Stridor

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Stridor is a harsh, coarse, grating respiratory sound with crowing quality, often audible without a stethoscope. Usually indicative of extrathoracic airflow obstruction, it represents the rapid, turbulent flow of air through a narrowed airway. At times described by parents as noisy breathing, congestion, wheezing, or hard breathing, stridor is an important symptom and physical finding that should prompt a directed and expedient evaluation.

Stridor can originate from any of the different areas of the airway, which can be divided into 3 zones: (1) the supraglottic zone, which includes the pharynx; (2) the extrathoracic tracheal zone, which includes the glottis, subglottic area, and proximal trachea; and (3) the intrathoracic tracheal zone, which can extend to the primary and secondary bronchi.

The phase of stridor may indicate the level of airflow obstruction. Inspiratory stridor implies a dynamic extrathoracic obstruction, whereas biphasic stridor suggests a fixed airflow obstruction. Expiratory stridor, termed a *homophonous wheeze* by many authorities, is most likely the result of a tracheobronchial obstruction.

Pediatric patients, especially infants with smaller airways, are especially susceptible to large airway obstruction from inflammation, secretions, and edema. Their small airway diameter results in a larger change in resistance and reduction in airflow than a larger patient as demonstrated by the Poiseuille law, stated $\dot{v}=P\pi r^4/8nl$, where $\dot{v}$ indicates flow; P, pressure difference across the length (l); n, coefficient of viscosity; and r, radius. Because flow varies with the radius to the fourth power, a small reduction in airway radius in a small airway can have a profound effect on airflow. The reduction in airflow increases the energy expended to move air across the airway, which, in turn, increases the likelihood of turbulent airflow, resulting in stridor and respiratory distress.

Stridor can occur as an acute event or present as a chronic phenomenon. Table 1 details the features of both.

A careful history and physical examination will guide the evaluation of stridor, both chronic and acute, and differentiate children who need aggressive evaluation from those who can be safely observed. The history should include birth history, onset of the stridor, change with position and sleep, relationship to feedings, associated symptoms of reflux, feeding problems, and episodes of apneas or cyanosis. Many children who are growing well and are comfortable but noisy can often be observed prospectively. Reports of poor growth, distress, or cessation of respiration require an immediate and in-depth evaluation.

The physical examination should include a detailed observation of the patient's breathing pattern with particular attention to the quality of the stridor and its phase, degree of respiratory distress, use of accessory muscles, and retractions. Again, patients with distress must be identified and treated immediately. The clinician should also carefully examine the head, including nasal cavity, pharynx, soft and hard palates, and the tongue, to identify possible sites of obstruction. The

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TABLE 1. **Causes of Stridor**

CAUSES OF ACUTE STRIDOR	
Foreign body	Most common between 1 and 3 years, often sudden onset with cough, stridor, or wheezing
Viral croup (laryngotracheobronchitis)	Most common cause of acute stridor, most commonly caused by parainfluenza virus but can also be cause by influenza type A or B, respiratory syncytial virus infection, and rhinovirus Age of presentation of 6 months to 6 years, often preceded by upper respiratory tract infection symptoms, child presents with low-grade fever and barking cough
Epiglottitis	Most commonly caused by *Haemophilus influenzae* type B; occurs in children 2 to 7 years of age, peak incidence at 3 years of age; incidence reduced markedly by the use of the *H influenzae* type B vaccine
Bacterial tracheitis	Toxic appearing with fever and respiratory distress; most common cause is *Staphylococcus aureus,* can also be caused by *H influenzae* type B and *Moraxella catarrhalis*
Spasmodic croup (acute spasmodic laryngitis)	Acute onset at night, not preceded by upper respiratory tract infection; possibly triggered by allergy, psychological factors, or gastroesophageal reflux
Laryngeal papillomatosis	Caused by vertical transmission of human papillomavirus, most common laryngeal neoplasm in children
Angioneurotic edema	Acute swelling of the upper airway: swelling of the tongue, face, and pharynx
Hypocalcemic tetany	Can cause laryngospasm and stridor, can cause irritability, tremors, twitching, and carpopedal spasm
Psychogenic stridor	May be caused by emotional stress or conversion disorder
Intubation, trauma (acquired subglottic stenosis)	Can produce laryngotracheal stenosis, subglottic edema, and laryngospasm
Other causes	Adenotonsillar hypertrophy, retropharyngeal or peritonsillar abscess
CAUSES OF CHRONIC STRIDOR	
Nasal and pharyngeal malformations,	Choanal atresia or stenosis, intranasal masses
Craniofacial abnormalities, hypotonia	Pierre Robin sequence, Treacher Collins syndrome, macroglossia, micrognathia; hypotonia worse with sleep
Laryngomalacia	Most common cause of congenital stridor; inspiratory stridor; onset within the first 2 weeks after birth Exacerbating factors: feeding, agitation, supine position Spontaneous resolution by the age of 12 to 24 months; conservative management; patients with recurrent cyanosis, failure to thrive, hypoxemia, or apnea are candidates for surgical correction
Laryngeal web, cyst	Most located in glottis area, usually biphasic stridor Complete webs cause respiratory distress, partial webs produce stridor, weak cry, and some respiratory distress
Vocal cord paralysis	Inspiratory stridor; history of weak cry; may be idiopathic, iatrogenic, or secondary to neurologic abnormalities; may mimic asthma Unilateral: stridor, feeding difficulties Bilateral: life-threatening airway obstruction
Subglottic cyst	Rare, history of intubation; biphasic stridor Identified by direct laryngotracheobronchoscopy
Subglottic stenosis	Congenital or acquired secondary to intubation Usually located 2 to 3 mm below the glottis
Subglottic hemangioma	Biphasic stridor, age of 6 weeks to 6 months Associated hemangiomas in other parts of the body
Gastroesophageal reflux	Can present with stridor, can worsen laryngomalacia and cause laryngospasm
Vascular malformation	Esophagram reveals compression

face should be evaluated for possible micrognathia and other evidence of a genetic syndrome. The entire skin should be examined to look for cutaneous hemangiomas that could suggest an airway obstruction.

Further evaluation, guided by the history and physical examination, includes radiography of the chest and soft tissues of the neck, which can be helpful in cases with potential foreign bodies or subglottic swelling. Esophagography is an excellent screen for possible vascular ring, although magnetic resonance imaging or computed tomography angiography would be necessary to confirm the diagnosis.

Direct visualization of the airway by bronchoscopy frequently yields the diagnosis in a patient with chronic stridor. Ideally, the patient will be sedated and breathing spontaneously to allow a dynamic evaluation of the airway in motion. A fiberoptic laryngoscopy can be performed while the patient is awake and can confirm a diagnosis of laryngomalacia. This diagnosis does not, however, exclude the possibility of pathologic findings below the vocal cords, requiring a complete evaluation.

In summary, stridor is a common presenting sign and symptom in pediatric medicine. Its presence indicates large airway obstruction, which can be life-threatening. A careful history and physical examination can guide the clinician in judicious use of imaging and consultative services while maintaining a careful regard for patient safety. Stable patients with chronic stridor that is inspiratory only can often be observed, whereas patients with distress or biphasic stridor should have a prompt evaluation.

COMMENT: The most common cause of stridor in young children is viral croup. Although most often a mild and self-limited illness, croup has kept many a parent awake all night (me included) fearfully watching a stridulous child in distress with a barking cough and labored breathing. For years the treatment we advised, warm mist and cool air, in truth had nothing more than tradition to support it. However, evidence trumps anecdote, and we have reasonable evidence to support the effectiveness of nebulized epinephrine and dexamethasone in alleviating symptoms, presumably by reducing inflammation and thus obstruction. Perhaps there was a clue in the typical pattern of croup to suggest that a corticosteroid would help: almost always symptoms are at their worst overnight, when endogenous glucocorticoid levels are at their lowest.

– Henry M. Adam, MD
Editor, *In Brief*

Parent Resources from the AAP at HealthyChildren.org

- http://www.healthychildren.org/English/health-issues/conditions/chest-lungs/Pages/Croup-Treatment.aspx
- Spanish: http://www.healthychildren.org/spanish/health-issues/conditions/chest-lungs/paginas/croup-treatment.aspx

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