Dysphagia 8:215–224 (1993)

Dysphagia

The Evaluation of Pediatric Feeding Abnormalities

Sandra S. Kramer, MD and Peggy Monahan Eicher, MD

Department of Radiology and Division of Child Development and Rehabilitation, Department of Pediatrics, Children's Hospital of Philadelphia; and University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, USA

Abstract. An appreciation of normal age-related feeding behavior is a prerequisite for the study of pediatric feeding disorders. When faced with a child with dysphagia, the diagnostic evaluation including history, physical and developmental exam, and feeding observation, followed by appropriate diagnostic tests forms the basis for understanding the underlying pathophysiology and the anticipated developmental progression. It makes possible the formulation and implementation of an optimal treatment strategy, which can be monitored for results and modified when needed. Four case examples are provided.

Key words: Dysphagia — Feeding problems in children — Deglutition — Deglutition disorders.

It is hard to be a pioneer at the end of the twentieth century. The easy things have long ago been done; what remains is the difficult, the obscure, and the complex. This was fertile territory for Dr. Martin Donner and appropriate for his talents. And when he founded, along with his associates, the first multidisciplinary Swallowing Center in the country at Johns Hopkins, he took care to include Pediatrics. We, his pediatric friends and colleagues, are honored in turn to contribute to this journal issue dedicated to him.

Dr. Donner, a pioneer in the study of dysphagia, understood the complexity of most feeding difficulties and the necessity of a coordinated approach by specialists from different disciplines to correctly diagnose the problem, appreciate its effect on the patient's daily life, and construct a successful treatment plan. He realized that the various viewpoints represented in the team could compound our knowledge in each individual case, and new understanding could be applied to other patients.

The multidisciplinary approach is particularly pertinent to pediatric feeding problems which are often more complicated because systems are already stressed by the demands of growth and development; nutritional needs are usually greater; anatomic structures grow and their relationships change; and maturation of the central nervous system is evolving. Although there may be similarities, dysfunctional feeding in children is very likely to be different from that of adults. For example, in congenital problems, feeding skills must be learned, without a background of existing skills, by children who may have neurological impairment and/or anatomic anomalies [1]. Such a situation is often further complicated by the child's inability to communicate clinical information or fully comprehend instructions.

The purpose of this paper is to review the diagnostic evaluation of children with dysphagia, emphasizing certain aspects of presentation, history, physical exam, and observation of feeding performance which will aid in the development of a working impression or hypothesis. Diagnostic exams used to clarify the hypothesis, including videofluoroscopic swallowing studies, will be briefly covered. The goal is to understand the underlying pathophysiology and expected developmental progression so that an optimal treatment strategy can be designed and monitored.

Normal Age-Related Feeding Behavior

Normal adult swallowing function is complex, but being fully mature, is stable. In children, growth and development create an undercurrent of change which dynamically affects swallowing behavior as it evolves from suckle feeding in infancy to the complete competence of

Address offprint requests to: Sandra S. Kramer, M.D., Department of Radiology, Children's Hospital of Philadelphia, 34th Street and Civic Center Boulevard, Philadelphia, PA 19104, USA

the adult [2–20]. An appreciation of this maturation process is basic to the understanding of all children's feeding behavior.

Swallowing in utero has been observed in fetuses as young as 16 weeks gestational age, and it plays an important role in the maintenance of amniotic fluid balance [21, 22]. In the second and third trimesters, the infant progressively displays elements of suckling activity, which have been shown by ultrasound to increase towards the end of gestation.

In premature infants, oral sucking behavior is poorly coordinated, inefficient, and intermittent, depending on weight and maturity [19, 23]. The pharyngeal swallow is present, however. Although a premature infant may be able to suckle feed at 34 weeks gestational age, he may have difficulty coordinating swallowing and breathing. This can result in a decrease in tidal volume, respiratory frequency, and minute ventilation leading to a fall in pO_2 [24]. Feeding competence in prematures evolves with growth and neuromuscular maturation.

Infantile suckle feeding, reflex behavior regulated at a subcortical level, probably in the medulla and/or pons, is fully developed in the full-term newborn. The infant's anatomy is well suited for nipple feeding: the tongue filling most of the oral cavity, discrete sucking pads (fatpads) in the cheeks, the relatively small pharynx, the prominent soft palate and arytenoid mass, and the hyoid bone and larynx located more anteriorly and superiorly than in the adult [5, 12].

Oral sucking results from the rhythmic compression of the nipple by the lower jaw and tongue against the upper jaw and palate. The pattern of tongue motion, best described as peristaltic, has been recently studied by ultrasound techniques [25]. A peristaltic wave moves posteriorly in the medial portion of the tongue, compressing the nipple and propelling the expressed liquid posteriorly toward the pharynx. The lateral portions of the tongue appose the palate and enclose the nipple and bolus laterally, creating a medial pathway through which to channel the bolus. Suckling is further aided by the development of a repetitive negative intraoral pressure alternating with nipple compression [26]. The accumulated liquid from one or more suckles fills a posterior reservoir between the soft palate and the tongue or the soft palate and epiglottis (the valleculae). After enough is collected posteriorly, the bolus is propelled backward, the pharynx is filled, and a normal pharyngeal swallow is initiated. Breathing stops, the soft palate elevates to seal off the nasopharyngeal aditus, the laryngeal airway closes, and a peristaltic contraction wave strips the pharyngeal lumen from top to bottom, moving the bolus into the esophagus, past the reflexly relaxed upper esophageal sphincter (cricopharyngeal sphincter). This pharyngeal phase of deglutition resembles that of the adult, except that the pharyngeal stripping wave may be more prominent and the pharyngeal swallow occurs with greater frequency and speed [2, 3]. Immediately after the pharyngeal contraction wave passes into the esophagus where it continues as the primary peristaltic wave, the cricopharyngeal sphincter returns to its tonically contracted state to prevent regurgitation back from the esophagus. The airway also usually reopens immediately.

A primary peristaltic wave carries the bolus down the esophagus to the stomach. In rapid infant swallowing, new pharyngeal contractions interfere with propagation of the primary esophageal peristaltic wave associated with the preceding swallow, leading to stasis and a "filled column" appearance in the esophagus. However, the last swallow in the series culminates in a peristaltic wave that strips the esophagus.

Oral suckling, pharyngeal filling and contraction, esophageal transport, and breathing between swallows usually occur in a smooth and stable pattern of events.

As higher centers relative to feeding develop in the midbrain, thalamus, and then in the cerebral cortex, more voluntary control over oral function becomes possible. Thus, it is in the oral phase of deglutition that the greatest changes occur in feeding behavior with growth and development. Between 6 months and 3 years, transitional feeding evolves, paralleling the maturation of the central nervous system. Midline mashing of purees and pablums by the tongue gives way to more competent manipulation of the bolus and lateralized processing of textured foods, employing vertical biting and chewing. The eruption of teeth accompanies this process.

More adultlike feeding behavior develops with further evolution of neural function. Coordination of mastication matures between 3 and 6 years of age. Anatomic changes also occur: growth of the face and skull base, enlargement of the oral cavity and pharynx, and descent of the larynx and hyoid bone. As more voluntary control over oral function is gained, pharyngeal and esophageal stage events remain involuntary and relatively unchanged.

In the normal situation, these maturational changes occur as a sequential progression and in a coordinated and closely integrated fashion. However, if a medical, neurological, or environmental problem impedes the feeding process, the integration of its components may be disrupted and the progression skewed or even halted.

Feeding Pathology: Initial Evaluation

In pediatric dysphagia, children are referred for evaluation primarily because of failure of alimentation: they do not eat enough. This may manifest itself in many ways; for example, as food selectivity or refusal, gagging or vomiting related to feeding, failure to thrive or weight

loss [22, 27]. Another frequent cause for referral is suspicion of airway compromise or aspiration because of coughing or choking during feeding, recurrent pneumonias, chronic congestion, recurrent bronchospasm, or apnea [27–29]. In each of these cases, the primary problem may be in the swallowing process or any of a host of factors influencing acquisition of feeding competence [30, 31].

The initial evaluation should reflect a multidisciplinary approach whether it is done by a team of specialists or one specialist coordinating the other components. The team members may vary but should include expertise in pediatric medicine, nutrition, development, psychology and family function, positioning and oral motor skills, and diagnostic studies [1]. The perspective gained from the expertise of many disciplines enables more efficacious assessment and coordinated treatment.

History

Feeding enables nourishment which directly influences the well-being of the child. In turn, the well-being of the child, namely, the integrated functioning of all its organ systems, influences the process of feeding [22]. A thorough history is central to successful evaluation of a feeding problem and must cover all organ systems as well as information on the child's pattern of growth. It is helpful to organize this medical information into abnormalities in structure and function [30]. For example, abnormalities such as cleft lip and palate or tracheoesophageal fistula are structural problems that can be successfully treated by surgery. Abnormalities of function such as the motor incoordination of cerebral palsy or gastroesophageal reflux are dynamic problems whose influence on feeding may change over time, depending on other factors such as growth and development [32–35].

Any medical problem, from otitis media to constipation, may have an effect on feeding. Its influence may be better understood in the context of other factors—medical, developmental, or psychosocial—affecting the child at the time. The developmental history should include information on the child's developmental progression, current developmental level, and any qualitative difficulties such as problems with attention, tolerance, or transitions. The developmental history will facilitate accurate interpretation of the child's response to past events and future interventions.

The feeding history should start at birth and attempt to record the child's progress through each transition phase of oral feeding—introduction of bottle, spoon, and cup; and through the introduction of advancing textures—purees, junior foods, and table foods. If difficulty is encountered at any step, it can temporally be correlated with medical and developmental events. It is worthwhile reviewing the child's current feeding regimen, including associated problems that occur with eating such as coughing, gagging, emesis, or wheezing. A description of what a typical mealtime is like elucidates the mealtime social environment and the caretaker's response to the feeding problem.

Exam and Observation

From the history, hypotheses are developed about the components contributing to the feeding problem. Information from the physical exam, including observations of a meal (Fig. 1), either substantiates or refutes these hypotheses. Obviously, the general physical exam should include present growth parameters, as well as assessment of the structure and function of the head and neck, oral cavity, chest, and abdomen. Dysmorphic findings should prompt investigation into associated syndromes or genetic disorders. The neurodevelopmental examination highlights neurological dysfunction and its effect on gross motor, fine motor, and language skills [1]. The child's performance on several developmental tasks gives an indication of his developmental level as well as how he approaches a problem—impulsively, easily frustrated, etc.

The oral motor exam looks at structure of the component parts, their relationship to each other, and their coordinated function [8, 9]. Watching a meal gives information on level of competency with demonstrated oral skills, coordination, and fatigue. Cervical auscultation of a swallow with different textures allows some insight into pharyngeal function and the coordination of the oral transport phase with the pharyngeal phase and respiration [36–38]. If the child behaves typically, the evaluator can see firsthand the antecedents to any associated problems. Also, the caretaker's response can be observed.

Clinical Impression

Original hypotheses are then reviewed in light of information gleaned from the exam and feeding observation. Those that can be confidently excluded on the basis of the clinical information are discarded. The remaining hypotheses focus the direction of laboratory investigation. This approach helps decrease the number of tests the child will undergo and enables the clinician to get the most from each test. The test can substantiate the working hypotheses and document the impact of a potential intervention.

Diagnostic Studies

Barium Studies

The videofluoroscopic barium swallowing examination recorded dynamically remains the best procedure for

Fig. 1. Observation of feeding skills of an infant (**A**) and child (**B**) with appropriate food, implements, and environment.

demonstrating the swallowing mechanism. It is widely available although technical quality may vary. It requires time, effort, proper personnel, and special equipment to perform well [2, 3]. Afterwards, the real time recording must be studied carefully with slow motion, back up, and stop-frame viewing.

The fluoroscopic swallowing study demonstrates the anatomy of the oral cavity, pharynx, larynx, and esophagus and its function (Fig. 2). It can document oral motor dysfunction, pharyngeal incoordination, nasopharyngeal reflux, and laryngeal penetration/aspiration in all its forms. It is helpful in identifying situations where oral feeding is unsafe and cases where feeding may proceed with certain modifications (e.g., bolus: texture, consistency, size; patient positioning [41]), and thus plays a central role in diagnosis, development of treatment plan, and monitoring of results [39].

The remainder of the upper gastrointestinal tract may be examined in the same or, as is frequently the case, a second sitting. Esophageal anatomy and mucosal pattern, peristalsis, gastroesophageal reflux, hiatal hernia, stomach and duodenal anatomy, gastric emptying, inflammatory lesions, malrotation, and other obstructing lesions can all be assessed by this technique [40].

Ultrasound Examination

Ultrasonography is a noninvasive technique that can be used to image the oral cavity. From the submental approach, a transducer can demonstrate in several planes the motion of the tongue and floor of the mouth during feeding [25]. Observations made with this technique have added to our understanding of oral motor function, peristaltic tongue motions during suckle feeding and the coordination of suckling, swallowing, and breathing [42]. Unfortunately, visualization of the pharynx is limited due to acoustic shadowing from adjacent bones and air bubbles in the lumen. It remains primarily a research tool.

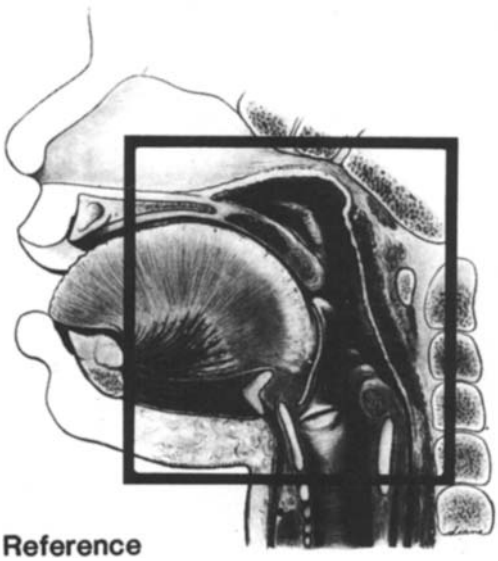
pH Probe Study

Many children with neuromuscular dysfunction and swallowing impairment also have gastroesophageal reflux (GER) [27]. It may manifest itself by further complicating swallowing function, by vomiting, regurgitation, refusal of feeds, irritability, and/or gastrointestinal bleeding. Respiratory problems including recurrent pneumonia and tracheobronchitis may be caused or exacerbated by GER. Bronchospasm, laryngospasm, and apnea, either as a result of aspiration or stimulation of vagally mediated irritant receptors, have in some cases been linked to GER.

A pH probe study of the lower esophagus is a sensitive method for determining the occurrence and severity of GER. It may be used to correlate reflux events to symptoms. Barium upper GI series is a less sensitive technique for the evaluation of GER. Endoscopy and biopsy are the "gold standard" for the diagnosis of reflux esophagitis.

Manometry

Manometry has been used to document esophageal motor function, and more recently to study pharyngeal peristal-



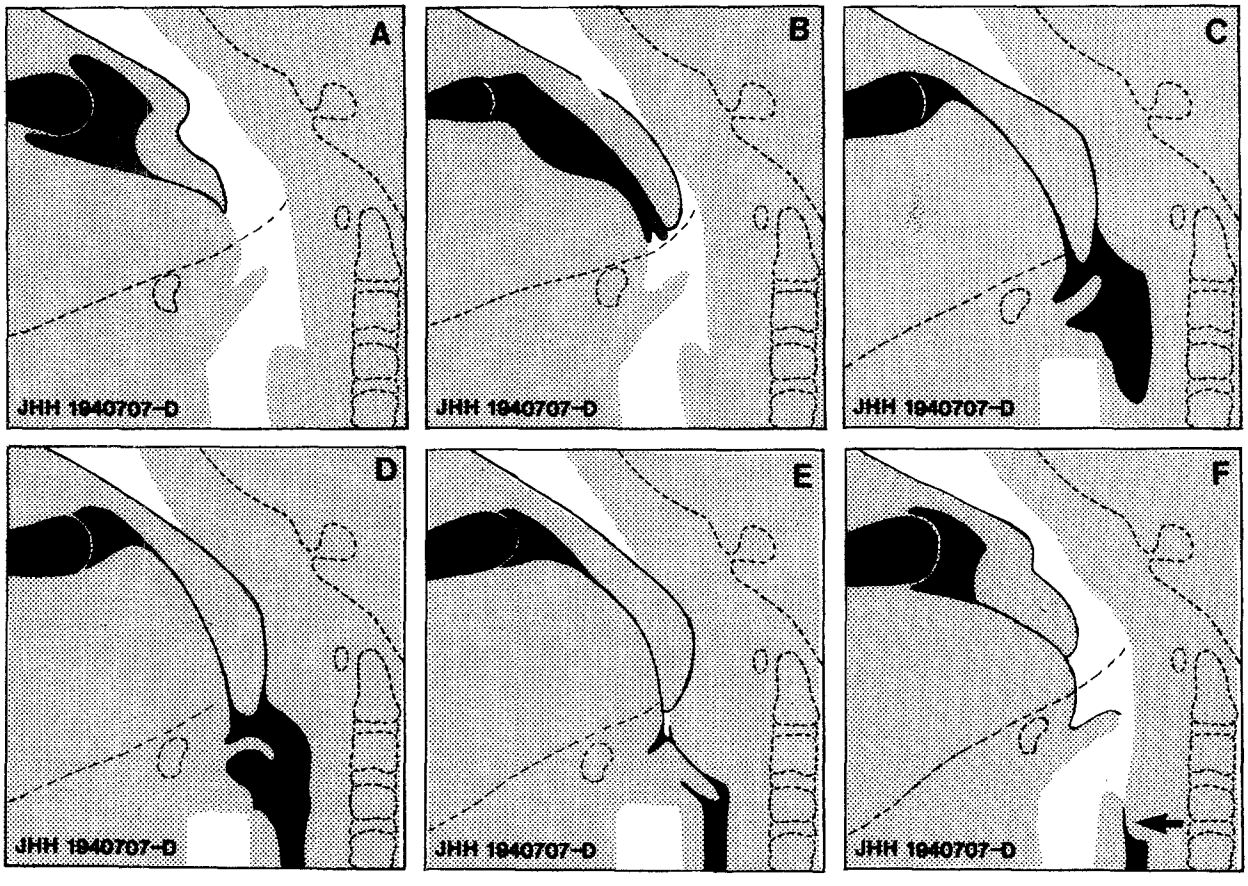


Fig. 2. Drawing of normal infantile suckle feeding (lateral projection). During oral suckling, barium (*black*) expressed from the nipple collects posteriorly in the mouth, but is confined by the apposition of the soft palate and the tongue (**A**). With vigorous suckling, the soft palate configuration changes and, at times, may bow forward as the soft palate-tongue relationship is maintained. As the tongue moves the collected liquid into the pharynx (**B, C**), the pharyngeal swallowing sequence is initiated. The soft palate elevates, apposes the posterior pharyngeal wall, and closes off the nasopharynx. Pharyngeal contraction begins as a posterior pharyngeal wave in the superior pharyngeal constrictor. The larynx is elevating, the epiglottis is beginning to tilt, and the laryngeal vestibule is closed (**C**). Pharyngeal contraction seen as a prominent posterior pharyngeal wave transports the barium through the pharynx and the open cricopharyngeal sphincter into the esophagus (**D**). No barium enters the closed, elevated larynx. At the end of pharyngeal constriction, the pharynx is completely empty (**E**). After barium has passed into the esophagus and the cricopharyngeal sphincter has closed, the airway (*white*) immediately reopens (**F**). Note barium outlining lower margin of cricopharyngeal sphincter (*arrow*) [2, reprinted with permission].

sis and upper esophageal sphincter responsiveness via a specially designed catheter or probe. Intraluminal pressure recordings made simultaneously at various levels in the pharynx, cricopharyngeal sphincter, and esophagus are correlated temporally to provide a picture of motor activity and coordination during swallowing. The procedure is moderately invasive, technically demanding, and requires sophisticated equipment and trained personnel [27, 43].

Nuclear Medicine Studies

The technetium-labeled milk scan can demonstrate and quantitate pharyngeal and esophageal transit, GER epi-

sodes occurring and clearing during the scanning period, and gastric emptying. Occasionally, aspiration of ingested or refluxed material can be verified.

Case Reports

Case 1

DB is a 10-year-old boy followed for severe cerebral palsy resulting from a viral illness at 3 months of age, mental retardation, and marginal weight gain with oral feedings. His clinic attendance had always been sporadic, but over the last year a different person brought him to clinic each time and knew very little about him. Simple recommendations made to facilitate weight gain were not followed. When planned orthopedic surgery made increased weight gain urgent, he was admitted into the hospital for further evaluation.

Physical examination revealed a thin boy, alert and interactive, but with limited vocabulary. Cardiopulmonary exam was unremarkable. Cranial nerves II–XII were intact. He had diffusely increased tone, brisk deep tendon reflexes, and multiple contractures in the pattern of spastic quadriplegia. He was able to sit supported in his wheelchair although hip abduction contractures made seated positioning difficult. Oral structures were intact. Isolated oral motor exam with a cookie showed tongue lateralization to both sides and a munching jaw pattern. He was able to drink thin liquids with a straw. He had an occasional tongue thrust.

Because of their inconsistent compliance documented in the clinic, it was suspected that DB's family was not spending the time necessary to feed him. In the hospital he received three regular meals a day and three milk shakes. Although he had some intermittent gagging and emesis, he gained 1 kg the first week. His weight gain slowed over the next few weeks and the gagging and vomiting increased. Gastroesophageal reflux was suspected. A milk scan showed no gastroesophageal reflux and normal gastric emptying.

DB's feeding skills were reevaluated over an entire meal. Although he used tongue lateralization and a munching pattern, higher textured foods prompted increased tongue and jaw thrusting with more anterior loss than when eating soft foods such as yogurt. He had increased upper airway congestion as the meal progressed and several episodes of emesis. A modified barium swallow with videofluoroscopy was obtained to better evaluate what was happening in the pharyngeal phase. This showed poor pharyngeal peristalsis but a competent swallow with thin barium. Thickened barium to the texture of pudding and the barium cookie were both aspirated before they could be completely transported through the hypopharynx. On the basis of this study, DB's diet was changed to pureed food, with each bite followed by a sip of liquids to facilitate transport. His PO intake increased by 10% with improved rate of weight gain. Gagging and emesis markedly decreased.

Case 2

KT is a 6-week-old male infant admitted to the hospital for evaluation of increasing congestion and cyanosis associated with feeding. He was the former 7 lb 3 oz product of a full-term, uncomplicated gestation, delivered vaginally without problem. On the first day of life, he was noted to have congestion with feedings but this was thought to be secondary to mucus. A heart murmur was detected but chest x-ray film and EKG were reportedly normal. He was discharged home with his mother at 3 days of age.

The mother reports "congestion" since birth. She has noted intermittent cyanosis during feedings associated with retractions, gasping, and coughing which can continue up to 10–15 min after a feeding. He has no diaphoresis and no tiring with feedings. Occasionally he will have a small amount of regurgitation after feedings but no emesis. The mother describes "pellet-like" stools, for which he is getting dilute formula. He is also receiving Mylicon for gas. Except for his "congestion," he has been in good health.

Physical examination was remarkable for appropriate growth parameters for age. A cardiac murmur consistent with peripheral pulmonic stenosis was audible. Although he had truncal hypotonia, he demonstrated increased extensor tone with arching of his back. Auditory and visual responses were appropriate for his age.

A history of chronic congestion suggests either aspiration or gastroesophageal reflux. The history of coughing and retractions during feeding points more to aspiration than reflux, although we can be suspicious of reflux because of the history of "regurgitation" and the frequent arching posture in the absence of other neurological signs to explain it (Sandifers syndrome). Cyanosis may be cardiac or airway related; the absence of diaphoresis and tiring with feedings in this child makes cardiac involvement less likely. Feeding observation showed a vigorous suck and swallow, but difficulty coordinating these with respirations, with notable stridor, and increasing congestion on cervical auscultation. Postulating that slower oral transport might allow more time to control the swallow, his feeds were thickened which decreased his congestion.

His hospital evaluation included an upper gastrointestinal (UGI) exam showing gastroesophageal reflux (Fig. 3A), normal gastrointestinal anatomy to the Ligament of Treitz, and no obstruction. A milk scan showed mild gastroesophageal reflux, no aspiration, and normal gastric emptying. Airway films showed tracheal narrowing consistent with tracheomalacia. An apnea study employing a nasal thermistor did not show central or obstructive apnea. Cardiac evaluation documented peripheral pulmonic stenosis. A modified barium swallow with videofluoroscopy showed mild dyscoordination of the pharyngeal swallowing mechanism with intermittent laryngeal penetration, aspiration, and sporadic nasopharyngeal reflux on thin barium (Fig. 3B). As the barium was thickened with rice cereal, the penetration, aspiration, and reflux decreased in frequency. A barium thickness equal to 1 oz of cereal in 3 oz of formula resulted in consistently competent swallows during the exam and afterwards, clinically.

Putting all information together, we have a 6-week-old infant with mild hypotonia, gastroesophageal reflux, tracheomalacia, and mild dyscoordination in the pharyngeal swallow resulting in intermittent aspiration and cyanosis. The gastroesophageal reflux is likely contributing to the arching posture. Whether the dyscoordination is somehow associated with tracheomalacia or a manifestation of mild neural abnormalities is not well understood. The child was treated with antacids for his reflux and thickened formula as indicated. His arching posture resolved quickly; the coughing and cyanosis disappeared.

Case 3

JG is a 31-month-old boy with extrapyramidal cerebral palsy and mental retardation, referred for evaluation of inadequate caloric intake, recurrent emesis, and failure to thrive. He was the product of a 27-week gestation complicated only by premature labor. He received ventilatory support for 6 weeks and was discharged home at 3 months of age with mild reactive airway disease requiring no medications. His medical history was remarkable for frequent bouts of bronchitis and pneumonia with recent difficulty clearing oral thrush and a candidal diaper rash.

P.O. feedings were initiated at 7 weeks of age. Although he required no tube supplements by 8 weeks, the parents recall he had a weak suck with intermittent bradycardia during feeding. Each meal took 90 min. Pureed foods were introduced at 6 months without problem. At 10 months, when junior foods were introduced, he started

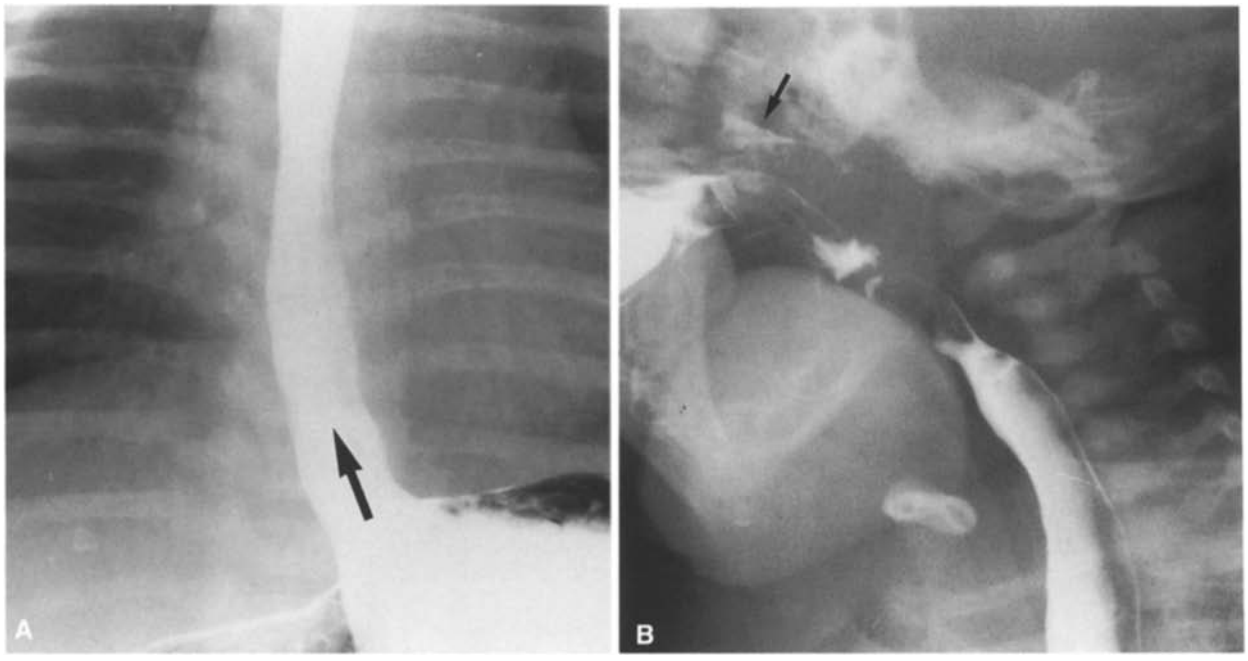


Fig. 3. A spot film from the UGI series clearly shows gastroesophageal reflux arrow (**A**). The swallowing exam (**B**) demonstrates nasopharyngeal reflux (arrow) caused by swallowing incoordination. The infant also had intermittent laryngeal penetration and aspiration.

vomiting. Parents recall chronic congestion from this point on with an episode of aspiration pneumonia. His growth curve also leveled off after 10 months. At the time of the evaluation, he was eating four to five jars of pureed food and 8–12 oz. of apple juice per day. Parents were limiting him in an effort to reduce emesis. They reported increased coughing 10 min after each meal.

On physical examination, height, weight, and head circumference were all well below the 5 percentile for age. Weight was in the 50 percentile for a 12-month-old. He had oral thrush. Tone was quite variable with persistent primitive reflex patterns consistent with extrapyramidal cerebral palsy. He had a tendency to arch his back when supine. Developmentally, his language skills were at a 5–7 month level, and motor skills were at a 3 month level. He still had poor head control. During a mealtime observation, his mother fed him heaping tablespoons in order to speed up the meal. He demonstrated a suckle pattern with difficulty coordinating the swallow with respirations. Cervical auscultation suggested a delay in triggering of the pharyngeal swallow with liquids, and poor pharyngeal clearance with purees. Aspiration was suspected because of the increasing congestion heard with feeding. However, the history of emesis and coughing 10 min after the meal strongly suggested gastroesophageal reflux as well.

JG was admitted to expedite his evaluation and implement interventions. His workup included a modified barium swallow with videofluoroscopy which showed poor oral transport with thicker foods (Fig. 4A) but no aspiration on thin or thickened barium; an UGI study showed gastroesophageal reflux and a dilated esophagus with poor peristalsis; a milk scan showed severe GER and delayed gastric emptying (Fig. 4B). Because of the prolonged oral thrush and the dilated esophagus, an esophagogastroduodenoscopy was done which verified candidal esophagitis. Antacid and prokinetic medications were started and JG received a 10-day course of ketoconazole and nystatin. Within 8 weeks, a repeat UGI study showed resolution of the esophageal dilatation. A repeat milk scan suggested decreased frequency of GER episodes.

JG had intermittent stridor at night, initially thought to be secondary to mild tracheomalacia exaggerated by his low tone during sleep. Over the course of several weeks, he developed increasing intermittent stridor. Airway films showed a nasopharyngeal airway obstructed by large adenoids (Fig. 4C) and a normal subglottic airway. An apnea study documented an obstructive pattern. He underwent tonsillectomy and adenoidectomy with resolution of his nighttime stridor.

Throughout his hospitalization, efforts were directed toward improving JG's upright positioning and oral motor skills. Despite clinical improvements in both, JG did not gain weight. He needed supplemental tube feeding overnight to initiate weight gain. His oral skills advanced to a munching pattern and soft solid foods were introduced.

The father was concerned that the mother would not be willing to change the way she fed JG unless we could prove to her it was helpful. Further discussions with both parents revealed marital tension, and maternal isolation and depression because of the time it took to care for JG. The mother was provided more help in the home which enabled her to comply with the recommended feeding protocol. Four months after discharge, the NG tube was discontinued. Weight continues to be between the 10 and 25 percentile for age.

Case 4

DL is a 10-month-old former 27-week premature male infant, referred for evaluation because of profuse drooling, lack of swallowing, and gagging with any P.O. feeding attempts. His neonatal course was complicated by bronchopulmonary dysplasia and tracheomalacia necessitating tracheostomy at 3 months of age (Fig. 5A) and prolonged ventilatory support. DL developed nutritional rickets and severe failure to thrive because of recurrent emesis with nasogastric (NG) tube feedings and severe diarrhea with gastrostomy tube feedings. GI evaluation with an UGI and milk scan documented gastroesophageal reflux and delayed gastric emptying. A prokinetic agent and antacid were started

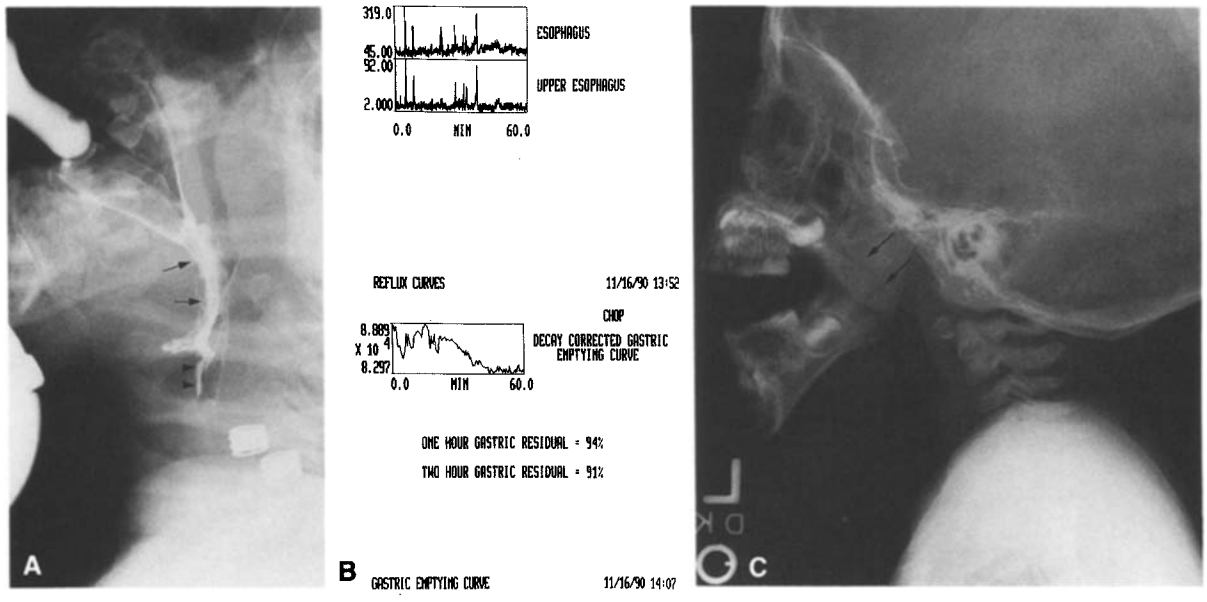


Fig. 4. Case 3. During the videofluoroscopic swallowing study (**A**), the child displayed poor oral transport and premature leak of the oral bolus into pharynx. Barium is seen dribbling over the back of the tongue (small arrows) and along the aryepiglottic folds (arrowheads). No aspiration occurred, however. (**B**) A graphic demonstration of data from the technetium milk scan. In the upper tracings, spike-like inflections on the graph reflect radioactivity counts increasing with reflux of material from the stomach and measured at different levels in the esophagus. Multiple episodes of gastroesophageal reflux occurred over the 60-min scanning period. The lower tracing portrays the gastric emptying curve. Emptying was delayed with a gastric residual at 1 h of 94% and at 2 h of 91% of the radioactivity originally in the stomach. (**C**) A lateral view of the oral cavity and pharynx shows occlusion of the nasopharyngeal airway caused by enlarged adenoids (arrows) which touch the soft palate. Also of note is the straightened mandibular angle, prominent antegonial notch, and the relatively short hard palate.

with improved toleration of bolus NG feedings. Although emesis continued, his growth rate was acceptable on 170 kcal/kg per day.

On physical examination, DL was a small but appropriately proportioned infant male with a tracheostomy. His neurological exam was remarkable for mild hypotonia, but no asymmetries. Developmentally, he demonstrated skills in the 4- to 5-month range. Oral motor examination revealed low facial tone, and an absent suckle reflex. He used a suckling pattern with a pacifier but had no tongue movements at all with a bottle or spoon. As the food dispersed in his mouth, he would gag and vomit. Cervical auscultation documented swallowing activity at times outside of the feeding session, but not while eating.

Because he was having recurrent emesis on medications, a pH probe study was done to further evaluate his GER. The study documented ongoing GER but suggested that it was minimized with small amounts of thickened feedings. He was started on continuous NG feedings of thickened formula. He was also weaned from theophylline in hopes of decreasing gastric irritation further. Emesis frequency did decrease. With improved GI stability, DL was able to better tolerate things in his mouth without gagging. A barium swallow documented mild oral transport incoordination with mild laryngeal penetration on thin liquids (Fig. 5B). Feeding attempts were initiated with a dipped spoon and laryngeal penetration was monitored via the tracheostomy. Under these conditions, acceptance of the spoon increased and his oral transport pattern improved. There was no deterioration in his respiratory status.

Summary of Cases

The children described here are typical of those presenting at a pediatric dysphagia clinic. Although 3 of the 4 have inadequate oral intake, the underlying reasons differ. These children could not have been effectively served by one discipline alone. The multiple pathophysiological influences acting on the feeding process could not have been identified without the benefit of a thorough history, physical, neurodevelopmental, and oral motor exam, and mealtime observation, followed by focused diagnostic tests.

Conclusion

The diagnostic evaluation including history, physical and developmental exam, and feeding observation, followed by appropriate diagnostic studies forms the basis for understanding the underlying pathophysiology and anticipated developmental progression in the individual child. This makes possible the formulation and implementation of an optimal treatment strategy, which can be monitored for results and modified when needed.

Upfront identification of the pathophysiological factors acting on the feeding process enables them to be dealt with in a timely and coordinated fashion, increasing the likelihood of treatment success. However, in formulating a treatment strategy one must consider what changes the child is medically and developmentally capa-

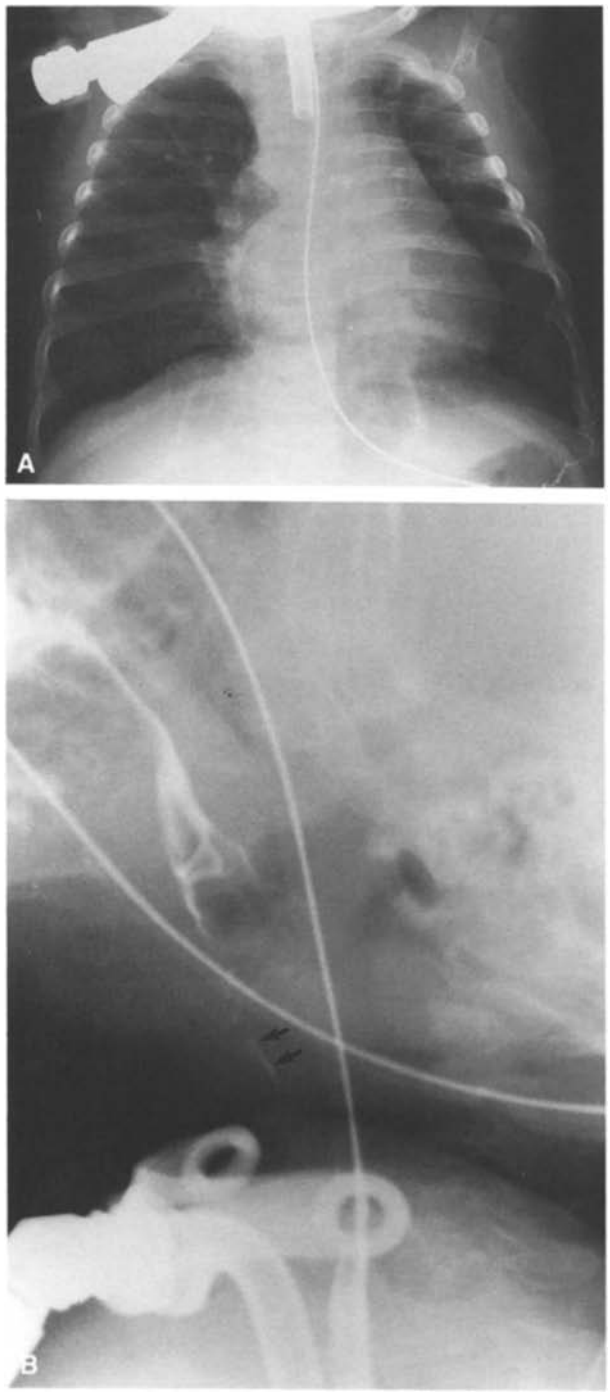


Fig. 5. Case 4. (**A**) On the chest x-ray film, a tracheostomy tube and NG tube are in place. The lungs are unevenly aerated with scattered areas of linear atelectasis as a result of bronchopulmonary dysplasia. The child also has a surgical clip in the left mediastinum and thoracotomy rib changes from a previous PDA ligation. (**B**) A lateral view from the barium swallowing study shows mild laryngeal penetration (arrows) with thin liquids. Mild incoordination in oral transport was also noted during the exam.

ble of and how much the family can accommodate, as it is imperative that the child's treatment goals are shared by the caretaker. Often, the treatment strategy must be approached in a stepwise fashion to prevent overwhelming the family. We can help prioritize with the caretakers what is essential and possible to do now and what to work on later.

As with all good treatment plans, the child's response must be monitored. With growth and development, things change; one needs to keep an open mind when interpreting the responses, and always question whether there may be a better way to achieve the same result in future cases.

References

1. Christensen JR: Developmental approach to pediatric neurogenic dysphagia. *Dysphagia 3:*131–134, 1989
2. Kramer SS: Special swallowing problems in children. *Gastrointest Radiol 10:*241–250, 1985
3. Kramer SS: Radiologic examination of the swallowing-impaired child. *Dysphagia 3:*117–125, 1989
4. Ardran GM, Kemp FH: The mechanism of changes in form of the cervical airway in infancy. *Med Radiogr Photogr 44:*26–38, 1968
5. Bosma J: Oral and pharyngeal development and function. *J Dental Res 42:*375–380, 1963
6. Bosma JF: Anatomic and physiologic development of the speech apparatus. In: Tower DB (ed.): *The Nervous System*, vol. 3, New York: Raven Press, 1975, pp 469–481
7. Bosma JF: Introduction to the symposium. In: Bosma JF, Showacre J (eds.): *Symposium on Development of Upper Respiratory Anatomy and Function: Implications for Sudden Infant Death Syndrome*. Washington DC: US Government Printing Office, 1976, pp 5–49
8. Bosma JF: Physiology of the mouth, pharynx, and esophagus: physiology of the mouth. In: Paparella MA, Shumrick DA (eds.): *Otolaryngology*, 2nd ed. Philadelphia: WB Saunders, 1980, pp 319–331
9. Bosma JF, Donner MW: Physiology of the mouth, pharynx, and esophagus: physiology of the pharynx. In: Paparella MA, Shumrick DA (eds.): *Otolaryngology*, 2nd ed. Philadelphia: WB Saunders, 1980, pp 332–345
10. Ardran GM, Kemp FH, Lind J: A cineradiographic study of bottle feeding. *Br J Radiol 31:*11–22, 1958
11. Bosma JF: Postnatal ontogeny of performances of the pharynx, larynx, and mouth. *Am Rev Respir Dis 131*(Suppl):S10–S15, 1985
12. Bosma, JF: Development of feeding. *Clin Nutr 5:*210–218, 1986
13. Bosma JF: Oral-pharyngeal interactions in feeding. Presented at symposium on dysphagia. The Johns Hopkins Medical Institutions, February 27, 1986
14. Logan WJ, Bosma JF: Oral and pharyngeal dysphagia in infancy. *Pediatr Clin North Am 14:*47–61, 1967
15. Illingworth RS: Sucking and swallowing difficulties in infancy: diagnostic problems of dysphagia. *Arch Dis Child 44:*655–665, 1969
16. Ardran GM, Kemp FH: Some important factors in the assessment of oropharyngeal function. *Dev Med Child Neurol 12:*158–166, 1970

17. Fisher SE, Painter M, Milmoe G: Swallowing disorders in infancy. *Pediatr Clin North Am 28:*845–853, 1981
18. Franken EA: Swallowing disorders. In: *Gastrointestinal Imaging in Pediatrics*. Philadelphia: Harper and Row, 1982, pp 26–32
19. Gryboski J, Walker WA: Suck and swallow. In: *Gastrointestinal Problems in the Infant*. Philadelphia: WB Saunders, 1983, pp 12–29
20. Cohen SR: Difficulty with swallowing. In: Bluestone CD, Stool SE (eds.): *Pediatric Otolaryngology*, vol. 1, Philadelphia: WB Saunders, 1983, pp 903–911
21. Pritchard JA: Fetal swallowing and amniotic fluid volume. *Obstet Gynecol 28:*606–610, 1966
22. Tuchman DN: Dysfunctional swallowing in the pediatric patient: clinical considerations. *Dysphagia 2:*203–208, 1988
23. Gryboski JD: Suck and swallow in the premature infant. *Pediatrics 43:*96–102, 1969
24. Shivpuri CR, Martin RJ, Carlo WA, Fanaroff AA: Decreased ventilation in preterm infants during oral feeding. *J Pediatr 103:*285–289, 1983
25. Bosma JF, Hepburn LG, Josell SD, Baker K: Ultrasound demonstration of tongue motions during suckle feeding. *Dev Med Child Neurol 32:*223–229, 1990
26. McGowan JS, Marsh RR, Fowler SM, Levy SE, Stallings VA: Developmental patterns of normal nutritive sucking in infants. *Dev Med Child Neurol 33:*891–897, 1991
27. Tuchman DN: Cough, choke, sputter: the evaluation of the child with dysfunctional swallowing. *Dysphagia 3:*111–116, 1989
28. Loughlin GM: Respiratory consequences of dysfunctional swallowing and aspiration. *Dysphagia 3:*126–130, 1989
29. Plaxido DT, Loughlin GM: Nasopharyngeal reflux and neonatal apnea. *Am J Dis Child 135:*793, 1981
30. Stevenson RD, Allaire JH: The development of normal feeding and swallowing. *Pediatr Clin North Am 38:*1439–1453, 1991
31. Itani Y, Fujioka M, Nishimura G, Niitsu N, Oono T: Upper GI examinations in older premature infants with persistent apnea: correlation with simultaneous cardiorespiratory monitoring. *Pediatr Radiol 18:*464–467, 1988
32. Morris SE: Developmental implications for the management of feeding problems in neurologically impaired infants. *Semin Speech Lang 6:*293–315, 1985
33. Morris SE: Development of oral-motor skills in the neurologically impaired child receiving non-oral feedings. *Dysphagia 3:*135–154, 1989
34. Monahan PS, Shapiro B, Fox C: Effect of tube feeding on oral function. *Dev Med Child Neurol 57*(Suppl):7, 1988
35. Monahan-Eicher PS: Feeding the child with disabilities. In: Batshaw ML, Perret YM (eds.): *Children with Disabilities, a Medical Primer*. Baltimore: Paul H. Brooks Publishing, Inc, 1992, pp 387–406
36. McPherson KA, Kenny DJ, Koheil R, Bablich K, Sochaniwskyj A, Milner M: Ventilation and swallowing interactions of normal children and children with cerebral palsy. *Dev Med Child Neurol 34:*577–588, 1992
37. Vice FL, Heinz JM, Giiuriati G, Hood M, Bosma JF: Cervical auscultation of suckle feeding in newborn infants. *Dev Med Child Neurol 32:*760–768, 1990
38. Bamford O, Taciak V, Gewolb IH: The relationship between rhythmic swallowing and breathing during suckle feeding in term neonates. *Pediatr Res 31:*619–624, 1992
39. Jones B. Kramer SS, Donner MW: Dynamic imaging of the pharynx. *Gastrointest Radiol 10:*213–224, 1985
40. Kramer SS: Pediatric radiology corner: complications following esophageal atresia repair. *Dysphagia 3:*155–156, 1989
41. Ekberg O: Posture of the head and pharyngeal swallowing. *Acta-Radiol [Diagn] (Stockh) 27:*691–696, 1986
42. Bu'Lock F, Woolridge MW, Baum JD: Development of coordination of sucking, swallowing and breathing: ultrasound study of term and preterm infants. *Dev Med Child Neurol 32:*669–678, 1990
43. Staiano A, Cucchiara S, DeVizia B, Andreotti MR, Auricchio S: Disorders of upper esophageal sphincter motility in children. *J Pediatr Gastroenterol Nutr 6:*892–898, 1987