Vocal Fold Motion Impairment

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Vocal fold motion abnormalities in children are the second most common form of laryngeal pathology seen in children, and often present in the first 24 months of life. A thorough evaluation of the aerodigestive tract will include an examination of the vocal folds, and a proper diagnosis is essential in order to decipher the etiology of swallowing, voicing and breathing abnormalities. This article reviews the workup, management options and clinical outcomes of unilateral and bilateral vocal fold motion impairment in the pediatric population.

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Introduction

Vocal fold paralysis (or impairment) is the second most common laryngeal abnormality in children and typically presents within the first 24 months of life.[1,2] The etiology of vocal fold immobility can be categorized as iatrogenic, neurologic or idiopathic.

In the unilateral vocal fold paralysis group, the iatrogenic causes are most common. Cardiac surgery in children poses a risk to the recurrent laryngeal nerve due to the course of this very small nerve in the neck and chest. The ligation of a patent ductus arteriosus (PDA) is the most common surgical procedure associated with a unilateral vocal fold paralysis. The rates reported in the literature are variable and range from 1% to 25%.[2,3] The recurrent laryngeal nerve is also at risk of injury following thyroidectomy, tracheoesophageal fistula (TEF) repair and excision of lesions that may lie along the course of the nerve in the tracheoesophageal groove in the neck.

Unilateral vocal fold immobility in children is mostly due to iatrogenic causes from cardiac and thyroid/neck surgeries.

The most common etiology of bilateral vocal fold paralysis is related to obstetric trauma, hypoxia and prematurity.

Neurological causes of unilateral vocal fold immobility have been described. These include peripheral nerve disease, agenesis of the corpus callosum and intracranial bleed.[2,4] Compression of the recurrent laryngeal nerve by abnormally enlarged cardiovascular structures may cause paralysis as well (this is known as Ortner's syndrome).

Idiopathic vocal cord paralysis is a diagnosis of exclusion when no other cause can be found. In the Daya review, which spanned a 10-year period at a tertiary referral Children's Hospital in London, 10 of 56 patients had an idiopathic vocal fold paralysis.

The most common etiology of bilateral vocal fold paralysis is related to obstetric trauma, hypoxia and prematurity.[5] The pathogenesis is likely related to a direct nerve injury (such as from forceps delivery) or secondary to intracranial injury and the symptoms are typically noted shortly after birth.

A significant proportion of bilateral vocal fold paralysis is idiopathic in origin, and the rate of recovery is about 50% within the first 1–2 years.[2,6] Delayed recovery of vocal fold motion in this group has been reported as late as 5–11 years postnatally and

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the proposed cause of this delayed recovery is delayed maturation of the vagal nuclei.[2,4,7]

It is important to recognize that even when no obvious etiology can be deciphered by history, some occult process may be a cause for the injury. Brain MRI studies of 23 neonates with a diagnosis of congenital idiopathic bilateral vocal cord paralysis showed non-specific findings including evidence of white matter injury ($n = 2$), abnormal white matter signal ($n = 1$), subdural bleed (3), cerebral swelling (1) and perisylvian polymicrogyria ($n = 1$). Although non-specific, these may be an indication of unrecognized perinatal intracranial injury that may explain some idiopathic cases. A brain MRI should be considered as part of the diagnostic workup of bilateral vocal fold immobility.[8]

Neurogenic causes may be central or neuromuscular. When considering central causes, the Arnold-Chiari malformation accounts for about 30% of the cases, and other pathologies such as hydrocephalus, cerebral hemorrhage, subdural hematoma and meningomyelocele may have an impact by potentially stretching or compressing the vagus nerve or damage its nuclei.[2,7] There is a high rate of recovery from bilateral vocal cord paralysis in this group, but there is also a higher need for surgical intervention to achieve resolution compared to the idiopathic group.

Signs and Symptoms

Unilateral cases of vocal fold paralysis are often accompanied by a weak cry, feeding difficulties, and aspiration.Bilateral paralysis, on the other hand, is associated with more severe stridor, possible airway compromise, cyanosis, and apneic episodes. Dysphonia and aspiration are rarely present in bilateral paralysis as most cases are abductor palsies, and therefore the vocal folds are closer to each other, allowing for good phonation and protection of the airway. Though these patients have higher short-term morbidity, they are more likely to recover than unilateral cases.

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Diagnostic Evaluation

Children with bilateral paralysis should have a thorough neurological work up as many have neurogenic causes. This should include an MRI to rule out any central cause that may be decompressed or otherwise addressed.

The laryngeal electromyography (L-EMG) has been established as a useful technique for determining the extent of injury, prognosis for recovery, and may help guide treatment. It is not a well-studied modality in children and has multiple challenges, most importantly due to the need to perform this procedure under anesthesia. The risk of placing a young child under anesthesia for a diagnostic procedure, and the loss of information obtained from the EMG due to the anesthesia, render this test in children not as effective as in the adult population. Because the time to recovery from paralysis can be highly variable, and since the surgical intervention may have long-term consequences, a method to determine the likelihood of recovery in children is desirable.

Children with paralysis should undergo a thorough neurological evaluation including Magnetic Resonance imaging and laryngeal Electromyography (EMG) depending on the circumstances.

In children who undergo a L-EMG under anesthesia, the electrode is inserted into the thyroarytenoid (TA) muscle, comparing the right and left responses. If the child is under a lighter plane of anesthesia that allows for motion of the vocal folds with respiration, the posterior cricoarytenoid (PCA) muscle insertion may provide information about the abductors of the larynx, but the results are not as accurate as in the awake patient. The PCA is an abductor muscle, which allows the vocal folds to move apart from one another, and the TA muscle is important in phonation and adduction of the vocal folds toward the midline. The electric activity and integrity of these muscles, and comparison of the rate of firing during inspiration and expiration in comparison to the intercostal muscles (or phases of respiration) are especially important in the evaluation of bilateral vocal fold immobility whereas paradoxical firing during inspiration will lead to airway obstruction (stridor upon inspiration). The standardization of L-EMG testing in children is a topic of clinical investigation.