Approach to Common Chief Complaints

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Introduction

Some of the most frequent indications to perform airway endoscopy in children include noisy breathing, chronic wheezing unresponsive to therapies for asthma, and chronic cough [1, 2]. The decision to perform bronchoscopy is based on a combination of several factors, including history and physical examination findings, impact of the problem on the child's health and well-being, results of prior studies, anticipation of how the information gained will affect future care of the patient, and understanding of the natural history of the problem. For instance, flexible bronchoscopy might be delayed or avoided in an infant with intermittent vibratory stridor who is growing and developing normally, since the likelihood that the infant would outgrow the problem without intervention is high. In contrast, bronchoscopy is warranted in an infant with vibratory stridor who has feeding difficulty, poor growth, and episodes of apnea.

Several reviews report excellent diagnostic efficiency of flexible bronchoscopy [3–7]. When evaluating children with noisy breathing or wheezing, the examination involves anatomic or structural assessment as well as observation of airway dynamics [8]. The latter is state dependent; some problems arise only during sleep while others might occur only with exercise. Thus, accurate diagnosis requires a recognition of the conditions under which the problem exists and an understanding of how the airways behave under normal and pathologic conditions. Ideally, those conditions can be reproduced during the examination so that the cause of the problem can be identified. Equally as important, dynamic findings that do not correlate with the child's presentation can be ignored. For instance, dynamic collapse of supraglottic structures after anesthesia or after administration of topical lidocaine in a child with no history of stridor is most likely a reflection of the effect of the anesthesia and does not reflect a pathological condition [9–11]. Similarly, tracheal collapse noted endoscopically during coughing or crying in an infant with no history of wheezing reflects normal airway dynamics [12, 13] and should not be labeled as tracheomalacia.

Airway Dynamics: General Considerations

While the airways serve as a conduit for gas exchange between the atmosphere and alveoli, they are not rigid tubes: they are exposed to transmural pressures ($P_{tm} = P_{intraluminal} - P_{pleural}$ for

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intrathoracic airways and $P_{intraluminal} - P_{atmospheric}$ f or extrathoracic airways) that cause changes in length and width with each respiratory cycle (Fig. 16.1). Inside the thorax, intrapleural pressure becomes more negative during inspiration, causing a relative positive transmural pressure that results in lengthening and widening of the intrathoracic airways as a subject inhales. During exhalation, the pleural pressure is greater than intraluminal pressure and so the airways shorten and narrow. The opposite occurs above the sternal notch, in the extrathoracic airway. There, atmospheric pressure (considered 0 cmH_2O) is more positive than intraluminal pressure during inspiration, causing the extrathoracic airway to narrow during that phase of breathing. During exhalation, intraluminal pressure in the extrathoracic airway is higher than atmospheric, and so the airway dilates slightly. These relationships explain why signs and symptoms of extrathoracic obstruction are accentuated on inspiration, while those of intrathoracic obstruction are more prominent on exhalation.

The normal change in airway cross-sectional area that occurs with change in transmural pressure will also be accentuated if transmural pressure increases. Thus, if an infant uses abdominal accessory muscles to exhale because of peripheral airway obstruction, transmural pressure across the central airways will be more positive than at rest and the airway may appear collapsible. Alternately, if a subject is heavily sedated and breathes with a shallow pattern, transmural pressure across the airway wall will be minimized and significant collapse can be overlooked.

The relative change in airway caliber is determined not only by the direction and magnitude of the transmural pressure across it but also by the characteristic stiffness of the airway wall. The trachea and main bronchi are comprised of C-shaped cartilages whose tips are spanned by a membrane of contractile and connective tissue. The cartilage is fairly stiff, but the posterior membrane is not as stiff and can invaginate into the lumen or evaginate depending on the direction and magnitude of the transmural pressure. The pressure–volume

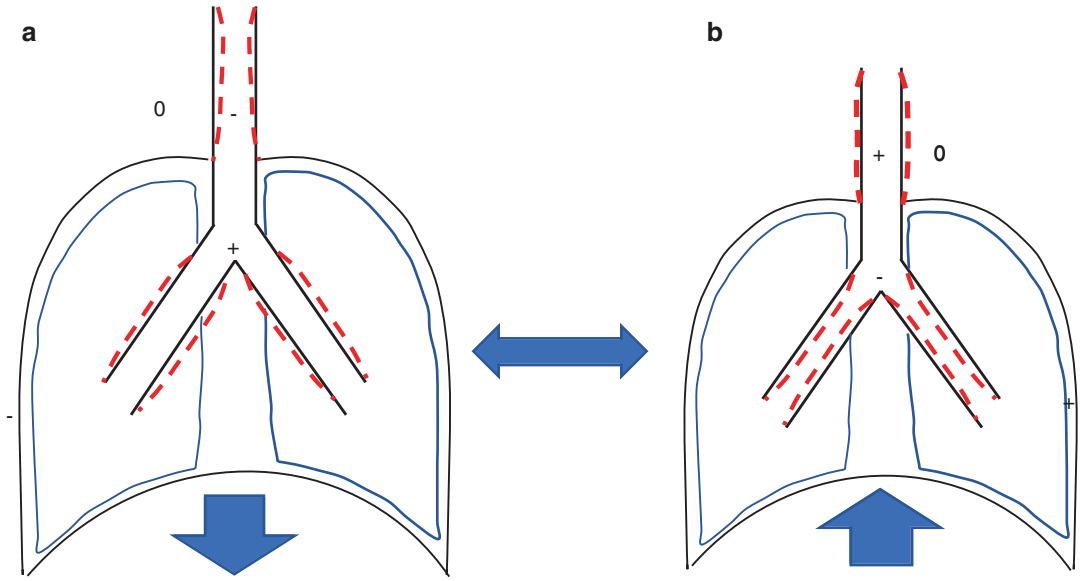

Fig. 16.1 (**a**) During inspiration, pleural pressure becomes more subatmospheric to draw air into the alveoli. Within the thorax, pleural pressure is lower than pressure within the airway lumen. As a result, transmural pressure (P_{TM}) acts as a distending force to dilate the intrathoracic airways. In the extrathoracic airway, intraluminal pressure falls below atmospheric pressure, so that P_{TM} favors narrowing of the extrathoracic airway. (**b**) During exhalation, pleural pressure becomes more positive than intrathoracic intraluminal pressure, resulting in narrowing of the intrathoracic airway. In the extrathoracic airway, however, intraluminal pressure is greater than atmospheric pressure, so that the extrathoracic airway dilates

relationship, or compliance of the airways changes with maturation. Tracheae from newborns are more compliant than those of infants and children, which in turn are more compliant than those of adults [14]. The increase in stiffness involves both the cartilaginous and contractile components of the airway wall [15, 16]. Thus, under normal circumstances, for the same change in transmural pressure the airway of a younger subject will have greater changes in cross-sectional area than that of an older one. The tone of airway smooth muscle also impacts on airway stiffness: contraction of the trachealis muscle will stiffen the airway and prevent collapse, while relaxation of airway smooth muscle can enhance the collapsibility of the central airway [17–19].

In addition to pressure across the airway wall, one other set of pressures that must be considered when assessing airway dynamics relates to the driving pressure necessary to move air from the atmosphere to the alveoli. That pressure must overcome frictional losses secondary to resistance through the airways, and it must also expand the elastic element of the respiratory system above its resting volume. The relationship between this driving pressure and the forces it must overcome is described by the Equation of Motion of the Respiratory System, which portrays the lung as an elastic–resistive series model, like a balloon attached to a straw (Fig. 16.2). The equation states that the pressure required to move air into the alveoli is determined by the sum of the product of the desired volume change (i.e., tidal volume) and magnitude of how much the respiratory system (lungs and chest wall together) resists the resulting stretch (that is, the *Elastance* of the respiratory system), together with the product of the flow rate of air and resistance to flow through the airways. A third "pressure cost" relates to acceleration of gas molecules down the airway, but this pressure is trivial at normal respiratory rates and can be ignored under normal breathing conditions for simplicity. Thus, the simplified Equation of Motion for a spontaneously breathing person is written as follows:

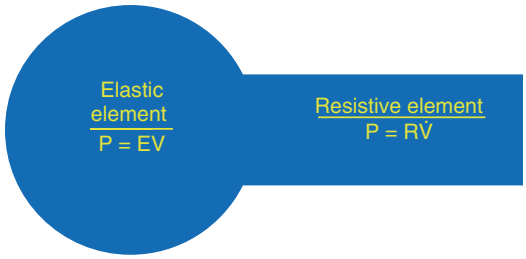

Fig. 16.2 The simplified Equation of Motion portrays the respiratory system in a series elastic model, akin to a balloon attached to a straw. The balloon represents the elastic component, while the straw is the resistive element. The pressure required to move air from the atmosphere into the alveoli is the sum of two products: the elastance (E) of the elastic component X the volume change (V) and airway resistance (R) X the flow of air through the airways ($\dot{V}$)

$$P_{mus} = EV + R\dot{V}$$

where P_{mus} is the pressure generated by the respiratory muscles, E is the elastance of the respiratory system, V is the desired volume change of the breath, R is the resistance through the respiratory system, and $\dot{V}$ is flow through the respiratory system. In other words, the equation states that there are two major loads in series, an elastic and a resistive one, that applied pressure must overcome to move air into the lungs.

In separating the different forces needed to inspire, the Equation of Motion also states that there must be a pressure difference down the airways in order to generate flow, and the pressure "cost" depends not only on how fast the air moves, but also on how much resistance in the respiratory system there is. Resistance describes frictional forces arising from both tissue movement and airflow through the airways. Frictional airway resistance occurs during breathing because of air molecules flowing through airways, and accounts for about 80% of total respiratory system resistance in adults [20]. Tissue resistance, which is usually a much smaller component of total respiratory system resistance, occurs because of displacement of tissues of the respiratory system during breathing.

When considering airway resistance, the relationship of individual airways to each other will influence the total airway resistance greatly. When airways are situated in parallel, as are small airways, individual resistances

down each airway are added reciprocally: $\frac{1}{R_{tot}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_n} \ldots$. Here, the resistance of all of the airways together is much smaller than the resistance of any single airway, so it would require an increase in resistance of many individual airways to increase the total resistance. In contrast, from the tip of the nose to the distal end of the trachea, resistances of intervening airway segments are arranged in series: that is, nasal, nasopharyngeal, oropharyngeal, hypopharyngeal, glottic, subglottic, and tracheal resistances are aligned one after the other. As such, those resistances are additive ($R_T = R_1 + R_2 + R_n \ldots$), so that each individual resistance is less than total resistance across that part of the airway. It also means that increasing the resistance in one part of that airway will directly increase total resistance (Fig. 16.3). Thus, increasing resistance in a proximal segment of the airway will require a greater total pressure drop to maintain flow, and so can exacerbate airway collapse in more distal segments. For example, an infant with moderate laryngomalacia can develop severe laryngeal obstructive symptoms when nasal congestion occurs because of the increase in nasal resistance: total respiratory system resistance increases, the intraluminal pressure drop across the nose will be greater, and simultaneously the infant will generate greater negative intrathoracic pressure to overcome that resistance. That combination will magnify the transmural pressure difference across the airway at the level of the supraglottis (as well as along all of the airway segments distal to the nose), favoring greater collapse of the supraglottic structures.

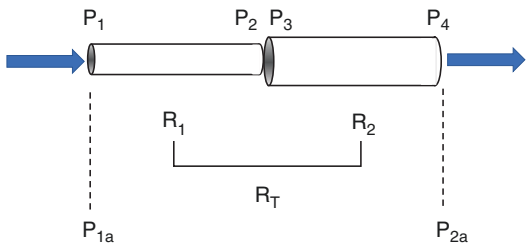

Fig. 16.3 In order for air to flow through an airway, pressure at the proximal end of the airway must be higher than pressure at the distal end. For a given flow rate, that pressure difference is determined by the resistance of the airway ($P1 - P2$ = Flow · Resistance). The larger airway in the diagram has a lower resistance across it than the smaller airway. If the two airways are placed in series, however, resistance across both airways will be greater than resistance through either one, so that the new proximal pressure (P_{1a}) will have to be higher than the proximal pressure through either of the individual airways (P_1 or P_3), and the pressure difference ($P_{1a} - P_{2a}$) has to be greater if flow is to remain constant

Airway Dynamics: Specific Considerations

The presence of airway narrowing that leads to stridor or wheezing often is state specific. When airway endoscopy is being considered, reproducing the conditions under which the noisy breathing occurs will increase the diagnostic yield and can make the difference between successfully determining the cause of the problem or not. For instance, adenotonsillar hypertrophy is considered to be the greatest risk factor for children to develop Obstructive Sleep Apnea (OSA) [21]. Nevertheless, an important mechanism for controlling patency of the pharynx is activation of the genioglossus muscle when intraluminal pressure becomes negative, but that reflex is lost or diminished in patients with obstructive sleep apnea [22]. Excessive sedation can result in collapse of the airway that might not be clinically relevant, but sedation titrated to effect can help identify the correct site of obstruction of the airway in patients with OSA [23]. At least 2 studies in children have shown that drug-induced sleep endoscopy has the potential to alter surgical approach based on findings of the studies [24, 25].

The state of the child during bronchoscopy is critical in interpreting findings of airway collapse. Airway caliber varies with respiratory cycle only slightly in a healthy infant breathing quietly, but the airway can narrow by as much as 50% if the infant cries or strains [26]. Similarly, small airway obstruction from bronchospasm or inflammation can produce cyclical intrathoracic

large airway dynamic collapse because the subject will increase pleural, and therefore transmural pressure to overcome the airway obstruction [26]. In adults, tracheobronchomalacia (TBM) is distinguished from this excessive dynamic airway collapse (EDAC) because TBM involves collapse of the cartilaginous portion of the airways, whereas EDAC refers to invagination of the pars membranacea (posterior membrane) while the cartilaginous rings maintain their shape [27]. Because the central airway is more compliant in infants than adults under normal circumstances, that differentiation may not be valid in infants and young children. These considerations are critical in the assessment of former preterm infants with severe bronchopulmonary dysplasia, who are at risk for central airway deformation and TBM because of exposure to positive pressure ventilation [28, 29], and who can also have severe small airway obstruction. Similarly, invagination of the posterior membrane during coughing is considered a normal finding during the maneuver [13, 30]. Excessive stimulation of the airway wall or inadequate topical anesthesia of the airway therefore can produce cough-induced airway collapse that could be misconstrued as abnormal.

Some conditions, like exercise-induced laryngeal obstruction, require replication of the stimuli that cause symptoms to yield the best chance of identifying a dynamic airway lesion [31, 32]. The larynx normally opens widely during exercise to increase flow across it while reducing resistance, but in some subjects, the larynx paradoxically narrows. Under resting conditions, there is usually no indication of an abnormality, and so ideally, laryngoscopy is performed under the same conditions that evince symptoms. Because there may also be a psychological component to laryngeal obstruction during exercise, a careful history must include all of the facts associated with exercise-induced dyspnea. One report, for instance, detailed a competitive swimmer who could cycle or run without difficulty, but upon entering a pool he would become dyspneic almost immediately [33]. Laryngoscopy during volitional hyperventilation was normal. When the patient was asked to hyperventilate while smelling chlorinated bleach, however, which simulated the odor of swimming in a chlorinated pool, he demonstrated paradoxical motion of the vocal cords almost immediately.

History

Certainly, all children who present with noisy breathing, chronic cough, or recurrent wheezing do not require bronchoscopic assessment. Some aspects of the history, however, can narrow the possible causes of a particular complaint, and contribute to the decision about the necessity for bronchoscopy. One group has created a mnemonic, SPECS-R, to determine the need for bronchoscopy in patients presenting with stridor (Table 16.1) [34]. Determining the cause of noisy breathing or wheezing from parental description of the sound is notoriously inaccurate [35–37], and physicians not specifically trained in airway disorders may also have difficulty characterizing the type of sound produced [38]. There are, however, other aspects of the history that can narrow the possible etiologies (Table 16.2). Broadly, the history designed to determine the cause of the problem includes timing, persistence, triggers, and predisposing factors for the problem. In addition to that information, details that would favor bronchoscopic evaluation include coexisting apnea, cyanosis, poor growth, and difficulty feeding [39].

Timing: Timing of symptoms includes age at which the problem began, whether the onset was abrupt or gradual, and whether the problem is

Table 16.1 Mnemonic for assessment of stridor [34]

S	*Severity* of airway obstruction according to parents' subjective impression
P	*Progression* of the obstruction
E	*Eating* or feeding difficulties, aspiration, failure to thrive
C	*Cyanotic* episodes, apneas, apparent life-threatening events
S	*Sleep* – obstruction so severe that sleep is disturbed
R	*Radiology* – specific abnormalities detected by radiographs

Table 16.2 Historical "2 Ts and 2 Ps"

Timing	Age at onset Abrupt or gradual
Triggers	Infectious or environmental factors Activities Sleep, eating, exercise
Persistence	Acute Chronic Intermittent Recurrent
Predisposing factors	Birth and obstetrical history Underlying conditions Prior surgeries

acute or chronic. Complaints that begin at or shortly after birth raise a concern for a congenital lesion of the airway [40]. Symptoms of congenital laryngomalacia, for instance, appear soon after birth. They worsen between 4 and 8 months, improve by 12 months, and typically resolve by 12–18 months [41]. Similarly, as many as 90% of airway hemangiomata present by 6 months of age [40, 42]. An infant who wheezes soon after birth is unlikely to have asthma but is more likely to have a lesion that causes airway narrowing from extrinsic compression, intraluminal obstruction, or abnormal airway collapsibility. Wheezing in an otherwise healthy toddler that develops abruptly and is accompanied by respiratory distress without a viral prodrome should raise concern for a retained foreign body. Some central airway lesions can be present but provide only subtle findings until the child acquires an acute respiratory illness, after which they become more clinically apparent. Inducible laryngeal obstruction, often referred to as Vocal Cord Dysfunction, does not typically occur in children younger than school age [43].

Persistence: Symptoms can be acute, chronic, persistent, intermittent, or recurrent. Lesions that cause intermittent symptoms probably result from dynamic airway narrowing rather than structural abnormalities. The intermittent nature of symptoms also can reflect severity of airway compromise. For instance, with minor degrees of airway narrowing, there may be no noisy breathing at rest, but, with increased effort, stridor can develop [39]. Recurrent problems can occur with viral illnesses or upon repeated exposure to an appropriate trigger but be absent during periods of wellness. The character of the persistence of symptoms often influences the need for or timing of bronchoscopy: since persistent chronic symptoms typically reflect a greater degree of airway narrowing, bronchoscopic evaluation is more likely to be considered.

Triggers: The most common trigger for infants and young children with recurrent wheezing is viral respiratory infection [44]. Viral upper respiratory infections can also exacerbate stridor or noisy breathing from any etiology because of the effect of mucosal edema and increased secretions on resistance throughout the extrathoracic airway. Similarly, infants with tracheobronchomalacia will have greater symptoms when any potential trigger results in an increase in expiratory effort, like crying or straining to pass a stool. Infants and toddlers who cough primarily during the act of drinking or eating, rather than after a meal, are at risk for swallowing dysfunction or a laryngeal cleft, or less commonly an H-type tracheoesophageal fistula. Exercise and emotional stress can be a trigger for inducible laryngeal obstruction, and the timing and duration of noisy breathing and associated dyspnea are distinct from those of exercise-induced bronchospasm [45].

Predisposing Factors: For children with noisy breathing, the search for predisposing factors often begins at birth. Clues to the etiology include information about the method of delivery and whether excessive traction on the neck was required. Presence of a shoulder dystocia would support this, although its absence would not preclude injury to the recurrent laryngeal nerve. Need for airway instrumentation and presence and duration of airway intubation would raise the concern for acquired glottic and subglottic lesions. A maternal history of perineal condylomata could help explain dysphonia or abnormal chest findings related to airway papillomas. Beyond a birth history, a history of prior neck or thoracic surgeries could point toward causes of stridor. Other known conditions that are associated with airway lesions, like Chiari

malformation (vocal cord paralysis), tracheoesophageal fistula with esophageal atresia (intrathoracic tracheomalacia), ventricular septal defect with large left to right shunt (left vocal cord paralysis and/or left main or lower lobe bronchus compression), all increase the risk of abnormal findings on bronchoscopy if the patient has stridor or wheezing.

Physical Examination

The physical findings that must be considered are directed toward the quality and characteristics of the abnormal sound, any associated changes to voice, clinical features that could predispose toward the problem, and the impact of the problem on the patient's breathing effort and overall growth and development. Together, these factors address the etiology and location of the problem as well as its impact on gas exchange.

In children with noisy breathing or recurrent wheezing, the type of noise that is generated reflects the site of obstruction (Table 16.3). With careful attention, the character of the noise can give important clues to the cause of noisy breathing or wheezing. Stridor reflects obstruction that is typically extrathoracic, and so it is usually an inspiratory sound because of the accentuated airway narrowing that occurs on inspiration in the extrathoracic airway. It can be bi-phasic when it is caused by a fixed lesion-like subglottic stenosis, when airway caliber does not vary with the phase of respiration. Stridor can be of varying pitch: some authors divide stridor into "voiced," describing a sound comprised of pure tones and overtones, and "fricative," referring to a noise-like sound [39]. Fricative stridor can be confused with stertor, a low-pitched, wet noise akin to snoring. Stertor is typically caused by obstructing lesions of the nasopharynx, oropharynx, and hypopharynx [34], although the quality of sound from those lesions has also been described as fricative stridor [39]. Pharyngeal-derived stertor occasionally can be biphasic. High-pitched, voiced stridor typically reflects lesions in the glottis or supraglottis, although laryngomalacia can also cause a low-pitched and fluttering stridor [34]. Longitudinal traction of the extrathoracic airway associated with neck extension will stiffen the airway to some degree, and so can make stridor related to extrathoracic tracheomalacia and laryngomalacia better. Conversely, neck flexion will exacerbate the stridor from those causes. A jaw thrust will ameliorate stridor or stertor related to glossoptosis and perhaps that due to hypopharyngeal hypotonia or pharyngomalacia. Similarly, prone positioning can improve the airway obstruction related to these problems as well as to laryngomalacia [46].

Wheezing is a musical sound that reflects intrathoracic airway obstruction. It occurs more commonly during exhalation because of the tendency for intrathoracic airways to narrow during that phase of breathing. It is caused by turbulent airflow through a narrowed airway; thus, there must be adequate flow to hear wheezes. Even when obstruction occurs primarily in small airways, there must be narrowing of medium-sized airways in order for wheezes to be generated. This can be the result of the same process that caused the small airway obstruction (bronchospasm, airway wall edema, secretions) or from dynamic compression resulting from increased pleural pressure generated to overcome the obstruction of the small airways. Infants and children with small airway obstruction often will breathe with a rapid and shallow breathing pattern, and wheezing can be overlooked unless the child is asked to breathe deeply and exhale forcefully. In subjects too young to follow such directions, the examiner can exert pressure on

Table 16.3 Noises, voice, and site of obstruction

Noise	Site
Snoring, gurgling	Pharynx, hypopharynx
High pitched	Supraglottic, glottic
Homophonous wheeze	Intrathoracic central airways
Heterophonous wheeze	Peripheral airways
Voice/cry	**Nasopharynx**
Hyponasal	Supraglottic
Muffled	Glottic
Hoarse/aphonia	Subglottic
Weak/soft	Intrathoracic
Normal	

the chest wall in the anterior–posterior direction in synchrony with an expiratory effort ("squeeze the wheeze"). When airway obstruction is caused by disease processes that affect small- and medium-sized airways, there are regional differences in the degree of airway narrowing. As a result, different sets of notes are generated in different regions of the chest. Thus, these "polyphonic" or "heterophonous" wheezes reflect wheezing that results from small or peripheral airway obstruction. In contrast, when obstruction occurs from a lesion in a central airway, the set of notes generated by that single obstruction will be the same throughout the chest, although their amplitude can vary depending on the distance away from the obstruction the observer listens. This type of wheezing is called "monophonic" or "homophonous" and reflects a large airway lesion like an endobronchial mass, airway compression, or tracheo- or bronchomalacia. Some authors refer to this type of wheezing as expiratory stridor, but that is a confusing term, which should be avoided. Every effort should be made by the examiner to determine whether wheezes are polyphonic/heterophonous or monophonic/homophonous, as the latter are much more frequently associated with lesions that should be evaluated bronchoscopically.

In addition to the character of the noise under investigation, alterations in voice can give important clues as to the level of obstruction. Hyponasal speech is associated with nasopharyngeal obstruction, like adenoidal hypertrophy. A muffled or "hot potato" voice reflects supraglottic obstruction like tonsillar hypertrophy or a supraglottic cyst. In contrast, patients with glottic obstruction like a glottic web or vocal cord paralysis can produce a hoarse voice or be aphonic. Children with subglottic lesions like subglottic stenosis will have a weak voice or a soft cry. Those children with intrathoracic lesions typically will have a normal voice.

Beyond findings related to the respiratory complaint under investigation, there may be other physical clues to the diagnosis or findings that predispose the patient to the respiratory difficulty under investigation (Table 16.4). Patients who present with stridor should have a careful examination of craniofacial structures that include patency of the nasal passages, assessment of the midface for malar flattening or "adenoidal facies," visualization of the oropharynx to rule out tonsillar hypertrophy, macroglossia, or a crowded oropharyngeal vault, and an assessment of the mandible to rule out micrognathia or retrognathia that could predispose to glossoptosis and upper airway obstruction. The examiner should perform a general evaluation for findings consistent with syndromes that are associated with airway obstruction. The skin should be examined for hemangiomata, as 50% of children with an airway hemangioma have a cutaneous lesion as well [47]. The association may be even stronger if the cutaneous hemangioma occurs in a "beard" distribution that includes the preauricular areas, lower lip, chin, and anterior neck [48]. The evaluation should also include an assessment of the child's resting tone, as infants with pharyngomalacia also often have generalized hypotonia and delayed motor development [34]. In children with chronic cough or recurrent wheezing, the presence of digital clubbing raises concern for a pyogenic process in the chest like bronchiectasis, and diseases like cystic fibrosis or primary ciliary dyskinesia should be considered.

Table 16.4 Physical examination: general considerations

Type of noise	Stridor, stertor, homophonous/monophonic wheeze, heterophonous/polyphonic wheeze
Phase of respiration	Inspiratory, expiratory, biphasic
Other findings	Craniofacial problems, syndromes, cutaneous hemangiomata, digital clubbing
Degree of distress	Retractions, accessory muscle use

Finally, there are physical findings that reflect the severity of obstruction and the impact of the respiratory problem on the overall status of the child. Intercostal, suprasternal, supraclavicular, and sternal retractions reflect a need for the child to generate increased negative intrathoracic pressure to achieve inspiration. This can be the result of decreased lung compliance or increased resistance anywhere along the airways, so that their presence alone does not distinguish between intrathoracic and extrathoracic disease. For

example, those retractions could be present in an infant with severe laryngomalacia or a child with bronchiolitis. Their presence and severity directly mirror the child's breathing effort and degree of respiratory distress. Subcostal retractions, however, reflect caudal displacement or flattening of the diaphragm because of hyperinflation, and their presence is associated with intrathoracic airway obstruction. Nasal flaring and head bobbing are signs of inspiratory accessory muscle use and they also reflect the child's degree of respiratory distress. Abdominal expiratory accessory muscle use signifies more severe intrathoracic airway obstruction; occasionally, it is accompanied by expiratory bulging of the intercostal or suprasternal spaces, reflecting the high pleural pressures being generated to overcome the obstruction and facilitate exhalation. If chronic airway obstruction is severe enough, the child may not be able to eat adequately. Additionally, use of accessory muscles increases the amount of work required by the muscles of respiration and the metabolic cost of breathing. The combination of decreased caloric intake and increased metabolic expenditure can lead to growth failure. When airway obstruction is severe enough to disrupt sleep and interfere with nourishment, it can also cause developmental delay. While these findings do not necessarily provide insight into the cause of a child's noisy breathing, wheezing, or chronic cough, they do reflect the severity of the problem and so contribute to the decision for and timing of airway endoscopy.

Conclusion

Airway endoscopy has become a powerful tool in the armamentarium of healthcare providers who care for infants and children with respiratory disorders. Nevertheless, it should be used selectively to minimize risks and cost of care. A careful history and physical examination can help the practitioner identify those problems that would be most amenable to bronchoscopic examination. Furthermore, understanding the conditions under which the respiratory abnormality occurs in a given patient can allow the endoscopist to reproduce or closely simulate similar conditions during the airway evaluation to enhance the diagnostic yield of the procedure. Importantly, an understanding of the physiology of dynamic airway mechanics during tidal breathing, forced exhalation, and cough can help the endoscopist distinguish between normal and abnormal phenomena.

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