Stridor in the Newborn

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KEYWORDS
- Stridor
- Newborn
- Neonate
- Neonatal
- Laryngomalacia
- Larynx
- Trachea

KEY POINTS
- Stridor originates from laryngeal subsites (supraglottis, glottis, subglottis) or the trachea; a snoring sound originating from the pharynx is more appropriately considered stertor.
- Stridor is characterized by its volume, pitch, presence on inspiration or expiration, and severity with change in state (awake vs asleep) and position (prone vs supine).
- Laryngomalacia is the most common cause of neonatal stridor, and most cases can be managed conservatively provided the diagnosis is made with certainty.
- Premature babies, especially those with a history of intubation, are at risk for subglottic pathologic condition,
- Changes in voice associated with stridor suggest glottic pathologic condition and a need for otolaryngology referral.

INTRODUCTION

Families and practitioners alike may understandably be alarmed by stridor occurring in a newborn. An understanding of the presentation and differential diagnosis of neonatal stridor is vital in determining whether to manage the child with further observation in the primary care setting, specialist referral, or urgent inpatient care. In most cases, the management of neonatal stridor is outside the purview of the pediatric primary care provider. The goal of this review is not, therefore, to present an exhaustive review of causes of neonatal stridor, but rather to provide an approach to the stridulous newborn that can be used effectively in the assessment and triage of such patients.

Definitions

The neonatal period is defined by the World Health Organization as the first 28 days of age. For the purposes of this discussion, the newborn period includes the first 3 months of age.

Stridor consists of a harsh whistle that is classically produced when air emanates from a narrowed portion of the airway. It can be low or high pitched, depending on the caliber and shape of the airway and the amount of respiratory effort. In most cases, the management of neonatal stridor is outside the purview of the pediatric primary care provider. The goal of this review is not, therefore, to present an exhaustive review of causes of neonatal stridor, but rather to provide an approach to the stridulous newborn that can be used effectively in the assessment and triage of such patients.

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cases, it is readily distinguished from *stertor*, which is a flapping or snoring sound generated by redundant soft tissue or secretions in the nasal passages, nasopharynx, oral cavity, or oropharynx. The only location in which stridor and/or stertor often present together is the supraglottis. Although stridor may be inspiratory or expiratory, most clinicians refer to expiratory stridor as *wheezing*, which is generally softer, higher pitched, multitone, and most audible over the lower lung fields.

*Operative endoscopy* refers to endoscopic evaluation of the airway under anesthesia in the operating room. *Flexible fiber-optic laryngoscopy* (FFL) is usually performed in the office or at the bedside and does not require anesthesia; however, the examination is usually limited to the supraglottis and glottis.

**Anatomy**

The airway can be broadly divided into clinically relevant segments. Obstruction of the nasal passages, nasopharynx, oral cavity, and oropharynx can produce obstructive breathing sounds that are usually stertorous in nature and are beyond the scope of this review. This discussion focuses on the anatomic regions that produce classic stridor; namely, the larynx and trachea (**Fig. 1**). The larynx is divided into the supraglottis (including the cartilages and folds above the true vocal folds), the glottis (true vocal folds), and the subglottis (1 cm of airway below the vocal folds). The trachea begins below the subglottis at the inferior border of the cricoid cartilage and ends by branching into the right and left main bronchi.

**CLINICAL EVALUATION**

**History**

The history of a neonate’s stridor is critical in considering its likely cause and the need for potential operative endoscopy. The following are features of the stridor about which caregivers should be questioned:

**Time of initial onset**

In most cases, stridor in a newborn will be due to a congenital anomaly and therefore presents at birth. Even laryngomalacia, which may be reported as manifesting in

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**Fig. 1.** Anatomy of the larynx and trachea. Note division of larynx into supraglottis, glottis, and subglottis. (For the National Cancer Institute © 2012 Terese Winslow LLC, U.S. Govt. has certain rights.)
the first week or 2 after birth, is usually present to some degree soon after delivery. In rare cases, masses or vascular compression of the airway, brainstem abnormalities, or perinatal iatrogenic injury to the recurrent laryngeal nerve may result in stridor in the newborn that presents postnatally. Examples include masses within the airway, mediastinal masses, vascular compression of the trachea, Chiari malformation, and recurrent nerve injury during congenital heart surgery or ligation of a patent ductus.

**Quality of the stridor**

In general, extrathoracic pathologic condition presents with inspiratory stridor, whereas intrathoracic pathologic condition presents with expiratory stridor (also known as wheezing). This rule holds up well in differentiating laryngeal abnormalities from those in the trachea. However, lesions of the glottis and subglottis both may present with biphasic (inspiratory and expiratory) stridor, making it difficult to distinguish lesions within the larynx based on phase of breathing alone. Diagnosing laryngeal pathologic condition is further complicated by the fact that some supraglottic pathologic conditions (primarily laryngomalacia and vallecular cysts) can present with stridor rather than, or in addition to, stridor. Most lesions below the supraglottis present with coarse stridor, whereas laryngomalacia and vallecular cysts may present with a more musical (“ascending glissando”) quality.

**Progression of the stridor**

Stridor that increases in severity or frequency over time implies an evolving pathologic condition, such as compression from growing mass (most commonly subglottic infantile hemangiomas or cysts), progressive stenosis following endolaryngeal or endotracheal injury, or increasing vascular compression of the airway. Stridor that is improving is more likely due to inflammation, or to laryngomalacia that is spontaneously resolving.

**Changes with position (prone vs supine) or state (sleep vs awake)**

Changes due to position and/or state are usually associated with lesions in dynamic, as opposed to static, portions of the airway, namely the supraglottis and trachea. When changes occur with positioning, the stridor is usually worse supine; this occurs primarily with laryngomalacia and supraglottic pathologic conditions, in which the symptoms are exacerbated by retropositioning of the tongue due to gravity. Stridor that changes with state is usually worse awake than asleep, and due to dynamic disorders such as laryngomalacia and tracheomalacia. Obstruction at the tongue base is generally worse asleep.

**Changes in voice**

Dysphonia, manifested as a hoarse cry, is associated almost exclusively with glottic pathologic conditions, such as vocal fold paralysis or laryngeal web.

**Associated symptoms**

Cough may be present in a variety of pathologic conditions, but is seen most commonly in lesions that narrow the subglottis or trachea. Cough with feeding is often suggestive of abnormalities of vocal fold mobility or dyscoordination between swallowing and breathing as is seen in laryngomalacia, but may also suggest a cleft of the larynx or tracheoesophageal fistula. Gastroesophageal reflux has been implicated as both a cause and a sequela of airway pathologic condition and is commonly associated with laryngomalacia.

Additional history is useful in decision-making regarding the need for operative endoscopy or surgical intervention. Many practitioners may be familiar with the
“SPECS” algorithm, as delineated in Table 1. The presence of sternal retractions, progression, feeding difficulty or failure to thrive, and/or acute life-threatening episodes often suggests a need for further intervention. Birth history, intubation history, medical and surgical history, and family history are also helpful in determining possible causes.

**Physical Examination**

Assessment should first rule out severe respiratory distress, focusing on vital signs, and noninvasive assessment of level of consciousness, skin color, and accessory muscle use. If the patient is unstable, further management will focus on assessing and supporting the patient’s airway, breathing, and circulation, with urgent transfer to a higher level of care once secure.

Most patients will present in a far less urgent manner. Examination of the stable newborn with stridor should focus on nasal and oral patency as well as many of the features previously discussed: the quality of breathing sounds on inspiration and expiration, the presence of retractions, changes in the pattern of breathing with positioning, and assessment of voice. A complete neonatal examination, including recognition of syndromic features, overall neuromuscular tone, and auscultation of the heart and lungs, may provide important additional information regarding possible cause of the stridor. Assessment of the child’s growth will provide further information about the child’s overall functioning and ability to balance feeding with respiratory effort and caloric consumption.

**Imaging**

Imaging is rarely necessary in the assessment of newborn stridor, with the exception of those cases in which intrathoracic pathologic condition is suspected. In such cases, the imaging can usually be deferred to specialist assessment, but should be a part of the evaluation. A chest radiograph will occasionally demonstrate vascular compression of the trachea or mediastinal masses. A plain radiograph of the larynx is more useful beyond the newborn period, particularly in the diagnosis of croup, subglottic masses, supraglottitis, and foreign bodies; however, the utility of laryngeal plain films is very dependent on technique. Computed tomography (CT) scans with contrast can be used in the assessment of suspected masses of the airway, neck, or mediastinum, and CT angiography is diagnostic in cases of vascular compression of the airway. MRI is particularly helpful in the assessment of vascular anomalies, or if there is concern for Chiari malformation in the case of bilateral vocal fold paralysis. Some practitioners also recommend MRI to image the course of the recurrent laryngeal nerve in cases of unilateral vocal fold paralysis. The primary care provider should consider that an inappropriately ordered scan will subject the patient to unnecessary expense and/or radiation, without providing clinically relevant information.

<table>
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<tr>
<th>SPECS algorithm for evaluation of stridor</th>
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**Endoscopic Assessment**

In most cases, initial endoscopic assessment of the neonatal airway may be performed with a flexible endoscope in the otolaryngology office or at the hospital bedside. The procedure does not require anesthesia and, in most cases, can be performed in less than 2 minutes. Flexible fiber-optic endoscopy allows the examiner to visualize the pharynx as well as the supraglottic and glottic larynx. Examination of the subglottic larynx and trachea is more safely and accurately performed as an operative endoscopy procedure under anesthesia in the operating room. Operative endoscopy uses rigid endoscopes that provide excellent visualization and high-resolution images of the larynx and trachea (Fig. 2). The procedure is usually performed with the child ventilating spontaneously to allow the child to protect the airway during the procedure and to better simulate the conditions under which the stridor is observed.

**CAUSES OF NEWBORN STRIDOR**

Stridor classically originates from the larynx and/or the trachea. Table 2 lists the most common causes of neonatal stridor by anatomic subsite.

**Supraglottic Obstruction**

Supraglottic obstruction is characterized by inspiratory stridor, because the negative pressure generated during inspiration tends to collapse relatively soft supraglottic structures. Laryngomalacia is the most common cause of supraglottic obstruction and the most common cause of neonatal stridor in general. The cause of laryngomalacia remains unestablished, although most investigators implicate neuromuscular immaturity as the likely cause. Mild laryngomalacia is the only cause of newborn stridor that can be appropriately managed without specialist referral, but must be diagnosed with a high degree of certainty. Symptoms classically include a high-pitched, sometimes staccato stridor that worsens when the child is agitated and/or supine. Endoscopic evaluation typically reveals prolapse of the epiglottis and/or the

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Fig. 2. Operative endoscopy before a tracheotomy. The examiner, anesthetist, and room staff are able to visualize lesions obstructing the airway as a magnified, high-resolution image.
cuneiform cartilages into the airway. Other common findings include curling of the epiglottis upon itself and webbing of the aryepiglottic folds (Fig. 3). The severity of laryngomalacia is judged primarily by the degree of impairment of feeding and weight gain, and the presence of respiratory distress and/or apneic events. Symptoms usually resolve spontaneously by 12 to 24 months of age, but active management can reduce symptoms. Side positioning or semiprone positioning may be helpful in reducing sleep symptoms. Medical treatment of laryngopharyngeal reflux may also be considered, because there is an association between the 2 disorders. However, there has yet to be a randomized controlled trial examining the effect of acid-reducing medication on laryngomalacia symptoms, and there is no clear evidence that surgery for laryngomalacia reduces the frequency of reflux.5,6 Providers may also consider instituting a high-calorie formula for patients with poor weight gain. In rare cases, supraglottoplasty is recommended to divide the aryepiglottic folds and reduce the cuneiform cartilages.7 Patients with symptoms of aspiration and those being considered for supraglottoplasty should undergo a preoperative video swallow assessment, because aspiration may occur because of the pathologic condition or occasionally because of the surgical intervention.

Congenital cysts of the larynx are another supraglottic cause of neonatal stridor that can mimic laryngomalacia by causing prolapse of the epiglottis into the airway. Such lesions may originate in the vallecula (Fig. 4) or in the saccule of the laryngeal ventricle (Fig. 5). These cysts can easily be decompressed or marsupialized, but have a propensity to recur, and complete resection at the time of diagnosis is preferable. They are usually treated by excising the cyst and cauterizing the base deep to the vallecular mucosa.

Table 2

<table>
<thead>
<tr>
<th>Anatomic Subsite</th>
<th>Pathologic Condition</th>
<th>Notes</th>
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<tbody>
<tr>
<td>Supraglottic larynx</td>
<td>Laryngomalacia</td>
<td>Most common cause of newborn stridor. Mild cases are self-limited and do not require surgical consultation.</td>
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<tr>
<td></td>
<td>Vallecular cysts</td>
<td>Lymphatic malformations most common.</td>
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<td></td>
<td>Saccular cysts</td>
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<td></td>
<td>Masses</td>
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<tr>
<td>Glottic larynx</td>
<td>Webs</td>
<td>Hoarseness and stridor.</td>
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<tr>
<td></td>
<td>Clefts</td>
<td>Stridor present only when associated with significant redundancy of mucosa.</td>
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<td></td>
<td>Vocal fold immobility</td>
<td>May be associated with Chiari malformation.</td>
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<tr>
<td>Subglottic larynx</td>
<td>Subglottic stenosis or cysts</td>
<td>Can be congenital or acquired (after prolonged intubation).</td>
</tr>
<tr>
<td></td>
<td>Masses</td>
<td>Hemangiomas most commonly present at 3–4 mo of age.</td>
</tr>
<tr>
<td>Trachea</td>
<td>Tracheomalacia</td>
<td>Because of abnormal ratio of membranous to cartilaginous tracheal wall.</td>
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<tr>
<td></td>
<td>Primary</td>
<td>Extrinsic compression from vascular anomalies (anomalous innominate, double aortic arch, pulmonary artery sling).</td>
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<td></td>
<td>Secondary</td>
<td>Collapse due to presence of tracheoesophageal fistula.</td>
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<tr>
<td></td>
<td>Stenosis</td>
<td>Congenitally complete cartilaginous tracheal rings, or intubation injury.</td>
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Glottic obstruction is usually due to a fixed lesion such as bilateral vocal fold immobility or laryngeal web. The associated stridor will usually be biphasic. Bilateral vocal fold paralysis is usually idiopathic, but may be due to an abnormality of the central nervous system, such as Chiari malformation, cerebral palsy, or hydrocephalus. In fact, stridor due to vocal fold paralysis can occasionally be the first presenting symptom of Chiari malformation. In such cases, the stridor may improve with the correction of the central nervous system pathologic condition. Cases in which the paralysis is not reversible often require surgical intervention, which may include some combination of posterior laryngeal grafting, posterior vocal fold resection, or tracheotomy. In contrast, unilateral vocal fold paralysis results in stridor that, if present, is very mild and often improves in idiopathic cases. This disorder has been
reported to occur in more than 30% of patent ductus ligations examined postoperatively regardless of symptoms.\textsuperscript{9} Laryngeal webs result from failure of the larynx to completely recanalize during embryogenesis (Fig. 6). In contrast to bilateral vocal fold paralysis, dysphonia is a prominent presenting symptom. This disorder may be associated with 22q11 deletion syndrome.\textsuperscript{10} Management usually includes division of the web and stenting as well as laryngeal expansion with cartilage grafting.

**Subglottic Obstruction**

Subglottic obstruction will usually result in biphasic, predominantly inspiratory stridor. Subglottic stenosis may be congenital or acquired, even in the newborn period. In congenital cases, the cricoid cartilage is often elliptical (Fig. 7). Such cases can often
be managed conservatively if the stridor is not severe, because the narrowed segment of the airway grows with the patient. Occasionally, division and grafting of the cricoid are necessary and may be performed using cartilage from the rib or thyroid cartilage. Acquired cases, most often due to prolonged endotracheal intubation in premature infants, have a less favorable prognosis and more often require some combination of serial balloon dilation, laryngotracheal cartilage grafting, and tracheotomy (Fig. 8).

Preterm infants who have been intubated may also develop progressive biphasic stridor due to subglottic cysts that slowly increase in size (Fig. 9). These lesions may be multiple, but usually respond quite well to laser or mechanical excision. The subglottis is also a site of predilection for infantile hemangiomas (Fig. 10). These lesions will rapidly increase in size during the first few weeks of age, with a presentation similar to that of the lesions above. Once diagnosed, subglottic hemangiomas usually respond well to medical therapy with propranolol, although refractory lesions may need to be addressed with steroid injection or surgical excision.11

Fig. 7. Congenital subglottic stenosis. Patient has no history of endotracheal intubation. Note the elliptical shape of subglottic (cricoid cartilage).

Fig. 8. Acquired subglottic stenosis in a patient with history of prematurity and endotracheal intubation.
Tracheal Obstruction

Tracheal obstruction will usually result in a predominantly expiratory stridor as increased intrathoracic pressure predisposes the trachea to collapse during expiration. Tracheal obstruction may be due to primary tracheomalacia, secondary tracheomalacia (compression of trachea, usually vascular), and tracheal stenosis. Primary tracheomalacia results from a reduced ratio of cartilaginous to membranous trachea, resulting in flattening of the affected area due to lack of support. This finding is particularly common in cases of tracheoesophageal fistula (Fig. 11). Although it often improves over time, severe primary tracheomalacia occasionally must be managed with a tracheotomy to bypass the collapsing segment. Secondary tracheomalacia is most commonly due to vascular ring (double aortic arch, or right arch with left ligamentum arteriosum; Fig. 12), aberrant innominate artery, aberrant right subclavian artery, or pulmonary artery sling. Depending on the pathologic condition, the compression can be relieved either by suspension of the offending vessel or by surgical correction.

Fig. 9. Cysts in subglottis of a patient with a history of prematurity and intubation. (A) Left-sided cyst visible from above glottis. (B) Closeup view reveals second subglottic cyst slightly deeper on right side.

Fig. 10. Subglottic hemangioma. Red spots on surface mucosa and spongy texture are the key to diagnosis. Biopsy is rarely necessary, but intraoperative steroid injection is a good adjunct to medical therapy with propranolol.
of the vascular anomaly. It should be noted that the aberrant right subclavian artery may also cause significant esophageal compression, and that the association of pulmonary artery sling with tracheal stenosis has been reported to be as high as 50%.¹² Tracheal stenosis is usually due to a tracheal segment in which complete tracheal rings replace the usually membranous posterior wall (Fig. 13). Unless exceedingly mild, this condition must be surgically corrected by slide tracheoplasty. Tracheal sources of stridor often require multidisciplinary treatment from a variety of aerodigestive specialists, including pediatric otolaryngologists, pulmonologists, gastroenterologists, cardiothoracic surgeons, and speech and swallow therapists.

REPRESENTATIVE CASES

Case 1

A concerned mother brings her 4-week-old son to clinic with noisy breathing. He is the product of a full-term birth to a primipara mother, with no perinatal complications or concerning family history. He has had noisy breathing since birth, which is most noticeable when feeding or laying on his back. He does have relatively frequent spit ups, but is able to take 4 ounces of formula in under 10 minutes, and does not have coughing episodes while feeding. He has not had any cyanotic episodes and is gaining weight appropriately. He does have noisy breathing when sleeping in the supine position.

Fig. 11. Tracheomalacia in a patient with tracheoesophageal fistula. The trachea has a classic “fish-mouth” appearance.

Fig. 12. (A) Posterior tracheal compression superior to carina due to double aortic arch. (B) Airway appears more patent immediately inferior to the area of vascular compression.
position, but has not been noted to have pauses during sleep. On examination, he is of above-average body mass index and is well appearing, with no dysmorphic features, no skin lesions, no murmurs or rubs, clear lung fields, and a grossly normal neurologic examination. He does have intermittent high-pitched inspiratory stridor when supine or excited, in addition to mild stertor and mild subcostal retractions. His stridor and stertor do appear to diminish when he is placed on his side or held in the prone position.

This example is classic for mild laryngomalacia. The child’s successful feeding and steady weight gain, as well as the absence of cyanotic episodes and sleep disturbance, are reassuring. If the diagnosis is in doubt, it may be confirmed in the otolaryngology office by FFL, and the patient can subsequently be followed in the outpatient primary care setting until the issue resolves with further growth and maturation. Conversely, poor weight gain and persistent airway distress should prompt further evaluation by the otolaryngologist.

**Case 2**

A 2-month-old girl is brought to clinic with 3 weeks of increasingly loud high-pitched breathing. This stridor had initially been associated with low-grade fever, cough, and rhinorrhea; however, these symptoms subsided, whereas the noisy breathing has continued to progress. She has already been taken to an urgent care clinic 2 weeks ago and treated with azithromycin and albuterol nebulizers. Her parents report a normal voice, snoring but no apnea, and no cyanotic episodes or respiratory distress. The remainder of her birth history, family history, and social history is unremarkable. Her physical examination reveals body weight at the 20th percentile, moderate generalized hypotonia, biphasic stridor that is more pronounced during the inspiratory phase, normal cry, and mild supraclavicular retractions. There are no notable skin lesions.

This case differs in several important ways from case 1. This patient’s stridor is of a different character (constant, biphasic), is delayed in its onset, and is progressive. She also has the additional finding of hypotonia. Given this constellation of symptoms, a central nervous system abnormality, such as Chiari malformation, should be high

Fig. 13. Tracheal stenosis due to complete tracheal rings. (A) Endoscopic appearance. (B) Three-dimensional CT reconstruction.
in the differential. Management options for her would include semiurgent outpatient referral to an otolaryngologist to include FFL, or inpatient admission for further monitoring during which otolaryngology consultation may be obtained.

Case 3

A 6-week-old baby boy is brought to clinic by his parents for concerns of poor feeding and weight gain. His prenatal course had been complicated by premature rupture of membranes, with delivery at 32 weeks’ gestation and subsequent 5-week stay in the neonatal intensive care unit. He had been intubated for 2 weeks after birth, before being extubated and weaned to room air. He was discharged without evidence of stridor, but has since had 2 episodes of croup that improved with courses of steroids. Even with treatment, however, his symptoms of noisy breathing and barky cough have never completely cleared. He has not gained any weight since leaving the hospital. Examination is significant for weight below the fifth percentile after adjusting for prematurity, mild subcostal retractions at rest, biphasic (predominantly inspiratory) stridor, and raised red lesions at the right mandibular angle and left parotid area.

Any newborn with a history of intubation and subsequent stridor, especially with barky cough, likely has fixed subglottic pathologic condition. The differential diagnosis in this case should include subglottic stenosis or subglottic cysts based on the history of prematurity and intubation, as well as subglottic hemangioma, given the prematurity and the skin lesions. All of these lesions present with a delay in onset, and none can be ruled out based on clinical presentation. In particular, the presence of hemangioma in the “beard” distribution increases the likelihood of such a lesion in the airway. Regardless of the precise origin of his subglottic lesion, this child has a history and physical examination inconsistent with benign laryngomalacia and should be referred for expedient otolaryngologist assessment. The fact that the child also has failure to thrive suggests that he would benefit from admission to the hospital, with inpatient otolaryngologic consultation at that time.

Case 4

A 2-month-old girl is evaluated in clinic for chronic cough, intermittent fevers, spit ups, and noisy breathing. She was born full term without complication, has no history of intubations, and has no significant family history. On further questioning, her cough is most severe during and after feeding. Her noisy breathing is intermittent, “panting” in nature, and is exacerbated by supine positioning, by feeding, and during crying. She has had several cyanotic episodes during which she appears to struggle to breath for intervals of approximately 10 seconds, and she additionally has pauses in her breathing during sleep. She has had poor weight gain and is below the fifth percentile for weight. On examination, she appears underweight, has moderately increased work of breathing, and has a low-pitched, sputtering stridor during expiration, with copious secretions. The remainder of her examination is remarkable for a systolic murmur.

In this case, the presence of failure to thrive and cyanotic spells already mandates urgent otolaryngology referral and/or inpatient admission. These symptoms coupled with (low-pitched) expiratory stridor, should raise concern for tracheal pathologic condition, such as primary tracheomalacia or extrinsic compression of the trachea. An H-type tracheoesophageal fistula can be associated with chronic aspiration as well as tracheomalacia in the absence of esophageal atresia, and might account for her expiratory stridor as well as her chronic cough and cyanotic episodes. Another diagnostic possibility is a vascular ring compressing the trachea that could additionally contribute to her systolic murmur. Appropriate consultations with otolaryngology
and pulmonology, operative endoscopy, and appropriate imaging should be ordered as an inpatient.

SUMMARY

Newborns with stridor who present to an outpatient pediatric office may be effectively evaluated, triaged, and diagnosed by applying history-taking principles, and a directed physical examination guided by a firm anatomic understanding of the origins of stridor. By recognizing the features that distinguish the various causes of stridor in the newborn, referrals for assessment may be appropriately triaged to either the emergency department or, less urgently, the office-based otolaryngologist.

REFERENCES