Management of oropharyngeal dysphagia in the neurologically intact and developmentally normal child

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Purpose of review

An enigmatic group of children with normal development and intact neurologic function display clinical, radiographic, and endoscopic evidence of persistent laryngeal penetration and/or aspiration during feeds, known as oropharyngeal dysphagia (OPD). Identifying the etiology of OPD in this patient population and designing the appropriate management has been difficult. As a result, many healthy children with OPD remain on thickened diets late into childhood.

Recent findings

Reports of OPD in neurologically intact children have emerged but are limited in providing clinical guidelines. Oropharyngeal dysphagia is best detected by instrumental evaluations of swallowing function but a correlation with clinical signs and symptoms is critical for therapy. The novel, but controversial, 3-oz. water swallow challenge in children can be a useful tool in clinic for persistent OPD. Physiologic conditions linked to pharyngolaryngeal sensory blunting may explain OPD in many neurologically intact children. Unexplained OPD in these children often improves with time and reflux therapy. Proving subtle anatomic anomalies as the source of OPD, such as type I laryngeal clefts, cricopharyngeal achalasia, and tonsillar hypertrophy, remains difficult. Surgical management for these entities may be the final step in the therapeutic tree. Despite the etiology and treatment, the return to a normal diet in children with OPD requires a graduated approach. This allows systematic neuromuscular training of the pharyngeal phase of swallowing.

Summary

OPD in the neurologically intact child is underrepresented in the literature. This review examines pediatric dysphagia and recent reports of OPD in developmentally normal children to extrapolate basic clinical guidelines for managing OPD in this population.

Keywords

aspiration, dysphagia, neurologically intact, oropharyngeal, pediatric, unexplained

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Introduction

Oropharyngeal dysphagia (OPD) is a dysfunction of the pharyngeal phase of swallowing that allows laryngeal penetration and tracheal aspiration of small volumes of solid food or liquids. Respiratory complaints associated with meals and frequent lower respiratory infections suggest the presence of OPD in children. OPD is best detected by instrumental evaluations of swallowing function. OPD in both children and adults is commonly associated with neurologic conditions that disrupt the normal mechanism of airway protection during swallowing. A recent rise in the rate of children with OPD has been correlated with increased survival of infants with a history of prematurity, neurologic disorders, and complex medical conditions [1,2]. However, there is a large proportion of children in whom reasons for OPD and aspiration are unclear [3]. These children pose a diagnostic and therapeutic challenge, are often placed on thickened diets for years, and followed with periodic swallowing evaluations until OPD is resolved.

Parental anxiety, strained parent–child relationships and potential long-term physical (lung disease) and psychological risks accompany pediatric OPD [4•]. Interestingly, however, the impact of OPD rarely affects each patient equally [5]. This is particularly true for neurologically intact children and further complicates their management. This report therefore examines the physiology of OPD to tailor a comprehensive approach to the assessment and management of unexplained OPD in children who appear otherwise developmentally normal.

Pharyngolaryngeal swallowing physiology

Swallowing is a complex process, involving both voluntary and involuntary actions, that occurs in four phases. Identifying the etiology of OPD requires understanding

the pharyngeal phase of swallowing. The pharyngeal phase of swallowing occurs in less than three seconds as relaxation of the upper esophageal sphincter (UES), formed primarily by the cricopharyngeal muscle, is synchronized with pharyngeal contraction and elevation of the larynx. This mechanism pulls the larynx forward, retroflexes the epiglottis, opens the UES, and allows tongue propulsion and pharyngeal direction of the bolus through the UES [6,7]. Coordination with respiration and closure of the laryngeal inlet, known as the pharyngoglottal closure reflex, occurs to protect the airway. Thus, deviations from normal upper aerodigestive anatomy and respiratory patterns can subsequently lead to OPD.

Pharyngoglottal closure, believed to be the first step in the pharyngeal phase of swallowing, occurs during both awake and sleep states near the onset of bolus transit. This reflex is the predominant mechanism of airway protection and is manifested by adduction of the true vocal cords and approximation of the arytenoid cartilages through contraction of the thyroartenoid and interarytenoid muscles [7–9]. Pharyngolaryngeal sensory and chemoreceptors trigger pharyngoglottal closure [8,9,10••]. Sensory feedback involves neural contributions from the glossopharnygeal (IX) and vagus (X) nerves located along the posterior oral cavity, pharynx, and supraglottic larynx (superior laryngeal nerve) [11–13]. The summation of sensory input determines the onset of spontaneous swallow and airway protection [9,12]. Deficits in neurosensory and neuromotor function can subsequently cause aberrancies in the pharyngeal phase of swallowing [14]. Similarly, abnormal laryngeal chemoreceptor sensitivity can deregulate laryngeal opening and cause aspiration [15].

Assessment and management

The management of OPD in the developmentally normal and neurologically intact child requires a holistic approach with careful integration of the child's history, physical exam, instrumental swallowing evaluations, and endoscopy. Despite recent research in this patient population, therapeutic protocols are currently not available and management of OPD likely varies among clinicians [3,16••]. Several disciplines (otolaryngology, pulmonology, gastroenterology, speech therapy) are often involved in the management of these patients. The decision to advance a child's diet may address the therapeutic goals of one specialty while counteracting those of another. For example, the desire to wean from thickened to thin liquids should be tempered by any risks to the child's pulmonary status and the treatment goals of the pulmonologist. Multidisciplinary input is thus critical before management decisions are made. This following material provides some interdisciplinary guidelines, based on the limited knowledge available, to address OPD in the neurologically intact child, but should be balanced with the conditions affecting each individual patient.

Figure 1 provides a rudimentary approach for the initial assessment of a child with unexplained OPD. Flexible laryngoscopy, chest radiograph and a good history and physical should be performed at the first clinic visit. Children with suspected laryngeal anomalies should proceed to microlaryngoscopy with bronchoscopy (MLB), while pulmonary infiltrates on chest radiograph (CXR) may be better elucidated by high-resolution spiral computed tomography (HRCT) and pulmonary consultation. Bronchial thickening, bronchiectasis, air trapping, and centrilobar opacities found on HRCT are suggestive of recurrent peripheral pulmonary injury from chronic aspiration. Neurologically intact children with OPD presenting older than 2 years of age should be explored for anatomic sources of OPD with MLB until proven otherwise.

Assessment and management of gastroesophageal reflux disease/laryngopharyngeal reflux (GERD/LPR) in a child with OPD can be initiated when signs, symptoms, and laryngoscopy suggest its presence. GERD/LPR has been implicated as a factor involved in OPD, but its role remains unclear. Spillage of gastric acid and irritants on the respiratory mucosa has been shown to decrease laryngeal sensation [17,18]. Theoretically, this can impact the precision of the pharyngoglottic closure reflex and allow laryngeal penetration and aspiration of thinner, and consequently faster-moving, material.

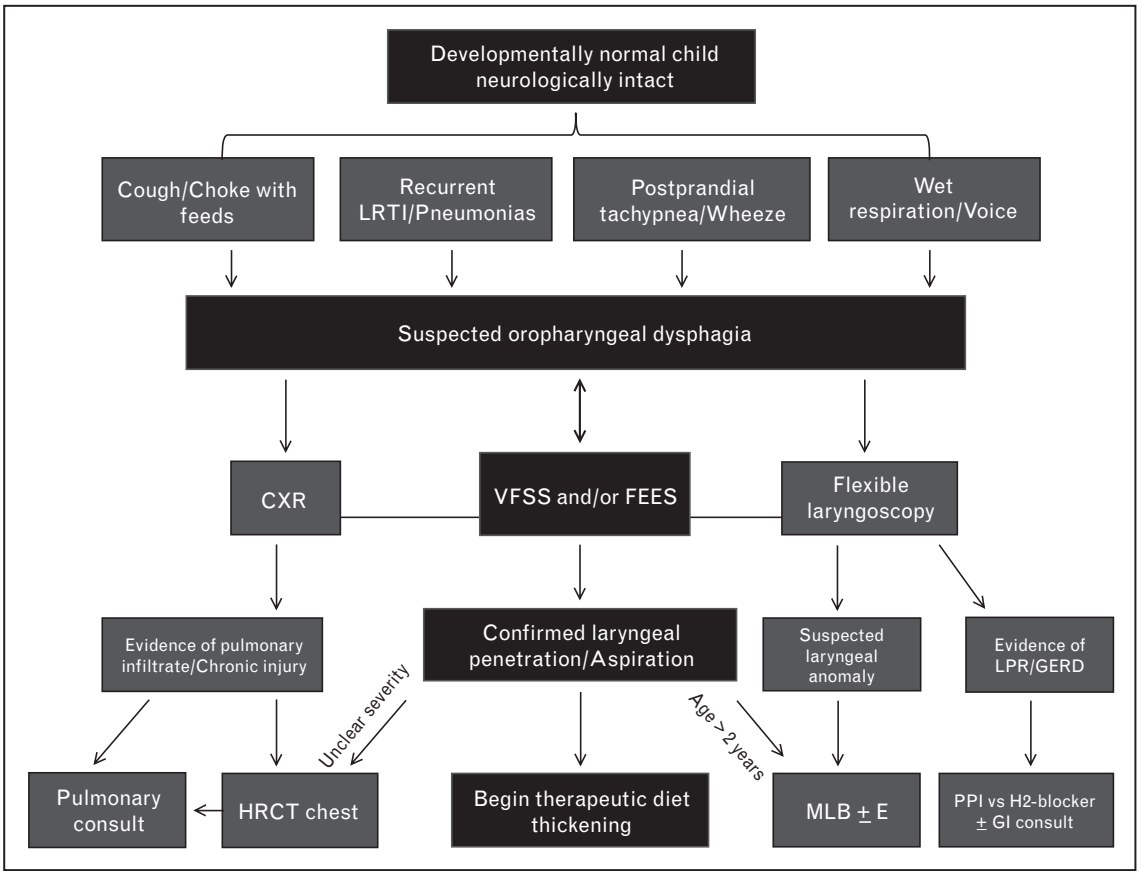
Instrumental swallowing evaluations

When a child presents with OPD, the safest initial management, until potential etiologies can be addressed, is best dictated by the results of instrumental swallow evaluations. Instrumental studies allow observation of the pharyngeal and upper esophageal phases of swallowing. Interpretations of these studies help determine the source of dysfunction and its relationship to the swallowing phase as seen in Table 1 [19,20]. The Videofluoroscopic Swallow Study (VFSS, a.k.a. modified barium swallow study) and the fiberoptic endoscopic evaluation of swallowing (FEES) are now considered gold standards for evaluating swallowing function in children. A delayed pharyngeal phase is reported to occur in the neurologically intact child with OPD [16••]. Advantages and disadvantages are associated with each assessment modality. Unfortunately, interpretation of VFSS and FEES exams in children still lack universally accepted standards.

Three-ounce water swallow challenge

In the attempt to provide an expedient and sensitive bedside tool for evaluating risk of aspiration in children, Suiter *et al.* [21••] examined the 3-oz. water swallow challenge. Based upon previous work in adults, these

Figure 1 Diagnostic algorithm for neurologically intact child with suspected oropharyngeal dysphagia



CXR, chest radiograph; E, esophagoscopy; FEES, fiberoptic endoscopic evaluation of swallowing; GERD/LPR, gastroesophageal reflux disease/laryngopharyngeal reflux; GI, gastroenterology; HRCT, high resolution spiral computed tomography; H2, histamine-2-receptor; MLB, operative microlaryngoscopy with bronchoscopy; PPI, proton pump inhibitor; VFSS, Videofluoroscopic Swallow Study.

authors demonstrated that OPD can sensitively be detected in children if coughing, choking, or tearing occurred during or shortly after drinking 3 oz. of water. The results were compared with FEES data and showed sensitivity for predicting aspiration status to be 100%. This correlated well with clinically relevant evidence (symptom-specific) of aspiration in children with pharyngeal dysphagia but was associated with a high false-positive rate. The inability of such a test to assess silent aspiration may limit applicability to all children with dysphagia. However, the exam may be particularly important as an adjunctive swallowing test for neurologically intact and developmentally normal children with VFSS evidence of dysphagia. Its role in the more common neurologically affected child with dysphagia remains unclear.

Initial treatment

Integrating data from instrumental swallowing evaluations with clinical history is paramount before treating OPD in the developmentally normal child. Cardiopulmonary status, number and degree of lower respiratory infections, and the extent of penetration, aspiration, and ability to clear secretions should all be considered. The rate and degree of laryngeal penetration and aspiration on VFSS or FEES exams often determine the temporary treatment regimen as these are believed to be good indicators of the patient's risk for aspiration. In children, the initial approach is to adjust diet consistencies. For example, an 18-month-old who aspirates thin liquids and has deep penetration with nectar consistency will be advised to thicken liquids to honey consistency until the next VFSS. In contrast, a child with laryngeal penetration with thin liquids without aspiration or a history of pulmonary infections will likely tolerate intermittent thin liquids at minimal risk. The speech pathologist, radiologist, and treating physician should confer on VFSS results and aspiration risk before concluding the best management option.

Compensatory feeding strategies to improve pharyngeal clearance are recommended as an adjunct to dietary changes in children with OPD. These strategies include, but are not limited to, upright positioning during feedings

Table 1 Findings on Videofluoroscopic Swallow Study and fiberoptic endoscopic evaluation of swallowing associated with suspected disorders during the pharyngeal phase of swallowing

Study finding	Clinical significance
Videofluoroscopic Swallow Study	
Penetration to laryngeal surface of epiglottis	Incoordination, ↓ pharyngeal constriction
Penetration into laryngeal vestibule	↓ Airway closure
Residue in pyriform sinus with laryngeal penetration	↓ Pharyngeal contraction, ↓ UES opening
Residue in pharyngeal recess	↓ Tongue base retraction, ↓ pharyngeal contraction, ↓UES opening
Aspiration before swallow	Delayed pharyngeal swallow initiation
Aspiration during swallow	Incoordination, delayed pharyngoglottal reflex Unilateral true vocal cord paralysis
Aspiration after swallow	Reduced pharyngeal pressure
Functional endoscopic evaluation of swallowing	
Preswallow penetration/aspiration	Delayed swallow initiation Impaired oral bolus handling Oropharyngeal anomaly redirecting bolus
Penetration/aspiration during swallow	Absent vocal fold closure (? paralysis) Delayed pharyngoglottal reflex Laryngeal cleft
Postswallow penetration/aspiration	Residue spillage Cricopharyngeal dysfunction Laryngopharyngeal sensory deficits
Residue valleculae after swallow	↓ Tongue base contraction ↓ Hyolaryngeal elevation ↓ Pharyngeal contraction
Residue in pyriform sinus	↓ Pharyngeal contraction ↓ UES opening
Absent/diminished pharyngeal squeeze	Neuromuscular weakness/incoordination

UES, upper esophageal sphincter. Data from [19,20].

along with reduced rates and lower volumes at more frequent intervals. When neurologic and anatomic etiologies for OPD are not found in the developmentally normal child, LPR should be seriously considered. Initial medical therapy for GERD/LPR has been well described in otolaryngology literature [22]. First line treatment includes the use of histamine(H)2-receptor blockers at 2–5 mg/kg twice daily and reflux precautions. Persistent symptoms beyond 1–2 months may require a therapeutic change to proton pump inhibitors (PPI) at 1–1.5 mg/kg separated into twice daily dosing. Refractory GERD/LPR or nighttime breakthrough reflux can benefit from nightly H2-blocker therapy. Treatment regimens for LPR may be impacted by local insurance provider requirements.

A follow-up in 3–6 months with another instrumental evaluation of swallowing will allow time for improved neuromuscular coordination, improved LPR, or the emergence of subtle neurologic or anatomic contributors to OPD. Failure to achieve developmental milestones, low tone, weak cry, or worsening symptoms should prompt neurologic evaluation and an MRI of the head. This may be the only presentation of minor cerebellar infarcts, chiari malformations, vascular anomalies, or tumors impacting the central nuclei (nucleus tractus solitarius, ventral medial reticular formation) involved in swallowing function [23••]. Patients with subtle neuromuscular conditions can then be recategorized.

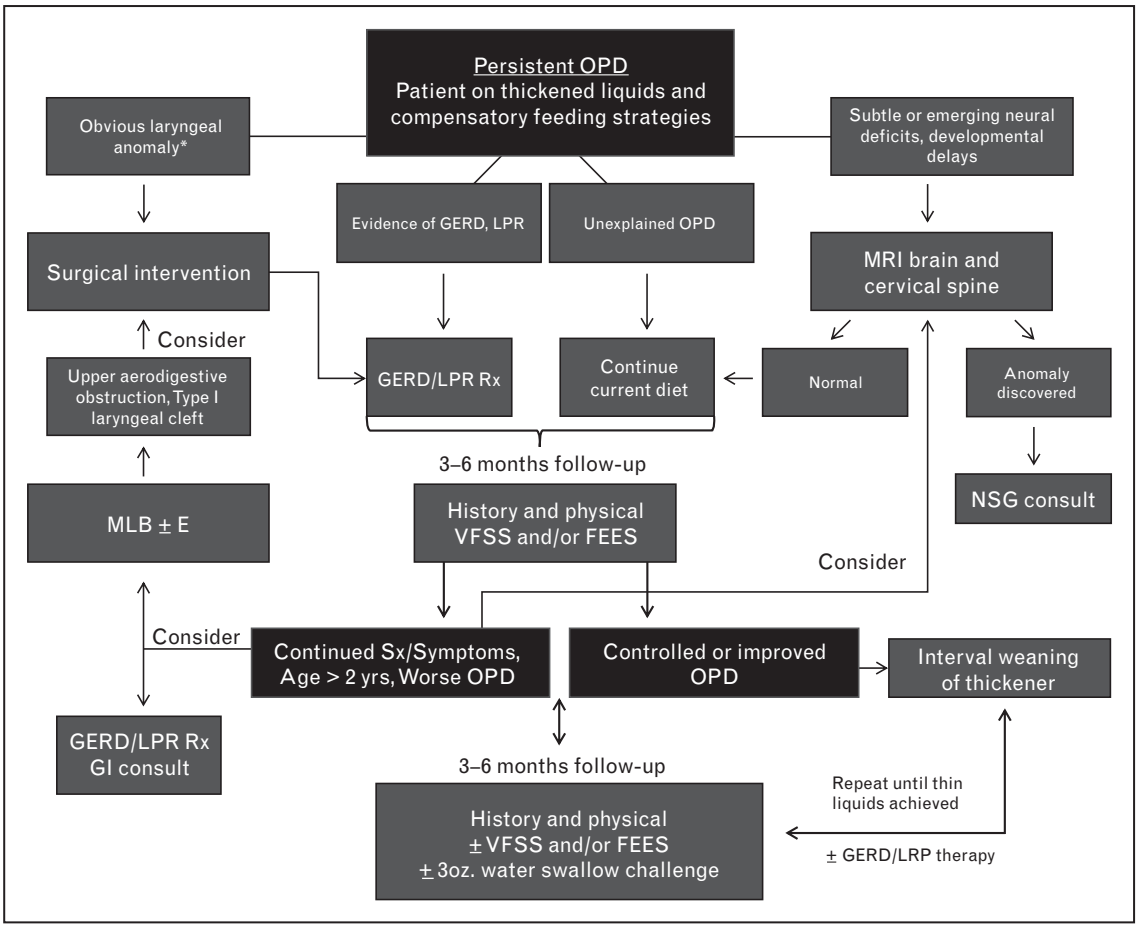
Dysphagia associated with prematurity or GERD/LPR frequently improves with time. During this time, children can proceed with their safest diet without inordinate parental and patient stress. Worsening signs and symptoms, however, should prompt further testing. Figure 2 outlines additional maneuvers to assess neurologically intact children with continued signs and symptoms of OPD. Despite a detailed exam and history, isolated OPD may still be present without a recognizable cause [23••,24,25].

When obvious anatomic explanations for OPD are discovered, such as deep laryngeal clefts (type II–IV) or severe laryngomalacia, then surgical intervention is performed. However, swallowing dysfunction should not be expected to improve immediately, as coordination of repaired structures requires time to reset. This is evident from prior studies. For example, Bakthavachalam *et al.* [26••] demonstrated that the mean time to resolution of dysphagia following type I laryngeal cleft repair was 7.8 months. Similarly, Richter *et al.* [27] noted a median 3.8-month interval between supraglottoplasty and improvement of aspiration in infants with severe laryngomalacia. Parental counseling regarding a slow return to normal swallowing while maintaining a safe diet is appropriate until further evidence (VFSS or FEES) or clinical signs of aspiration show improvement.

Persistent oropharyngeal dysphagia in the developmentally normal child

The workup of persistent OPD in the neurologically intact child should consist of exploring less common and potentially controversial etiologies of OPD

Figure 2 Management algorithm for persistent oropharyngeal dysphagia in the neurologically intact and developmentally normal child



*Type 2 or higher laryngeal cleft, severe laryngomalacia. E, esophagoscopy; FEES, fiberoptic endoscopic evaluation of swallowing; GERD/LPR, gastroesophageal reflux disease/laryngopharyngeal reflux; H and P, history and physical; MLB, operative microlaryngoscopy with bronchoscopy; NSG, neurosurgery; OPD, oropharyngeal dysphagia; PPI, proton pump inhibitor; VFSS, Videofluoroscopic Swallow Study.

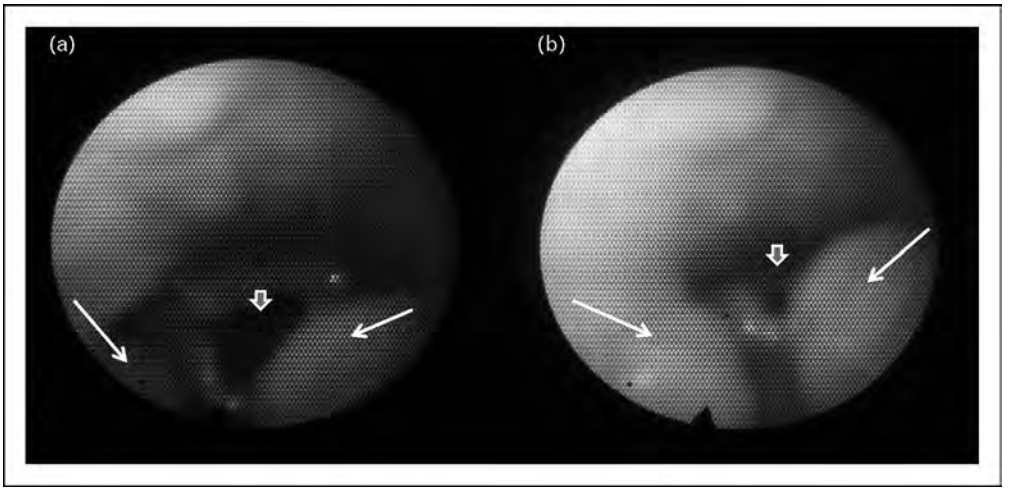
(Fig. 2). Potential sources of persistent OPD in the presumed neurologically intact and developmentally normal child are as follows:

(1) anatomic
    (a) laryngeal cleft
    (b) laryngomalacia
    (c) tonsillar hypertrophy
    (d) cricopharyngeal achalasia,
    (e) vocal cord weakness
(2) physiologic
    (a) GERD/LPR
    (b) eosinophilic esophagitis
    (c) sinonasal secretions
    (d) food allergy
(3) subtle neuromuscular
    (a) type I chiari malformation
    (b) rare intracranial pathology
    (c) prematurity
    (d) mild developmental delays.

Sources of upper airway obstruction should be examined, including choanal atresia, severe adenoid hypertrophy, lingual or palatine tonsillar hypertrophy, laryngomalacia, and tracheomalacia. Although not specific to these anatomic entities, evidence suggests that subtle discoordination of breathing with eating (suck–swallow–breathe) may contribute to OPD when anatomic obstruction is present [27,28••,29]. Laryngoscopy performed during a clinic visit can also detail vocal cord weakness or chronic irritation of the supraglottis and postcricoid area. Sinonasal secretions, GERD/LPR, or other inflammatory conditions like eosinophilic esophagitis may be the source of laryngeal inflammation and sensory blunting [17,18,30•,31].

A brief FEES examination during laryngoscopy in the cooperative child may provide new information. For example, hypopharyngeal pooling and aspiration from cricopharyngeal achalasia may be present. Similarly, the relationship between laryngomalacia and microaspiration

Figure 3 Laryngeal penetration and aspiration in a child with palatine tonsillar hypertrophy



(a) Flexible laryngoscopic view of 4-year-old with progressive dysphagia and palatine tonsillar hypertrophy (long arrows) impeding epiglottis inversion. (b) Compressed tonsils can displace thin and nectar thick liquids into the laryngeal vestibule (small grey arrow) with intermittent aspiration during the pharyngeal phase of swallowing.

has been reported using FEES [27,32]. Impediments to laryngeal elevation and epiglottic inversion as seen with lingual and palatine tonsillar hypertrophy can also be detected (Fig. 3) [29,33]. Redirection of the liquid bolus into the larynx by prominent palatine tonsils before vocal cord closure can also be viewed by FEES. In the toddler who presents with typical signs, symptoms, and VFSS evidence of OPD without prior history, tonsillar hypertrophy should be closely scrutinized.

Utilization of both VFSS and FEES is recommended when OPD persists. In these enigmatic patients anatomic and functional sources of OPD need to be explored, which are best identified by FEES and VFSS, respectively. In collaboration with speech pathology, the otolaryngologist is better equipped to locate anatomic contributors to OPD during FEES. Some evidence suggests that sensitivity is also improved with FEES compared with VFSS [34]. On the contrary, VFSS with speech pathology provides a better examination of pharyngeal function and squeeze, which is obscured during the 'white out' phase of a FEES exam. Thus, together VFSS and FEES provide complete upper aerodigestive visualization and the extent of pharyngolaryngeal function during the pharyngeal phase of swallowing.

Because results from VFSS may be the primary reason for an encounter with a dysphagia expert, direct observation of the study by the treating physician is ideal. More importantly, instrumental swallowing studies in this patient population should be interpreted with several concepts underscored. The first tenet is that the same degree of laryngeal penetration and aspiration does not affect each patient equally [5]. Second, healthy patients can display occasional and transient laryngeal penetration [19,35]. Third, laryngeal penetration and aspiration is an episodic phenomenon and may be exacerbated in children, or only be evident, during the stress of instrumental swallowing evaluations. Finally, VFSS and FEES are confounded by poor intra-rater and inter-rater reliability as normative data for these tools remain unavailable for comparison [36]. When the clinical history and exam does not support VFSS results, then persistent OPD in the otherwise normally developing child should be reassessed. In this setting, a 3-oz. water swallow challenge may prove valuable. A normal HRCT of the chest, without evidence of chronic pulmonary disease, may also be useful.

A complete MLB is recommended in a neurologically intact child when persistent symptoms and VFSS/FEES evidence of OPD exist beyond 2 years of age. Children who initially present to a dysphagia expert at an age of more than 2 years should also be considered for MLB. OPD secondary to prematurity and GERD often resolves before this age. Thus, some authors advocate that anatomic abnormalities shall be the presumed cause of OPD in older children who are otherwise healthy [37••]. Examination for a deep interarytenoid notch (a.k.a. grooves), type I laryngeal cleft, and cricopharyngeal achalasia should be part of the assessment when persistent OPD is present. Shallow laryngeal clefts are best identified when the interarytenoid space is palpated [37••]. A tonsillectomy (palatine or lingual), under the same anesthetic, can be considered if suspected to be the source of OPD. Similarly, esophagoscopy with biopsies will allow identification of cricopharyngeal achalasia or eosinophilic esophagitis. Eosinophilic esophagitis is a known source of

laryngeal inflammation and dysphagia but a direct link, although suspected, to aspiration has not been determined [30•,38,39].

The utility of bronchial alveolar lavage (BAL) for lipid-laden macrophages during MLB to assess for chronic pulmonary aspiration remains controversial and is no longer routinely performed for OPD [40]. However, BAL may be a useful marker of disease severity and therapeutic progress in the enigmatic patient with OPD who appears otherwise developmentally normal. As an example, the mean lipid-laden macrophage index in children with type II laryngeal clefts was shown by Keiran *et al.* [41•] to be significantly higher than in those with type I laryngeal clefts.

Once subtle etiologies are discovered, then medical or surgical intervention can proceed with confidence. Calculated treatment of subtle inflammatory and anatomic anomalies can be performed safely in the child with OPD of unclear etiology. Nasal steroids and medical treatment of GERD/LPR (H2-receptor blockers, proton pump inhibitors) may improve laryngeal sensitivity and the pharyngoglottic closure reflex. Elimination diets for suspected food allergies as directed by GI may also be helpful in some children. Surgical management of sources for airway obstruction will also presumably lead to better coordination and space for bolus handling during the pharyngeal phase of swallowing. This should only be performed once other anatomic and inflammatory sources of OPD are systematically ruled out.

Reassessment of safe alimentation should be performed with a 3–6-month interval between therapy (medical or surgical) and instrumental swallow evaluations. This allows time for healing, neuromuscular rehabilitation, and pharyngolaryngeal coordination to develop after surgical intervention. Inflammatory conditions may take more time to resolve during medical treatment and require repeat assessments.

Introducing safe alimentation

When anatomic sources of dysphagia cannot be clearly demonstrated and inflammatory conditions are not readily apparent, then management of OPD in the neurologically intact child may consist primarily of reflux therapy, observation, repeated instrumental evaluations of swallowing, and the gradual introduction of thinner diet consistencies. These patients are often ex-premature infants (<37 weeks) with LPR who generally resolve their OPD before school age. Delayed or aberrant laryngeal chemoreceptor sensitivity in these patients may explain this finding [15,42••]. Dysphagia in children due to cow's milk protein allergy has been previously reported and should be addressed [43]. Interestingly, a significant percentage of children at our institution with unexplained OPD have been found to have cow's milk protein intolerance that improves with a change in formula (unpublished data). Nonetheless, a step-wise and systematic approach to graduating patients to safer alimentation is necessary and may expedite the return to a normal diet.

Diet weaning is also important for patients in whom a cause of OPD has been determined and addressed. Training of the involuntary swallowing reflex and pharyngoglottic closure seems critical to successful reduction of thickened consistencies despite the etiology of OPD. Even with repair or resolution of the inciting pathology (i.e. laryngeal cleft, LPR, cricopharyngeal achalasia), development of swallowing coordination will take several months [26••,27,37••]. In essence, function follows form with interval training necessary in dysphagia management in children. Many children will have been on a thickened diet for years. Common sense suggests that time for training pharyngeal and laryngeal neuromuscular coordination is required. Research on successful weaning parameters is currently not available. The management of the neurologically intact child with OPD should be vigilant but with the expectation, by the physician and caretakers, that it may take months or years to resolve. Some research suggests that the average time to return to normal diet in the developmentally normal child with OPD is 3.2 years [16••].

Signs and symptoms of aspiration are the best predictors of successful weaning and return to normal swallowing function. When silent aspiration is not present on VFSS, then symptoms of coughing and choking during swallow shall help parents and practitioners determine the best diet consistency. Other symptoms, such as watering eyes, wet respiration, and tachypnea, will help gauge poor alimentary control in silent aspirators. Interestingly, this is common in neurologically intact children with OPD [3]. Bronchopulmonary events, such as worsening asthma, increased use of respiratory medications, recent pneumonia, and recurrent lower respiratory infections, are also clear indicators of unsuccessful dietary weaning.

The initial diet consistency recommended for a child with OPD depends upon VFSS or FEES results. This is usually a consistency one level greater than that which is penetrating the larynx or aspirated below the vocal folds. Dietary weaning should then consist of slow introduction of thinner consistencies than that first proposed. Broad strokes in dietary changes (i.e. from honey to nectar) are typically unsuccessful and OPD symptoms will recur. Therefore, dietary trials comprising half-step increments in consistency training are recommended. For example, a child on a honey-thickened diet can begin weaning on 75% honey (a.k.a. thick nectar). This is introduced and continued until the next clinic visit unless signs or

symptoms of penetration/aspiration occur. The original consistency is reintroduced to the child (for 1-week duration) only if symptoms (cough, choking, and wet respiration) recur. When the child is symptom-free again, then a return to the new, thinner consistency is recommended.

This intermittent introduction of thinner consistencies allows safe and graduated improvement in swallowing function through interval training. When on a weaning program, patients can return to the clinic for periodic evaluations at 3–6 months. Patients can also be weaned from reflux medications for GERD/LPR if they are improving in their diet consistency without signs and symptoms of penetration, aspiration, or LPR. If the child with OPD is tolerating the new consistency, then another half-step increment in consistency weaning can be performed. This process can be repeated every 1–2 months as long as signs and symptoms of aspiration remain absent.

VFSS and FEES exams can be repeated at 9–12 month intervals to check the status of the weaning protocol in successful patients. This can reduce the amount of radiation exposure (especially when FEES is employed) and potentially traumatic testing in children while allowing the practitioner to treat the patient based on symptomatic improvement rather than diagnostic studies. When successful weaning does not occur, then a repeat VFSS and FEES is appropriate at earlier intervals. With astute parents and detailed planning, weaning protocols can be instituted at home and repeat VFSS and FEES exams are unnecessary in the absence of signs and symptoms.

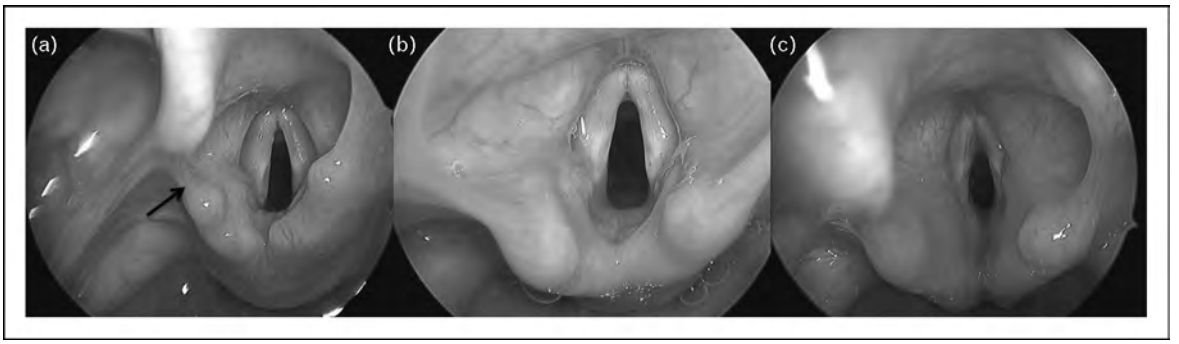
The ideal candidate for early dietary weaning attempts is the child with laryngeal penetration. Some evidence suggests that these patients are at no greater risk for pneumonia or lower respiratory tract infections than children within the community. Using multivariant analysis, Weir *et al.* [44••] demonstrated that children with aspiration on VFSS had the same risk of developing pneumonia as other children in the community when other risk factors for pneumonia were excluded. However, cautious introduction to thinner consistencies should be performed in children with underlying respiratory conditions.

Laryngeal clefts and deep interarytenoid notches

The diagnosis and management of type I laryngeal clefts and deep interarytenoid notches is a remarkable challenge to the pediatric otolaryngologist. A confident standard for detection has not been established. Shallow laryngeal clefts may be present in many neurologically intact children with OPD and may require repeated endoscopy for identification. A thorough examination for this defect is particularly important in the older child. Type I laryngeal clefts have also been attributed to infants with congenital stridor, OPD, and laryngeal anatomy similar to laryngomalacia [45]. To complicate the diagnostic picture, children with unexplained OPD present similarly to those with laryngeal clefts in terms of signs, symptoms, and VFSS results (aspiration with thins, laryngeal penetration with nectar). Sometimes only subtle differences in anatomy are apparent (Fig. 4).

As evidenced by recent reports on shallow laryngeal clefts (type I and type II), some children return to normal swallowing function without surgical intervention [26••,37••]. However, this recovery is often delayed relative to those children undergoing repair and highlights the importance of a thorough risk assessment prior to surgical correction [26••]. Surgical intervention for subtle laryngeal clefts should be tempered with the patient profile. Serious consideration for cleft repair might include toddlers with persistent OPD not responding to medical management and infants with recurrent pneumonia and a worsening pulmonary status.

Figure 4 Anatomic findings on intraoperative microlaryngoscopy in neurologically intact children less than 2 years of age with the same Videofluoroscopic Swallow Study results and oropharyngeal dysphagia



(a) No evidence of laryngeal cleft as the posterior cricoid mucosa rises to the level of the false vocal folds. Anatomic correlates to laryngomalacia, such as short aryepiglottic folds and prominent cuneiform cartilage, are found (small arrow). (b) A child with type I laryngeal cleft with superior portion of cricoid below the level of true vocal folds. (c) 15-month-old with type II laryngeal cleft.

Conclusion

Laryngeal penetration and aspiration in the neurologically intact and developmentally normal child is underreported. Systematic evaluation of this patient population has proved difficult, leaving only a few research reports available for review. Extrapolation from these reports and examination of the dysphagia literature for children can provide some rudimentary clinical guidelines for patient management. Although provided in this text, additional studies are necessary to examine the effectiveness of the proposed algorithms.

Subtle anatomic and inflammatory conditions can explain OPD in otherwise healthy children and can be treated appropriately. Surgical therapy of anatomic sources will expedite the return to normal diet. Swallowing training with gradual introduction of thinner consistencies seems necessary for the safe reintroduction to thin diets despite the etiology of OPD in the neurologically intact child.

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 582).

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