CHAPTER 12

Clinical Assessment and Diagnostic Approach to Common Problems

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For millennia the mark of the healer or physician was the ability to discern the nature of a patient's illness through careful questioning, observation, and examination. However, over the past half century or more, practitioners have for a variety of reasons come to rely more and more on technologic means of diagnosis. Although always important, the art of physical diagnosis will likely reassume greater importance in clinical practice in the future because of the growing emphasis on cost containment and the likelihood of limited access to certain technologies. This chapter focuses on clinical assessment of the respiratory system in children. There is much overlap between the respiratory examination and that of other systems, and it is assumed that the reader has mastered basic physical examination skills. Several excellent resources for the general physical examination are listed in the references.[1,2]

HISTORY

The extent and focus of the history (and physical examination) are dictated by the patient's pressing complaint. With few exceptions, there is no such thing as a "routine history and physical," both those activities being tailored to fit the particular complaint that the patient has. An extended history may not be necessary in every case. For example, it would not be necessary to inquire into the stool characteristics of a patient presenting for evaluation of snoring. Careful attention should be paid to the patient's narrative, and probing, nonleading questioning and clarification of key points follow. The exact order of elicitation is not as important as a consistent general routine covering all aspects pertinent to the patient's complaint.

Most physicians begin with the history of present illness, although in younger pediatric patients, it may be appropriate to begin with the antenatal and birth histories. Often the first step is to elicit the chief complaint with an open-ended statement or question. It is generally better not to accept a diagnosis as the reason for seeking consultation. The clinician should insist on hearing the symptoms that promoted concern in the patient's own words. Obviously, information such as the circumstances at onset, frequency, duration, and severity is important. Adapting the mnemonic device PQRST (provocation/palliation; quality; radiation; severity; timing) may be helpful in systematically characterizing a symptom. Associated symptoms such as fatigue, exercise induction or intolerance, and viral syndrome are important to note as well. The results of prior evaluations should be solicited and every effort made to obtain the actual reports or images of previous procedures, including those from pulmonary function tests. Information about previous therapies used and the response or lack thereof can provide important clues as to possible etiologies and may allow an assessment of compliance as well.

The antenatal, birth, and neonatal histories in general should be reviewed; the detail necessary depends on the individual. The duration of the pregnancy, together with any complications, including maternal medications and substance use or abuse (as well as tobacco), should be noted. The circumstances of the delivery and the neonatal course should also be reviewed.

Previous respiratory problems, including previous respiratory illnesses, hospitalizations, and pulmonary injuries (e.g., chest trauma or surgery, smoke inhalation), should be explored in detail, especially as they relate to airway instrumentation (e.g., endotracheal intubation, bronchoscopy). A history of recurrent pneumonia may suggest immunodeficiency, cystic fibrosis, anatomic abnormality, dysfunctional swallowing, or bronchiectasis. The child with a history of tracheoesophageal fistula repair is prone to tracheomalacia and gastroesophageal reflux–related disease.[3] Survivors of adult respiratory distress syndrome initially have restrictive lung disease followed later by peripheral airway obstructive disease.[4,5] Evidence of atopy, such as eczema, atopic dermatitis, hay fever or known allergies, may be important in the child with chronic cough or difficulty controlling asthma. A history of frequent infections, blood product transfusion, parental substance abuse, or poor growth may be a clue to an underlying immunodeficiency. Risk factors for human immunodeficiency virus, both iatrogenic and behavioral, should be carefully explored because this virus is the most clinically relevant immunodeficiency in many countries.

The family history may provide valuable information. It is often fruitful to probe using a variety of terms; for example, *chronic bronchitis, wheezy bronchitis,* and *asthmatic bronchitis* are all frequently used to describe asthma. There may be terms in the local vernacular, especially in areas where segments of the population use traditional healers, with whom the practitioner should become familiar. It is also important to elicit a family history of illnesses unlikely in the child, such as a parent or grandparent with recent lung cancer, because this may disclose a cause of undue anxiety about a cough or another respiratory symptom.

The social history is always important, if for no other reason than it provides a better understanding of the patient's circumstances, potentially yielding information helpful in both making a diagnosis and planning therapy (e.g., assessing the likelihood of compliance problems). Specific items to be elicited include the makeup (number, age) of the household unit and the family's living arrangements (house, trailer, apartment). School or day-care attendance or child care arrangements should be reviewed, with attention paid to the environment there as well (see later section). Hobbies may also be important, especially those involving exposure to dusts, paints,

and other fumes. Even hair spray use can be clinically relevant. In a setting that preserves confidentiality, the clinician should discretely ask older children and adolescents about inhaled substances of abuse, such as tobacco, marijuana, and solvents (e.g., paint, glue, correction fluid). Of course, contact with ill individuals and travel are also pertinent.

A careful environmental history is important. The type of heating and cooling system in place should be noted. Other information such as the age of the dwelling, the presence of a basement, and recent renovations may also be useful. The number and type of animals present should be established. Many families do not consider animals kept outside, such as farm animals or birds, to be pets, so it may be better to ask about "animals" rather than "pets." It is important to inquire about exposure to potential irritants. The most common of these is smoke, either from tobacco use or use of wood for heating, cooking, or both. New composite furniture (manufactured from particle board and veneers), waterbeds, carpets, and ceiling tiles may contain volatile aldehydes that may incite asthma.

Often neglected in the pediatric patient, a review of systems can provide important information. Headache may be a sign of sinus disease or, especially if occurring in the early morning, a result of obstructive sleep apnea. Ocular symptoms such as conjunctivitis and blepharitis, as well as nasal symptoms, may indicate an atopic predisposition or in the infant a chlamydial infection. Recurrent mouth ulcers or thrush can be associated with immunodeficiency, as may chronic or recurrent ear drainage. Poor feeding, edema, shortness of breath, and exercise tolerance can be clues to the presence of congestive heart failure. Stool characteristics, abdominal bloating, and fatty food intolerance are important features of cystic fibrosis. Neurologic symptoms such as seizures or developmental delay are important in evaluating the child with apparent life-threatening events or suspected chronic or recurrent aspiration.

PHYSICAL EXAMINATION

This section focuses on the chest and respiratory system, with pertinent findings in other systems included as appropriate. For examination of other systems the reader is referred to one of the general physical examination texts listed in the references.[1,2] It is best to establish a consistent pattern for the physical examination so that part of it is not omitted. The order in which the components of the examination are presented here is arbitrary. At all times the privacy of the patient should be respected, the examination being conducted out of view (and preferably out of hearing) of other patients. In the case of adolescents the use of another staff member, the same gender as the patient, as a chaperon may be appropriate.

Upper Airway

Although not truly an airway or gas-exchanging tissue, the ear is considered part of the respiratory tract for several reasons. The middle ear and eustachian tube develop embryologically from the first pharyngeal pouch and share a contiguous mucosal surface with the respiratory tract.[6] The lining of the eustachian tube consists of ciliated pseudostratified columnar epithelium identical to the remainder of the respiratory tract.[7] Although the middle ear is lined predominantly with simple squamous or cuboidal epithelium, patches of ciliated pseudostratified columnar epithelium have been described there as well.[7] There are also cough receptors located in the external auditory canal. Thus it is important to examine the ears for foreign bodies and for signs of middle ear infection or another abnormality as a source of chronic cough.

The nasal passages are uniquely configured to perform their role as the portal for inspired air. The turbinates, and to some degree the paranasal sinuses, warm and humidify inspired air from ambient temperature and humidity to roughly body temperature and 100% relative humidity. Careful inspection of the nose can identify subtle changes indicative of local and sometimes systemic disorders. Children with inhalant allergies frequently develop a transverse nasal crease, the result of repetitive up-and-down rubbing to relieve itching and discomfort. This may be accompanied by other signs associated with allergic disease, such as dark circles under the eyes ("allergic shiners") and Dennie's sign, which is skin creases radiating from the inner canthus of the eye to approximately two thirds the length of the lower lid margin. The nasal bridge is normally straight. A deviation of the bridge may indicate a congenital abnormality or previous trauma and should prompt careful inspection of the septum for deviation and obstruction. Widening of the nasal bridge can be seen in individuals with extensive nasal polyps (Fig. 12-1). The relative patency of the passages can be assessed by asking the child to sniff (or simply listening in the younger child) while manually occluding one naris. A question of complete obstruction can be clarified by passage of a feeding tube or red rubber catheter. With congenital or acquired absence of the alar cartilage the nares may collapse with each inspiration.

The nasal passages themselves can often be visualized through the use of an otoscope and a large (4- to 5-mm) ear speculum by placing the free hand on the top of the patient's head and with the thumb gently lifting the tip of the patient's nose. Alternatively a nasal speculum can be used. The nasal mucosa, normally pink and glistening, should be inspected for edema and changes in color (inflamed or pale, boggy or gray), and the color, consistency, and odor of any secretions are noted. Obviously inflamed mucosa suggests infection, whereas pale, boggy mucosa is frequently seen in allergic rhinitis. With chronic rhinitis the mucosa may take on a grayish appearance. Foul-smelling and sometimes bloody secretions suggest a foreign body or chronic sinus disease, whereas clear secretions may occur in allergic rhinitis or early in the course of an uncomplicated upper respiratory infection. A smear of nasal secretions, stained with Hansel's stain, may be helpful, a predominance of eosinophils suggesting allergic disease and a predominance of polymorphonuclear leukocytes (especially when accompanied by a single bacterial morphology) suggesting bacterial sinus disease. No conclusions regarding the causative organism should be drawn from these results, however. The septum should be inspected for deviations, perforations, and sites of bleeding. (The nasopharynx is a common source of perceived hemoptysis.) Foreign bodies, polyps (see Fig. 12-1), and masses within the nares should be carefully sought out and inspected.

Examination of the paranasal sinuses is difficult in children younger than 10 years of age. Techniques such as transillumination and percussion not only are impeded by lack of cooperation but also may be difficult to interpret because of the relative thickness and density of the overlying soft tissues. However, it may be possible to localize the source of purulent secretions in the nose by direct inspection. Most commonly this is the middle meatus, which is located between the middle

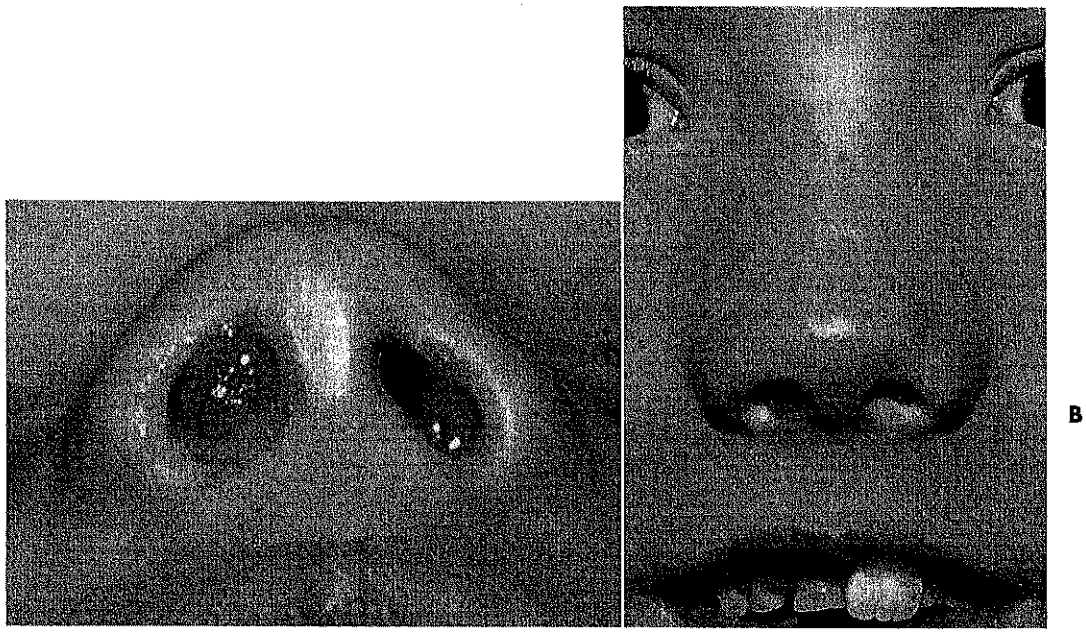

Fig. 12-1. A, Child with cystic fibrosis and a large nasal polyp. **B,** Note the widening of the nasal bridge and the polyp projecting from the right naris.

and inferior turbinates; the middle meatus drains the frontal, maxillary, and anterior ethmoid sinuses. The confluence of these three meatuses is called the *osteomeatal complex.* Obstruction from edema, a foreign body, or a polyp in this region is a frequent cause of chronic sinus disease. However, this may not be readily identified on examination; computed tomography (CT) is a more reliable means of diagnosis.

The profile of the mandible should be inspected carefully for the presence of retrognathia or micrognathia, either of which may lead to airway obstruction, especially during sleep. The state of oral hygiene, including not only of the teeth but also the oral mucosa, should be noted. The integrity of the palate should be ensured either by visualization or preferably by gentle palpation because a submucous cleft palate can easily be missed on simple inspection. The size and shape of the uvula is noted. A long uvula may cause chronic cough, whereas a bifid uvula may be a clue to an occult submucous cleft palate. The motion of the uvula and soft palate during phonation and gagging is important to note, especially in children with known neurologic abnormalities. Poor or abnormal motion may suggest palatal insufficiency or cranial nerve palsy that may be associated with dysfunctional swallowing and an increased risk of aspiration. The clinician should also note the presence and size of the tonsils as well as any other masses, especially unilateral enlargement, which can be seen in retropharyngeal or tonsillar abscess or lymphoma. Adenoidal tissue visible on the posterior pharyngeal wall ("cobblestoning") is abnormal and implies hypertrophy in association with allergic disease.

The presence or absence of foul breath should be noted. Fetid breath may indicate poor dental hygiene, a nasal foreign body, anaerobic infection, or even pneumonia.

The position of the trachea is important to note during examination of the neck. Deviation to one side may be associated with pneumothorax, neck mass, unilateral pulmonary agenesis or hypoplasia, or unilateral hyperinflation such as with foreign body or congenital cystic lung disorders. With the exception of unilateral pulmonary agenesis or hypoplasia, which causes deviation toward the involved side, the trachea is deviated away from the abnormality. The neck should be palpated for masses, thyromegaly, and adenopathy.

The character of the voice often provides important information as well. Hoarseness with or without stridor suggests an abnormality of the vocal cords such as edema, dysfunction (e.g., paresis, paralysis), or injury. A weak voice accompanied by a high-pitched inspiratory stridor but no hoarseness can result from a subglottic obstruction, whereas a muffled voice associated with a low-pitched stridor but no hoarseness suggests a supraglottic obstruction. Narrowing of the glottis itself results in hoarseness with a high-pitched stridor only on inspiration. Hoarseness or a muffled cry in a newborn is very suggestive of a congenital glottic or subglottic abnormality and should prompt further investigation, especially in infants at risk for laryngeal papillomatosis because of maternal genital papillomatosis.

Chest

Inspection

Examination of the chest, as with other areas, should begin with inspection. The general shape of the chest and the presence of any deformities are noted. The circumference of the chest, as measured at the nipple line, should be roughly equal to the head circumference in infants and is larger in older children. Barrel chest deformity, an increase in the anteroposterior dimension of the chest, is associated with obstructive lung disease. There is a good correlation between the degree of severity of this deformity and both increased lung volumes (functional residual capacity, residual volume, total lung capacity, functional residual capacity/total lung capacity, and residual volume/total lung capacity) and radiographic findings of hyperinflation in children with poorly controlled asthma.[8]

Asymmetry of the chest can be seen in children with cardiomegaly (especially with right-sided ventricular hypertrophy), pneumothorax, and scoliosis. Pectus carinatum (pigeon

breast) or pectus excavatum (funnel chest) can be present to a variable degree. The latter may falsely accentuate the severity or even mimic the presence of sternal retractions. Harrison's groove or sulcus, a horizontal depression in the lower thoracic cage at the site of diaphragmatic attachment, may be seen in patients who have chronically increased work of breathing, as in pulmonary fibrosis, cystic fibrosis, or poorly controlled asthma.

Work of breathing is assessed mainly through inspection. The respiratory rate, preferably noted with the child at rest or asleep, is a fairly sensitive clinical indicator of pulmonary health (Table 12-1). However, fever and metabolic acidosis can lead to an increased respiratory rate in the absence of pulmonary disease. Nasal flaring, an attempt to reduce nasal resistance to airflow, is a manifestation of increased work of breathing, as is the use of accessory muscles of respiration such as the sternocleidomastoid muscles. Retractions or indrawing of the skin of the neck and chest are signs of increased work of breathing as well. Areas of retraction include the suprasternal notch (suprasternal retractions), the subxiphoid region (infrasternal retractions), and the costal interspaces (intercostal retractions). In infants and toddlers the sternum itself draws in during inspiration, a manifestation of the increased chest wall compliance in this age group. Because of this, other sites of retraction may be absent in this age group, whereas in the older child, suprasternal and intercostal retractions predominate.

Children with evidence of increased work of breathing are said to have dyspnea, although complaints of shortness of breath are subjective and may not be related to a true respiratory pathologic condition. Children with neuromuscular disease, quadriplegia, paralyzed hemidiaphragm, and other such conditions may complain of dyspnea associated with metabolic acidosis or fever because of their inability to effectively increase their minute ventilation, the normal response in such a setting. Dyspnea may be semiquantitated by noting the number of words a child is able to speak before having to take a breath or by asking the child to count and noting the highest number reached. Both the use of accessory muscles and dyspnea correlate closely with lung function as measured by the 1-second forced expiratory volume and oxyhemoglobin saturation in children with acute exacerbations of asthma.[9]

The respiratory pattern may also provide valuable information. It is important to remember that the respiratory pattern is set by the respiratory centers in the brain stem. Changes in the pattern can reflect responses to oxygenation state, acidosis, or alkalosis or can indicate a primary abnormality of the respiratory centers themselves. The depth of respiration should also be noted. One author has suggested that each physician establish informal "norms" for depth of respiration in children of various ages by noting the distance from the nose at which the breath can be felt on the hand.[10]

Individuals with restrictive lung disease may have shallow, rapid respirations. Hyperpnea, rapid and deep respiration, can be associated with a number of underlying problems, including hypoxia and metabolic acidosis. Alkalosis may result in slow, shallow breaths. Biot's respiration, a pattern of very irregular respirations with alternating periods of hyperpnea and apnea, can be seen in meningitis, encephalitis, and other central nervous lesions involving the respiratory centers. Cheyne-Stokes respirations are a repetitive pattern of gradually increasing and decreasing respirations over 30 seconds to 1 minute and are generally associated with coma. The relative length of the respiratory phases (the inspiratory/expiratory ratio) is significant, with the inspiratory and expiratory phases normally being approximately equal. Prolonged expiration is seen in obstructive diseases such as bronchiolitis, acute exacerbations of asthma, and cystic fibrosis. Some degree of paradoxic respiration, or abdominal ("belly") breathing, may be normal, especially in children up to 6 or 7 years of age. Prominent respirations of this type in any child, however, generally reflect a pulmonary abnormality such as pneumonia, upper airway obstruction, or obstructive lung disease.

Table 12-1 Respiratory Rates of Normal Children

	SLEEPING		AWAKE	
AGE	MEAN	RANGE	MEAN	RANGE
Waring				
6-12 mo	27	22-31	64	58-75
1-2 yr	19	17-23	35	30-40
2-4 yr	19	16-25	31	23-42
4-6 yr	18	14-23	26	19-36
6-8 yr	17	13-23	23	15-30
AGE	BOYS	GIRLS		
Iliff and Lee				
0-1 yr	31	30		
1-2 yr	26	27		
2-3 yr	25	25		
3-4 yr	24	24		
4-5 yr	23	22		
5-6 yr	22	21		
6-7 yr	21	21		
7-9 yr	20	20		
9-13 yr	19	19		
13-14 yr	19	18		
14-15 yr	18	18		
15-16 yr	17	18		
16-17 yr	17	17		
17-18 yr	16	17		

Adapted from Waring WW. In Kendig EL, Chernick V, eds: *Disorders of the respiratory tract in children*, Philadelphia, 1983, WB Saunders, pp 57-78; and Iliff A, Lee VA: *Child Develop* 23:237-245, 1952.

Palpation

Although more generally thought of in terms of the abdominal examination, palpation is important in the respiratory examination as well. It is used to confirm the visual observations of chest wall shape and excursion. Palpation is performed by placing the entire hand on the chest and feeling with the palm and fingertips. Friction rubs may be felt as high-frequency vibrations in synchrony with the respiratory pattern. Tactile fremitus, the transmission of vibrations associated with vocalization, is at times difficult to assess in children because of a lack of cooperation and a higher-pitched voice; lower-pitched vocalization is more effectively transmitted. It is best felt with the palmar aspects of the metacarpal and phalangeal joints on the costal interspaces. Decreased fremitus suggests airway obstruction, pleural fluid, or pleural thickening, whereas increased fremitus is associated with parenchymal consolidation. Occasionally a "thud" can be felt high in the chest or in the neck, a finding suggestive of a free tracheal foreign body. One can also assess chest excursion by placing the hands with the fingertips anterior and thumbs posterior and noting the degree of chest wall movement, comparing excursion of one side with the other by noting the movement of the thumbs away from

the midline (the spinous processes). The point of maximal impulse, frequently shifted to the left in cardiac disease, may be shifted to the right in severe asthma, a large left-sided pleural effusion, or a tension pneumothorax. With massive left-sided atelectasis, it may be shifted to the left.

Percussion

Much like its counterpart in the musical world, percussion of the chest relies on differences in vibratory characteristics, in this case using various tissues, to produce characteristic sounds. First described by Leopold Auenbrugger in Vienna in 1761, the technique was largely ignored by the medical community until around the turn of the next century, when it was revived by Napoleon's personal physician, Corvisant. It is widely thought that Auenbrugger adapted the technique from that used by his innkeeper father to determine the level of wine in barrels, though it is not known for certain how Auenbrugger developed the idea. There are two different methods of performing percussion: direct (or immediate), in which the chest is struck directly with the finger, and indirect (or mediate), in which sound is generated by striking a finger laid on the chest. This discussion involves the indirect method only. A discussion of direct percussion can be found elsewhere.[11]

Correct technique is critical in both performing and interpreting percussion of the chest, especially in small children. Percussion is best performed with the child upright with the head in a neutral position. A single finger from one hand (the pleximeter) is placed on an interspace; care is taken to avoid contact of the other fingers and palm with the chest because contact between the chest and any other part of the nonstriking hand dampens the sound generated and leads to erroneous interpretation. The finger is then struck with a single finger from the other hand (the plexor) by holding the hand fixed and pivoting at the wrist, quickly removing the striking finger, again to avoid dampening the sound. Many examiners find it comfortable to use the long fingers of each hand for this technique. Generally the clinician strikes 2 to 3 times in each position. The force used should be consistent with each strike and should not be too strong. Excessive force may lead to an erroneous impression of hyperresonance, especially in a small child. Some have suggested the use of a reflex hammer as the plexor; this should not be done in children because it may lead to increased resonance. Sounds commonly elicited by percussion of the chest are listed in Table 12-2.

The clinician can delineate the level of the diaphragmatic leaves anteriorly and posteriorly by carefully percussing along the lower thoracic cage (Table 12-3). This can be helpful in guiding auscultation. The clinician may even be able to assess diaphragmatic excursion in older children and adults with suspected diaphragmatic dysfunction by percussing during inspiration and expiration; in adults this is normally 5 to 6 cm. The extent of the mediastinal structures can also be delineated.

Auscultation

After development of the stethoscope by Rene Laennec in 1816 and its improvement by Piorry, Williams, Cammann, and others, physicians had the ability to recognize changes in sound characteristics in the chest and to correlate these changes with specific pathophysiologic events in health and disease. (An excellent review of the history and physics of the stethoscope is available.[12]) Although the standard stethoscope does not amplify sound, by excluding extraneous environmental sounds and to some degree localizing sounds, it allows the clinician to assess gas movement within the lungs and relate changes to known associations with specific abnormalities. Thus developing expertise in interpreting auscultatory findings is very much an experiential process, and as such, there is no substitute for having listened to a large number of patients, both with and without lung disease. Audiotape programs, such as one available from the American College of Chest Physicians,[13] can be helpful in establishing a base on which to build this skill.

Table 12-2 Sounds Elicited by Percussion of the Chest

TERM	DEFINITION
Tympany	This sound is usually heard only in the abdomen; massive pneumothorax is suggested if it is heard in the chest.
Hyperresonance	This sound is associated with emphysema or free intrapleural air.
Bellmetal resonance	Also called the *coin test,* this is a clearly transmitted metallic sound heard with a stethoscope when tapping a coin that is held flat against the chest with another coin; it indicates a pneumothorax.
Skodiac resonance	This peculiar, high-pitched sound is obtained by percussion just above the level of a pleural effusion.
Resonance	This is the normal state in the chest; it is sometimes called *vesicular resonance.*
Dullness	This sound is associated with pleural fluid or parenchymal consolidation.
Flatness	This sound can be heard by percussing over muscle; its presence in the chest suggests massive effusion.

Table 12-3 Usual Level of Diaphragm as Assesssed by Percussion

TERM	LEFT	RIGHT
Anterior	Ribs 8-10	Ribs 6 (midaxillary line)
Posterior	Ribs 8-10	Ribs 8-10

For most physicians the standard binaural stethoscope is adequate as long as it is in good repair. The earpieces should fit well to exclude environmental sounds. The tubing should not be cracked or kinked and ideally should be no longer than 30 cm, although many physicians accept longer lengths for ease and comfort in examining patients. The bell should be fitted with a rubber ring, and the diaphragm should be intact. Pulmonologists may find it more convenient to use a differential stethoscope, a stethoscope with two chest pieces, one connected to each earpiece, allowing simultaneous auscultation and direct comparison of sounds in different locations. However, use of the differential stethoscope requires even more practice than the standard binaural stethoscope for effective use, so it is probably not practical for the general pediatrician or family physician.

The diaphragm, which filters out low-pitched sounds, thereby isolating high-pitched sound, should be pressed tightly against the skin. In contrast, the bell should be placed lightly on the skin to preferentially isolate low-pitched sounds. If excessive pressure is applied when using the bell, the skin below the bell may be stretched taut, thereby functioning as a diaphragm and filtering out the low-pitched sounds being sought. A loud, roaring sound generally indicates inadequate contact between the chest piece and skin, especially when the bell is

used. This can be especially problematic when examining an infant or small child unless a stethoscope with appropriate-sized chest pieces is used. Instruments with chest pieces appropriate for premature infants, infants, children, and adolescents and adults are available. The clinician should avoid listening through clothing or bed clothes and should listen (if possible) with the patient breathing slowly and deeply through the mouth in a neutral position, either upright or prone or supine.

As always, it is best to develop a consistently used pattern of examination to avoid missing areas (Fig. 12-2). The upper lobes are best heard by listening anteriorly in the infraclavicular regions, the lower lobes by listening posteriorly below the scapulae, and the right middle lobe and lingula by listening anteriorly lateral to the lower third of the sternum. All lobes can be heard in the axillae.

When auscultating, the clinician should note the amplitude of the sounds produced. It is also important to specify the timing (continuous, early, or late), pitch (high, medium, or low), and character (fine, medium, or coarse) of sounds. These sounds can be divided into breath sounds (produced by the movement of gas through the airways), voice sounds (modifications of phonation not heard distinctly in the normal state), and adventitious sounds (neither breath or voice sounds). Table 12-4 lists the most commonly heard sounds.

Breath Sounds. Vesicular breath sounds are the sounds heard during respiration in a healthy individual. They have a low-pitched, "whishing" quality with a relatively longer inspiratory phase and a shorter expiratory phase and are louder on inspiration. These sounds emanate from the lobar and segmental airways and are then transmitted through normal parenchyma.[14]

Bronchial breath sounds are usually louder than vesicular sounds and have short inspiratory and long expiratory phases. They are higher pitched and louder during expiration. They may be the result of consolidation or compression (i.e., airlessness) of the underlying parenchyma. A similar sound can be heard by listening directly over the trachea.

Bronchovesicular breath sounds, as the name implies, are intermediate between vesicular and bronchial sounds. The respiratory phases are roughly equal in length. This sound is felt to be indicative of a lesser degree of consolidation or compression (airlessness) than bronchial sounds. Bronchovesicular (and sometimes bronchial) breath sounds can occasionally be heard in normal individuals in the auscultatory triangle (the area in the back bound by the lower border of the trapezius, the latissimus dorsi, and the rhomboideus major muscles) and the right upper lobe.

Wheezes are continuous musical sounds, more commonly expiratory in nature, and usually associated with short inspiratory and prolonged expiratory phases. They can be of single (monophonic) or multiple (polyphonic) pitches, which are higher pitched than vesicular sounds. These can often be very difficult to distinguish from snoring and upper airway sounds such as stridor.

Stridor is a musical, monophonic, often high-pitched sound, usually thought of as inspiratory in nature; it can be expiratory as well, produced by partial obstruction of a central, typically extrathoracic airway. Its presence in both inspiration and expiration suggests severe, fixed airway obstruction.

A cardiorespiratory murmur is a localized vesicular sound that appears to be synchronized with the heartbeat, mimicking a cardiac murmur or bruit. It can be heard anywhere in the

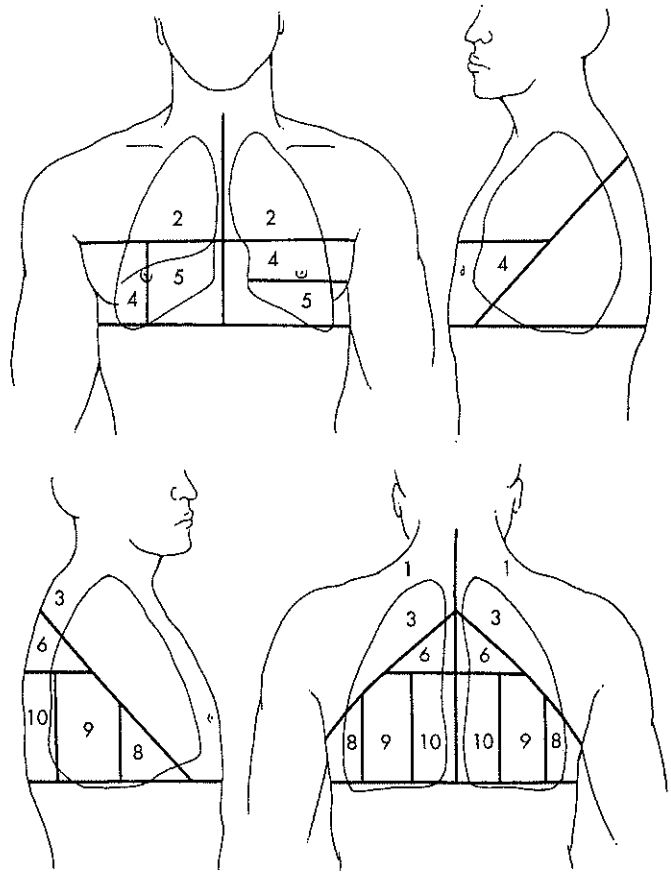

Fig. 12-2. Projections of the lobar/segmental pattern on the surface of the chest. *1,* Apical segment of the upper lobes; *2,* anterior segment of the upper lobes; *3,* posterior segment of the upper lobes; *4,* superior lingular *(left)* and lateral *(right)* segments of the middle lobe; *5,* inferior lingular *(left)* and medial *(right)* segments of the middle lobe; *6,* superior segment of the lower lobes; *8,* anterior basal segment of the lower lobes; *9,* lateral basal segment of the lower lobes; *10,* posterior basal segment of the lower lobes.

Table 12-4 Pulmonary Auscultatory Sounds*

COMMON TERMINOLOGY	ACCP-ATS "PREFERRED" TERMINOLOGY
Breath sounds	**Breath sounds**
Vesicular sounds	Normal
Bronchial sounds	Decreased
Bronchovesicular sounds	Absent
Wheeze	
Stridor	
Cardiorespiratory murmur	
Voice sounds	
Whispered pectoriloquy	Clarity increased or decreased
Bronchophony	Intensity increased or decreased
Egophony	
Adventitious sounds	**Adventitious sounds**
Fine (subcrepitant) crackles or rales	Crackles or rales (no subclassifications)
Coarse (crepitant) crackles or rales	Wheezes or rhonchi (varying pitch, quality, intensity)
Rhonchi	Mediastinal crunch
Squawk	Pleural rub
Pleural friction rub	Pleuropericardial rub
Peristalsis	

ACCP, American College of Chest Physicians; *ATS,* American Thoracic Society.
*See text for description of terms.

chest but is frequently very dependent on body position, often disappearing with position change. It may be heard in systole, diastole, or both during quiet respiration.

Voice Sounds. The normal lung parenchyma filters vocalization so that whispered sounds are not usually heard during auscultation and normally spoken syllables are indistinct. Bronchophony is the distinct transmission of spoken syllables as the result of an underlying consolidation or compression. More severe consolidation or compression results in the transmission of whispered sounds or whispered pectoriloquy. Egophony is very similar to bronchophony but has a nasal quality as well. It may reflect an underlying effusion, consolidation or compression, or both conditions.

Adventitious Sounds. The nomenclature for adventitious sounds is perhaps the least standardized of all physical findings and therefore is prone to confusion. Synonymous terms such as *rales* and *crackles, subcrepitant* and *fine,* and *crepitant* and *course* are widely used in a variety of combinations. Because past attempts at standardization have met with variable success,[12,15] the authors have chosen to identify these sounds using several descriptors, allowing the reader to choose which to use and hopefully allowing him or her to recognize others when used by colleagues.

Fine (subcrepitant) crackles are thought to be the result of the explosive reopening of alveoli that closed during the previous exhalation or exhalations.[16] These occur exclusively during inspiration and are associated with conditions such as bronchitis, pneumonia, pulmonary infarction, and atelectasis. They can also be normal when heard in the posterior lung bases during the first few breaths on awakening. They may be imitated by rolling several strands of hair between the thumb and forefinger in front of the ear or by pulling apart Velcro. Hamman's sign, also called a *mediastinal crunch,* is the finding of crackles associated with systole and is suggestive of pneumomediastinum.

Coarse (crepitant) crackles are popping sounds likely produced by the movement of thin fluids in bronchi or bronchioles.[16] They occur early in inspiration and occasionally in expiration as well, may be audible at the mouth, and may clear or change pattern after a cough. They can sometimes be heard in the anterior lung bases during exhalation to residual volume. An example of these sounds are the crackles typically heard in patients with cystic fibrosis.

Rhonchi (sometimes more descriptively called *large airway sounds*) are gurgling or bubbling sounds usually heard during exhalation. These sounds are the result of movement of fluid within larger airways.

A squawk is a short inspiratory wheeze often heard in association with fine crackles. It is thought to result from the explosive opening and fluttering of a large airway.

In individuals with decreased or absent pleural lubricating fluid, a pleural friction rub may be heard. This loud, grating sound may come and go over a short period of time. It is usually associated with a subpleural inflammatory process.

Finally, peristalsis may sometimes be heard within the thorax, especially over the left lung base because of the proximity of the stomach and large bowel. The clinician must be alert to the possibility of acquired or congenital diaphragmatic hernia.

Other Signs and Symptoms

Occasionally, pulmonary disease is manifest by changes or signs in other organ systems. An example is digital clubbing, the broadening and thickening of the ends of the fingers and toes that occur as the result of connective tissue hypertrophy and hyperplasia[17] and increased vascularity[18] in the distal phalanges (Fig. 12-3). It may be quite subtle but can be confirmed clinically by checking for Schamroth's sign (Table 12-5). Although clubbing can be a primary finding (either idiopathic or inherited), it is usually seen in association with lung disease, heart disease, or liver or other gastrointestinal diseases as well (Box 12-1). The degree of clubbing can be quantitated by several methods as a way of following the progression of lung disease.[19,20] Clubbing may occur acutely (e.g., with a bout of severe pneumonia) but may also regress if the underlying cause is corrected. When associated with a usually painful periostosis, clubbing is one component of hypertrophic osteoarthropathy.

The pathophysiology of clubbing associated with lung disease is unclear. It may be the result of the lungs' failure to remove or inactivate a circulating fibroblast growth factor,[21] although the arachidonic acid metabolites prostaglandin F_{2a} and E have been implicated in patients with cystic fibrosis.[22] Still another theory proposes that clubbing is the result of peripheral impaction of megakaryocytes and platelets in the digits, with subsequent release of platelet-derived growth factor, which induces the histologic and anatomic changes associated with clubbing.[23]

Cyanosis, another abnormality that may be associated with lung disease, is the bluish discoloration of tissues caused by increased concentrations of reduced (unoxygenated) hemoglobin, which is purple (Box 12-2). It occurs more readily in tissues with low blood flow or higher oxygen extraction than tissues with higher flow or lower oxygen extraction. This accounts for the traditional interpretation that peripheral cyanosis (or acrocyanosis) reflects less severe hypoxemia than central cyanosis.

The use of cyanosis as a clinical indicator of hypoxemia is confounded by a number of factors. Simply identifying the cyanotic patient can be problematic because of variations in skin pigmentation, poor lighting, the presence of nail polish, or temperature extremes (especially cold). Even when cyanosis is unequivocally present or absent, inferences made regarding the oxygenation state of the patient may not be correct. Cyanosis occurs when the concentration of reduced arterial hemoglobin exceeds 3 g/dl. At this level the concentration of reduced hemoglobin in the capillary beds is generally 4 to 6 g/dl. However, the blood's oxygen-carrying capacity and therefore blood oxygen content depend primarily on total hemoglobin concentration. Thus the actual oxygen content may be normal in a cyanotic patient with polycythemia, but an anemic patient may have an abnormally low oxygen content in the absence of cyanosis. Clinical impressions of oxygenation, such as cyanosis, should therefore be verified by arterial blood gas analysis.

Pulsus paradoxus is another physical sign sometimes associated with pulmonary disease, particularly obstructive lung disease. Pulsus paradoxus is the fluctuation in arterial systolic blood pressure with the respiratory cycle, the pressure falling during inspiration and rising with exhalation. It is quantified as the difference between the systolic pressures measured during inspiration and expiration. It can be measured a number of ways, most easily by using a sphygmomanometer. It can also be qualitatively identified by observing the pressure tracing of an intraarterial catheter or the pulse tracing of a pulse oximeter. It may also be detected by palpation in patients with pulsus paradoxus greater than 20 mm Hg, signifying more severe obstructive lung disease (see later section).

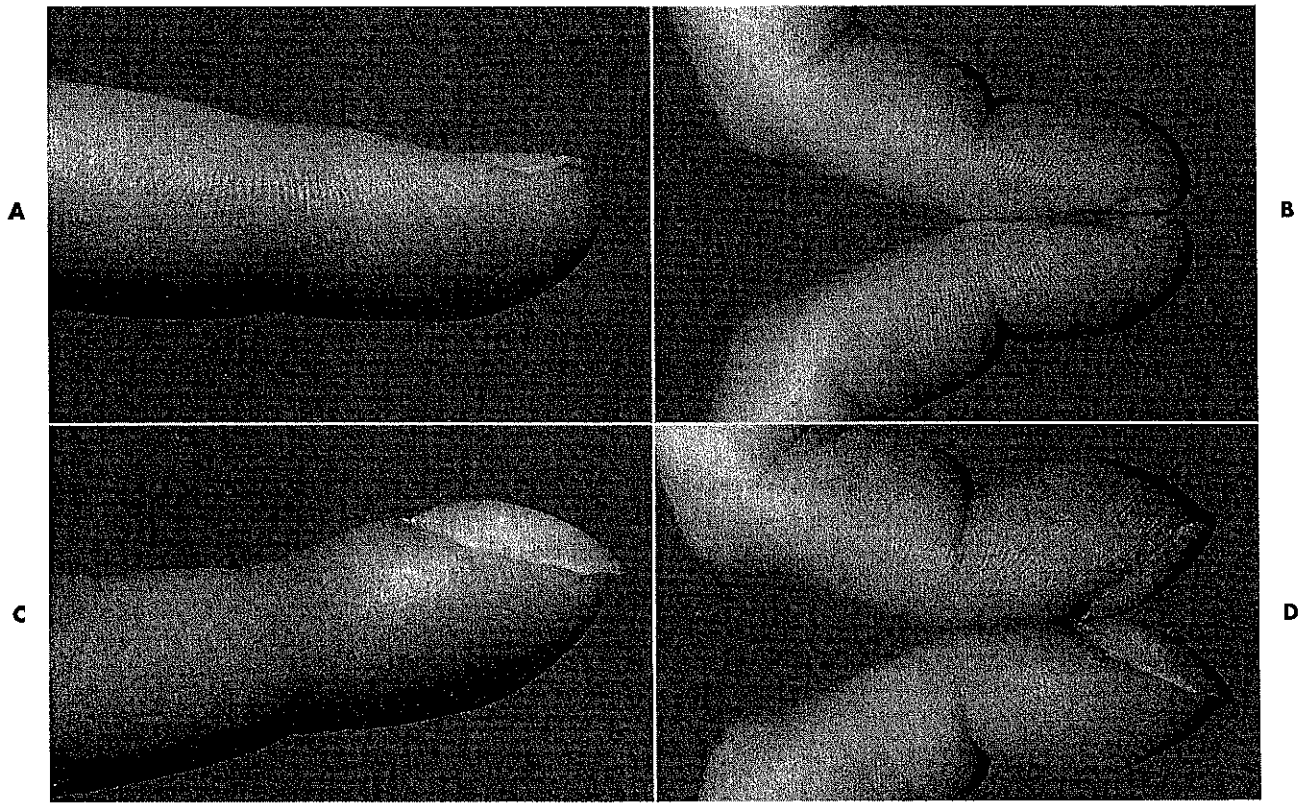

Fig. 12-3. Lateral view of the index finger and Schamroth's sign in a healthy individual (**A** and **B**) and in an individual with severe clubbing (**C** and **D**).

Table 12-5 Pulmonary "Signs"

SIGN	DEFINITION
Abrahams'	Rales and other adventitious sounds, changes in respiratory murmurs, and an increase in whispered sounds can be heard on auscultation over the acromial end of the clavicle for some time before they become audible at the apex.
Aufrecht's	Diminished breath sounds occur in the trachea just above the jugular notch in cases of tracheal stenosis.
Baccelli's	There is good conduction of a whisper in nonpurulent pleural effusions.
Bird's	There is a zone of dullness on percussion with the absence of respiratory signs in the presence of a hydatid cyst of the lung.
di'Espine's	In pulmonary tuberculosis, bronchophony over the spinous processes is heard at a lower level than in healthy people.
Ewart's	In large pericardial effusions, an area of dullness with bronchial breathing and bronchophony is found below the angle of the left scapula.
Ewing's	Dullness on percussion to the inner side of the angle of the left scapula denotes an accumulation of fluid in the pericardium behind the heart.
Fischer's	In a case of tuberculosis of the bronchial glands, if one bends the child's head as far back as possible, auscultation of the manubrium sterni sometimes reveals a continuous loud murmur caused by the pressure of the enlarged glands on the vena anonyma.
Hamman's	Crackles associated with systole is suggestive of pneumomediastinum; this sign is also called the *mediastinal crunch.*
Hoover's	A modification in the movement of the costal margins during respiration is caused by flattening of the diaphragm; this sign suggests emphysema or another intrathoracic condition causing a change in the contour of the diaphragm.
Jackson's	During quiet respiration, the movement of the paralyzed side of the chest may be greater than that of the opposite side, whereas in forced respiration, the paralyzed side moves less than the other.
Lorenz's	This sign is stiffness of the thoracic spine in early pulmonary tuberculosis.
Perez's	Rales are audible over the upper part of the chest when the arms are alternately raised and lowered; it is a common occurrence in cases of fibrous mediastinitis and aneurysm of the aortic arch.
Rotch's	Percussion dullness occurs in the fifth intercostal space on the right side in cases of pericardial effusion.
Schamroth's	In patients with clubbing, there are loss of the normal diamond-shaped aperture at the base of the nails and an increased angle at the nail tips when the dorsal surfaces of the terminal phalanges are approximated.
Skoda's	Skodaic resonance occurs.

The pathophysiology of pulsus paradoxus is likely multifactorial.[24] With wider swings in intrathoracic pressure associated with airway obstruction, there is a wider gradient between pressure within the intrathoracic and extrathoracic arterial vessels. Thus the left ventricle must generate increased force to keep the arterial pressure relatively constant. Because the ventricle does not do so in an instantaneous fashion, there is a drop in arterial pressure. The wider swing in intrathoracic pressure also results in greater right ventricular filling pressure, leading to increased right ventricular end-diastolic volume and displacement of the ventricular septum leftward. This reduces left ventricular filling, thereby reducing stroke volume and further decreasing arterial pressure during and immediately after inspiration.

BOX 12-1
Digital Clubbing

- Intrapulmonary shunting and inflammation
  - Bronchiectasis
  - Severe pneumonia, lung abscess, or empyema
  - Interstitial lung disease (autoimmune and infectious)
  - Pulmonary arteriovenous malformation
  - Hepatopulmonary syndrome
  - Pulmonary malignancy
- Cardiac and cardiovascular causes
  - Cyanotic congenital heart disease
  - Bacterial endocarditis
- Noncardiopulmonary causes
  - Inflammatory bowel disease
  - Thyrotoxicosis
- Familial

BOX 12-2
Cyanosis

- **Central cyanosis**
- Arterial hypoxemia
- Normal levels of arterial oxygen
  - Hematologic causes
    - Methemoglobin
    - Other hemoglobinopathies
  - Vascular cause
    - Superior vena caval obstruction
- **Peripheral cyanosis**
- Vascular causes
  - Peripheral cyanosis resulting from vasomotor instability or hypothermia
  - Venous obstruction
  - Shock or hypoperfusion with venous stasis
- Hematologic cause
  - Polycythemia

Pulsus paradoxus is useful in evaluating children with cystic fibrosis[25] and asthma, in which pulsus paradoxus of more than 15 mm Hg has been found to correlate with a 1-second forced expiratory volume of less than 60% of the predicted value.[26] It should be noted that the levels of pulsus paradoxus commonly seen with obstructive lung disease are much higher than those seen in individuals whose cardiac tamponade is the etiology of pulsus paradoxus.

EVALUATION OF THE CHILD WITH CHRONIC COUGH

Physiology

Cough is an extremely important component of pulmonary host defense. When functioning effectively, it clears bulk material from the airway. In patients with impaired mucociliary clearance either from acquired or congenital abnormalities of ciliary function or other mechanical factors, cough may be the only airway clearance mechanism available. The loss of effective cough in patients with advanced neuromuscular or neurologic disease is a critical factor in the morbidity and mortality of those disorders.

Although a seemingly simple action, cough is actually a very complex reflex involving afferent pathways in the vagus and efferent pathways in the somatic nervous system. Cough can be produced or suppressed volitionally, although it is not always completely suppressible. Although their existence has not yet been confirmed histologically (only inferred by physiology and suggested by electron micrographic studies), cough receptors are thought to be fairly widely distributed in the respiratory tract. They are found predominantly in the extrapulmonary airways (larynx, trachea, mainstem bronchi) but are also present in the external auditory canals, tympanic membranes, upper airway, pleura, pericardium, and diaphragm. Few, if any, are found in the lung parenchyma itself.

The sequence of events associated with a cough are well described. The initial phase consists of opening of the glottis and a short inspiration, which increases lung volume for the next phases. The glottis then closes, and the chest wall, abdominal, and perineal muscles contract, generating high intrathoracic and transpulmonary pressures. With the sudden opening of the glottis, there is rapid decompression of the airway with a high-velocity expulsion of gas and movement of airway contents (e.g., secretions and other solid material) proximally. In smaller airways the intrathoracic pressure generated may lead to airway closure, trapping some material distally. Thus cough primarily clears the larger, more central airways. Recognition of this phenomenon has led to alternative methods of airway clearance, such as autogenic drainage[27] and the use of positive expiratory pressure and flutter valve devices,[27,28] which are thought to be more effective at clearing the smaller, more distal airways.

Movement of material as the result of coughing occurs by three mechanisms. First, the high-velocity airflow results in a wavelike gas or liquid pumping of the mucous blanket and movement of loose mucus and other material. The increase in intrathoracic pressure causes airway compression, which squeezes some material proximally into larger airways. This is especially important peripherally, where gas velocities are insufficient to propel mucus. Finally, the vibration of the airway walls and the shearing force of the high-velocity gas flow dislodge mucus from the wall. The sounds produced by coughing are the result of the vibration of secretions and nonrigid respiratory structures.

In contrast to the beneficial airway clearing effects of cough, there are a number of potential deleterious effects as well. Extremely forceful coughing may induce bronchospasm in some individuals. With extremely forceful coughing, there may be injury to the larynx or development of an air leak such as a pneumothorax, a pneumomediastinum, or interstitial emphysema. The high intrathoracic pressures generated during coughing impede venous return to the heart, may result in transient systemic hypertension, or may induce dysrhythmias. Syncope can occur because of strenuous coughing. With very forceful coughing, rib fractures may occur. Other complications include rupture of the rectus abdominis muscles, urinary incontinence, pulmonary emboli, and kinking and knotting of venous catheters. An excellent in-depth review of cough is available.[29]

Evaluation

There are many etiologies of chronic cough in childhood (Box 12-3). Without some guidance in tailoring it to the individual, evaluation of this complaint could consume a tremendous amount of time and medical resources. The guidance needed can usually be provided by a careful history.

BOX 12-3
Persistent Cough*

- Congenital anomalies
  - Connection of the airway to the esophagus
    - Laryngeal cleft
    - Tracheoesophageal fistula
  - Laryngotracheomalacia
    - Primary laryngotracheomalacia
    - Laryngotracheomalacia secondary to vascular or other compression
  - Bronchopulmonary foregut malformation
  - Congenital mediastinal tumors
  - Congenital heart disease with pulmonary congestion
- Infection
  - Recurrent viral infection (infants and toddlers)
  - Chlamydial infection (infants)
    - Whooping cough–like syndrome
      - *Bordetella pertussis* infection
      - Chlamydial infection
      - *Mycoplasma* infection
      - Cystic fibrosis (infants and toddlers)
  - Granulomatous infection
    - Mycobacterial infection
    - Fungal infection
  - Suppurative lung disease (bronchiectasis and lung abscess)
    - Cystic fibrosis
    - Foreign body aspiration with secondary suppuration
    - Cilia dyskinesia
    - Immunodeficiency
      - Primary immunodeficiency
      - Secondary immunodeficiency (especially human immunodeficiency virus and acquired immunodeficiency syndrome)
  - Paranasal sinus infection
- Allergy and asthma
  - Asthma and cough-variant asthma
  - Allergic or vasomotor rhinitis and postnasal drip
- Aspiration (fluid material)
  - Dyskinetic swallowing with aspiration
    - General neurodevelopmental problems
    - Möbius syndrome
  - Bottle-propping and bottle in bed (infants and toddlers)
  - Gastroesophageal reflux
- Foreign body aspiration (solid material)
  - Upper airway aspiration (tonsillar, pharyngeal, laryngeal)
  - Tracheobronchial aspiration
  - Esophageal aspiration with an obstruction or aspiration resulting from dysphagia
- Physical and chemical irritation
  - Smoke from tobacco products (active and passive)
  - Wood smoke from stoves and fireplaces
  - Dry, dusty environment (hobbies and employment)
  - Volatile chemicals (hobbies and employment)
- Psychogenic or habit cough

*Longer than 3 weeks.

Onset of cough in the neonatal period is suggestive of a congenital airway malformation. In the perinatal period, abnormalities such as tracheal stenosis, laryngeal web, and tracheosophageal fistula may present with cough, whereas tracheomalacia typically results in cough later in the neonatal period. There may be an association with infectious symptoms such as TORCH (toxoplasmosis, other agents, rubella, cytomegalovirus, herpes simplex) syndrome, chlamydial infection, or pertussis; in older children, there may be an association with tuberculosis or sinusitis. The character of the cough can also provide important clues to the etiology. A continual cough, perhaps worse at night, may be found in asthma, cystic fibrosis, or other forms of bronchiectasis (especially if the cough is productive). Features suggestive of asthma (such as prolonged cough after upper respiratory tract infections, exercise, or exposure to environmental irritants) or the presence of risk factors for asthma (family history, history of prematurity) should prompt a careful evaluation for asthma or cough-variant asthma as a cause. A loud, honking cough absent during sleep is highly suggestive of a psychogenic cough, habit cough, or cough tic. History of a choking or gagging spell followed by chronic cough may promote concern over a possible aspirated foreign body, although there may be no such history, even in cases of documented foreign body aspiration. Chronic aspiration or gastroesophageal reflux as the cause of cough may be elicited by a careful neurologic and feeding history. Obviously, signs or symptoms of chronic illness, such as poor growth, recurrent fevers, and purulent sputum, should prompt a search for more severe pulmonary or systemic disease. Finally, the social history often provides information vital to elucidation of the cause. Factors such as exposure to environmental tobacco smoke, wood stoves, solvents, and dusts can explain chronic respiratory symptoms. The presence of family or school conflicts may support a suspicion of psychogenic cough.

The physical examination must be complete and carefully performed, with emphasis placed on the head and neck (transverse nasal crease, allergic shiners, boggy anasal mucosa, polyps, ear disease, foreign body in ear or nose, postnasal drip, long uvula, cobblestoning of posterior pharynx), chest (hyperinflation, wheezes, crackle, stridor), and heart (murmurs, gallops, signs of heart failure). The laboratory evaluation, which could easily be exhaustive, should be directed by findings elicited in the history and examination. Common tests include pulmonary function testing, including bronchoprovocation (pharmacologic, exercise, cold air); chest radiograph (two views, occasionally inspiratory and expiratory or lateral decubitus) and other imaging studies (CT, magnetic resonance imaging [MRI], sinus series and CT); barium esophagogram; esophageal pH monitoring; and bronchoscopy. The use of flexible vs. rigid bronchoscopy in evaluating pediatric patients has been reviewed recently[30] and bronchoscopy may be appropriate in selected patients. Unless foreign body aspiration is considered likely, flexible fiberoptic bronchoscopy is generally the procedure of choice. Laboratory studies that may be helpful include a complete blood count with differential (evaluating for leukocytosis, eosinophilia), total immunoglobulin E assay, purified protein derivative and control skin tests, sweat test, sputum culture (including culture for acid-fast bacillus and fungus), ciliary biopsy, and limited allergy skin testing (limited to locally common aeroallergens and animals and foods known to be in the child's environment). It may also be reasonable to perform an empiric trial of bronchodilators or a short course of systemic corticosteroids.

EVALUATION OF THE CHILD WITH AIRWAY OBSTRUCTION

Regardless of the etiology of the obstruction, wheezing and stridor with increased work of breathing are the cardinal manifestations of clinically significant airway obstruction. Usually the term *stridor* refers to a vibratory sound that is loudest on inspiration and is predominantly due to dynamic extrathoracic airway obstruction. In contrast, wheezing is usually produced by intrathoracic obstruction that worsens on expiration. At times, it can be difficult to distinguish between wheezing and stridor, and it should be remembered that critical airway obstruction can lead to stridor or wheeze in both phases of respiration (Box 12-4). A monophonic wheeze suggests obstruction

BOX 12-4
Airway Obstruction: Wheeze and Stridor

Inspiratory obstruction = extrathoracic

The vibratory sound produced by inspiratory obstruction is heard during inspiration, is usually monophonic, and may be high pitched as in croup or low to medium pitched as in snoring resulting from adenotonsillar hypertrophy.

- Congenital malformations
  - Nasal, nasopharyngeal, and oropharyngeal malformations
    - Retrognathia (Pierre Robin syndrome)
    - Nasal, choanal, or nasopharyngeal stenosis, tumor, mass
      - Craniopharyngioma
      - Anterior encephalocele
      - Teratoma
    - Adenotonsillar hypertrophy
      - Obesity or redundant pharyngeal tissue
      - Hypotonia (e.g., Down syndrome)
    - Oral cavity or pharyngeal tumor
      - Lingual tumor
        - Lingual thyroid tumor
        - Hemangioma
      - Neck masses
        - Bronchial cleft cyst
        - Cystic hygroma
  - Laryngeal or subglottic airway malformations
    - Laryngomalacia
    - Paralyzed vocal cords
    - Laryngeal or arytenoid cysts
    - Laryngocele
    - Subglottic stenosis
    - Subglottic hemangioma
- Infection
  - Nasal, nasopharyngeal, and oropharyngeal infection
    - Tonsillitis and peritonsillar abscess
    - Sublingual abscess (Ludwig's angina)
    - Retropharyngeal abscess
  - Laryngeal and subglottic infection
    - Epiglottitis
    - Croup (spasmodic)
    - Bacterial tracheitis (usually some expiratory wheeze)
    - Juvenile respiratory papillomatosis (early)
    - Tetanus with laryngospasm
- Foreign body or aspiration
  - Gastroesophageal reflux with edema, laryngospasm
  - Foreign body aspiration in pharynx, larynx, or subglottis
- Trauma
  - Laryngeal hematoma
  - Laryngeal burns or scalds
  - Stenosis secondary to instrumentation
  - Vocal cord paralysis after surgery
- Allergy and asthma
  - Anaphylactoid reaction to food or inhalant
  - Vocal cord dysfunction
- Metabolic problem
  - Hypocalcemia or hypomagnesemia
- Acquired tumor (rare)

Expiratory obstruction = intrathoracic

The vibratory sound produced by this obstruction is best heard on expiration and may be focal or monophonic and of low to medium pitch or may be diffuse or polyphonic and of medium to high pitch.

- Congenital malformations
  - Tracheobronchial tree malformations
    - Tracheobronchomalacia
      - Primary (focal or diffuse) tracheobronchomalacia
      - Tracheobronchomalacia secondary to compression by tumor (focal)

Expiratory obstruction = intrathoracic—cont'd

- Congenital malformations—cont'd
  - Tracheostenosis
    - VATER (vertebral defects, imperforate anus, tracheoesophageal fistula, radial and renal dysplasia) association
    - Complete tracheal rings
  - Vascular compression (ring or sling)
    - Aberrant subclavian vein
    - Pulmonary artery sling (aberrant left pulmonary artery)
    - Right-sided thoracic aorta with left ductus arteriosus
    - Left-sided thoracic aorta with right ductus arteriosus
    - Double aortic arch
  - Dilated cardiac chamber or dilated pulmonary artery with compression
- Infection
  - Intrinsic airway narrowing
    - Bronchitis
    - Bronchiolitis
    - Laryngotracheobronchitis
    - Bacterial tracheitis
    - Bronchiectasis
    - Cystic fibrosis
    - Juvenile respiratory papillomatosis (late)
  - Extrinsic airway compression
    - Mycobacterial or fungal infection with lymph node enlargement
    - Infection of congenital foregut malformations, cysts
    - Lung abscess
- Foreign body or aspiration
  - Gastroesophageal reflux with bronchitis
  - Foreign body in airway
  - Foreign body in esophagus
- Trauma
  - Tracheobronchial burns or scalds
  - Tracheobronchial injury (blunt or penetrating)
- Allergy and asthma
  - Anaphylactoid reaction to food or inhalant
  - Asthma with inflammation or bronchospasm
- Autoimmune disease
  - Bronchiolitis obliterans after lung or bone marrow transplant
  - Idiopathic bronchiolitis obliterans
- Tumor
  - Primary airway narrowing
    - Hamartoma
    - Benign tumors (e.g., lipoma, chondroma, myoblastoma)
    - Malignant tumor
      - Bronchial adenoma
      - Bronchogenic carcinoma
      - Sarcoma
  - Extrinsic airway compression
    - Hodgkin's lymphoma
    - T cell lymphoproliferative disease with mediastinal mass
    - Sarcoma
- Pulmonary edema

Inspiratory and expiratory obstruction

When obstruction is evident in both phases of breathing, the obstruction may be variable and may simultaneously occur in both the intrathoracic and extrathoracic airways (e.g., croup with laryngotracheobronchitis). If this has not occurred, the obstruction may have become critical in nature. This is particularly the case in extrathoracic airway obstruction in which the loss of obstruction during expiration is worrisome. In contrast, in intrathoracic airway obstruction resulting from asthma and bronchitis, wheezing commonly occurs in both phases of respiration but can usually be localized to the chest as opposed to the upper airway.

of a large central airway, whereas a polyphonic wheeze reflects peripheral airway obstruction.

Although asthma is certainly the most common disorder associated with wheezing, not every child with wheezing has asthma, nor does every child with asthma wheeze. The differential diagnosis of wheezing varies significantly with the age of the child. Congenital anatomic abnormalities that produce wheezing, like those associated with cough, are generally more likely to present in early infancy rather than later. Laryngotracheomalacia is an exception, usually presenting at several weeks of age or later. Laryngomalacia and extrathoracic tracheomalacia typically present as inspiratory stridor, whereas intrathoracic tracheomalacia and bronchomalacia are associated with low-pitched expiratory wheezing. Asthma, bronchiolitis, and bronchopulmonary dysplasia all may be associated with wheezing in infancy but can generally be distinguished on historical grounds. Along with asthma, cystic fibrosis and chronic aspiration (secondary to gastroesophageal reflux or a neurologic abnormality with dysfunctional swallowing) may present as wheezing at any age. Foreign body aspiration, most commonly pulmonary but also esophageal, classically presents as a monophonic, unilateral wheeze and is unusual before 6 months of age. This diagnosis, however, should be considered regardless of history (or lack thereof). Congestive heart failure may lead to wheezing secondary to lymphatic engorgement and resultant compression of the airway within the peribronchovascular sheath. Finally, wheezing may be produced by vocal cord opposition, either volitionally (often subconsciously) or because of vocal cord dysfunction.[31]

Evaluation of the child with wheezing starts with a careful history followed by thorough examination. When present, signs and symptoms of increased work of breathing or distress may dictate swift intervention before etiologic evaluation can take place. Depending on the age of the patient and the suspected etiology, ancillary tests may be helpful. These could include imaging studies (chest radiograph, CT, MRI, esophagogram, swallowing study), pulmonary function testing with bronchoprovocation or bronchodilator response, microbiologic studies (especially for respiratory syncytial virus in infants), and an empiric trial of bronchodilators. Bronchoscopic evaluation may also be helpful.

EVALUATION OF THE CHILD WITH EXERCISE INTOLERANCE

The majority of patients with chronic lung or cardiac disease and exercise intolerance usually have a clear reason for the inability to exercise; this may include deconditioning secondary to the primary illness. Instead of deconditioning, this section addresses the apparently normal child who has a difficult time exercising and develops dyspnea with a normal workload. These patients are commonly brought to their physician because they are unable to complete physical education at school or have a difficult time on sports teams. The approach to the apparently normal child with exercise intolerance involves delineating whether the child has a cardiorespiratory problem or is simply deconditioned (Box 12-5). The history is critical in this assessment. Data regarding symptoms compatible with asthma, cystic fibrosis, or another lung condition such as preexisting bronchopulmonary dysplasia need to be obtained. Similarly, a history of congenital or acquired cardiac disease needs to be reviewed.

BOX 12-5
Exercise Intolerance

- Chronic lung disease
  - Asthma
    - Exercise-induced bronchospasm
    - Vocal cord dysfunction
    - Deconditioning resulting from exercise-induced bronchospasm
  - Other pulmonary conditions
    - Bronchopulmonary dysplasia
    - Cystic fibrosis
    - Pulmonary fibrosis
    - Other
- Congenital or acquired cardiac disease
- Deconditioning with or without obesity
- Myopathy or muscular dystrophy
- Endocrine abnormalities
  - Thyroid dysfunction
  - Cortisol insufficiency
  - Diabetes mellitus
- Other chronic illnesses

Other than deconditioning, the leading cause of exercise intolerance is a variant of asthma, exercise-induced bronchospasm (EIB).[32] Children with EIB usually complain of a tightening or pain in the chest or submental triangle after vigorous exercise. This pain may be associated with frank wheezing or cough. Usually, patients complain of difficulty breathing that does not improve on stopping the exercise but that instead worsens after they sit down to rest. The symptoms then usually subside spontaneously. On cold or dry days, the tightness and cough are worse with exercise involving free running, such as soccer, football, and hockey. Swimming and cycling seem less prone to inducing bronchospasm. Some athletes notice that they can "run through" their bronchospasm or even prevent it by doing brief sprints before competing to obtain the protective effect of exercise on further EIB. Children with EIB may also have a history of spontaneous or prolonged wheezing and cough with colds. Collateral allergic symptoms should also be sought. The physical examination may be normal, but signs of allergy and asthma should be sought. Occasionally, wheezing or hyperinflation may be found; however, in children with these signs, usually asthma has already been diagnosed. Laboratory studies such as an exercise or cold-air challenge test may be conducted both to demonstrate airway hyperreactivity and to reproduce the symptoms so that the child can confirm their nature (see Chapter 15). In contrast, a trial of a β-agonist such as albuterol before exercise may be effective in diagnosing EIB as well as assessing a treatment modality.

Cardiovascular disease leading to exercise intolerance in an apparently normal child is uncommon and is usually diagnosed based on a history of diaphoresis and dyspnea with initiation of exercise. Furthermore, dyspnea resolves with resting compared to the persistence or worsening of EIB. A history of ankle edema, palpitations, fainting, chest pain, and nocturnal symptoms such as orthopnea or paroxysmal nocturnal dyspnea should be obtained but is positive only in children with relatively severe disease. Physical examination may reveal weight loss and fatigue, a hyperactive precordium, pathologic murmurs, and evidence of hypervolemia such as hepatomegaly and peripheral edema. Electrocardiography and chest radiography are central to the laboratory assessment; however, a child with dyspnea and signs of cardiac disease should be referred to a pediatric cardiologist for clinical assessment, echocardiography, and management.

It is relatively common for the pulmonary specialist to be asked to assess a child for exercise intolerance who has neither EIB nor heart disease. These children are commonly mildly to moderately obese, have a sedentary lifestyle, and do not readily engage in sports. They are commonly assessed because of an inability to keep up with school exercise programs. Their dyspnea and fatigue usually occur during exercise such as running laps. They usually do not have chest pain or cough and do not complain of any dysphoria or tightness in the submental region. They may complain of headache, leg pain, and cramping with exercise. Lacking the symptom complexes and findings previously noted, this group may most benefit from exercise testing. The clinician can use the test to reproduce the symptoms and demonstrate that the child does not have bronchospasm. Furthermore, the child may be unable to exercise vigorously enough to successfully complete an exercise challenge test. These clinical and laboratory findings combined can be useful to reassure the family that cardiorespiratory disease is not present and that deconditioning is the main problem. An exercise program and weight-control program can then be prescribed to help the child return to an active lifestyle.

EVALUATION OF THE CHILD WITH CHEST PAIN

The child with chest pain can present a challenge for the practitioner; parental anxiety is usually high because of the concern that the child may have heart disease (Box 12-6). In fact, the majority of children with chest pain have either EIB or a musculoskeletal cause that will respond to antiinflammatory medication or nonspecific therapies.[33,34] Chest pain resulting from cardiac disease is uncommon in an apparently healthy child without other cardiac symptoms. The history should be focused after the clinician determines that the child is generally well. The pain should be characterized using the PQRST approach. The pain is described as sharp, burning, or dull and aching. It is localized, and any radiation such as from the spine through an intercostal space should be noted. Radiation to the shoulder suggests diaphragmatic irritation. Worsening of the pain with breathing or movement should be noted, as should other provocative factors. The history should include a survey of activities compatible with muscular strain such as recent trauma, contact sports, and sports such as weight training. Surprisingly, many children do not associate anterior parasternal chest pain with the fact that they just began weight training to increase their pectoral muscle bulk. Also, many children carry schoolbooks in a pack or bag slung over one shoulder, leading to shoulder girdle strain. Patients with asthma, pertussis, and cystic fibrosis may develop chest pain associated with chronic cough and repetitive trauma to the ribs and muscles of the chest wall. The history should also review recent symptoms of lung infection, allergies, asthma, and EIB. Symptoms of arthritis or joint disease should be assessed, as should any recent skin changes or weight loss. Gastroesophageal reflux with esophagitis can also present as chest pain. A history of reflux after meals or on lying down with heartburn, a bitter taste in the mouth, water brash, and sensitivity to acid, high-fat foods, or coffee can be helpful. The physical examination should be relatively complete and include an assessment of general well-being and the respiratory, cardiovascular, gastrointestinal, and musculoskeletal systems. Changes on the chest wall with swelling or any mass, particularly over the costochondral and clavicular joints, should be specifically noted. Tenderness over the site of chest pain strongly implicates a musculoskeletal process. Although acute infection such as pneumococcal pneumonia with pleurisy is usually a clear diagnosis, other infections such as histoplasmosis, coccidioidomycosis, and tuberculosis may have a slow course and present with pleuritic pain. Thus a careful chest examination for reduced air entry, crackles, or a friction rub is important. The results of chest radiography are usually normal in musculoskeletal chest pain but may be reassuring to both the parent and practitioner. Electrocardiography or stress testing is only occasionally useful in cases without additional cardiac symptoms or signs.

BOX 12-6
Chest Pain

- Musculoskeletal or soft tissue problems (most common)
  - Chronic cough (asthma, cystic fibrosis, pertussis)
  - Sports or weight training that caused muscle or joint strain
  - Blunt trauma to the ribs or joints
  - Costochondritis
  - Tietze's syndrome
  - Rheumatoid arthritis
  - Breath development, inflammation
  - Diaphragmatic pain
  - Slipping rib syndrome
- Asthma
  - Acute bronchospasm, especially with exercise
  - Pneumomediastinum
  - Pneumothorax
- Pleural inflammation
  - Viral inflammation, Bornholm disease or pleurodynia
  - Bacterial, mycobacterial, or fungal infection with pleurisy
- Gastrointestinal or abdominal problems
  - Gastroesophageal reflux
  - Gastric or duodenal ulcer
  - Diaphragmatic irritation caused by an intraabdominal process
- Cardiac problems (uncommon)
  - Aberrant coronary problems
  - Pericarditis, myocarditis, or myopathy
  - Palpitations or dysrhythmias that are confused with pain
- Pulmonary vasculature
  - Pulmonary embolus
  - Sickle cell pulmonary crisis
- Psychogenic or psychophysiologic problems

EVALUATION OF THE CHILD WITH HEMOPTYSIS

The approach to diagnosing hemoptysis in a child depends on whether there is a known preexisting disease such as cystic fibrosis.[35,36] In the previously well child with hemoptysis, the history is critical (Box 12-7). Care should be taken to ensure that the red or purple material expectorated was actually blood and not coloring from food. Afterward, the most important point is to try to determine that the bleeding truly represents respiratory bleeding from the lower respiratory tract and is not due to nasal, pharyngeal, or gastrointestinal bleeding (Table 12-6). A history of recent epistaxis, acute or recurrent tonsillitis, or throat trauma focuses attention on the upper respiratory tract. Indeed, examination of the nasopharynx by a specialist is sometimes important in ruling out a bleeding site in the upper respiratory tract. A history of gastroesophageal reflux, vomiting, liver disease, or portal hypertension focuses concern on the gastrointestinal tract as the source of the bleeding.

Although some streaking of the sputum in bacterial bronchitis or pneumonia is relatively common, true hemoptysis in

BOX 12-7
Hemoptysis

Pulmonary origin of bleeding
- Infection
  - Acute tracheobronchitis or severe pneumonia
  - Bronchiectasis
  - Erosion by an infected lymph node (mycobacteria, fungi)
  - Lung abscess
  - Fungal infection, including secondary mycetoma
  - Parasitic infection
  - Pulmonary hemorrhage in severe viral pneumonia
- Foreign body aspiration
- Bronchial tumor
  - Primary tumor
  - Secondary tumor
- Autoimmune lung disease
  - Idiopathic pulmonary hemosiderosis
  - Goodpasture's syndrome
  - Milk allergy (Heiner syndrome)
  - Wegener's granulomatosis
  - Other vasculitis (e.g., Churg Strauss)
- Pulmonary vascular conditions
  - Pulmonary embolism
  - Primary pulmonary hypertension
  - Obstructed pulmonary veins
  - Raised left arterial pressure
    - Congestive heart failure or pulmonary edema
    - Mitral valve stenosis
    - Aortic valvular stenosis or obstruction
  - Arteriovenous malformations
  - Osler-Weber-Rendu disease
  - Sickle cell pulmonary crisis
  - Pulmonary hemorrhage in acute respiratory distress syndrome
- Bronchopulmonary foregut malformations
- Trauma
  - Blunt trauma with pulmonary contusion or airway disruption
  - Penetrating trauma

Nonpulmonary origin of bleeding
- Upper airway conditions
  - Epistaxis
  - Sinusitis
  - Adenoidal or tonsillar bleeding
  - Severe pharyngitis or pharyngeal trauma
  - Coagulopathy with trauma to the mouth or pharynx
- Gastrointestinal conditions
  - Esophagitis with gastroesophageal reflux
  - Esophageal varices secondary to portal hypertension
  - Gastric or duodenal ulcer
  - Mallory-Weiss syndrome or esophageal erosion with severe vomiting or bulimia
- Munchausen or Munchausen by proxy syndrome

Lack of bleeding
- Natural and artificial coloring in food
- Dyes in medicines
- Nasal foreign body with dye (crayon)

Table 12-6 Differential Features of Hemoptysis and Hematemesis

HEMOPTYSIS	HEMATEMESIS
Blood is coughed up, not vomited. Retching and nausea may come from pharyngeal irritation from blood.	Blood is vomited.
A portion of the blood should be frothy.	Blood is never frothy.
Blood is usually, but not always, bright red in color.	Blood is dark red in color.
Blood is alkaline in reaction.	Blood is acid in reaction.
Hemoptysis is preceded by a gurgling noise or a sensation stimulating a cough reflex. This may be absent in massive hemoptysis.	Hematemesis is preceded by nausea and vomiting.
There is sometimes a history of past cough.	There may be history of alcoholism and/or gastric disturbances, plus clinical findings of liver disease.
There is continued blood-tinged sputum, which lasts for several days.	Blood-tinged sputum is usually absent.
Blood is mixed with pus, organisms, or macrophages; some of the macrophages may contain hemosiderin particles.	Vomited blood may contain food particles.
Anemia may or may not be present.	There are often clinical and laboratory findings of blood loss before the actual hematemesis.

From Lyons HA: *Basics of RD: ATS news*, New York, 1976, American Lung Association.

the previously well child is rare. The hemoptysis should be characterized by the volume of blood (i.e., streaking vs. submassive [<240 ml] vs. massive [≥240 ml]). Whether the blood was a bright red liquid that clotted or simply old purple-brown clots should be noted. In the case of submassive and massive hemoptysis the patient may have a warm, bubbling feeling over the affected segment. The history should rigorously assess the possibility of foreign body aspiration. This may not have been a recent event because foreign bodies leading to bleeding must usually be in the respiratory tree long enough to cause chronic infection or irritation with mucosal erosion. Past respiratory illness such as remote foreign body aspiration, pertussis, and severe pneumonia can also be associated with hemoptysis related to bronchiectasis formation. A history of heart disease should be obtained because increased left atrial pressures or obstructed pulmonary veins can lead to bleeding. Usually, however, the cardiovascular history is negative. Physical examination is usually negative in the absence of acute lung infection or chronic lung problems such as cystic fibrosis. Focal lung changes such as reduced or lagged air entry and focal hyperinflation may suggest foreign body aspiration. Coarse crackles and reduced air entry may lead to the consideration of infection and, if accompanied by clubbing, bronchiectasis. Chest radiography is used to rule out pneumonia, gross bronchiectasis, and cavitary disease. Evidence of focal hyperaeration or atelectasis may suggest focal airway obstruction resulting from a foreign body, infected lymph node, or tumor. If the diagnosis of bronchiectasis is considered, a thin-section, high-resolution CT scan rapidly identifies the presence of these lesions. Bronchoscopy can also be used, but the site may be obscured in the presence of moderate bleeding. Bronchoscopy may be most useful after the bleeding has quieted, when lesions such as bronchial adenomas, lymph nodes eroding the mucosa, and foreign bodies can be better seen. Angiography may be used while echocardiography and cardiac catheterization may have a role in diagnosing recurrent hemoptysis with no apparent lesion. In this case the hemoptysis may result from an obstructed pulmonary vein.

EVALUATION OF THE CHILD WITH HYPOXIA

The approach to evaluating the child with evidence of tissue hypoxia requires the determination of whether the hypoxia is due

to a failure of oxygen delivery or an inability of the tissues to use oxygen (Box 12-8). Failure of oxygen delivery or use may be evidenced by an alteration in global metabolism, resulting in anaerobic glycolysis with the production of a lactic acidosis, or an end-organ dysfunction (e.g., confusion secondary to cerebral hypoperfusion). A common approach has been to classify tissue hypoxia as occurring in one of four manners. The first two abnormalities lead to reduced oxygen content in the blood. The most common cause in patients with lung disease, hypoxemic hypoxia, is due to a reduced arterial partial pressure of oxygen, leading to an inadequate saturation of hemoglobin (see later section). The second is anemic hypoxia. Even with a normal arterial partial pressure of oxygen and hemoglobin saturation, anemia (reduced functional hemoglobin) leads to reduced oxygen delivery resulting from reduced oxygen capacity in the blood. This occurs in carbon monoxide poisoning, in which the hemoglobin is bound with the carbon monoxide, reducing the amount available to carry oxygen. In addition, carbon monoxide poisoning increases the affinity of hemoglobin for oxygen, further reducing oxygen delivery to the tissues. If oxygen content is adequate but signs of tissue hypoxia are present, there are two possibilities. Either the oxygen is not being delivered to the tissues, or the tissues are unable to use oxygen in aerobic metabolism. The former is called *circulatory hypoxia* and may occur globally as in shock or locally as in vascular obstruction with ischemia. The latter, histotoxic hypoxia, occurs in sepsis and cyanide poisoning of aerobic metabolism when the cells are unable to conduct aerobic glycolysis.

The approach to hypoxemic hypoxia (see Box 12-8) is to divide potential causes into five categories. As in assessing tissue hypoxia, the clinician simply needs to work through the steps of the oxygenation of blood in the lung to delineate potential problems. First, a reduced inspired oxygen partial pressure leads to hypoxemia in the absence of compensatory hyperventilation. This may result from a reduced fractional concentration of oxygen secondary to oxygen consumption by combustion or of other gases in the environment. It may also occur with a reduced barometric pressure caused by increases in altitude. Hypoventilation with an increase in the level of alveolar carbon dioxide and decrease in the level of alveolar oxygen causes hypoxemia as a result of a failure to ventilate adequate oxygen into the lungs to meet the body's metabolic demands. These first two causes of hypoxemia are associated with a normal alveolar-arterial oxygen difference. Thickening of the alveolar-capillary membrane may cause the normal perfusion limitation of oxygen transfer to become diffusion limited and lead to hypoxemia. Increased cardiac output and reduced alveolar oxygen levels exacerbate this diffusion block. Areas of local hypoventilation in the lung resulting from either airway or airspace disease lead to hypoxemia secondary to a ventilation-perfusion imbalance with incomplete saturation of blood passing through these regions of the lung. Finally, blood from the systemic venous system may bypass the ventilation entirely, either because of intrapulmonary shunting with lung disease or arteriovenous fistulae or because of extrapulmonary shunting with congenital heart or great vessel malformation (cyanotic congenital heart disease). The arterial carbon dioxide level may be normal in all of these conditions except hypoventilation. This is because the healthy, well-ventilated lung can compensate for the dysfunctional lung by clearing excess carbon dioxide. Unfortunately, blood that is normally ventilated is already nearly completely saturated with oxygen, and thus healthy units cannot return arterial oxygen to normal by overcompensating for units with diffusion block, ventilation-perfusion imbalance, or shunt. Finally, shunt is commonly separated from the other causes of hypoxia because it does not respond to the administration of supplemental oxygen with a significant increase in arterial oxygen levels.

BOX 12-8
Tissue Hypoxia

- Hypoxemic hypoxia: low arterial partial pressure of oxygen
  - Low inspired oxygen concentration (low inspired oxygen partial pressure)
    - Low barometric pressure (high altitude)
    - Low inspired oxygen concentration (low fraction of inspired oxygen)
  - Cardiorespiratory disease
    - Hypoventilation
    - Diffusion block
    - Ventilation-perfusion imbalance
    - Shunting
      - Intrapulmonary shunting
      - Extrapulmonary shunting
- Anemic hypoxia
  - Anemia
  - Carbon monoxide poisoning
- Circulatory hypoxia
  - Shock or hypoperfusion
    - Hypovolemic shock
    - Obstructive shock
    - Cardiogenic shock
    - Distributive shock
  - Local vascular obstruction
- Histotoxic hypoxia
  - Sepsis with poor oxygen use
  - Cyanide poisoning

EVALUATION OF THE CHILD WITH HYPOVENTILATION

The definition of *hypoventilation* is an increase in the arterial carbon dioxide level above 45 mm Hg; it is, by definition, a respiratory acidosis. It may differ with the apparent minute ventilation, tidal volume, or respiratory rate. *Hyperpnea* and *hypopnea* refer to an apparent increase or decrease in overall breathing; however, *hyperventilation* and *hypoventilation* refer specifically to the level of arterial carbon dioxide achieved. The first step in assessing the child with hypoventilation is to try to determine whether the respiratory pump is functioning as well as expected in response to substantive lung disease or whether it is a primary or adjunctive cause of the increased arterial carbon dioxide (Box 12-9). Second, the pump may be functioning properly and delivering an adequate minute ventilation; however, there may be an increased ventilation of physiologic dead space with reduced alveolar ventilation. This may result either from a reduction in tidal volume with a fixed physiologic dead space or from an increase in physiologic dead space that is not matched by an increase in tidal volume. In either case, the dead space/tidal volume ratio increases, and alveolar ventilation is compromised, leading to carbon dioxide retention.

The differential diagnosis of airspace or airway disease that can lead to carbon dioxide retention is broad and is not addressed further here. The differential diagnosis of a failure of the respiratory pump is considered here because it may apply even in the cases in which lung disease is paramount. A useful approach is to work through the steps necessary for the maintenance of minute ventilation, starting centrally with respiratory

BOX 12-9
Hypoventilation

Reduced minute ventilation
- Respiratory pump failure as a primary cause
  - Central controller failures
    - Encephalopathy or brain stem dysfunction
      - Infection
      - Intoxication
      - Metabolic dysfunction or seizure
      - Tumor
      - Trauma, concussion, or hemorrhage
      - Malformation (Arnold-Chiari malformation)
    - Central hypoventilation syndrome
    - Metabolic alkalosis
  - Cervical spinal cord disruption (upper motor neuron)
    - Infection
    - Tumor
    - Trauma, concussion, or hemorrhage
    - Inflammation (transverse myelitis)
    - Compression (achondroplasia, Down syndrome)
    - Multiple sclerosis
  - Cervical spinal cord (lower motor neuron cell)
    - Infection (poliomyelitis)
    - Inflammation or degeneration (transverse myelitis)
    - Vasculitis or vascular accident
    - Werdnig-Hoffman disease
  - Phrenic or intercostal nerves (lower motor neuron; axon)
    - Trauma (thoracic surgery or penetrating injury)
    - Demyelinating neuropathies (Guillain-Barré syndrome)
    - Tumor
  - Neuromuscular junction failure
    - Myasthenia gravis
    - Botulism
    - Aminoglycosides
    - Pseudocholinesterase deficiency
  - Respiratory muscle failure
    - Muscular dystrophies
    - Extreme electrolyte abnormalities
    - Extreme starvation or metabolic imbalance
    - Familial paralysis syndromes (e.g., hypokalemia)
  - Chest wall disease or disruption
    - Flail chest
    - Restrictive chest wall disease
      - Kyphoscoliosis
      - Congenital chest wall malformation or dystrophy
      - Ankylosing spondylitis
    - Prune-belly syndrome (infancy)
- Increased respiratory work with muscle fatigue
  - Increased elastic work
    - Pulmonary fibrosis
    - Pulmonary edema
      - Cardiogenic edema
      - Noncardiogenic edema (adult respiratory distress syndrome)
    - Diffuse pneumonia
  - Increased resistive work
    - Upper airway obstruction
    - Lower airway obstruction
  - Mixed increase in elastic and resistive work

Increased minute or reduced alveolar ventilation
- Increased physiologic dead space
  - Increased anatomic dead space
    - Severe bronchiectasis
  - Increased alveolar dead space
    - Alveolar distention or overexpansion
      - Intrathoracic airway obstructive airway disease
      - Mechanical ventilation with inadvertent positive end-expiratory pressure
    - Shock with reduced pulmonary perfusion pressures
    - Pulmonary embolus
- Reduced tidal volume with normal physiologic dead space

control and ending with the respiratory muscles and chest wall (see Box 12-9). Failure may result from central controller failure or disruption of upper motor neuron function, such as in sedation or cervical cord damage. Lower motor neuron disease may occur at a cellular level, such as in poliomyelitis, or may be more peripheral resulting from damage to the phrenic nerves caused by trauma or demyelinating diseases. The neuromuscular junction may inadequately conduct the neural impulse, such as in botulism, or the muscle be unable to respond, such as in profound hypokalemia. Finally, even if the controller/feedback system is functioning and the respiratory muscles are able to respond, the chest wall itself must be able to function as a pump without reduced motion or inappropriate paradoxical motion.

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CHAPTER 13

Imaging of the Respiratory System

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Because new tests can be invented faster than it is decided that they are of no use, clinicians now have at their disposal a myriad of imaging methods.[1] The role of the radiologist has thus broadened from assurance of film quality, film interpretation, and reporting to the acquisition of the most correct examination by teaching physicians about new imaging techniques and their applications.

This chapter discusses the different imaging tools available and their uses in the investigation of respiratory tract problems. Specific disease entities are discussed elsewhere in this book. Rather, a basic systematic approach to the proper use of imaging is offered. In this chapter, the respiratory tract is arbitrarily divided into four anatomic regions—nasal airway, paranasal sinuses, extrapulmonary airway, and chest.

PHYSICAL PRINCIPLES OF IMAGING MODALITIES

Radiography

The x ray, the most common medium used in imaging, is a form of electromagnetic radiation. High-voltage generators produce an electron beam that, on striking a rotating anode, produces a spectrum of x rays of varying energies. The number of x rays (photons) and the shape of the energy spectrum can be manipulated with changes in output (the tube current in milliamperes [mA]), filtration, and maximum voltage across the x-ray tube (peak kilovoltage [kvP]). These x rays interact within the body in various ways depending on the thickness and density of the body part. The number of x rays that traverse the body and reach the film or another detector dictate the characteristics of the image produced.

Extrapolation from data identifying the risks of high-dose radiation (leukemia, cancer, cataract stimulation) implies that even low-dose radiation exposure from diagnostic examinations may not be innocuous. Therefore every effort must be made to limit radiation exposure, especially in the pediatric population (Table 13-1). The best way to do this is to limit the number of studies

Table 13-1 Average Radiation Dose

	DOSE (mR)
Newborn chest, two views	20
Digital newborn chest, two views	8
1-year-old chest, two views	50
Conventional chest, two views in older child	75
Digital chest, two views in older child	19
Neck, two views	100
Fluoroscopy/minute*	500-1000
CT chest standard/slice*†	1500
CT chest high-resolution/slice*†	2000
Ambient radiation/year/at sea level	100

Skin entry dose from National Council on Radiation Protection and Measurements: *Radiation protection in pediatric radiology,* NCRD Rep No 68, Bethesda, Md, 1981, The Council.
mR, Millirad (0.001 R).
*Data from Children's Hospital of Michigan, Detroit.
†Each slice is finely coned, so there should be no additive dose.