Tutorial

Pathologies That Threaten the Pediatric Airway

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There are many different congenital, traumatic, iatrogenic, inflammatory, and neoplastic causes of airway compromise or obstruction in children. Of the numerous symptoms exhibited by the neonate or young child with abnormal airway narrowing, stridor is perhaps the most alarming. This condition is characterized by a harsh, creaking, grating, or whistling sound that results from increased air turbulence through constricted, static and/ or dynamic head and neck pathways. There are 3 primary subtypes of stridor. The *inspiratory* form most often occurs during deep inhalation activity. It can be faint in volume or quite audible. Placement of a stethoscope diaphragm on the larynx of the child with suspected inspiratory stridor reveals the source of airflow impedance at or immediately above the glottis. Stridorous sounds during *exhalatory* activity are suggestive of airflow obstruction within the proximal or distal trachea. *Biphasic* stridor is characterized by noisy breathing behaviors during both inhalation and exhalation. Airflow obstruction within the glottis introitus, the immediate subglottic space, the proximal trachea, or altogether ordinarily cause this condition. All of these can be determined by timing the phase of respiration with the stridor.

Children with significant nasal obstruction often exhibit hyponasal resonance features, nasal flaring, and snorting behaviors. Rarely they will suffer from chest retractions during deep breathing. In neonates this problem can result in serious respiratory distress because they are ordinarily obligate nasal breathers.

When the dimensions of the nasopharyngeal or oropharyngeal cavity are compromised, inspiratory breathing is likely to be characterized by a coarse sound, also known as stertor. Whereas the voice of this child may be generally acceptable, it may possess throaty or guttural resonance features. Feeding can be difficult due to the obstructed swallowing pathway. Drooling is also a problem for the child because of difficulty swallowing saliva. Nec-

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essary mouth breathing merely exacerbates the drooling severity.

Children with swelling or obstruction involving the oropharyngeal tissues often exhibit a muffled or so-called hollow or "hot potato" sounding voice. Snoring is also common. Dysphagia is a frequent side effect in this clinical population because flexion of the epiglottis during swallowing is retarded by the associated pathology. As noted above, glottic obstruction usually results in significant dysphonia and inspiratory stridor. With progression of the condition, inspiratory stridor may convert to a biphasic variety; in severe cases, suprasternal, intercostal and substernal retractions can be observed during distressed breathing cycles. Masses, postoperative scar tissue formation, and congenital or acquired stenosis of the subglottis typically cause inspiratory and biphasic stridor. Although the voice characteristics may be within normal limits in some children, in some cases harsh-strained vocal quality occurs due to airflow resistance and high-pressure vocalization efforts. Although dysphagia is not usually observed in children with subglottic pathologies, a barking cough may be noted. In addition to expiratory stridor, associated with tracheal-bronchial obstructions, wheezing may be perceived during auscultation, along with a high-pitched cough.

Children with stridor often present with one or more co-existing signs and symptoms that may be equally concerning to parents and physicians. These include generalized agitation, dysphagia, drooling, dysphonia, sleep apnea, tachycardia, cyanosis, and failure to thrive. As such, these conditions must be promptly evaluated because they may be pathognomonic of a potentially life-threatening pathology.

The objectives of this introductory article are to provide comprehensive reviews of: (1) normal pediatric airway anatomy, (2) key physical examination techniques, and (3) the most common causes of stridor. Within the contexts of these discussions, appropriate yet variable differential diagnostic and treatment strategies for the stridorous child will be emphasized.

ANATOMY OF THE PEDIATRIC AIRWAY

Pediatric airway anatomy differs substantially from that of the adolescent or adult. The infant's head is relatively large, and its neck is quite short. Bones of the cranium form approximately two-thirds of the skull; the smaller and more numerous facial bones compose the remaining third. This ratio gradually reverses throughout early childhood as the cranium dimensions become less prominent with continuous growth of the facial bones, especially the mandible and to a lesser extent the maxillae. The pediatric larynx is much smaller than its adult counterpart, and in comparison it is located very high in the neck around the seventh cervical vertebrae. In infants and young children the epiglottis is in close anatomic proximity to the soft palate and can frequently be viewed upon routine examination of the pharynx. Whereas the adult thyroid cartilage rests in the neck at an angle of approximately 90 degrees, in the infant it has a more pronounced anterior tilt. With advancing age, the cartilages of the larynx become moderately calcified. In infants, however, all laryngeal cartilages are rather soft and supple, significantly limiting their vulnerability to life-threatening traumatic fracture. In the infant, the vocal folds are a third of an inch long (8 mm). The maximum width between the free edges of the folds during deep inspiration is 4 mm. The interior diameter of the signet ring-shaped cricoid cartilage is approximately 6 mm. The distance between the vocal folds and the carina is 4 cm in the young child, with a diameter of 4 mm. If this tiny glottal or subglottal region is compromised by any small space occupying pathology, significant stridor may result. It has been estimated that 1 mm of edema or obstruction within these regions can reduce normal respiratory airflow dynamics by as much as 66%. The infant's relatively large tongue occupies most of the oral cavity. This factor, along with the high position of the larynx in the neck, causes obligate nasal breathing activities for the first few months of life. Associated abdominal or diaphragmatic respiration is evident at rest, in part because of the infant's chest wall elasticity or compliance. Oral breathing behaviors are generally reflexive and normally do not emerge before two months of age or degree of neurologic development.

The oropharyngeal airway in children is quite narrow compared to adults, mostly due to the presence of bilateral, variably enlarged pharyngeal, palatal, and lingual tonsils (i.e.., Waldeyer's ring). Congenital and acquired pathologies that afflict these upper airway cavities and compromise their dimensions invariably cause the child to suffer from distressed respiratory activity and hypoventilation. During normal inspiratory-expiratory pulmonary cycles, pressures rarely exceed 3 mm Hg differential. Children with airway pathologies may generate pressures that reach 100 mm Hg.

HISTORY AND PHYSICAL EXAMINATION

General History

When a child presents with signs of airway difficulty, regardless of his or her age, the first task of the examiner is to assess as quickly as possible the urgency of the problem. Life-threatening respiratory distress must be immediately treated. Depending on the suspected cause, management may include: (1) watchful surveillance without specific treatment, (2) removal of a foreign body from the airway, (3) intubation, and/or (4) tracheotomy. Once the child is stable and the airway is secure a comprehensive medical history should be obtained and thorough physical examination conducted. If the airway concern is not severe, taking the history and performing the physical should begin at the time of presentation.

The onset of the problem is crucial to the differential diagnosis. For the newborn with respiratory distress the examiner must consider possible associated vocal fold paralysis, congenital web, ***complete tracheal rings*** (Figure 1) or congenital subglottal stenosis. Neonates (4 to 6 weeks) with stridor may be suffering from underlying laryngomalacia. If the 1- to 6-month-old infant presents with stridor, especially postintubation, the differential diagnosis is acquired subglottic stenosis until proven otherwise. Subglottic hemangioma may also account for the presentation at this age. Laryngotracheal bronchitis (croup) often causes breathing difficulties in very young children (0 to 2 years). Foreign body aspiration episodes should be considered in every patient regardless of age but are noted to be a more common cause of stridor in toddlers (1 to 3 years), as they advance their eating habits and explore environmental choking hazards with their mouths.

History of coughing with feeds may reveal a ***laryngeal cleft*** (Figure 2) or tracheoesophageal fistula as the reason for stridor. While, Airway edema from laryngopharyngeal reflux (LPR) should also be kept in mind as a differential.

As previously defined, the type of stridor exhibited by the child can often facilitate discovery of the site of airway compromise. Naturally, if the parent or guardian explains that an antecedent choking event occurred during eating strong suspicion of foreign body aspiration should arise. With no such precursor event, the age of onset is of paramount importance to the diagnosis. The birth history and any required intubation, surgical procedures, or respirator placement must be explored and discussed. Immediate postoperative stridor, for example, may be indicative of subglottal edema or vocal fold trauma secondary to intubation trauma. Delayed (14–21 days) postoperative airway distress

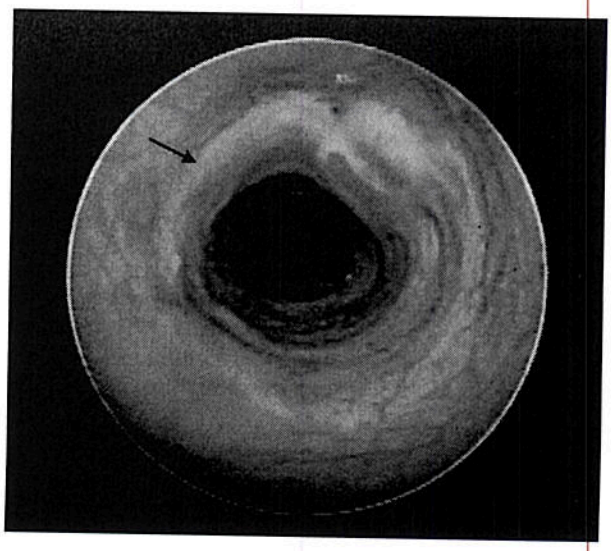


Figure 1. Complete tracheal ring.

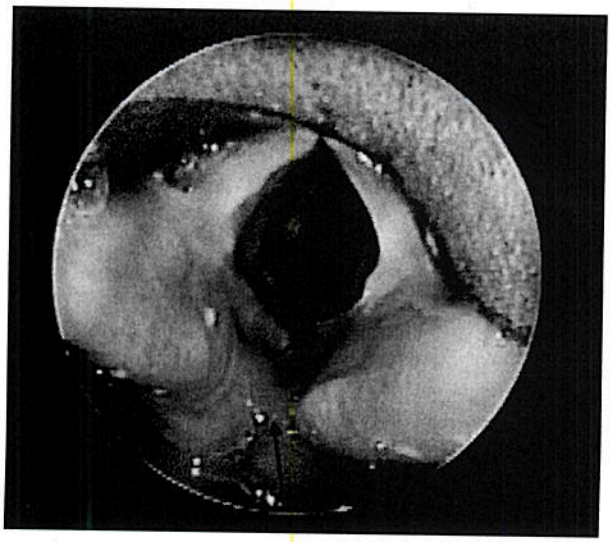


Figure 2. Benjamin Ingles classification Type 2 laryngeal cleft extending through interarytenoid area and past TVF into cricoid cartilage.

often points to the formation of glottal or subglottal scar or granulation tissue, which can narrow the airway dimensions, resulting in stridor. In the case of associated neck pain and fever, the origin of the airway obstruction is frequently caused by an inflammatory condition. Congenital pathologies are very common causes of respiratory distress in children, especially, if the problem begins and is persistent within the first six months of life.

Unlike stridor, wheezing in the infant or toddler is usually suggestive of lower airway pathology. In older children, asthma and allergy are very common causes of the problem; viral respiratory tract infection or gastroesophageal reflux are more likely culprits in very young children. In fact, only 30% of infants who struggle with wheezing eventually develop asthma or allergies. Therefore, pediatric reactive airway difficulties may be caused by many different abnormal anatomical or physiological conditions that should not be confused with asthma. If there is any associated cough, it is important to distinguish whether it is perceptibly wet or dry. With the latter subtype, the hacking, nonproductive sounds are commonly caused by asthma, particularly in older children. Conversely, a wet-gurgle cough is symbolic of airway purulence and standing mucus secretions, typically manifestations of an underlying bacterial or viral infection. Wet vocal quality and associated hoarseness often coexist with these conditions. The child with a chronic wet-sounding cough and voice and frequent upper respiratory infections should be considered for the diagnosis of cystic fibrosis. An impaired mucociliary clearance disorder such as primary ciliary dyskinesia (e.g., Kartagener's syndrome) may be a viable alternative diagnosis in some young children. Less common causes of chronic wet cough and voice quality in this population include persistent postnasal drainage with associated endo-laryngeal infiltration. Allergic or nonallergic rhinitis and sinusitis, and bronchitis secondary to the poisonous effects of environmental second hand or passive smoke on the airway cilia are often responsible for these sequelae. Questioning parents or guardians about their smoking habits can shed light on this possible differential diagnosis. In the child with a chronic wet cough disorder differentiation of the cause may be facilitated by considering these important factors: (1) most new diagnoses of cystic fibrosis are made in children who do not have a positive family medical history of asthma, (2) if there is a parental past history of COPD, CAD, CVA, and/or lung cancer the probability of a smoking causal relationship to the wet cough is high, (3) coughing that frequently awakens a child from sound sleep is usually characteristic of a significant pathology, (4) whereas Kartagener's syndrome is often associated with sinusitis, bronchiectasis, and recurrent otitis media, children with cystic fibrosis rarely suffer from the latter condition, and (5) if cystic fibrosis is the suspected cause of reactive airway disease in a child, investigation of the stool may reveal the presence of oil or grease (steatorrhea).

Infants with noisy breathing behaviors without evident respiratory distress usually exhibit normal oxygen and saturation levels. Conditions that may evoke this clinical profile may include tracheomalacia and bronchomalacia; that is, large airway obstructions. Hypoxemia, on the other hand, most often results when the lungs are not continuously ventilated with fresh air, which in turn depresses the oxygen tension level in the blood returning to the heart. In such cases of poor ventilation pulse oximetry reveals low saturation. These abnormal features are not uncommonly observed in infants with small airway pathology, such as bronchiolitis, wherein the lungs cannot ventilate or empty efficiently due to severe airflow resistance. Breathing is rapid (tachypnea) in the infant with respiratory problems, which interferes with the ability of the lungs to deflate completely during each expiratory cycle. With each subsequent breath, air trapping occurs as residual air in the lungs mixes with newly inspired air. This causes overexpansion of the lung-thorax unit and hypoxemia.

Inspection

The exam should start with observation of the child's breathing behaviors. This inspection can be accomplished when the child is being held by a parent, guardian or nurse, or on the examination table. The child's shirt should be removed so the clinician can examine the neck, chest, and abdomen directly for physical signs of retraction. The respiratory rate, degree of dyspnea and type of stridor should be assessed initially. Normative values vary with age. At 2 weeks the newborn's rate is up to 50 breaths per minute. In the 6-month-old child the rate rarely exceeds 44, and for the 1 year old, 32 breaths per minute occur. Older children approximate adult-like respiratory cycles at 16 to 20 breaths per minute. Abnormally rapid rates, accompanied by nasal flaring, use of accessory respiratory muscles, breathing fatigue, and cyanosis may collectively represent serious airway pathology. If these conditions are observed the examiner must proceed with concerns that immediate airway stabilization via intubation may be necessary.

Auscultation

The standard stethoscope may be used creatively to listen for signs of unusual turbulence and stridor, which may help localize the site of obstruction. When the diaphragm is placed on the nares, nasal airflow resistance can easily be heard through the nostrils. A small dental or laryngeal mirror can also be positioned under each nostril to confirm the auscultation impression of airflow retardation. When listening to the lungs inspiratory and expiratory durations are roughly equivalent, and the sounds are tubular; that is, they possess a bronchial quality. It is important to understand that as air fills the lungs there is little to no associated sound. Conversely, within the peripheral airway (ie., the mainstem bronchi and trachea) the sounds of inspiration are normally much louder than those of expiration. At the immediate glottis and subglottis airflow dynamics can be appreciated with a stethoscope. At these levels stridor can be differentiated from wheezing or crackling sounds produced by children with specific pulmonary pathologies, such as asthma or bronchiolitis. Whereas crackling sounds are usually discontinuously perceptible, wheezing sounds are usually more continuously detectable upon auscultation. A crackle has a fine or coarse popping quality that is causally related to the presence of airway fluid. It resembles the sound made when Velcro strips are pulled apart. Crackles are most detectable with auscultation during inspiration, and they are frequently signs of interstitial lung disease. Wheezes are more audible during expiration and they can be polyphonic or monophonic. The former subtype is characterized by different pitches, frequently caused by vibrations of inelastic and diffusely narrow air sacs within the lungs, as observed in asthma sufferers. Monophonic implies constant sound features, which usually result from partial obstruction within a single location of the airway. Children with histories of tracheomalacia, bronchomalacia, vascular rings, and bronchial foreign body aspiration often struggle with such wheezing signs and are often misdiagnosed with asthma by emergency department and primary care physicians secondary to this.

Bilateral abductor vocal fold paralysis can be mistakenly diagnosed as asthma if the examiner fails to differentiate glottal stridor, due to insufficient glottal width during breathing and speaking, from the wheezing caused by asthma. If the stethoscope is alternatively and strategically placed on the lungs, chest, and larynx such diagnostic confusion should promptly be eliminated. Finally, if the child with noisy breathing patterns is repositioned on the examination table or within the arms of the parent or guardian the perceptual characteristics and degree of the problem is significantly altered. If the child lies prone on the examination tabletop with neck extended and the noisy breathing behavior diminishes the differential increases for possible laryngomalacia or innominate artery compression. Children with micrognathia and macroglossia also benefit from such repositioning.

Palpation

Unilateral vibrations are often causally related to bronchomalacia, the presence of a foreign body within a main-stem bronchus, or a mucus plug within the trachea. Palpation of the lower rib cage area for signs of an unusual degree of retraction may highlight in such cases that the child is struggling to exchange air during respiratory cycles; a common finding in children with obstructive laryngeal or subglottic pathologies.

Head and Neck Examination

There are some examinations that can usually be performed in the office with children of all ages. For example, the anatomical and physiological integrity of the craniofacial complex, external auditory canals and eardrums, nasal septum, turbinates, tongue, teeth, hard and soft palates, tonsils, and mandible and neck, at the very least, canbe superficially appraised for pathologies or foreign bodies that may account for the presenting respiratory distress. When possible, flexible nasopharyngeal and laryngeal endoscopy should be performed in the office setting to avoid the ordeal of general anesthesia in the operating room. Because very young children may be at risk for apneic responses to this examination procedure, a bag ventilation kit with appropriate suction and oxygen supplies should be available. Target conditions on this examination that must be considered and correlated with the patient's age and clinical presentation include: (1) a nasal foreign body, (2) choanal atresia, (3) hypertrophied adenoids and palatal tonsils, (4) macroglossia, (5) edematous lingual tonsils, (6) epiglottitis, (7) diffuse and viscous secretions in the valleculae and/or piriform sinuses, (8) laryngomalacia (9) posterior glottic (interarytenoid) edema and granulation tissue, and (10) unilateral or bilateral vocal fold paralysis. A suspected subglottal or distal tracheobronchial pathology in children cannot easily be evaluated with flexible endoscope examination in the office setting. Instead, radiographic appraisal, and/or direct rigid endoscopic visualization in the operating room must be scheduled. In cases

with significant co-existing swallowing difficulties or significant signs and symptoms of gastroesophageal reflux disease it may be of diagnostic value to include esophagoscopy in the workup.

Diagnostic Imaging

In the final analysis, radiographic imaging, including plain film anteroposterior and lateral neck and chest x-rays, fluoroscopy, MRI with gadolinium, CT scans with contrast, and barium swallow studies, are useful in determining the causes of significant breathing problems in children. Which of these tests to order in any given case is usually based on the age of the child and the initial general clinical portrait Although plain films are frequently used to evaluate children with stridor, their consistent diagnostic value has not been unequivocally demonstrated. Low correlations between plain radiograph findings and the results of direct laryngoscopy and bronchoscopy may indeed be the rule rather than the exception.

Airway fluoroscopy is, on the other hand, a more reliable, low risk, quick (< 1 minute), noninvasive, dynamic evaluation tool for the entire breathing mechanism. Results of this examination are usually far superior to those of plain films, especially when investigating vocal fold mobility, and subglottal, tracheal, and bronchial lesions.

Vascular compression pathologies and mass lesions of the nasopharynx and mediastinum are best detected with MRI or CT scanning with contrast studies. The latter exam is often the most sensitive tool for the detection and differentiation of choanal atresia, choanal stenosis, and suspected supraglottal, glottal and subglottal masses.

To rule out aspiration episodes the modified barium swallow study is indicated, wherein the fluoroscope is positioned to evaluate lingual propulsion, velopharyngeal competency, pharyngeal motility, and laryngeal motion during the ingestion of various liquid and food bolus types. This study (± esophagram to follow the swallow to the stomach) is of paramount importance in the diagnostic workup of children with co-existing breathing and swallowing difficulties.

COMMON CAUSES OF AIRWAY OBSTRUCTION IN CHILDREN

Nasal Cavity

Obstructive nasal cavity pathologies only result in clinically significant respiratory distress in neonates because, as previously mentioned, they are obligate nasal breathers. Older children can compensate via mouth breathing behaviors, if necessary. The following conditions represent the most common causes of pediatric nasal obstruction: (1) foreign bodies inserted by the child during play and environmental exploration, (2) upper respiratory infections or rhinovirus with resultant nasal congestion, (3) severe allergic and nonallergic rhinitis with associated turbinate hypertrophy, (4) sinonasal polyps or other neoplasms, (5) septal deviation, and (6) choanal atresia or stenosis. Because only the latter condition is likely to cause an airway emergency it is discussed in detail.

Choanal atresia (Figure 3) occurs in approximately 1 out of 8,000 births. This congenital bony and membranous nasal occlusion pathology can be unilateral or bilateral. When it is bilateral, the neonate will present with severe respiratory distress at birth. Crying may momentarily alleviate the airflow difficulty with associated, intermittent breaths through the oral cavity. The actual cause is unknown, but it has been suggested that it may result embryologically because either the buccopharyngeal membrane fails to resorb naturally or the epithelial rest cells that help form the nasal cavities are over-abundantly produced. Not infrequently, choanal atresia can present as part of the ***CHARGE*** syndrome, wherein children afflicted may present with colobomas, heart anomalies, choanal atresia, growth retardation, genital and ear anomalies. At first, bilateral choanal atresia is treated urgently to rescue the neonate from this life-threatening condition. Oral intubation or a McGovern nipple and nasogastric feeding approach is applied initially, followed by more definitive surgical reconstruction once the airway is secure and the infant is stable. Although unilateral choanal atresia frequently will

Figure 3. Choanal atresia (*arrow pointing to bony occlusion*).

not cause respiratory difficulty and it often goes undiagnosed in otherwise healthy neonates.

Palatal and Pharyngeal Boundaries

There are several pathologic conditions involving these subsites of the pediatric airway, including adenotonsillar hypertrophy, parapharyngeal, retropharyngeal, and peritonsillar abscesses, and foreign bodies lodged in the valleculae or hypopharynx. With the possible exception of a retropharyngeal abscess or foreign body, which could significantly compromise the oropharyngeal aperture, most of these conditions are unlikely to result in urgent breathing difficulties. However, odynophagia and delayed aspiration episodes may be sequelae of each condition. In severe cases, pneumonia can result, which in turn can indirectly lead to substantial respiratory distress.

Peritonsillar abscess is the most common deep oral cavity infection in older or teenage children. Overall, approximately 3 out of 10,000 people experience this condition, and less than one half are children. Pathologically it is theorized that this progresses via the spread of bacteria from infected tonsils or minor salivary glands into the peritonsillar space. The involved tonsil and the adjacent superior constrictor muscles anatomically form this space. Proximal peritonsillar cellulitis forms initially and then slowly converts into an abscess, most commonly containing group A or group B streptococcal cells. The clinical portrait includes the following signs: (1) deviation and edema of the tonsil toward the palatal midline with associated rotation of the pillar, (2) odynophagia, (3) fever, (4) intermittent aspiration, (5) muffled voice, (6) trismus, and (7) poor appetite. If untreated, this condition can advance into a deep neck abscess with invasion into the parapharyngeal and retropharyngeal spaces. Worse yet, erosion of the carotid artery can occur in some cases, as can septic thrombosis of the internal jugular veins, mediastinitis, and sepsis. Treatment typically involves use of broad-spectrum antibiotic medication; incision and drainage may be initially indicated in severe cases. Older children with prior histories of recurrence often benefit from planned tonsillectomy.

With ***retropharyngeal abscess***, pus accumulates in the space between the posterior pharyngeal wall and the prevertebral fascia. Lymph node inflammation develops bilaterally along a vertical plane within this space. Because these nodes drain from the oropharynx, nasopharynx, teeth, maxillary sinuses, and eustachian tubes they provide a passage for infection from these sites to spread into the mediastinum. These nodes are most prominent in toddlers and naturally regress by 6 years of age. Due to the drainage patterns, upper respiratory infections are the most common trigger mechanism in this group. Less common causes of retropharyngeal abscesses include foreign body lacerations and iatrogenic trauma of the pharyngeal wall during nasopharyngeal endoscopy and endotracheal intubation. Intense inflammatory responses involving this space can lead to airway compromise, dysphagia, aspiration pneumonia, and odynophagia.

Oral Cavity

Abnormalities in the size or rest position of the tongue can have a profound negative impact on breathing. ***Lingual angioedema***, secondary to a severe allergic or anaphylaxis response, can compress the oropharyngeal aperture and threaten normal respiration. ***Macroglossia***, along with midface hypoplasia, generalized hypotonia, and obesity predisposition, may be observed in children with Down syndrome. These morphologic aberrances significantly contribute to upper airway obstruction in this population. The ***micrognathic*** profile of the child with Pierre Robin sequence results in glossoptosis. In severe cases, respiratory distress is exhibited within the first couple of months of life, especially when the neonate is in a supine position and the tongue obliterates the oropharynx. Beckwith–Weidmann syndrome as well as the mucopolysaccharidoses syndromes (e.g., Hurler's, Hunter's, and Marteaux-Lang) are diseases that may be characterized by generalized upper airway inflammation, macroglossia, a short and immobile neck, and TMJ dysfunction. These features can result in mild to moderate airway obstruction in the young child.

Pronounced hypertrophy of the lingual tonsils, which frequently co-exists with adenoid and palatal tonsil swellings, can collectively obstruct the upper airway and contribute to significant breathing, swallowing, and sleeping difficulties in children. Vallecular cysts, ***saccular cysts*** (Figure 4), laryngoceles and occasionally thyroglossal duct cysts or certain branchial anomalies can have similar debilitating effects.

Ludwig's angina or necrotizing fasciitis can cause profound upper airway compromise. First described in 1836, this fast spreading, cellulitis of the connective neck tissues and floor of mouth with gangrenous pathology in some cases may be fatal. By definition it involves the submandibular,

sublingual, and submaxillary spaces. Infection and associated swelling in these zones tend to expand upward and outward. This causes large full neck appearance and, more importantly, the base of tongue to be displaced posteriorly, causing obstruction to the airway. Dental abscesses involving the lower molar teeth are the most common cause of Ludwig's angina. In children, another common cause is laceration of the sublingual tissue with a sharp toy or object. Patient's typically present with dysphonia, odynophagia, drooling, fever and dyspnea. At the time of diagnosis, the airway should be secured and treatment must ensue soon after to reverse the progression of this disease. If left untreated, death may result from respiratory obstruction or, less commonly, pneumonia, mediastinitis, and sepsis. Broad-spectrum antibiotic therapy to combat aerobic and anaerobic bacteria has been shown to be effective in controlling this disease.

Larynx

There are numerous congenital and acquired laryngeal pathologies that can variably threaten the pediatric airway, including: (1) laryngomalacia, (2) glottic webs, (3) atresia, (4) laryngocele, (5) vocal fold paralysis, (6) subglottic hemangioma (7) subglottic cysts, (8) papillomatosis, (9) acute epiglottitis, (10) laryngo-tracheo-bronchitis, (11) benign and malignant neoplasms, (12) foreign body penetration and aspiration, (13) fracture, and (14) subglottal stenosis. Of these conditions, only the most common ones are addressed below in detail.

Laryngomalacia (Figure 5)

More than 50% of neonates who exhibit inspiratory stridor suffer from this congenital pathology. This is generally characterized by an omega-shaped epiglottis, shortened aryepiglottic folds and redundant mucosa of the arytenoid cartilages. There is a 2:1 male versus female incidence ratio, and signs and symptoms of low-pitched stridor with a fluttering vocal quality usually appear within the first 2 weeks of life. The breathing difficulty is often exacerbated by feeding activity, particularly when the neonate is agitated and lying in the supine position. There are two primary mechanisms that account for airway obstruction and stridor during inspiration: (1) the omega-shaped epiglottis minimizes the aperture of the laryngeal inlet, which increases the resistance to respiratory airflow dynamics, and (2) the shortened aryepiglottic folds and floppy arytenoid mucosa, which prolapses medially and anteriorly over and into the glottal introitus. Whereas the actual etiology is unknown, most theoretical explanations for the prenatal development of laryngomalacia focus on the persistence of an abnormally soft cartilaginous supraglottis coupled to an underlying immature laryngeal neuromotor control system. If atypical signs and symptoms, such as cyanosis, altered

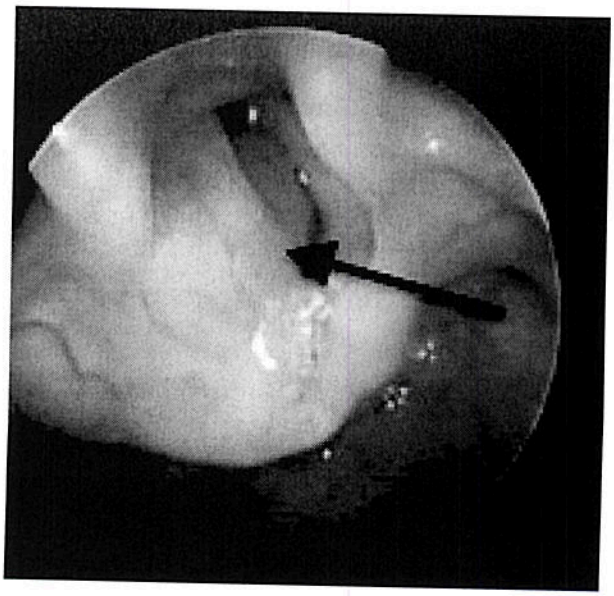


Figure 4. Saccular cyst obstructing glottal introitus.

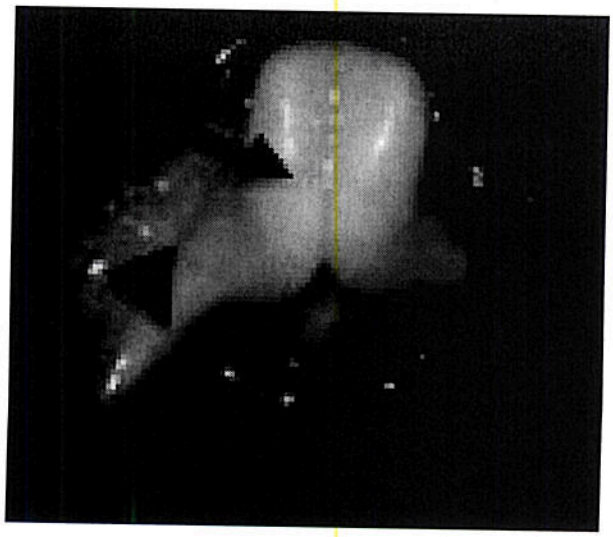


Figure 5. Laryngomalacia (short arytenoid-epiglottal folds and omega-shaped epiglottis).

cry, and aspiration episodes are observable the diagnosis of laryngomalacia may be challenged. In the absence of these problems, flexible laryngoscopy in the clinic or rigid fiberoptic laryngoscopy under general anesthesia with spontaneous breathing should be conducted for comprehensive appraisal of the upper airway. Adding to the differential diagnosis is the tendency for supraglottal collapse to occur during the waking phases of this procedure. In 90% of children with laryngomalacia it is a self-limiting condition, requiring no surgical intervention. In these cases, stridor may persist and even worsen until 9 months of age before gradually and completely resolving by 2 years of age. For those with concerning signs of obstructive sleep apnea, failure to thrive, or cor pulmonale, supragottoplasty is indicated. This may include: (1) division of the aryepiglottic folds to release constrictive tension and enlarge the dimensions of the laryngeal inlet, (2) trimming the epiglottis, 3) resection of the redundant arytenoid mucosa and/or corniculate cartilage, (4) suspension of the prolapsing epiglottis to emancipate the laryngeal introitus, and (5) laser arytenoidectomy. Complete resolution of stridor occurs in more than 80% of cases postoperatively. Because laryngopharyngeal reflux may prolong the resolution process and is often a co-existent pathology, proton-pump inhibitor and/or H2 antagonist medications should be prescribed routinely for precaution purposes. For completeness, it should be noted that there is a neurological subtype of laryngomalacia, which has been observed in children with muscular dystrophy and cerebral palsy. In cases of severe neurogenic airway collapse a tracheotomy is usually the treatment of choice.

Laryngeal Webs (Figure 6)

Although this condition may develop prenatally, it is most often a result of scar tissue formation in the anterior or posterior glottal commissure secondary to prolonged intubation associated with respirator placement. Premature infants are therefore at risk for this condition. If the web is broad based it causes inhalatory stridor, limited audible airflow in cases of complete webbing (atresia), and hoarse-breathy cry behaviors because vocal fold excursions during breathing and vocalization will be inhibited. In such cases, tracheotomy and keel placement may be necessary. If chronic recurrence occurs, laryngeal reconstruction must be considered for definitive relief. Minor webbing rarely results in respiratory distress or the need for immediate surgical management.

Laryngo-tracheo-bronchitis (LTB) (Figure 7)

The signs and symptoms of viral croup or LTB have historically been classified using many other diagnostic terms: False croup, spasmodic croup or laryngitis, pseudomembranous croup, acute subglottic laryngitis, acute infective LTB, and laryngotracheitis. The incidence of this disorder is unknown, in part because mild cases are probably never formally treated or recorded by healthcare providers. It is most prevalent in children between 1 and 2 years of age, with the vast majority occurring within the first five years of life. This inflammatory disorder of the upper airway usually begins with gradual symptoms of low grade fever, sore throat, a dis-

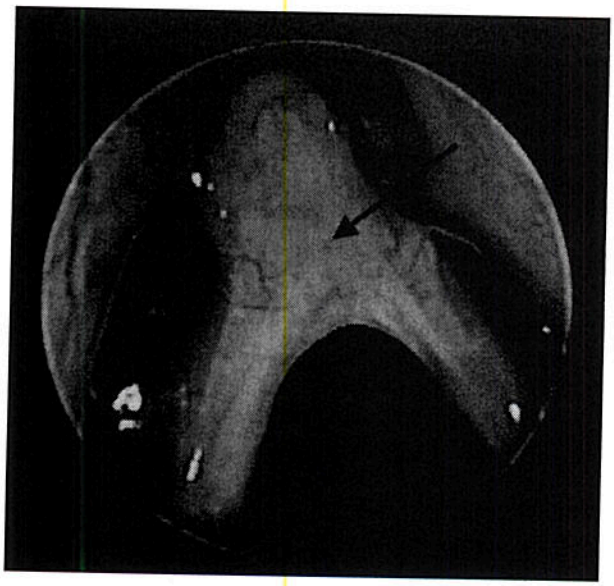


Figure 6. Anterior laryngeal web.

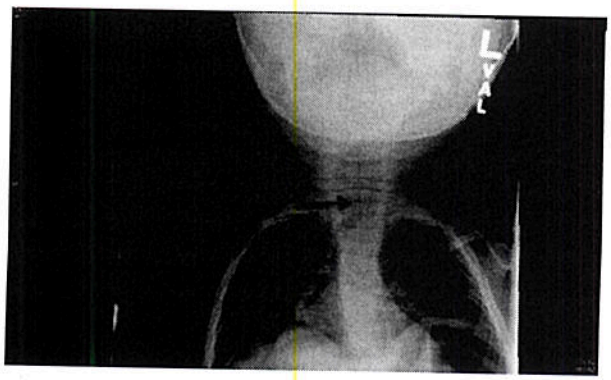


Figure 7. Anterior x-ray demonstrating laryngo-tracheo-bronchitis (croup). Arrow pointing to subglottic narrowing "steeple sign."

tinctive seal-like barking cough, rhinorrhea, high-pitched inspiratory stridor, and hoarseness. When these classic signs and symptoms are evident, radiography is not usually required for confirmation of the diagnosis. However, plain neck x-rays often reveal the "steeple sign," or hourglass narrowing of the subglottis. The degree of respiratory distress may vary from mild to severe. In the latter case, tachypnea, airflow resistance, suprasternal, substernal, and intercostal retractions are observable. If cyanosis, restlessness, and wheezing and crackling sounds are exhibited by the child hypoxemia and hypercarbia should be suspected. These signs and symptoms of LTB ordinarily are worse at night.

The microbiology of viral croup implicates the parainfluenza type 1 virus in approximately 50% of cases. Airway obstruction is a concerning sequelae in many of these children, owing to associated subglottic edema, tracheal and bronchial inflammation, and abundant mucus accumulation at the glottis. Hospitalization is only required if poor responses to standard use of inhaled racemic epinephrine, humidification therapy, and systemic or nebulized steroids occur and more frequent, carefully monitored treatments are indicated. Additionally, children with notable stridor may similarly be good candidates for admission. On rare occasions, careful, smooth, endotracheal intubation is required for the child with severe LTB.

Epiglottitis (Figure 8)

Also known as supraglottitis this condition primarily involves the epiglottis, and to a lesser extent other structures above the glottis, such as the aryepiglottic folds, bodies of the arytenoids cartilages, and the uvula. Whereas in children inflammatory reactions are usually confined to the epiglottis, in adults collateral involvement of the epiglottis, prevertebral soft tissue, valleculae, base of tongue, and velum may be observed. The child who presents with epiglottitis usually exhibits a course of rapid onset sore throat, fever, hoarseness, drooling, dyspnea, anterior neck tenderness, poor appetite, and dysphagia. Coughing is rare with this pathology. A clue to the differential diagnosis is the child's tendency to sit leaning forward with elbows on thighs (tripod position). This position elongates the laryngeal inlet dimensions for more comfortable airflow past the obstructing, edematous epiglottis. If not identified and treated within the first few hours of symptoms this condition can be life-threatening.

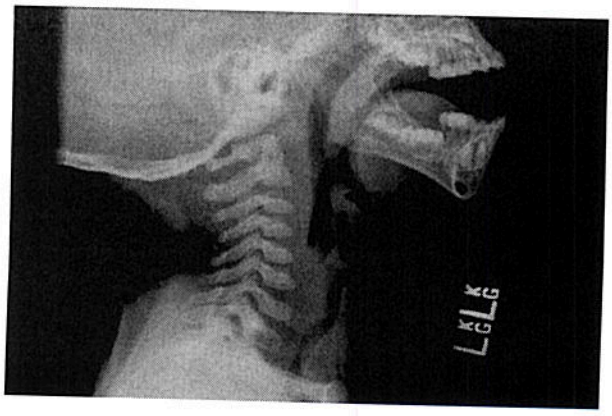


Figure 8. Lateral x-ray demonstrating epiglottitis with arrow pointing to edematous epiglottis "thumb sign."

Fewer than 1 in 100,000 children suffer from epiglottitis in their lifetime. The cause in most cases is the *H. influenzae* type B pathogen. With the advent of the *Haemophilus influenzae* vaccine (Hib) epiglottis is now frequently being observed in older children then in the past and is often associated with other pathogens, especially streptococcus pneumoniae. Other possible etiologies include *Staphylococcus aureus, Haemophilus parainfluenzae, Klebsiella pneumoniae,* varicella zoster, herpes simplex, group A, B, and C streptococcus, *Candida albicans*, immunodeficiency syndrome, and thermal trauma secondary to ingestion of hot food or exposure to the burning insult of crack cocaine. Radiography is not usually indicated to reach the diagnosis. If plain films are utilized epiglottic edema is evidenced by a classic "thumbprint image." Treatment ordinarily involves halothane and oxygen induction of anesthesia, placement of an intravenous line, direct laryngoscopy with blood and epiglottic cultures for the differential, followed by smooth and careful endotracheal intubation and IV antibiotic therapy with a beta lactamase stable antibiotic.

Recurrent Papillomatosis (Figure 9)

Most often a benign, acquired pathology, this condition is of viral origin. It is characterized by the formation of persistent or recurrent wart-like growths with intrinsic squamous papillomata cellular proliferation. These invasive and widespread lesions often challenge the upper airway, especially at the mucosquamous junction of the glottis. The most common pathogen is the human papilloma virus types 6 and 11. Those afflicted usually suffer from inspiratory stridor, shortness of breath and

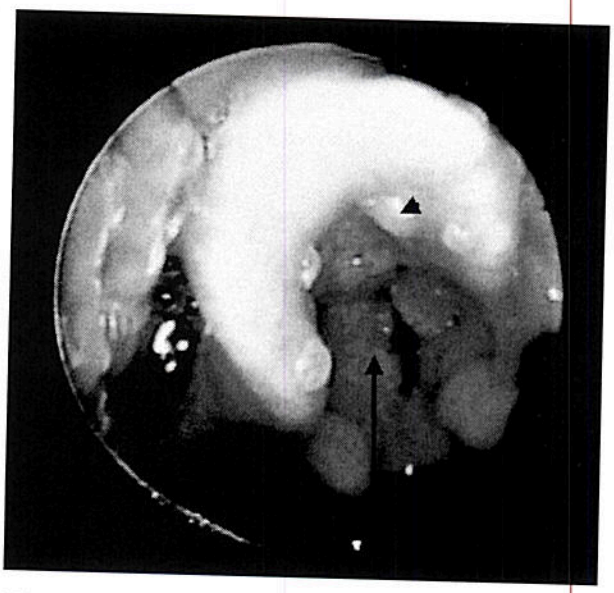


Figure 9. Diffuse papillomas involving the laryngeal aspect of epiglottis (arrowhead) and true and false vocal folds (arrow).

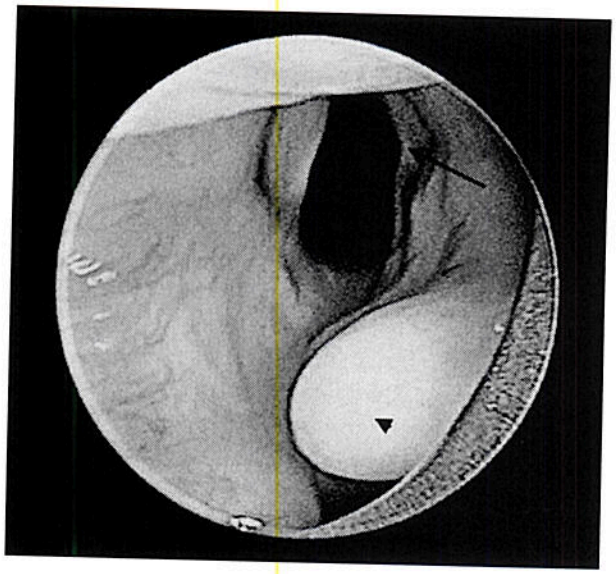


Figure 10. Right true vocal fold paralysis. Arrow pointing to atrophied and shortened right true vocal fold in the paramedian position. Arrowhead at prolapsed arytenoid.

hoarseness. If downstream invagination into the subglottis or tracheobronchial tree occurs the condition becomes more concerning and become life threatening. Signs of biphasic stridor will likely prevail in such a case.

Debulking surgical management is the treatment of choice in most cases with significant airway obstruction. Techniques include use of cold steel, microdebrider, CO_2 laser, and radiofrequency ablation. Although there are limited empirical data to support its therapeutic value, intralesional cidofovir injections continue to be proposed in addition to surgery. Because laryngeal papillomas have a strong propensity toward recurrence in the young child, long-term vocal fold scarring and fibrosis are not uncommon sequelae to the numerous required surgical treatments, irrespective of the operative methods employed. Currently, microdebrider, radiofrequency excision and CO_2 laser are being used to enable access to the papillomatous lesions within the proximal and distal trachea and to avoid spread of the virus to other areas of the tracheobronchial tree.

Vocal Fold Paralysis (Figure 10)

In children this condition is the second most leading cause of airway compromise. It can usually be attributed either to: (1) birthing trauma (2) vocal fold or recurrent laryngeal nerve trauma during prolonged or difficult intubation and extubation experiences, (3) neurologic abnormality, as may occur with vagus nerve compression secondary to Arnold-Chiari malformation, or (4) surgical complications, in particular cardiothoracic. Vocal fold paralysis can be unilateral or bilateral. In the former case, the presence or absence of stridor depends upon the glottal resting position of the paralyzed vocal fold. Hoarse-breathy voice or weakened cry and problems with episodic aspiration are likely in this population. If automatic, contralateral vocal fold compensatory voice and swallowing functions develop, formal treatments for the involved vocal fold can be delayed indefinitely. For the child with persistent dysphagia, feeding via a nasogastric tube may be necessary to prevent aspiration pneumonia and to afford the employment of specific therapeutic exercises to improve protective coughing behavior and vocalization efforts. Severe respiratory distress is rare in children with unilateral vocal fold paralysis.

Bilateral abductor vocal fold paralysis induces many more clinically significant abnormal signs and symptoms, including life-threatening airway occlusion. In this condition voice is typically unaffected because the vocal fold free edges are in close approximation near the midline of the glottis. Evaluation of the severity of stridor and status of

the vocal folds largely depends upon the age of the child and the degree of agitation and respiratory distress exhibited. When office setting examination is not feasible, rigid airway endoscopy under general anesthesia with spontaneous ventilation should be performed; vocal fold mobility is best appraised during the waking phase of this procedure. Passive motion can also be assessed at this time to rule out cricoarytenoid joint subluxation or fixation. MRI of the brain is almost always indicated in cases of bilateral vocal fold paralysis for the purpose of excluding an Arnold-Chiari malformation and/ or hydrocephalus.

Recovery of motion can be expected in as many as 70% of cases within 12 months postonset, especially if the etiology of vocal fold paralysis is either unknown or clearly attributable to local trauma. When possible, definitive surgical intervention of the bilaterally paralyzed larynx should therefore be delayed to allow for spontaneous healing. In the case of persistent unilateral vocal fold paralysis with aspiration difficulty, injection laryngoplasty may be an appropriate procedure to increase its bulk and improve glottal competency during voice and swallowing activities. This procedure usually results in temporary (<1year) gains, and may need to be repeated if recovery of motion does not occur. In the late teenage years, medialization thyroplasty may be required to achieve longer lasting improvement.

For bilateral vocal fold paralysis airway widening procedures may be attempted with the objective of eventual decannulation and preservation of swallowing and voice abilities. Unilateral medial arytenoidectomy, using laser technology, has been effective for such purposes in select individuals. Creating a small triangular divot in the posteromedial membranous section of one vocal fold has also been reported with similar objectives for this clinical population, with mixed outcomes.

Subglottic Stenosis (Figure 11)

This condition is the third most leading cause of airway compromise in children. Although it is a congenital anomaly, it can also occur as a result of abundant scar tissue and/ or granulation tissue formation following a prolonged or difficult intubation experience. The hazardously narrow dimensions of the immediate subglottis cause biphasic stridor, lower respiratory tract infections, dyspnea, and effortful phonation or cry. In the congenital variant, these presenting signs are usually evident within the first 3 months of age. Not infrequently, there is

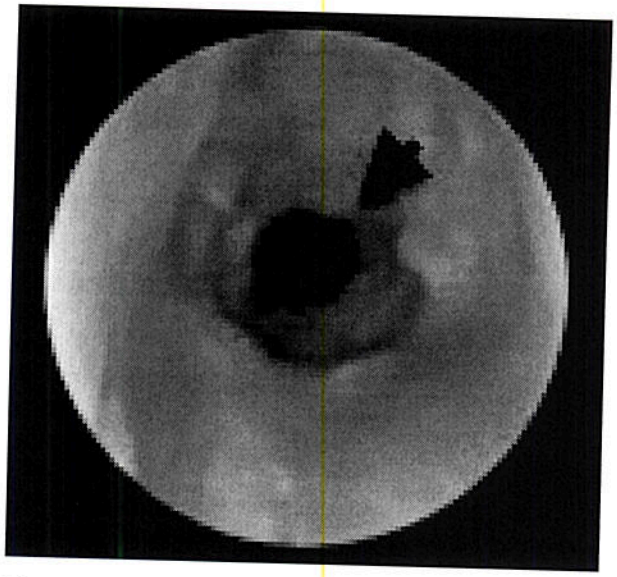


Figure 11. Subglottic stenosis.

an associated history of recurrent croup and laryngopharyngeal reflux. The diagnosis is made via conventional techniques, and variably sized endotracheal tubes are utilized for measurement purposes. Traditionally, a newborn subglottic lumen diameter of 6mm or greater has been defined as normal. The Myer and Cotton classification system was developed to categorize the degree of stenosis and facilitate treatment planning. Grade 1 represents stenosis equal to or less than 50%. Grade 2 symbolizes a 50 to 70% level of subglottic stenosis and airflow obstruction. In Grade 3 the stenosis ranges from 71 to 99%; and in Grade 4 there is no discernible lumen in the immediate subglottal space.

Depending on the stenosis grade, treatment may range from close surveillance to aggressive surgical intervention to enlarge the subglottis and relieve the aforementioned signs and symptoms. Laryngotracheal reconstruction is the most common approach to management, for children with severe Grade 3 and Grade 4 stenosis. In this surgery a costal or thyroid cartilaginous graft is harvested and fastened to a vertical laryngotracheal fissure. This technique is generally aimed at expanding the stenotic segment. Successful endoscopic carbon dioxide laser and microdebrider resection of the stenotic segment both with and without mitomycin application has been reported in cases of mild to moderate involvement (ie., Grade 1 to mild Grade 3). Less morbid treatment involving use of variably sized rigid or balloon dilators has also been investigated with mixed results. This is largely because

multiple procedures are typically required to ensure long-term airway stability.

Subglottic Hemangioma (Figure 12)

This is the most common laryngeal neoplasm in children. There is a 2:1 female to male incidence level. This represents a rapidly growing vascular tumor. It is always benign, and it is pathologically characterized by cellular hyperplasia of endothelial cells, mast cells, fibroblasts, and macrophages. Signs of inspiratory or biphasic stridor, barking cough, and effortful and harsh cry or phonation are usually evident within the first 6 months of life and their proliferative phase generally lasts for 6 to 12 months. Luckily, in most cases these lesions undergo spontaneous resolution or involution over a 2 to 10-year period at a rate of 10% per year. Plain film anteroposterior neck x-rays reveal asymmetric subglottic narrowing in most clinically significant cases. CT or MRI studies nicely outline the dimensions of the lesion and any extension into the neck or mediastinum. Airway endoscopy with spontaneous ventilation demonstrates a soft appearing submucosal mass that may have a bluish or reddish discoloration that often blanches in response to topical epinephrine.

Nonsurgical management in mild to moderate cases may include systemic or intralesional corticosteroids and now oral propranolol. Surgical options involve either CO_2 or KTP laser excision or open submucosal resection of the tumor, with or without concurrent tracheotomy for the more sever cases.

Trachea

There are several abnormal conditions involving the trachea that contribute to stridor and respiratory distress in children. These include tracheomalacia, bacterial and viral tracheitis, tracheoesophageal fistula, and foreign body aspiration.

Tracheomalacia (Figure 13)

When the cartilaginous framework composing the trachea remains abnormally soft postnatally the lumen may collapse and obstruct airflow dynamics. Occurring within the first year of life, tracheomalacia and tracheobronchomalacia cause expiratory or biphasic stridor, expiratory wheezing, and recurrent lower respiratory tract infections. This latter condition arises in response to excess mucus within the immediate subglottis and laryngeal inlet that can drain to and infect the lungs. These secretions accumulate because the abnormally narrow trachea or bronchi inhibits the upstream aerodynamic ejection or clearance mechanism.

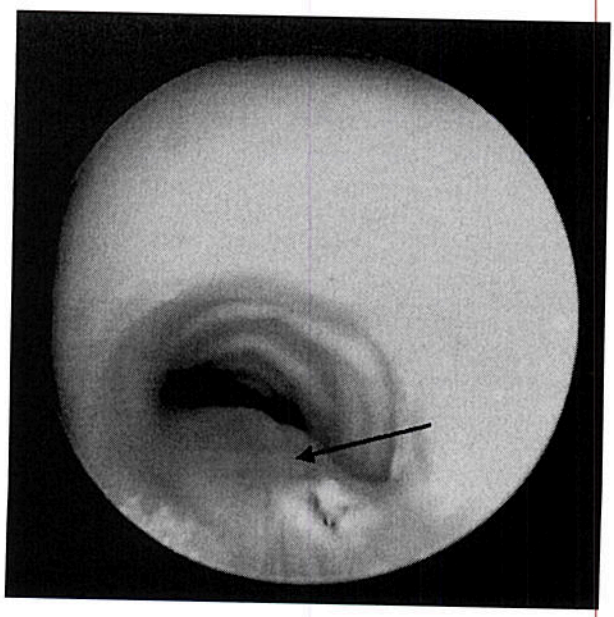


Figure 12. Subglottic hemangioma. Arrow pointing to left posterior subglottic hemangioma—most common location in the airway.

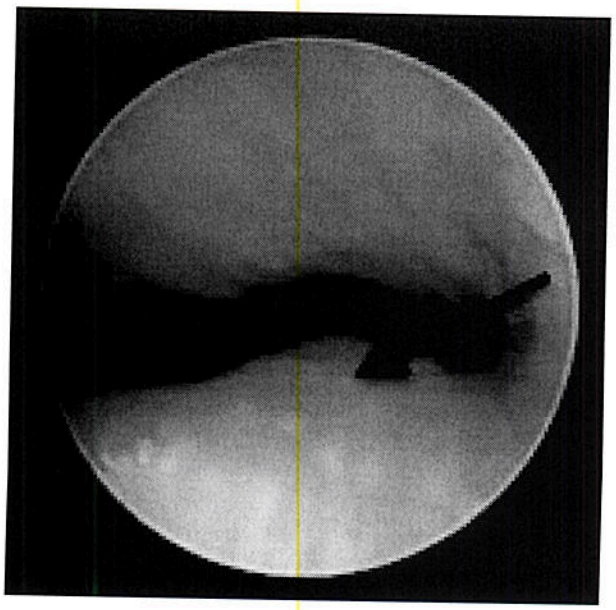


Figure 13. Tracheomalacia. Notice the lack of definitive tracheal rings and airway collapse.

Found in association with some congenital disorders, such as Down's syndrome, tracheomalacia usually spontaneously resolves by 2 years of age. Although, fluoroscopy can be used for investigation, bronchoscopy under general anesthesia is required for definitive diagnosis. In severe cases, tracheostomy and positive air ventilation may be required until natural resolution occurs.

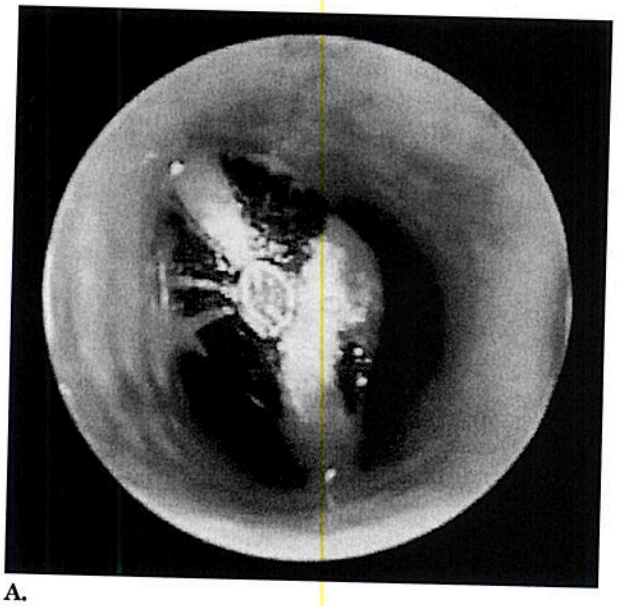
Bacterial Tracheitis

Viral croup can advance into this superinfection condition. Other classifications include bacterial laryngotracheobronchitis, membranous LTB, and pseudomembranous croup. *Staphylococcus aureus* is the most common causative pathogen. *Streptococcus pneumoniae*, *H. influenzae*, measles, and enterovirus species have been reported but are less likely etiologies. Presentation in children usually follows an upper respiratory infection with slowly progressive signs of exudates and copious purulent secretions, biphasic stridor, high fever, listlessness, dyspnea, and croupy cough. Of diagnostic significance is the fact that the child does not readily improve with the inhalation of racemic epinephrine and systemic steroids. The barking cough also helps to differentiate bacterial tracheitis from epiglottitis; coughing is not typically a sign of this latter pathology. Additionally, as previously described, epiglottitis not only progresses very rapidly it is also characterized by notable drooling and dysphagia, which are not pathognomonic of bacterial tracheitis. Auscultation of the lungs demonstrates rhonchi or wheezing, owing to standing tracheal exudates and lung infiltrates. Stressful chest retractions are usually evident. This clinical portrait is seen in children as young as 3 weeks of age and in adolescents as old as 16 years.

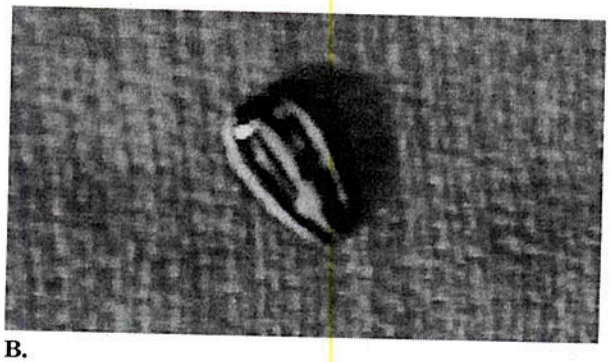
Frequent orotracheal suctioning of the bacterial membranes within the trachea is vital to control, without which the child may succumb to this disease because of a completely obstructed airway. Placement of an endotracheal tube for maintenance can be required until the infection subsides and the child is more stable. Use of IV antibiotics directed against the suspected causative agent is of paramount importance to the cure.

Foreign Body (Figures 14A and 14B)

Aspiration of a food item, common household object, or a toy into the laryngo-tracheo-bronchial axis is a cause of stridor and breathing difficulty in young children, especially those with advancing di-



A.



B.

Figure 14. A. Foreign body in proximal trachea. **B.** Sunflower seed after extraction.

ets and ambulation abilities. An antecedent acute episode of choking and gagging, as may be reported by a parent or guardian, points to this likely explanation for the aforementioned sequelae. The severity of these signs and symptoms may vary from intermittent coughing or wheezing to pronounced respiratory distress due to complete airway obstruction. Plain film x-rays are of limited diagnostic value in many cases. For instance, both plastic, organic and nonmetallic objects are nonradiopaque and therefore undetectable with such studies. In most instances of suspected foreign body aspiration, expiratory chest film and fluoroscopy produce good diagnostic evidence, with signs of mediastinal shift and air trapping. That being stated, history is the most important aspect to be considered in the decision-making process.

In the case of a negative imaging study, if the recent history supports the likelihood of foreign body aspiration, it is wise to proceed with ventilating rigid bronchoscopy under general anesthesia to rule out this potentially life-threatening condition. As many as 25% of negative film results prove to be false. During this procedure if a foreign object is discovered it can be safely removed and the proximal and distal airway can be thoroughly appraised for clearance. Intravenous steroids and inhaled bronchodilators are usually administered postoperatively, owing to both reactive and iatrogenic induced local edema.

If a large foreign body lodges in the hypopharynx or at the level of the cricopharyngeal muscle the child will exhibit symptoms of a globus sensation and gagging behaviors. Respiratory distress may develop if the foreign body encroaches upon the laryngeal inlet or converts into an airway emergency. Foreign bodies that lodge within the esophageal lumen can cause constant compression against the posterior tracheal airway with resultant dyspnea. Prompt removal is required, especially if the object can produce a toxic or erosive effect, such as a battery.

Neck Masses

Of all the possible neck pathologies that afflict children vascular malformations are the most likely to induce clinically significant respiratory distress. From a histologic point of view, vascular malformations can be classified as capillary, cavernous, and cystic with lymphatic micro and macro cystic subtypes. These malformations do not spontaneously involute and generally grow with the patient. ***Lymphatic cystic masses (lymphangiomas)*** (Figure 15) cause abnormal, partially isolated routes of lymphatic drainage, and they can result in secondary aerodigestive compressive signs and symptoms.

Lymphangiomas can grow into large, space occupying masses that compress the upper airway and cause notable breathing difficulty. They typically present within the first few years of life, and are generally soft to palpation, virtually painless, cosmetically obvious when they appear in the neck, and compressible. Because these masses do not usually resolve spontaneously, sclerotherapy has emerged as the treatment of choice for macrocystic lesions with surgical resection being reserved for microcystic and large lesions. Not uncommonly, these malformations recur, owing to poor encapsulation and difficult dissection planes.

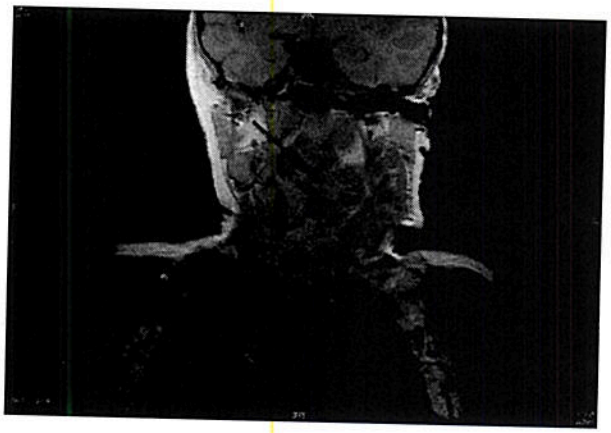


Figure 15. Coronal T1 MRI of large lymphatic malformation obstructing airway at hypophayngeal (*arrow*) and subglottic region.

Large neck masses have the greatest tendency to cause a congenital high airway obstruction syndrome (CHAOS). Survival of the newborn in such cases often depends on emergent airway maintenance soon after birth.

Lungs

If respiratory distress in a child cannot be attributed to nasal, oropharyngeal, supraglottal, glottal, subglottal, neck, or tracheal obstruction, attention should be directed to the lungs for a possible explanation. In children younger than 3 years, the intrapulmonary airway is very small. Thus, any lower respiratory tract infection will cause diminished airflow and breathing ability. Pulmonary auscultation often illustrates the presence of wheezing in these children and a decrease in airway responsiveness, which may be associated with bronchopulmonary dysplasia, viral respiratory diseases, asthma, environmental allergens, and certain hereditary factors.

Less than 50% of young children who exhibit signs of wheezing actually suffer from or develop asthma. Most of these infants struggle with reactive airway disease. However, when asthma is considered the probable diagnosis, it must meet specific criteria to support this conclusion: (1) the child should be at least 5 years of age, (2) airflow resistance or obstruction should be intermittent not constant, (3) such obstruction should be immediately reversible after administration of short acting beta 2–agonist medication, and (4) alternative causes, such as rhinovirus infection, environmental irritants, and allergic airway manifestations must have been excluded in the broad differential workup.

The risk of developing asthma is less than 10% if neither parent suffers from this condition. The risk respectively rises to 25 and 65% if one or both parents are afflicted.

There is a high correlation between asthma and allergy-induced lower airway reactivity abnormality. Bronchiole inelasticity may occur with each condition. Both co-exist in many children and give rise to very similar pathophysiologic respiratory signs and symptoms. Allergy testing often helps to delineate specific antigen sensitivities that may mediate or induce airway inflammation, edema, bronchospasm, excessive tracheal and pulmonary mucus accumulation, and labored breathing. If such testing fails to identify an underlying allergy, the diagnosis of asthma may be accurate. Naturally, effective allergy versus asthma medical therapy should be differentially employed, depending on the ultimate diagnosis.

CONCLUSION

The undeveloped pediatric airway is fragile, and therefore subject to compromise by many different pathologies. Comprehensive knowledge of these potentially threatening conditions should be of paramount importance to the otolaryngologist. Without detailed understanding of these factors, accurate differential, appraisal, and treatment may not be possible. Such failure may lead to serious complications and even death in some cases. The most common airway pathologies in children have been presented in this review. Certainly, the information contained here in is not an exhaustive account of all conditions that may place a child's life at risk because of respiratory distress. Pediatricians and pediatric otolaryngologist alike must utilize effective and broad-based algorithm to limit diagnostic mistakes and erroneous treatment decisions. It has been our intention to produce a solid foundation for the differentiation of pediatric pathologies and associated effective treatment strategies. To optimize clinically favorable outcomes, practicing clinicians should master the material and apply the information to their general fund of knowledge when evaluating children with abnormal breathing.

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