Stridor in Children

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Introduction

Stridor can be defined as the audible sign produced by the turbulent air flow through a narrow segment of the respiratory tract. According to the Venturi effect, air flow increases in a narrowed segment of a tube. This increase produces a negative pressure on the walls of the airway (Bernoulli principle), which probably explains the origin of the clinical sign and the tendency of airway callapse in this situation.

Stridor is the most important sign of airway obstruction in children, and it requires a precise diagnosis and sometimes immediate treatment. The degree of airway compromise – indicated by an increased ventilatory effort, tachypnea, supraclavicular or intercostal retractions and cyanosis – determines the urgency of the diagnostic workup and the beginning of therapeutic maneuvers.

Clinical Evaluation

History

In the absence of signs of severe respiratory distress, a detailed clinical history should be taken before any intervention. The stridor characteristics, and the phase of the respiratory cycle during which it is perceived help to determine the affected site. Obstructive lesions of the supraglottic segment commonly produce high pitch inspiratory stridor. In glottic and subglottic lesions, stridor tends to be of intermmediate pitch and biphasic, since the air flow is impaired during both inspiration and expiration. When the norrowing occurs at the tracheal or bronchial level, the positive pressure generated during expiration results in expiratory stridor.

The clinical history should also include information about the beginning of the symptoms, its duration, progression, variability, severity (subjective parent impression), presence of cyanosis episodes, effects of sleep, feeding, crying and posture. Other antecedents are equally important, such as prematurity, history of intubation (difficulty, duration, frequency, tube size, etc.), syndromic or non-syndromic malformations, gastroesophageal reflux disease and pulmonary, cardiac or neurologic impairment.

The influence of sleep and awakeness on the occurrence or intensity of the symptoms should also be noted. Generally, obstruction worse during sleep time is of pharyngeal origin (pharyngeal or palatine tonsillar hypertrophy), whereas the worsening of laryngeal, tracheal and bronchial obstructions happens during awakeness, and is exacerbated by physical straining.

Physical examination

The physical examination should start with the careful observation of the child. Increased respiratory rate, nose flaring, intercostal, subcostal and suprasternal

retractions, abdominal movements and cyanosis are indications of respiratory distress. If these signs are present in a child with progressive discomfort, airway stabilization measures might be needed before the examination proceeds.

The cardiopulmonry auscultation is performed before any other diagnostic maneuver causes crying. If there are associated dysphagia or aspiration, it is useful to watch the patient during swallowing. Congenital abnormalities should be actively looked for, which includes the examination of the palate, the observation of the relative size of the tongue and mandible and other craniofacial syndrome stigmata.

The examination of the nasal cavities, pharynx and larynx with a flexible endoscope is almost an extension of the physical examination. Its office-based performance is safe when done by a trained staff, with adequate precautions of aspiration and oxygen supplementation. Special attention is devoted to the dynamic evaluation of the supraglottic structures and the vocal chord mobility. The subglottis is not adequately visualized in most cases, and no attempt should be done to go beyond the level of the vocal chords without topical anesthesia, sedation and complete monitoring.

Complementary investigation

Radiology evaluation

The basic evaluation includes chest radiograms, to exclude pulmonary disease or mediastinal abnormalities. Cervical x-rays in posteroanterior and lateral projections, with adequate soft tissue penetration can be ordered. Their usefulness has been questioned because of the low correlation between its findings and the direct laryngotracheal endoscopy (18-20%) and the use restricted to the cases when there is permanent anatomic obstruction, such as the stenoses.

The videofluoroscopic study and the barium esophagogram are useful in the cases with associated swallowing difficulties, and can reveal laryngeal clefts, tracheoesophageal fistulas, vascular rings and gastroesophageal reflux.

If a vascular compression or other mediastinal abnormality is suspected, a contrast-enhanced computed tomography or a MRI scan with gadolinium (as an alternative to angiography) should be ordered.

Direct laryngotracheoscopy

Stridor can represent severe airway problems, but, in most cases, it is caused by benign and self-limited diseases. Therefore, the complete endoscopic evaluation is not indicated in all patients, being restricted to the cases with severity indicators (worsening stridor, cyanosis/apnea episodes), suspected synchronic lesions, recurrent episodes of unknown etiology, low weight gain or increased risk of office-based endoscopy due to pulmonary or congenital cardiac disease. It is also the gold-standard for the evaluation of laryngothracheal stenoses.

The exam is performed under general anesthesia with spontaneous ventilation, and the airway is evaluated with a microscope or a rod-lens endoscope. The use of the microscope allows the palpation of the posterior comissure to identify interarythenoid fibrosis or cricoarythenoid anchilosis. The trachea and bronchi are examined with a ventilating bronchoscope or a telescope introduced through the laryngoscope. At the end of the exam, the vocal chord mobility is evaluated during anesthetic recovery

The presence of macroglossia**,** tonsillar hypertrophy, pharyngeal collapse, peritracheostomal granulomas, tracheomalacia, extrinsic tracheal compression and inflammatory signs suggesting gastroesophageal reflux can be observed.

Most frequent etiologies

Table 1, Table 2 and **Table 3** list the main causes of respíratory stridor according to the obstructed site.

Table 1. Causes of pharyngeal obstruction

1. Pharyngeal and palatine tonsillar hypertophy
2. Foreign body
3. Craniofacial anomalies
4. Neurologic lesions
5. Rhinopharyngeal mass (encephalocele, teratoma, glioma)
6. Retropharyngeal mass

Table 2. Causes of laryngeal obstruction

Congenital
1. Laryngomalacia
2. Subglottic stenosis
3. Vocal chord paralysis
4. Laryngocele
5. Laryngeal webs
Aquired
1. Infectious
a. Supraglottitis
b. Viral laryngotracheobronchitis (croupe)
c. Bacterial tracheitis
2. Neoplastic
a. Malignant
b. Hemangiomas
c. Recurrent papillomatosis
d. Lymphangioma
e. Granulomas
3. Laryngotracheal angioedema
4. Trauma
5. Foreign body

Table 3. Causes of tracheobronchial obstruction

1. Tracheomalacia, bronchomalacia
2. Vascular anomalies
3. Stenoses
4. Tracheoesophageal fistula
5. Foreign body
6. Bronchitis, bronchiolitis

According to Holinger, congenital laryngeal anomalies are responsible for 60% of stridor cases in children before 2,5 years of age, and laryngomalacia stands out as the most common isolated cause. On the other hand, when one considers only the causes of acute onset stridor, the differential diagnosis turns to the infectious causes of airway obstruction (supraglottitis, viral croup and bacterial tracheitis) and foreign body.

Laryngomalacia

Laryngomalacia commonly presents within the first two weeks of life, with noisy breathing and inspiratory stridor that occurs during rest and worsens with straining – crying, agitation, feeding – and supine position. After a period of symptom exacerbation during the first months of life, a gradual improvement usually occurs beginning in the sixth month.

Although most patients present with a benign clinical course, with symptom resolution until 18-24 months of age, some children develop severe obstruction, cor pulmonale, pectus excavatum, obstructive apnea and low weight gain, with possible associated dysphagia and gastroesophageal reflux.

The diagnosis is done through history and physical examination, complemented by flexible laryngoscopy. The endoscope should be passed through both nostrils, to evaluate the nasal cavities, rhinopharynx and supraglottis. The obstruction mechanism is determined as frequently as possible. The association of two or more abnormalities (**Table 4**) is common, although one of them usually predominates.

Table 4. Laryngomalacia obstruction mechanisms

Type 1: Aryepiglottic fold collapse, with aspiration of the cuneiform cartilages into the larynx
Type 2: Long, tubular epiglottis, with exacerbated omega shape
Type 3: Anterior and medial collapse of the corniculate and cuneiform cartilages
Type 4: Posterior displacement of the epiglottis towards the posterior pharyngeal wall
Type 5: Short aryepiglottic folds

Direct laryngotracheoscopy is indicated in the cases with suspected synchronic lesions, due to either inconsistency between laryngoscopy findings and the obstruction severity or suggestive radiographic alterations.

The treatment is expectant in most cases, with continued observation of feeding, growth and development of cardiopulmonary symptoms. When obstructive apnea, low weight/height gain, cor pulmonale or other complications owing to the respiratory disturbance ensue, the surgical treatment should be considered.

The obstructive mechanism identified during the flexible laryngoscopic examination determines the necessary surgical maneuvers, that can include the resection of the lateral borders of the epiglottis, section of the aryepiglottic folds and removal of redundant mucosa over the corniculate or cuneiform cartilages. The approach should be as conservative as possible, considering the possible complications of stenosis, aspiration, edema and dysphagia.

Infectious etiologies

Each of these diseases (viral laryngotracheobrochitis, supraglottitis and bacterial tracheitis) has a distinct clinical presentation and treatment, and their main aspects are outlined on **Table 5**.

Table 5. Diagnosis and treatment of infectious etiologies for stridor

	Viral croup	Bacterial tracheitis	Supraglottitis
Age	3-6 months	3 months-16 years	3-5 years
Onset	Gradual	Unpredictable	Gradual
Cough	Barking	—	—
Drooling	—	—	+
X-rays	Steeple sign	Irregular tracheal lining	Thumb sign
Etiology	Parainfluenza	*S.aureus*	*H. influenzae*
Treatment IV Antibiotics +/- Intubation	Humidification Epinephrine Corticoids	Bronchoscopy +/- Intubation	IV Antibiotics

Modified from Johnson JT (ed) - Maintenance manual for lifelong learning 2002:427-433.

Viral laryngotracheobronchitis (croup)

Laryngotracheobronchitis (LTB) patients typically present with a history of upper airway infection that progresses to the characteristic "barking" cough, hoarseness and mild to moderate stridor. The presence of biphasic stridor, retractions, tachypnea and desaturations should alert to the risk of respiratory insufficiency.

Cervical X-rays in anteroposterior projection can help the diagnosis, demonstrating the typical subglottic narrowing (steeple sign), present in 50% of the cases, or an unsuspected foreign body. The flexible laryngoscopic examination is usually well tolerated when there is no severe respiratory distress, although the subglottis visualization is not always possible.

The treatment consists of support measures. Historically, humidification of the airway has been advocated as the first therapeutic measure, although there is no evidence of a better prognosis or benefitial effects on the subglottic mucosal inflammation. Patients that do not respond to humidification should receive inhalations of racemic epinephrine (0,5 ml of 1:1000 solution in 3 ml of saline). Even though it does not alter the course of the viral infection, the use of epinephrine can reduce the need to stablish an artificial airway. The use of cortico-steroids has been demonstrated to be useful in the treatment of severe croup, reducing the hospital stay when administered intramuscularly (dexamethasone 0,5 - 1mg/kg) or via inhalation (budesonide).

The stablishment of an artificial airway through intubation is essential when clinical therapy does not prevent the progression of the respiratory insufficiency. The nasotracheal approach is preferable, with a tube 0,5 mm smaller than the recommended for the patient´s age. Extubation should be attempted only when the child is not febrile, with minimal secretions and with air leak around the tube (confirmed by cough or vocalization), which generally occurs after 5 days.

Bacterial tracheitis

This infection extends from the subglottic region through all the tracheal length. The most accepted etiological mechanism is that bacterial invasion occurs secondary to an upper airway viral infection.

Affected patients belong to a wide age range, from 3 months to 13 years old, and have an initial presentation similar to that of LTB, with cough and hoarseness. However, they evolve, in a 8-10 hour period, to inspiratory stridor, retractions and cyanosis. X-rays can show irregularrities in the tracheal air column and concomitant pneumonia.

Rigid endoscopy under general anesthesia is diagnostic and therapeutic, because it

allows the visualization of the swollen and inflammed tracheal mucosa, as well as the removal of crusts and inspissated purulent secretions. Intravenous antibiotics should target the commonly isolated organisms: *S. aureus* and *H. influenzae*. Intubation is necessary in about 85% of the patients, with the advantages of better airway control and pulmonary hygiene.

Supraglottitis

Children with supraglottitis represent a true aiway urgency, caused by a bacterial invasion of the lingual aspect of the epiglottis, aryepiglottic folds, laryngeal vestibule and paraglottic spaces by *Haemophilus influenzae* type b.

Affected patients are 3 to 6 years old and present with a history of progressive soreness of the throat that progresses to drooling, respiratory distress and inspiratory stridor in a period of 4 to 8 hours. The child has a toxemic appearance and adopts the typical tripod posture, sitting with the neck in extension and the arms providing support.

The risk of worsening the respiratory distress with diagnostic maneuvers, such as examination of the mouth with a tongue blade and flexible endoscopy, should prevent their performance. X-rays in lateral projection can demonstrate the typical sign of epiglottis thickening (thumb sign), but they are not essential to the diagnosis and should not delay therapeutic intervention.

After the diagnosis is made, nasotracheal intubation should be attempted. This procedure is done in a setting that allows the performance of a tracheotomy if the attempt is unsuccessful. Blood and epiglottis cultures are drawn and antibiotics are directed towards the most common etiological agent: *H. influenzae* b.

Conclusion

Airway abnormalities frequently present with stridor in infancy. The quick recognition of its etiology leads to the most appropriate treatment.

Recommended readings

1. Stern Y, Cotton RT. Evaluation of the noisy infant. In: Cotton RT, Myer CM (eds) - Practical pediatric otolaryngology, Lippincott-Raven Publishers, 1999:471-476.
2. Stroud RH, Friedman NR. An update on inflammatory disorders of the pediatric airway: epiglottitis, croup, and tracheitis. Am J Otolaryngol 2001;22:268-275
3. Holinger LD. Evaluation of stridor and wheezing. In: Holinger LD, Lusk RP, Green CG (eds) – Pediatric laryngology and bronchoesophagology, Lippincott-Raven Publishers, 1997:41-48
4. Management of pediatric airway inflammatory conditions. In: Johnson JT (ed) - Maintenance manual for lifelong learning 2nd ed, Kendall-Hunt Publishing Company, 2002:427-433.
5. Graf J, Stein F. Tracheitis in pediatric patients. Semin Pediatr Infect Dis 2006;17:11-13.
6. Masters IB, Chang AB, Patterson L, Wainwright C, Buntain H, Dean BW, Francis PW. Series of laryngomalacia, tracheomalacia, and bronchomalacia disorders and their associations with other conditions in children. Pediatr Pulmol 2002;34:189-195.