ABSTRACT: This article presents an overview of the normal anatomy and physiology of the pediatric larynx, followed by some examples of pediatric voice disorders that were chosen to exemplify the alterations to the laryngeal anatomy and the subsequent modifications to laryngeal function. Vocal fold nodules are primarily reviewed due to their high incidence in the pediatric population. Three other disorders, including unilateral vocal fold paralysis, hyperfunction without the presence of a lesion, vocal polyps, contact ulcers, and vocal fold paralysis (McMurray, 2003). The alterations in laryngeal structure associated with congenital voice disorders are implied in their titles. On the other hand, acquired pediatric voice disorders evoke the structural and functional changes that are typically noted in their adult forms. More recent research would suggest that the nature of vocal disorders in adults and children differs, and this is related to anatomical and physiological differences in the larynx (e.g., McAllister, 1997; Netsell, Lotz, Peters, & Schulte, 1994; Stathopoulos & Sapienza, 1997).

Children differ from adults in the way they produce their voice. These functional differences need to be considered when counseling children and their families about the causes of voice disorders, as well as the rationale for choosing the therapies that are used for the rehabilitation of a particular disorder. The next section describes the laryngeal anatomical and physiological features of the child larynx that should be considered distinct from the adult’s.

KEY WORDS: voice, children, structure, function

LARYNGEAL ANATOMY AND PHYSIOLOGY IN CHILDREN

When evaluating the pediatric voice, there are a number of factors to consider. Among these factors are distinctions in laryngeal anatomy between children and adults. These...
distinctions require consideration in the evaluation of pediatric voice disorders, particularly when completing a visual examination of the larynx (laryngoscopy) to determine the normality of the laryngeal structure. The most obvious laryngeal anatomical difference between children and adults is the size of the larynx. The pediatric larynx is smaller than the adult larynx. In newborns, the length of the vocal fold is 2.5–3.0 mm, with continual growth as a function of age. The appearance of sex differences in vocal fold length occurs sometime between 10 and 14 years of age. Adult vocal fold length is approximately 17–21 mm in adult males and 11–15 mm in adult females (Hirano, Kurita, & Nakashima, 1983). Total vocal fold length for females and males increases until 20 years of age.

Further distinction between pediatric and adult laryngeal anatomy is found in the proportion of membranous versus cartilaginous structure that is present. In the newborn and young child, the membranous portion of the vocal fold makes up less of the total vocal fold length in comparison to that of the adult. Histological differences also exist between pediatric and adult vocal fold structure. The vocal fold mucosa is thinner in newborns and young children, with the ratio of the mucosa to the length of the membranous portion of the vocal fold greater as compared to the adult structure. Furthermore, the layered structure of the vocal folds is not differentiated in newborns and young children; the lamina propria is very uniform in structure. In adults, there is clear differentiation between the superficial, intermediate, and deep layers of the lamina propria. There is no ligamentous structure in newborns, with an immature one emerging between the age of 1 and 4 years. After the age of 4 years, a ligamentous structure is observable (Hirano et al., 1983). Additional histological distinctions include a greater percentage of collagen in the pediatric vocal fold muscle and less dense anterior and posterior macula flava fibers, which implies less anchoring strength of the laryngeal structures (Hirano et al., 1983).

Other important anatomic features of the pediatric larynx that may have implications for the interpretation of a laryngoscopic examination include high laryngeal position, the shape of the epiglottis, and the diameter of the glottal and subglottal spaces. The pediatric larynx lies between the first and third cervical level in comparison to an adult’s, which continues to lower with advancing age, with the lower border of the cricoid cartilage hovering between the sixth and seventh cervical vertebrae (Fried, 1983). In the pediatric larynx, the laryngeal framework descends into the neck with growth and elongates the oropharynx and hypopharynx, taking it from a conical shape to a more cylindrical contour. With development, laryngeal–pharyngeal widening occurs along with an increased diameter of the cricoid cartilage and expansion of the alar wings of the thyroid cartilage.

The epiglottis is omega shaped in approximately 50% of the pediatric population (Sylvan, 1983) and should not be considered anomalous. The presence of a pliant and soft epiglottis is also a characteristic of pediatric anatomy. It is often in direct contact with the soft palate, particularly in infants. Also, it is in more direct contact with the tongue base, which allows easier inversion into the laryngeal space, as is the case with laryngomalacia (i.e., epiglottis collapse into the airway).

Moreover, the subglottal space in a child’s airway is the narrowest of the airway in comparison to the adult airway, with the full-term diameter of the subglottal space cited as 4 mm. The narrowest portion of the adult airway is the glottis. Also, the subglottis in children is the least pliable part of the airway. The cricoid ring is the only portion of the airway that is surrounded completely by cartilage. It is this area that is most susceptible to the development of laryngeal trauma and laryngeal stenosis (Wiatrak, 2002). Finally, there is little calcification to the pediatric laryngeal cartilages. The whole laryngeal framework in children is much softer than in adults, which makes it less susceptible to blunt trauma but more susceptible to collapse due to negative inspiratory pressures developed during breathing.

Figure 1 shows an anterior view of the larynx (Hirano et al., 1983) depicting the cartilaginous framework of an immature versus a mature structure. Note that the hyoid bone in the immature larynx assumes a much lower anatomical position and actually overlaps the thyroid cartilage. Additionally, there is no vertical prominence in the thyroid cartilage. The presence of a vertical prominence (i.e., Adam’s apple) does not occur until substantial changes happen in vocal fold length (approximately between the ages of 10 and 14 years). The thyroid cartilage does not assume its adult configuration until adolescence. From Figure 1, it is also apparent that the cricothyroid membrane is a slit rather than a palpable space. Because of these anatomical distinctions, the internal examination of a child’s larynx will be unlike that of an adult’s. In addition, the external examination of the larynx and anatomical landmarks will be distinctly unique in children.

Figure 1. Anterior view of the cartilaginous laryngeal framework showing a depiction of an immature larynx and a mature larynx.

A posterior view of the cartilaginous framework of the immature larynx (see Figure 2) reveals a furled epiglottis and proportionately larger arytenoids than those seen in adults. The large arytenoids, the shape of the epiglottis, as well as large aryepiglottic folds may obscure the view of the true vocal folds and glottis during laryngoscopic examination. Figure 3 shows an endoscopic view comparing the immature and mature laryngeal structures. In children, there is a more anterior insertion of the vocal folds into the vocal process of the arytenoids. Again, this endoscopic view also depicts the proportionately larger arytenoids as compared with the other laryngeal structures. Furthermore, the pediatric vocal folds also have a downward slant from posterior to anterior, which is a characteristic that is not found in the adult larynx. The posterior glottis of a pediatric client is approximately 50% of the adult length. This has been termed by Hirano and colleagues as the respiratory glottis, and in the infant particularly, the respiratory and protective functions have a larger role than in phonation (Hirano et al., 1983).

One other influence that may affect the ability of the endoscopist to successfully obtain a view of the pediatric larynx is laryngeal alignment (Fried, 1983). The trachea, pharyngeal, and oral axes must be aligned to assist with endoscopy. The back of the head of the infant and young child should be elevated slightly to help with this alignment. Too much cervical flexion or improper elevation of the head will result in incorrect positioning and create difficulty in passing an endoscope. Without proper alignment, structures will be obscured, and there is a possibility of causing discomfort to the child during an examination.

As the larynx grows, the anatomical distinctions between the pediatric and adult larynx become less significant. There is an unfurling of the epiglottis, increased stiffness of the cartilages, and a decrease in the relative size of the

**Figure 2.** Posterior view of the cartilaginous laryngeal framework showing a depiction of an immature larynx and a mature larynx.

---

**Figure 3.** Endoscopic view of the larynx depicting distinctions in laryngeal anatomy between an immature and a mature larynx.

---


---

arterytoids and areyepiglottic folds with development/maturation. This maturation process helps reduce the consequences of certain disorders, such as laryngomalacia, and improves the chances of completing a successful examination of the larynx. It is the awareness of this course of laryngeal maturation and its effect on laryngeal growth that can assist in evaluating the larynx and determining the overall treatment plan and prognosis of a child’s condition.

**Laryngoscopic Examination**

Generally, the laryngoscopic procedures used to examine a pediatric larynx are quite similar to those employed with adults. There are, however, some differences in the preparation of the pediatric client that must be considered given the distinctions in laryngeal anatomy that were just reviewed. First, the flexible scope selected for examination of the larynx is smaller, approximately 2.7–3.0 mm diameter for the pediatric client as compared to 3.4 mm diameter for the adult. Children may also have more anxiety about the procedure than adults do, and clinicians should be prepared to change how they introduce the procedure and how they coach the child through the procedure. The school speech-language pathologist (SLP) can help prepare the child before the procedure by explaining the process and showing the child how the procedure is done.

Of course, the endoscopic examination of the larynx is only one part of a comprehensive evaluation of the pediatric client presenting with dysphonia. There are other clinical observations that accompany the visual examination of the larynx that provide a record about a suspected dysfunction. These observations include, but are not limited to, medical history, which includes information from the primary care physician and other physicians regarding associated symptoms with the digestive, pulmonary, and neurological systems; an oral peripheral examination; observation of body posture; elicitation of reflexes, such as
sensory reflexive testing or motor reflexive testing; auditory–perceptual evaluation; and instrumental analysis. Within each of these categories of examination, the differences between pediatric and adult anatomy and physiology should be considered. However, the scope of this article does not offer a full presentation of this information. A brief summary of some of these evaluation procedures and their diagnostic role is provided in Table 1.

When determining the effects of pediatric laryngeal dysfunction/pathology on vocal fold vibration, clinicians are advised to turn to the appropriate and available pediatric normative literature. Adult normative laryngeal function data should not be used as a comparison for children’s function, anymore than a child should be asked to do tasks that are adult-like. It is the clinician’s responsibility to bring his or her knowledge of pediatric structure and function to the clinic. This, in addition to patience, time, and creativity, will help ensure an accurate evaluation of the pediatric voice patient.

### EFFECTS OF ALTERED LARYNGEAL ANATOMY AND PHYSIOLOGY IN PEDIATRIC VOICE DISORDERS

#### Laryngeal Hyperfunction

The loud voice of a child is recognizable. It is not uncommon to hear the outcome of laryngeal hyperfunction in a school-aged child or see the extrinsic laryngeal muscle tension in the child’s neck as he or she speaks. False vocal fold (FVF) adduction and compression of the arytenoid cartilages to the petiole (the stalk of the epiglottis that serves as the attachment to the larynx) of the epiglottis in an anterior to posterior (A-P) direction are thought to characterize hyperfunctional voice disorders with abnormally increased muscle tension or effort (see Figure 4).

Anatomically, there are immediate effects of hyperfunction, but over time, if these behaviors are maintained, the vocal fold irritation turns into edema (swelling), causing irregular vocal fold edges, or possibly the development of vocal fold nodules, vocal fold polyps, or contact ulcers. Laryngeal hyperfunction is usually caused by vocal behaviors that are not healthy for the vocal fold layers, particularly the mucosa of the vocal folds, and prevents the vocal folds from functioning optimally. This condition is normally reversible with the elimination of laryngeal hyperfunction coupled with a program of vocal health.

**Table 1.** General overview of relevant information gathered during assessment.

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Purpose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical history</td>
<td>To gather relevant information regarding gestation and birth that are contributory to vocal function/dysfunction.</td>
</tr>
<tr>
<td>Overall health</td>
<td>To reveal any chronic/common conditions (i.e., ear infection, cold, excessive coughing) that can contribute to vocal abuse/misuse.</td>
</tr>
<tr>
<td>Examination of the head and neck</td>
<td>Usually performed by an otolaryngologist. This assessment will reveal any abnormalities that may relate to the voice and speech mechanisms.</td>
</tr>
<tr>
<td>Examination of the cranial nerves</td>
<td>To determine if there is a lesion associated with the cranial nerves that would alter laryngeal function.</td>
</tr>
<tr>
<td>Oral peripheral</td>
<td>To evaluate the relative size, shape, and function of the structure within the oral cavity.</td>
</tr>
<tr>
<td>Family history</td>
<td>To determine if there are any significant medical issues in the family that may precipitate laryngeal dysfunction.</td>
</tr>
<tr>
<td>Neurological exam</td>
<td>To rule out any neurological or neuromotor disorders that would affect the overall function of the larynx and its support system.</td>
</tr>
<tr>
<td>Psychological exam</td>
<td>To determine if any mental health issues contribute to the voice problem. Parents may need probing for information regarding the client’s personality that may be contributory.</td>
</tr>
<tr>
<td>Nutrition</td>
<td>To determine if any significant eating habits may contribute to the voice problem (i.e., reflux, bulimia, dehydration).</td>
</tr>
<tr>
<td>Current medication</td>
<td>To obtain a list of current medications to evaluate if there is any effect on vocal function.</td>
</tr>
<tr>
<td>Recreational/social</td>
<td>To determine if social factors are relevant to the client’s overall vocal health (i.e., sporting events, social groups).</td>
</tr>
</tbody>
</table>

**Figure 4.** Endoscopic view of the larynx depicting a case of pediatric laryngeal hyperfunction.
**Pediatric Vocal Fold Nodules**

Vocal fold nodules are the most frequently occurring voice disorder encountered in school-aged children (Benjamin & Croxson, 1987; Moran & Pentz, 1987), with an incidence report of more than 1 million school-aged children in the United States (von Leden, 1985). Vocal nodules have been described as small benign swellings along the margins of the vocal folds, which usually occur at the junction of the anterior and middle third of the vocal fold as a result of vocal trauma (von Leden, 1985; see Figure 5 for an example). They have also been described as small, white or grayish protuberances on the free margin of the vocal fold at the junction of the anterior and middle third of the vocal fold (Allen, Pettit, & Sherblom, 1991). Benjamin and Croxson (1987) characterized vocal nodules as a fusiform thickening of the mucosal surface. Others have described nodules as a localized edema and submucosal hemorrhage (Tucker, 1993) or an edema of the subepithelial tissue (von Leden, 1985). The general consensus of opinions points to vocal nodules as an edema or hemorrhage of some part of the mucosal layer resulting from persistent trauma at the anterior third and posterior two thirds of the vocal fold, the area of greatest mechanical stress.

Pediatric vocal nodules are typically of the same color as the vocal folds. von Leden (1985) suggested that the color can vary from pale to pink, with the nodule’s size varying from cone-shaped to round. “Simple nodules” have been characterized as soft and flexible, as opposed to “larger nodules,” which may appear as hard and fibrous. The size of the nodule is related to the length of time the vocal nodule has been present and the length of time the vocal fold has been exposed to irritation.

Vocal nodules are defined as benign bilateral lesions. When there is a case of a unilateral nodule, magnification typically reveals a small partner or irritation located directly on the opposite vocal fold (von Leden, 1985). Kotby, Nassar, Seif, Helal, and Saleh (1988) studied the ultrastructural features of vocal fold nodules to determine their distinct structural differences in comparison to other histologically common benign lesions, like vocal fold polyps. They concluded that nodules, in comparison to polyps, had a thicker epithelium, and that epithelial cellular degeneration was more frequent in nodules. Kotby and colleagues also found that the basal lamina was partly disrupted with intracellular deposits and gapping between the polyhedral cells of the nuclear membrane/cytoplasm, as well as hyaline degeneration unlike that found in polyp formation. So, although the two may appear to be similar in their superficial appearance, there are actual histological differences between nodules and polyps. In an article that addressed pediatric voice disorders, Gray, Schneider, and Smith (1996) described vocal nodules as basement membrane zone disorganization and abnormal extracellular matrix of the lamina propria. Batza (1970) described vocal nodules in children as benign fibrous and layered epithelial growths, with the earliest changes as thickening of the epithelium, along with submucosal edema. With continued irritation, fibroblasts proliferate, making it harder for the vocal fold to vibrate.

The formation of vocal nodules is most often correlated with hyperfunction. It seems that a child’s emotional/psychological and behavioral characteristics are factors that reportedly contribute to the development and/or maintenance of the vocal nodules. These characteristics may include aggression, disturbed peer relations, distractibility, screaming, immaturity, difficulty managing stressful situations, depression, anxiety, anger, loud talking, hyperactivity, frustration, and interpersonal problems (von Leden, 1985). The direct relationship, however, between psychological characteristics and the development of vocal fold nodules is still unclear (Greene & Mathieson, 1989). Physiological factors influencing vocal nodule development may include, but are not limited to, too high or too low blood circulation, too high or too low inspirational capacity and reserve volume, a high level of subglottal air pressure, an increase in laryngeal effort, increased tension in the thyrovocalis muscle, and dehydration (Greene & Mathieson, 1989). Gastroesophageal reflux may result in vocal fold edema and chronic throat clearing (leading to vocal abuse), which leads to the development of vocal fold nodules in children.

Voice quality disturbances that can occur with vocal nodules include hoarseness, excessive coughing, breathlessness, voice breaks, aphonia, dysphonia, and monotone or varying pitch (Hufnagle, 1982; Sapienza & Stathopoulos, 1994). These voice quality disturbances are not necessarily unique to vocal nodules, which reinforces the need for an endoscopic image to confirm their presence. A clinician who chooses to merely treat a voice symptom without verifying the disease process is in direct violation of ethical care.

The size of the nodule is somewhat related to the ability to sustain a stable tone. In other words, the larger the nodule, the shorter the sustained tone due to the increased mass of the vocal fold(s) and or irregular vibration of the vocal fold caused by the presence of the vocal fold nodule.

---

**Figure 5.** Endoscopic image of pediatric vocal nodules.
Sapienza and Stathopoulos (1994) found an increase of airflow in pediatric cases of vocal nodules. Without complete closure of the vocal fold and irregular vibratory motion, the result is a higher than average airflow and a greater amount of subglottal air pressure produced during speech tasks. The increased airflow may contribute to the perception of a breathy voice quality. Fundamental frequency or the pitch of the voice changes due to the presence of vocal nodules. This change occurs because there is an alteration to the vocal fold stiffness and mass.

Unilateral Vocal Fold Paralysis

The next type of voice disorder that is reviewed is unilateral vocal fold paralysis (UVFP). The reason this disorder was chosen is because of its complex nature, which affects not only voice quality, but also airway patency. This condition results in an alteration to laryngeal structures and subsequently changes how the vocal folds function to produce voicing.

UVFP occurs in children and is classified into congenital and acquired etiologies. This type of VFP can be caused by head and neck injuries, disease, or surgery (most commonly related to cardiothoracic surgery). Nerve damage to the vagus nerve, which has branches that run from the brainstem to the larynx and regulates the movement of the vocal folds, is the specific cause of VFP. The symptoms presented with UVFP can include breathy or hoarse vocal quality and difficulties generating normal vocal loudness levels. Although symptoms of stridor and dyspnea are more common in children with bilateral VFP, these symptoms are found in cases of UVFP as well (Daya, Hosni, Bejar-Solar, Evans, & Bailey, 2000). Stridor is defined as noisy breathing that may be present on both inspiration and expiration. Stridor and dyspnea may be exacerbated during increased exertion levels, and pose significant functional limitations for speaking while participating in even light exercise (i.e., speaking while walking with a group of friends). Children with a UVFP may have great difficulty “yelling” to peers in playground and sports activities.

The incidence of UVFP in neonates is one of the more common neurological or traumatic conditions, and the treatment is either surgical, behavioral, or a combination of both. In the preschool or elementary school years, the SLP’s role will be to treat the UVFP or deal with the residual effects of the surgically treated UVFP, completed when the child was young. These residual effects may include continued difficulties with voice quality. UVFP is more common than bilateral VFP and has less impact on airway patency, although stridor can be a symptom, particularly if the paralyzed vocal fold is positioned in a paramedian position (closer to the healthy fold), as compared to a lateral position. The diagnosis of UVFP needs to be made by a certified otolaryngologist similar to any dysfunction that is affecting the larynx. When UVFP is diagnosed, a waiting period of 3 months to a year may occur as the client’s laryngeal function is monitored for the potential return of function. Return of function, and its probability, is related to the cause of the disorder.

Daya et al. (2000) reported the recovery times of patients with VFP and indicated that neurological UVFP had the highest rate of recovery.

Bilateral Abductor VFP

Although typically thought to be less common, in the Daya et al. (2000) study, there was an almost equal distribution of UVFP as compared with bilateral VFP.

Bilateral abductor VFP can result from damage to the recurrent laryngeal nerves. The causes of this disorder are varied; however, the symptoms are fairly homogeneous. Children with bilateral VFP often present with two primary categories of problems that cause distress: breathing and voice. Often, it is the breathing symptoms that limit the function of these children socially and medically.

Damage to the recurrent laryngeal nerves can happen a number of ways and can be congenital or acquired (Daya et al., 2000). Congenital recurrent laryngeal nerve dysfunction is typically associated with other conditions, including hydrocephalus, meningomyelocoele, and meningocoele, which may be associated with the Arnold-Chiari malformation, an illness whereby the medulla and cerebellum protrude through the foramen magnum into the spinal column. Acquired cases of bilateral abductor VFP may be due to a surgical trauma where one or both of the recurrent laryngeal nerves are damaged. Surgical trauma, according to Benninger, Gillen, and Altman (1998), is the primary manner in which paralysis of this type occurs. One example of this is damage to the recurrent laryngeal nerve during thyroid surgery (Echeverri & Flexon, 1998). This damage may be bilateral or unilateral. When the nerve is damaged bilaterally, severe breathing and voice symptoms may result due to a limited glottal airway. When this occurs, a tracheostomy during the initial stages of the thyroid surgery recovery may be required due to significant airway obstruction. Although a tracheostomy is an effective treatment option, it is generally unacceptable as a long-term solution for maintaining breathing and voice (Segas, Stravroulakis, Monolopoulo, Yiotakis, & Adamopoulos, 2001). Finally, idopathic causes are linked to many cases of childhood bilateral VFP. With an idiopathic cause, spontaneous recovery will most likely occur over time. One study shows that recovery can occur up to 11 years post diagnosis of the paralysis (Daya et al., 2000).

Some researchers believe that there is an immaturity in the development of the abductor fibers of the recurrent laryngeal nerve or a fragility to those fibers that may result in dysfunction of the abductor muscles that, with development, eventually resolves (Gacek, 1976). In the past, paralysis was caused by infections from whooping cough, encephalitis, polio, tetanus, and other sources, although these causes have diminished substantially as a result of the progressive treatments seen in the current care of pediatrics.

In cases of bilateral abductor paralysis (whereby the vocal folds cannot open), airway patency is the first order of priority. Figure 6 shows the restriction to the airway in this client where the vocal folds are maximally abducted but the glottal area is limited. The vocal folds need to be
lateralized in order to increase the width of the glottal space. This is because the glottal width is compromised due to the position of the paralyzed folds in an adductor or near-adductory state. Lateralization can be accomplished by cordectomy and/or CO₂ laser endoscopic arytenoidectomy. These techniques are often complicated but attempt to preserve phonatory capabilities while improving respiratory function. When reviewing the child’s medical history with the parent, it is important to realize that there is no clear difference between surgical outcomes in cordectomy versus arytenoidectomy. Both procedures appear to be equally effective and reliable in the management of the restricted airway. The phonatory outcomes are less predictable with either surgical procedure. Cordectomy is the easier and faster procedure to perform. As a result of either procedure, the voice will likely be soft, raspy, or hoarse. Obviously, articulation will not be affected.

Children who have bilateral VFP require specific attention from the SP because of potential effects of this condition on both breathing and vocal function. The degree of vocal fold mobility needs to be assessed and interpreted along with determining the size of the airway and the functional problems that the child is having. Often, the voice quality that is presented by a child with bilateral VFP cannot be remediated any further because of the surgical alterations that occurred with the purpose of developing a patent airway. The child also may have physical activity limitations due to shortness of breath, particularly if and when decannulated. Schoolteachers and physical education instructors should realize these restrictions.

Case presentation. In a previous study, we used a unique respiratory strength training program to help a female adolescent with bilateral abductor paralysis deal with her sensation of breathlessness, or shortness of breath (Baker, Sapienza, Davenport, Martin, Hoffman, & Woodson, 2003). Voice quality was hoarse and of lower than normal loudness, but was self-reported as functional. The paralysis stemmed from a history of papillary thyroid carcinoma. She had undergone a thyroidectomy with bilateral neck dissection 4 years before the study. Following this surgery, she presented with bilateral abductor paralysis of the vocal folds suggestive of damage to the recurrent laryngeal nerves. Her primary complaint was dyspnea or breathlessness, not voice quality.

Dyspnea is an uncomfortable sensation that is experienced when the impediments to breathing are greater than the person’s respiratory function capabilities (Salzman, 1997). Dyspnea is often associated with airway obstruction. Significant dyspnea may severely limit an individual’s functional abilities. The sensation of breathlessness may limit speech duration, particularly during physical exertion. Coupled with inadequate inspiratory pressure and flow, more frequent breaths during speech are often needed. These breaths are often audible and stridulous, disrupting the quality of the voice and the fluency of connected speech. Depending on the severity of the obstruction caused by the position of the paralyzed vocal folds, walking a short distance may be challenging because of symptoms of breathlessness.

Although the adolescent underwent two left cordectomies to increase the glottal airway, little to no decrease in dyspnea occurred. A medial arytenoidectomy with CO₂ laser was performed later, but the glottal airway was still quite limited along the length of the vocal folds. It should be noted that defining normal values for glottal width is difficult because of imaging constraints. When the vocal folds are imaged, the distance of the scope tip to the vocal folds cannot be defined routinely, and there is distortion to the image due to the angle of the scope tip relative to the viewing plane. Clinicians typically define limitations in glottal width subjectively when they are engaged in routine clinical care. With this client, glottal width was impaired because of the bilateral VFP. Vibratory amplitude was substantially reduced, and her airway size was, in the clinician’s opinion, small relative to that seen in a normal examination. The assessment of a reduction in glottal width agreed with the client’s subjective complaint of dyspnea.

Because of the uncertainty of the effectiveness of another lateralization procedure and the continuation of her functional limitations, the experimental treatment option of respiratory muscle strength training was explored. By increasing the strength of the inspiratory muscles, it was thought that her dyspnea would decrease. The rationale for respiratory muscle strength training in a case of bilateral abductor paralysis can be found in Baker et al. (in press), and the outcomes for a variety of different client groups, including other pediatric cases, is reported in numerous publications (see Kellerman, Davenport, & Martin, 2000; Sapienza, Brown, Martin, & Davenport, 1999; Sapienza, Ruddy, Davenport, Martin, & Lehman, 2001).

The respiratory strength training protocol for this client consisted of a 5-week training period. The muscles that were targeted during training were inspiratory, including the diaphragm and external intercostals. She trained with a spring-loaded respiratory trainer (see Baker et al., 2003) 5 days per week. Each training session consisted of five sets of five breaths at a threshold set at 75% of her maximum ability to generate an inspiratory pressure. In this short
period of training time, she increased her ability to generate maximum inspiratory pressures by 47%, and her perception of dyspnea was substantially reduced. She was able to engage in more physically challenging activities and available pediatric normative literature. A number of pediatric voice disorders that alter the normal course of development exist, thus affecting the normal physiological integrity of the vocal folds. A few types of voice disorders were brought forth in this discussion to highlight the effects of laryngeal hyperfunction on tissue structure and provide evidence that voice disorders affect voice quality as well as breathing function. In order to treat children with voice disorders effectively, it is imperative to know exactly how the disorder has altered the laryngeal structure and how dysfunction of the larynx can in turn cause changes in airway resistance. With this information, more appropriate therapy targets can be developed by the SLP and met by the child.

**REFERENCES**


Received March 5, 2003
Accepted September 18, 2003
DOI: 10.1044/0161-1461(2004/029)

Contact author: Christine M. Sapienza, PhD, 63 Dauer Hall, Gainesville, FL 32611. E-mail: sapienza@csd.ufl.edu