stimulation of laryngeal chemoreceptors without evidence of aspiration or as a consequence of hypoxemia. Hypoxemia may result from the effects of direct aspiration on gas exchange, from apnea triggered by laryngeal and nasopharyngeal chemoreceptors, or in patients with compromised lung function as a result of normal decrease in minute ventilation that occurs with the suckle feeding [53–55]. Symptoms such as chronic recurrent coughing, choking, and postprandial congestion or wheezing generally indicate the occurrence of aspiration. Infants, especially premature infants, appear to be at an increased risk of respiratory disease from dysfunctional swallowing [9]. Clinical manifestations of dysfunctional sucking/swallowing in infants are primarily apnea and bradycardia during feeding, although chronic/recurrent respiratory problems (congestion, cough, and wheezing) are also seen [52]. Congested or noisy breathing during and following feeding is also a common complaint of parents in infants with dysfunctional swallowing. Dysphagia can also be an important but under-recognized cause of chronic/recurrent bronchitis, asthma, and pneumonia in infants [9].

Respiratory disease secondary to dysphagia in an older child is typically seen in a neurologically impaired host [56, 57]. Apnea and bradycardia are uncommon in older children. Bronchitis, pneumonia, atelectasis, and recurrent wheezing are more likely to be seen in this population. Feeding and swallowing evaluation should be considered in those with CNS injury affecting cranial nerve function and difficult-to-control chronic/recurrent bronchitis, wheezing, pneumonia, or asthma. Tracheobronchomalacia, a complication of chronic inflammation of the major airways, occurs commonly. Dysfunctional swallowing is also encountered in children with a tracheostomy. The tracheostomy may interfere with normal laryngeal function during swallowing and predispose to aspiration [50].

Aspiration may also occur in children with disorders of swallowing after an episode of gastroesophageal reflux; also, acid reflux may result in bronchospasm, pneumonia, or apnea [58, 59].



**Fig. 20.1** Flowchart for the investigation and management of dysphagia in children. (Adapted by permission from BMJ Publishing Group Limited, from Ref. [50] and reprinted with permission from Ref. [13])

The most obvious sign that a person may have aspirated is the post-swallow cough, but in the swallowing-impaired children other more insidious indicators may be present. “Silent aspiration” with no clinical signs can account for over half of all cases of radiologically defined aspiration [50, 60].

## Diagnosis

Feeding disorders and dysphagia in infants and children can be both physiological and behavioral in nature [61].

The evaluation of feeding and swallowing dysfunction is best performed as a multidisciplinary process with coordinated input from a variety of team members, including pediatricians, pediatric gastroenterologists, developmental pediatricians, speech–language pathologists, occupational therapist, and pediatric dietitians [62]. The goals of this

evaluation including the following: (1) ascertain whether oropharyngeal dysphagia is likely and identify the likely etiology, (2) identify structural etiologies of oropharyngeal dysfunction, (3) ascertain the functional integrity of the oropharyngeal swallow, (4) evaluate the risk of aspiration pneumonitis, and (5) determine if the pattern of dysphagia is amenable to therapy [63].

The investigation and management of swallowing disorders are summarized in Fig. 20.1 [13, 50].

## History

A comprehensive history, obtained from individuals directly involved in caring for the child (e.g., parents, feeding specialist), is essential in evaluating children with swallowing disorders. The evaluation begins with a focused feeding

history, including current diet, textures, and route and time of administration, modifications, and feeding position. Medical comorbidities that may affect swallowing need to be investigated.

Child's caretakers also should be questioned regarding associated symptoms such as oral aversion, weak sucking, irritable behavior, gagging and choking, and disruptions in breathing or apnea. Postural or positional change during feeding also may be reported in children with dysphagia. Odynophagia and emesis may be related with pharyngeal and/or esophageal disorders. A history of recurrent pneumonia may indicate chronic aspiration; a history of stridor in relation to feeding may indicate a glottic or subglottic abnormality contributing to feeding disorders. Determining whether these symptoms occur before, during, or after the swallow helps localize the affected phase [27, 28].

In addition, nutritional and psychological assessment should be evaluated. Many patients with swallowing disorders have concurrent illness that may increase metabolic needs. Psychological assessments help to identify behavioral and parental factors that may be contributing to a feeding disorder. Psychosomatic causes of dysphagia should be considered in adolescents with dysphagia [7, 64, 65].

## Physical and Clinical Evaluation

In dysphagic patients, physical examination aims to: (1) characterize the underlying systemic underlying systemic or metabolic disease when present; (2) localize the neuroanatomical level and severity of a causative neurological lesion when present; and (3) detect adverse sequelae such as aspiration pneumonia or nutritional deficiency [29].

There are four key questions that physicians and nurses in primary care can ask parents when an infant or young child presents at the office or clinic with parental concerns related to feeding [66].

The answers help determine whether a comprehensive clinical feeding and swallowing assessment is needed, even though the answers do not necessarily define the problem:

How long do mealtimes typically take? If more than about 30 min on any regular basis, there is a problem. Prolonged feeding times are major red flags pointing to the need for further investigation:

- Are mealtimes stressful? Regardless of descriptions of factors that underlie the stress, further investigation is needed. It is very common for parents to state that they "just dread mealtimes."
- Does the child show any signs of respiratory stress? Signs may include rapid breathing, gurgly voice quality, nasal congestion that increases as the meal progresses, and panting by an infant with nipple feeding. Recent upper

respiratory illness may be a sign of aspiration with oral feeds, although there may be other causes.

- Has the child *not* gained weight in the past 2–3 months? Steady appropriate weight gain is particularly important in the first 2 years of life for brain development as well as overall growth. A lack of weight gain in a young child is like a weight loss in an older child or adult.

The physical examination views the whole child and specifically focuses on the upper aerodigestive tract, beginning with an examination for structural and functional abnormalities. Oral cavity anatomic abnormalities, such as ankyloglossia, cleft lip or palate, or macroglossia, need to be excluded [7]. The palatal gag is perhaps the most assessed reflex and should be evaluated [51]. A hyperactive gag can result in significant feeding difficulties, and in the past an absent gag reflex was viewed as an indication to stop oral feeding [9, 56].

It is critical that observation of the feeding process be included [62]. This part of the examination is best performed in conjunction with a feeding and swallowing specialist, such as a speech–language pathologist or an occupational therapist. This examination includes assessments of posture, positioning, patient motivation, oral function, efficiency of oral intake, and clinical signs of safety. During the feeding trial, the presence of abnormal movements such as jaw thrust, tongue thrust, tonic bite reflex, and jaw clenching is noted. A variety of therapeutic positions, techniques, and adaptive feeding utensils may be used [6, 27].

A variety of assessment scales may be used to detail and quantitate results of the swallowing evaluation. However, all assessments are based on similar observation of feeding structure and function [67].

Usually, a careful developmental, medical, and feeding history provides clues to the diagnosis that guide the selection of further diagnostic tests. Only after all reasonable physical causes have been ruled out should a feeding or swallowing disorder be attributed to a purely behavioral cause [7].

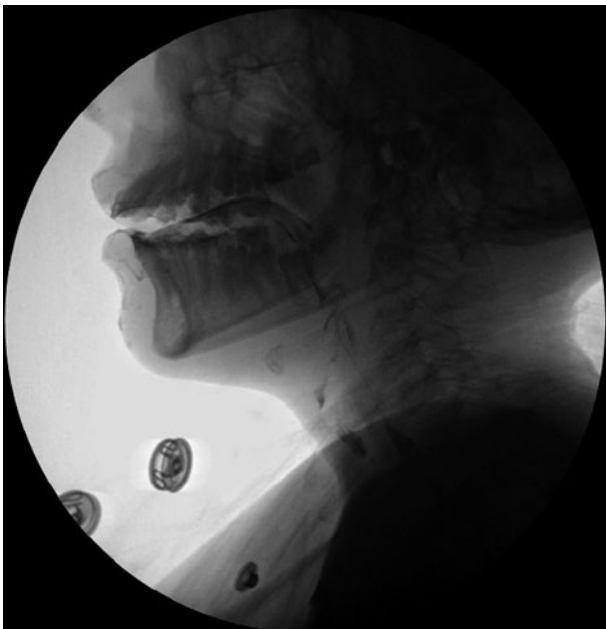
## Diagnostic Tests

**Radiographic Assessment** The videofluoroscopy represents the gold standard method for evaluation of children with swallowing disorders. A videofluoroscopic swallow study is ideally performed by a consultant radiologist and specialist and speech and language therapist [68]. A series of swallows of varied volumes and consistencies of contrast material are imaged in a lateral projection, framed to include the oropharynx, palate, proximal esophagus, and proximal airway. Studies are recorded on videotape to permit instant replay, in slow motion if necessary, and examination of both the presence and mechanism of swallowing dysfunction.



As such, the videofluoroscopic study provides evidence of all four categories of oropharyngeal swallowing disorders: (1) inability or excessive delay in initiation of pharyngeal swallowing, (2) aspiration of ingestate, (3) nasopharyngeal regurgitation, and (4) residue of ingestate within the pharyngeal cavity after swallowing. Furthermore, the procedure allows for testing of the efficacy of compensatory dietary modifications, postures, swallowing maneuvers, and facilitatory techniques in correction or observed dysfunction. Generally, the videofluoroscopic evaluation is completed by esophagography to evaluate the esophageal phase of deglutition (Fig. 20.2) [29]. A nasogastric tube does not alter the findings of videofluoroscopic swallowing study and does not increase the risk of aspiration; however, it might increase the incidence of respiratory compromise when aspiration is present [69].

**Ultrasonography** Ultrasound imaging has been used to a limited extent in the assessment of oral phase dysphagia. Using a transducer positioned in the submental region, ultrasonography allows observation of the motion of structures in the oral cavity such as the tongue and floor of the mouth during feeding and deglutition, but lacks sensitivity in visualizing pharyngeal motion and determining whether aspiration has occurred. Ultrasonography represents the only method of imaging that can study infants during breastfeeding and may be particularly useful in distinguishing an infant's inability to latch on from maternal factors contributing to feeding difficulties [14]. Unfortunately, laryngeal



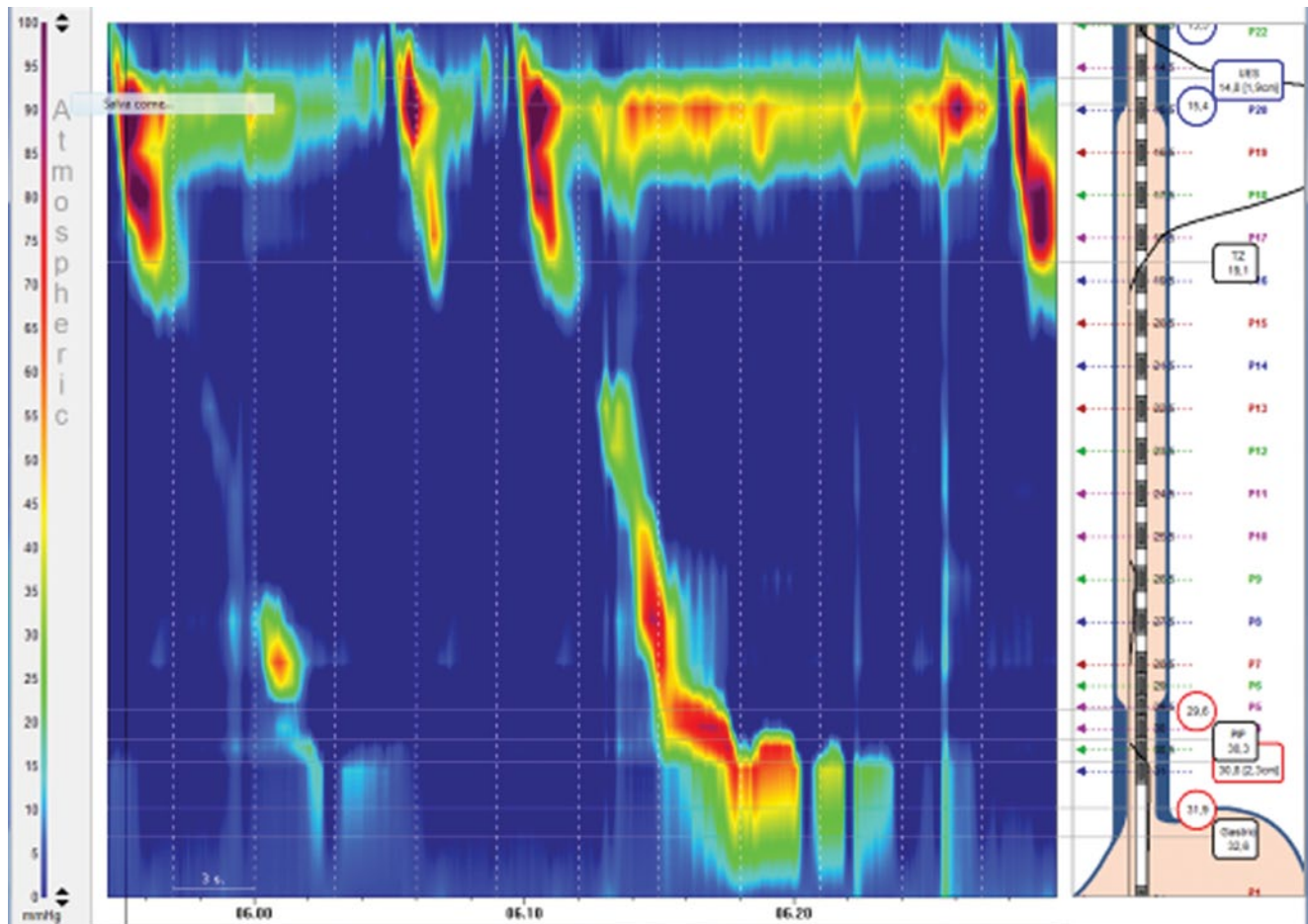
**Fig. 20.2** Lateral fluoroscopic projection of a child showing contrast material in the valleculas, pyriform sinuses, laryngeal vestibule, and esophagus

penetration and aspiration are not easily detected because of the shadows cast by the laryngeal structures [9, 34, 70].

**Pharyngeal Manometry** Intraluminal manometry, performed using a transnasally positioned manometric assembly, can quantify the strength of pharyngeal contraction, the completeness of UES relaxation, and relative time of these two events. Most studies indicate that the manometry of the UES and pharynx provides useful information primarily in patients that have symptoms of oropharyngeal dysfunction. The coordination of muscle activity at various levels can be obtained by simultaneous recording of pressure in the pharynx, at the level of cricopharyngeus, and in the esophagus. Anatomic references are not available with this technique [49, 71]. Recently, manometry equipment has evolved to allow more precise and detailed evaluation of esophageal function with high-resolution manometry and esophageal pressure topography plotting [72] (Fig. 20.3).

**Fiberoptic Endoscopic Examination** Pediatric fiberoptic endoscopic examination (FEES) is a relatively new diagnostic method to add to and complement the current armamentarium of techniques for evaluating dysphagia and/or aspiration. FEES is performed by passing a flexible laryngoscope into the oropharynx after anesthetizing the nares and nasopharynx [73]. It provides the ability to diagnose many of the laryngeal disorders that may affect the child, while at the same time evaluating the swallowing mechanism itself. The procedure involves five components: assessment of anatomy as it affects swallowing, evaluation of movement and sensation of critical structures, assessment of secretion management, direct assessment of swallowing function for food and liquid, and patients' response to therapeutic maneuvers. In experienced hands, this test can be performed in children with minimal discomfort [74, 75].

**Scintigraphy** Scintigraphy is a radionuclide evaluation using Technetium-99m-labeled sulfur colloid mixed in the infant's formula. It has been proposed as an alternative and perhaps more sensitive way of quantifying aspiration, transit times, gastroesophageal reflux, and pharyngeal residue. Based on a case report, the radionuclide salivagram has also been used to document aspiration of saliva. The major limitations of this technique are the poor definition of the anatomy and the poor sensitivity for detecting aspiration during swallowing in known aspirators. At the present, the use of this technique in pediatric age is limited [75, 76].



**Fig. 20.3** Esophageal pressure topography plotting: complete peristaltic chain studied with a 21-lm catheter. The three *inter-segmental* troughs are indicated in the figure, and the pressure amplitudes repre-

sented by the isobaric contour regions are shown in the *color legend* (in mmHg above gastric baseline pressure; pressures below the first isobaric contour are shown in *blue*). *SW* swallow

## Treatment Options

Optimal management strategies are critical for infants and children with feeding and swallowing problems. The management of swallowing dysfunction involves a team approach. Individuals involved in addition to the medical team include a swallowing expert (speech–language pathologist or occupational therapist), a nutritionist, and the family. Since swallowing abnormalities arise from a diverse group of underlying disorders, management techniques must be individualized. This heterogeneity is also reflected in the fact that patients have different potentials to recovery [6].

Although total oral feeding may not be a realistic goal, it is the universal hope of caregivers. Professionals are obligated to point out prerequisites for oral feeding and to discuss the probability that an individual child may reach the goal. These management decisions are typically made on the basis of clinical observations and assessments.

In addition, important information is obtained through an instrumental assessment by videofluoroscopic swallows study. A methodical videofluoroscopic swallowing study: (1) defines the anatomy of the oropharynx; (2) detects dysfunction as evident by aspiration, poor clearance, or poor control of the bolus; (3) determines the mechanism responsible for the dysfunction; and (4) examines the short-term effects of the therapeutic strategies designed to eliminate or compensate for that dysfunction [77]. Management decisions may incorporate nutritive recommendations, medical and surgical decisions, position guidelines, oral-motor/swallow practice, and behavioral intervention [78].

The clinical and instrumental evaluation of children with sucking and swallowing disorders should allow for the recognition of treatable anatomic or inflammatory lesions.

A child may refuse to eat even if his anatomic abnormality has been corrected because of learned aversion to feeding. Behavior therapy often can overcome this type of conditioned food refusal [7, 65].

**Table 20.4** Swallowing strategies for pediatric dysphagia. (Reprinted with permission from Ref. [13], Table 15.4, p. 244)

Behavioral training	
Dietary modification	Thickened liquids Thin liquids
Proper intrabolus placement	Modify feeding utensils and bolus presentation
Swallowing exercises	Supraglottic swallow Supersupraglottic swallow Effortful swallow Mendelsohn maneuver
Modification in body tone posture seating and positioning	Head tilt Chin tuck Head rotation Lying on side, elevation
Suckle-feeding-valved feeding bottle	–
Cricopharyngeal myotomy	–
Facilitatory techniques	Biofeedback Thermal stimulation Gustatory stimulation
Provide alternate means of enteral nutrition	Nasogastric feeding Gastrostomy tube (surgical or endoscopic)

Various therapeutic approaches may improve the efficiency and safety of feeding. Management techniques involve devising compensatory strategies to minimize swallowing-related complications [79].

These include changing the textures of foods; pacing of feeding; changing the bottle or utensils; and changing the alignment of the head, neck, and body with feeding (Table 20.4) [13, 72].

Frequently, children with severe anatomic disorders but normal neurological function develop their own adaptive strategies to allow for safe oral feeding. Unfortunately, many children with feeding disorders have non-correctable neurologic or anatomic abnormalities that make oral feeding difficult or unsafe. Some patients cannot obtain adequate nutrition by mouth because of a risk for aspiration. Thus, supplying a portion of patient's nutrition by nasogastric or gastrostomy feeding may be beneficial [7]. For those children who have been intubated, management includes teaching techniques that will facilitate transitioning from non-oral to oral feeding. However, there is little evidence that non-oral feeding reduces or eliminates the risk of aspiration [80–82].

The strongest evidence-based recommendation that can be made pertains to diet modification. Furthermore, the literature provides reasonable evidence of the plausibility of swallowing therapy but minimal evidence of efficacy. Nonetheless, although no hard evidence supports its efficacy, the available data are inconclusive and swallowing therapy has not been proven ineffective. Thus, the current weight of

opinion, combined with the convincing demonstration of biological plausibility for specific techniques and the consistency of low-grade evidence, is the basis to recommend that swallowing therapy should be used. Large-scale randomized, controlled trials are needed to clarify the current recommendations [29].

## Prognosis

Prognosis depends on underlying conditions that predispose to impaired sucking and swallowing. However, the early recognition of feeding problems, diagnosis of underlying disorders, and appropriate intervention improve outcomes for the child and the family.

## References

1. da Costa SP, van den Engel-Hoek L, Bos AF. Sucking and swallowing in infants and diagnostic tools. *J Perinatol.* 2008;28:247–57.
2. Delaney AL, Arvedson JC. Development of swallowing and feeding: prenatal through first year of life. *Dev Disabil Res Rev.* 2008;14:105–17.
3. Presse J, Kikano G. An overview of pediatric dysphagia. *Cin Pediatr.* 2009;48:247–51.
4. Arvedson J, Clark H, Lazarus C, Schooling T, Frymark T. Evidence-based systematic review: effect of oral motor interventions on feeding and swallowing in preterm infants. *Am J Speech Lang Pathol.* 2010;19:321–40.
5. Logemann JA. Evaluation and treatment of swallowing disorders. 2nd ed. Austin: Pro-Ed; 1998.
6. Tuchman DN. Disorders of deglutition. In: Walker WA, Durie PR, Walker-Smith JA, Watkins JB, editors. *Pediatric gastrointestinal disease.* 3rd ed. Hamilton: BC Decker; 2000. p. 277–88.
7. Rudolph CD, Link DT. Feeding disorders in infants and children. *Pediatr Clin North Am.* 2002;49(1):97–112.
8. Del Giudice E, Staiano A, Capano G, Romano A, Florimonte L, Miele E, Ciarla C, Campanozzi A, Crisanti AF. Gastrointestinal manifestations in children with cerebral palsy. *Brain Dev.* 1999;21(5):307–11.
9. Slattery J, Morgan A, Douglas J. Early sucking and swallowing as predictors of neurodevelopmental outcome in children with neonatal brain injury: a systematic review. *Dev Med Child Neurol.* 2012;54:796–806.
10. Miller CK. Updates on paediatric feeding and swallowing. *Curr Opin Otolaryngol Head Neck Surg.* 2009;17:194–9.
11. Loughlin GM, Lefton-Greif MA. Dysfunctional swallowing and respiratory disease in children. *Adv Pediatr.* 1994;41:135–62.
12. Cohen SR. Difficulty with swallowing. In: Bluestone CD, Stool SE, Arjona SK, editors. *Pediatric otolaryngology.* Philadelphia: WB Saunders; 1983. p. 903–11.
13. Miele E, Staiano A. Disorders of sucking and swallowing. In: Guandalini S, editor. *Textbook of pediatric gastroenterology and nutrition.* London: Taylor & Francis; 2004. p. 233–46.
14. Wolff Ph. The serial organization of sucking in the young infant. *Pediatrics.* 1968;42:943–56.
15. Mizuno K, Ueda A. The maturation and coordination of sucking, swallowing, and respiration in preterm infants. *J Pediatr.* 2003;142(1):36–40.
16. Mathew OP. Science of bottle-feeding. *J Pediatr.* 1991;119:511–9.



17. Derkay CS, Schecter GL. Anatomy and physiology of pediatric swallowing disorders. *Otolaryngol Clin North Am.* 1998;31:397-404.
18. Mistry S, Hamdy S. Neural control of feeding and swallowing. *Phys Med Rehabil Clin N Am.* 2008;19:709-28.
19. Barlow SM, Lund JP, Estep M, Kolta A. Central pattern generators for speech and orofacial activity. In: Brudzynski SM, editor. *Handbook of mammalian vocalization.* Oxford: Elsevier; 2009. p. 1-3317.
20. Lund JP, Kolta A. Generation of the central masticatory pattern and its modification by sensory feedback. *Dysphagia.* 2006;21(3):167-74.
21. Barlow SM. Central pattern generation involved in oral and respiratory control for feeding in the term infant. *Curr Opin Otolaryngol Head Neck Surg.* 2009;17(3):187-93.
22. Miller AJ. Deglutition. *Physiol Rev.* 1982;62:129-84.
23. Morrell RM. The neurology of swallowing. In: Groher ME, editor. *Dysphagia and management.* Boston: Butterworths; 1984. p. 3.
24. Bosma JF. Postnatal ontogeny of performances of the pharynx, larynx, and mouth. *Am Rev Respir Dis.* 1985;131(5):S10-5.
25. Tuchman DN. Dysfunctional swallowing in the pediatric patient: clinical considerations. *Dysphagia.* 1988;2(4):203-8.
26. Stevenson RD, Allaire JH. The development of normal feeding and swallowing. *Pediatr Clin North Am.* 1991 Dec;38(6):1439-53.
27. Gewolb IH, Vice FL, Schweitzer-Kenney EL, Taciak VL, Bosma JF. Development patterns of rhythmic suck and swallow in preterm infants. *Dev Med Child Neurol.* 2001;43:22-7.
28. Moore KL. The developing human: clinically oriented embryology. 4th ed. Philadelphia: WB Saunders; 1988.
29. Morris SE, Klein MD. Pre-feeding skills: a comprehensive resource for feeding development. Tucson: Therapy Skill Builders; 1997.
30. Lau C, Schanler RJ. Oral feeding in premature infants: advantage of a self-paced milk flow. *Acta Paediatr.* 2000;89(4):453-9.
31. Milla PJ. Feeding, tasting, and sucking. In: Walker-Smith WA, Watkins JB, editors. *Pediatric gastrointestinal disease.* Philadelphia: BC Decker; 1991. p. 217-23.
32. Lemons PK, Lemons JA. Transition to breast/bottle feedings: the premature infant. *J Am Coll Nutr.* 2001;2:126-35.
33. Palmer MM, Crawley K, Blanco I. The neonatal oral-motor assessment scale: a reliability study. *J Perinatol.* 1993;13(1):28-35.
34. Bu'Lock F, Woolridge MW, Baum JD. Development of co-ordination of sucking, swallowing and breathing: ultrasound study of term and preterm infants. *Dev Med Child Neurol.* 1990;32(8):669-78.
35. Darrow DH, Harley CM. Evaluation of swallowing disorders in children. *Otolaryngol Clin North Am.* 1998 Jun;31(3):405-18.
36. Illingworth RS, Lister J. The critical or sensitive period, with special reference to certain feeding problems in infants and children. *J Pediatr.* 1964;5:839-48.
37. Mennella JA, Beauchamp GK. Development and bad taste. *Pediatr Asthma Allergy Immunol.* 1998;12:161-4.
38. McFarland DH, Tremblay P. Clinical implications of cross-system interactions. *Semin Speech Lang.* 2006;27(4):300-9.
39. Cook IJ, Kahrilas PJ. AGA technical review on management of oropharyngeal dysphagia. *Gastroenterology.* 1999;116(2):455-78.
40. Logemann JA. Evaluation and treatment of swallowing disorders. San Diego: College Hill; 1983.
41. Dodds WJ, Stewart ET, Logemann JA. Physiology and radiology of the normal oral and pharyngeal phases of swallowing. *AJR Am J Roentgenol.* 1990;154(5):953-63.
42. Mendelsohn M. New concepts in dysphagia management. *J Otolaryngol.* 1993;22 Suppl 1:1-24.
43. Doty R, Bosma JF. An electromyography analysis of reflex deglutition. *J Neurophysiol.* 1956;19:44-60.
44. McConnel FMS. Analysis of pressure generation and bolus transit during pharyngeal swallowing. *Laryngoscope.* 1988;98:71-8.
45. McConnel FM, Cerenko D, Mendelsohn MS. Dysphagia after total laryngectomy. *Otolaryngol Clin North Am.* 1988;21(4):721-6.
46. Curtis DJ, Cruess DF, Dachman AH. Normal erect swallowing. Normal function and incidence of variations. *Invest Radiol.* 1985;20(7):717-26.
47. Sivarao DV, Goyal RK. Functional anatomy and physiology of the upper esophageal sphincter. *Am J Med.* 2000;108 (Suppl 4a):27S-37.
48. Palmer ED. Disorders of the cricopharyngeus muscle: a review. *Gastroenterology.* 1976;71(3):510-9.
49. Staiano A, Cucchiara S, De Vizia B, Andreotti MR, Auricchio S. Disorders of upper esophageal sphincter motility in children. *J Pediatr Gastroenterol Nutr.* 1987;6(6):892-8.
50. Leslie P, Carding PN, Wilson JA. Investigation and management of chronic dysphagia. *Br Med J.* 2003;326(7386):433-6.
51. Myer CM. Sialorrhea. *Pediatr Clin of North Am.* 1989;36:1495-500.
52. Loughlin GM. Respiratory consequences of dysfunctional swallowing and aspiration. *Dysphagia.* 1989;3(3):126-310.
53. Durand M, Leahy FN, MacCallum M, Cates DB, Rigatto H, Chernick V. Effect of feeding on the chemical control of breathing in the newborn infant. *Pediatr Res.* 1981;15(12):1509-12.
54. Thach BT. Maturation and transformation of reflexes that protect the laryngeal airway from liquid aspiration from fetal to adult life. *Am J Med.* 2001;111 (Suppl 8A):69S-77.
55. Hoekstra RE, Perkett EA, Dugan M, Knox GE. Follow-up of the very low birth weight infant (less than 1251 grams). *Minn Med.* 1983;66(10):611-3.
56. Tuchman DN. Cough, choke, spitter: the evaluation of the child with dysfunctional swallowing. *Dysphagia.* 1989;3(3):111-6.
57. Rogers BT, Arvedson J, Msall M, Demerath RR. Hypoxemia during oral feeding of children with severe cerebral palsy. *Dev Med Child Neurol.* 1993;35(1):3-10.
58. Berquist WE, Ament ME. Upper GI function in sleeping infants. *Am Rev Respir Dis.* 1985;131(5):S26-9.
59. Boyle JT, Tuchman DN, Altschuler SM, Nixon TE, Pack AI, Cohen S. Mechanisms for the association of gastroesophageal reflux and bronchospasm. *Am Rev Respir Dis.* 1985;131(5):S16-20.
60. Smith CH, Logemann JA, Colangelo LA, Rademaker AW, Pauloski BR. Incidence and patient characteristics associated with silent aspiration in the acute care setting. *Dysphagia.* 1999 Winter;14(1):1-7.
61. Sonies BC. Swallowing disorders and rehabilitation techniques. *J Pediatr Gastroenterol Nutr.* 1997;25 (Suppl 1):S32-3.
62. Kramer SS, Eicher PM. The evaluation of pediatric feeding abnormalities. *Dysphagia.* 1993;8(3):215-24.
63. American Gastroenterological Association. American gastroenterological association medical position statement on management of oropharyngeal dysphagia. *Gastroenterology.* 1999;116:452-4.
64. Kovar AJ. Nutrition assessment and management in pediatric dysphagia. *Semin Speech Lang.* 1997 Feb;18(1):39-49.
65. Babbitt RL, Hoch TA, Coe DA, Cataldo MF, Kelly KJ, Stackhouse C, Perman JA. Behavioral assessment and treatment of pediatric feeding disorders. *J Dev Behav Pediatr.* 1994 Aug;15(4):278-91.
66. Arvedson JC. Swallowing and feeding in infants and young children. *GI Motility Online.* 2006. doi:10.1038.
67. Gisel EG, Alphonse E, Ramsay M. Assessment of ingestive and oral praxis skills: children with cerebral palsy vs. controls. *Dysphagia.* 2000 Fall;15(4):236-44.
68. Ekberg O, Olsson R, Bulow M. Radiologic evaluation of dysphagia. *Abdom Imaging.* 1999;24(5):444.
69. Mutaz Alnassar, Kamaldine Oudjhane, Jorge Davila. Nasogastric tubes and videofluoroscopic swallowing studies in children. *Pediatr Radiol.* 2011;41:317-21.
70. Rudolph CD. Feeding disorders in infants and children. *Pediatrics.* 1994;125(6 Pt 2):S116-24.
71. Hila A, Castell JA, Castell DO. Pharyngeal and upper esophageal sphincter manometry in the evaluation of dysphagia. *J Clin Gastroenterol.* 2001;33(5):355-61.

72. Goldani HA, Staiano A, Borrelli O, Thapar N, Lindley KJ. Pediatric esophageal high-resolution manometry: utility of a standardized protocol and size-adjusted pressure topography parameters. *Am J Gastroenterol*. 2010;105(2):460–7.
73. Broniatowski M, Sonies BC, Rubin JS, Bradshaw CR, Spiegel JR, Bastian RW, Kelly JH. Current evaluation and treatment of patients with swallowing disorders. *Otolaryngol Head Neck Surg*. 1999;120(4):464–73.
74. Langmore SE, Schatz K, Olsen N. Fiberoptic endoscopic examination of swallowing safety: a new procedure. *Dysphagia*. 1988;2(4):216–9.
75. Hamlet SL, Mutz J, Patterson, R., Jones L. Pharyngeal transit time: assessment with videofluoroscopic and scintigraphic techniques. *Dysphagia* 1989;4:4–7.
76. Silver KH, Nostrand DV. Scintigraphic detection of salivary aspiration: description of a new diagnostic technique and case reports. *Dysphagia*. 1992;7:45–9.
77. Logemann JA. Role of the modified barium swallow in management of patients with dysphagia. *Otolaryngol Head Neck Surg*. 1997;116:335–8.
78. Arvedson JC. Management of pediatric dysphagia. *Otolaryngol Clin North Am*. 1998;31:453–76.
79. Helfrich-Miller KR, Rector KL, Straka JA. Dysphagia: its treatment in the profoundly retarded patient with cerebral palsy. *Arch Phys Rehabil*. 1986;67:520–5.
80. Croghan JE, Burke EM, Caplan S, Denman S. Pilot study of 12-month outcomes of nursing home patients with aspiration on videofluoroscopy. *Dysphagia*. 1994 Summer;9(3):141–6.
81. Groher ME. Bolus management and aspiration pneumonia in patient with pseudobulbar dysphagia. *Dysphagia*. 1987;1:215–6.
82. Shaker R, Easterling C, Kern M, Nitschke T, Massey B, Daniels S, Grande B, Kazandjian M, Dikeman K. Rehabilitation of swallowing by exercise in tube-fed patients with pharyngeal dysphagia secondary to abnormal UES opening. *Gastroenterology*. 2002;122(5):1314–21.

---

### Additional Educational Resources:

Resource center: The Dysphagia Research Society is organized exclusively for charitable, educational and scientific purposes (<http://www.dysphagiaresearch.org/>) -this multidisciplinary website has a wealth of information on dysphagia, references to texts, archives, and links to other related sites; user friendly and very comprehensive.