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The History and Physical Examination

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At the beginning of the 21st century the diagnosis of disease still requires a detailed medical history and a thorough physical examination. For the majority of patients in many areas of the world, additional information from laboratory tests and other data are of rather limited availability. Modern science and technology have changed the situation considerably in the industrialized nations, but we are paying a high price. Cost containment in health care has become essential, and physicians have to be skillful in their history taking and physical examination techniques to collect a maximum of information before ordering expensive medical-technical investigations. The relevance of these skills in pediatric respiratory medicine is exemplified by clinical severity scores that are widely used in care maps for asthma, bronchiolitis, and croup in children, or in scores developed to manage patients suspected to have severe acute respiratory syndrome (SARS).

The diagnosis of disease in children even more than in older patients has to rely on the patient's history and on observations gathered during the physical examination. Young children cannot follow instructions and participate in formal physiologic testing, and physicians hesitate before subjecting their pediatric patients to invasive diagnostic procedures. Diseases of the respiratory tract are among the most common in children, and in the majority of cases they can be correctly identified from medical history data and physical findings alone. The following review of the medical history and physical examination in children with respiratory disease includes some observations that were made with the help of modern technology. These technological aids do not lessen the value of subjective perceptions but rather emphasize how new methods may further our understanding, sharpen our senses, and thereby advance the art of medical diagnosis.

■ THE HISTORY

■ GENERAL PRINCIPLES

The medical history should be taken in an environment with comfortable seating for all, a place for clothing and belongings, and some toys for younger children. Formula should be on hand to help quiet infants and toddlers. Privacy has to be assured, without the usual interruptions by phone calls and other distractions. If possible, the physician should see one child at a time because the presence of young siblings or other children in the room tends to be distracting. Data that should be recorded at the beginning include the patient's name and address, the parents' or guardians' home and work phone numbers, the name of the referring physician, and information on the kindergarten or school if this is relevant. In many cases the history will be given by someone other than the patient, but the physician should still ask even young children directly about their complaints.

When asking about the history of the present illness, the physician should encourage a clear and chronologic narrative account. Questions should be open ended, and at intervals the physician should give a verbal summary to confirm and clarify the information. Past medical data and system review are usually obtained by answers to direct questions.

■ STRUCTURE OF THE PEDIATRIC HISTORY

The source of and the reason for referral should be noted. On occasion, the referral may have been made by someone other than the patient or the parents, such as a school teacher, a relative, or a friend. The *chief complaint* and the person most concerned about it should be identified. The *illness at presentation* should be documented in detail regarding its onset and duration, the environment and circumstances under which it developed, its manifestations and their treatments, and its impact on the patient and the family. Symptoms should be defined by their qualitative and quantitative characteristics as well as by their timing, location, aggravating or alleviating factors, and associated manifestations. Relevant past medical and laboratory data should be included in the documentation of the present illness.

This general approach is also applicable when the emphasis is on a single organ system, such as the respiratory tract. The onset of disease may have been gradual (e.g., with some interstitial lung diseases) or sudden (e.g., with foreign body aspiration). The physician should ask about initial manifestations and who noticed them first. The age at first presentation is important because respiratory diseases that manifest soon after birth are more likely to have been inherited or to be related to congenital malformations. Depending on the duration of symptoms, the illness will be classified as acute, subacute, chronic, or recurrent. These definitions are arbitrary, but a disease of less than 3 weeks duration is generally called acute, between 3 weeks and 3 months subacute, and longer than 3 months chronic. If symptoms are clearly discontinuous, with documented intervals of well-being, the disease is recurrent. This distinction is important because many parents may perceive their child as being chronically ill, not realizing that young normal children may have six to eight respiratory infections per year, particularly during the first 2 years if the child is in a daycare setting or if they have older siblings.

Respiratory diseases are often affected by environmental factors. There should be a careful search for seasonal changes in symptoms to uncover possible allergic causes. Exposure to noxious inhaled agents, for example, from industrial pollution or more commonly from indoor pollution by cigarette smoke, can sustain or aggravate a patient's coughing and wheezing. Similarly, a wood-burning stove used for indoor heating may be a contributing factor. The physician should therefore obtain a

detailed description of the patient's home environment. Are there household pets, such as dogs, cats, and hamsters, or birds, such as budgies, pigeons, or parrots? What are the plants in and around the house? Are there animal or vegetable fibers in the bedclothes or in the floor and window coverings (e.g., wool, feathers)? Are there systems in use for air conditioning and humidification?

There may be a relationship between respiratory symptoms and daily activities. Exercise is a common trigger factor for cough and wheezing in many patients with hyperreactive airways. A walk outside in cold air may have similar effects. Diurnal variation of symptoms may be apparent, and attention should be paid to changes that occur at night. These changes may also be related to airway cooling, or they may reflect conditions that are worse in the recumbent position, such as post-nasal drip or gastroesophageal reflux. Food intake may bring on symptoms of respiratory distress when food is aspirated or when food allergies are present.

A large proportion of children presenting with respiratory symptoms will be suffering from infection, most often viral. It is important to know whether other family members or persons in regular contact with the patient are also affected. When unusual infections are suspected, questions should be asked about recent travel to areas where exotic infective organisms may have been acquired. Drug abuse by parents or by older patients and others with high-risk lifestyles may lead the physician to consider the possibility of the acquired immunodeficiency syndrome (AIDS).

Descriptions of respiratory disease manifestations may come from the parents or directly from an older child. Common symptoms are fever, cough and sputum production, wheezing or noisy breathing, dyspnea, and chest pain. Most of these are discussed in more detail at the end of this chapter.

The previous medical history will provide an impression of the general health status of the child. First, the birth history should be reviewed, including prenatal, natal, and neonatal events. The physician should inquire about the course of pregnancy, particularly whether the mother and fetus suffered from infections, metabolic disorders, or exposure to noxious agents, such as nicotine. The duration of pregnancy, possible multiple births, and circumstances leading to the onset of labor should be noted. Difficult labor and delivery may cause respiratory problems at birth (e.g., asphyxia and meconium aspiration), and the physician should ask about birth weight and Apgar scores. The neonatal course has to be reviewed carefully because many events during this period may have an impact on the patient's respiratory status in later years. Were there any signs of neonatal respiratory distress, such as tachypnea, retractions, and cyanosis? Treatment with oxygen or endotracheal intubation should be recorded. Some extrathoracic disorders provide valuable clues for diagnosis, such as the presence of eczema in atopic infants or neonatal conjunctivitis in a young patient with chlamydia pneumonia, particularly if there was a documented infection of the mother.

Much is learned from a detailed feeding history, which should include the amount, type, and schedule of food intake. The physician should ask whether the child was fed by breast or bottle. For the newborn and young infant, feeding is a substantial physical exercise and may, in the presence of respiratory disease, lead to distress, much as climbing stairs does in the older patient. The question of exercise tolerance in an infant is therefore asked by inquiring how long it takes the patient to finish a meal. The caloric intake of infants with respiratory disease

is often reduced despite an increased caloric need, to support the work of breathing. This reduced caloric intake commonly results in a failure to thrive. Older patients with chronic respiratory disease and productive cough may suffer from a continuous exposure of their taste buds to mucopurulent secretions and may quite understandably lose their appetites, but medical treatment (e.g., with certain antibiotics) may have similar effects. Patients with food hypersensitivity may react with bronchospasm or even with interstitial lung disease on exposure to the allergen (e.g., to milk). Physical irritation and inflammation occur if food is aspirated into the respiratory tract. This happens frequently in patients with debilitating neurologic diseases and deficient protective reflexes of the upper airways but may also occur in neurologically intact children.

The physical development of children with chronic respiratory diseases may be retarded. Malnutrition in the presence of increased caloric requirements is common, but the effects of some long-term medical treatments (e.g., with steroids) should also be considered. Previous measurements of body growth should be obtained and plotted on standard nomograms. Psychosocial development may be affected if chronic lung diseases, such as asthma or cystic fibrosis, limit attendance and performance at school or if behavioral problems arise in children and adolescents subjected to chronic therapy. More severely affected patients may also be delayed in their sexual development.

Many diseases of the respiratory tract in children have a genetic component, either with a clear mendelian mode of inheritance (e.g., autosomal recessive in cystic fibrosis, homozygous deficiency of α_1 -antitrypsin, sex-linked recessive in chronic granulomatous disease, and autosomal dominant in familial interstitial fibrosis) or with a genetic contribution to the cause. Examples of familial aggregation of respiratory disease are chronic bronchitis and bronchiectasis or familial emphysema in patients with heterozygous α_1 -antitrypsin deficiency, in which the susceptibility of the lung to the action of irritants (e.g., cigarette smoke) is increased. A mixed influence of genetic and environmental factors exists in polygenic diseases, such as asthma or allergic rhinitis.

When inquiring about the family history, the physician should review at least two generations on either side. The parents should be asked whether they are related by blood, and information should be obtained about any childhood deaths in the family. The health of the patient's siblings and also of brothers and sisters of both parents should be documented. Particular attention should be paid to histories of asthma, allergies and hay fever, chronic bronchitis, emphysema, tuberculosis, cystic fibrosis, and sudden unexpected infant death.

A detailed report of prior tests and immunizations should be obtained. Quite often this requires communication with other health care providers. Results of screening examinations (e.g., tuberculin and other skin tests, chest radiographs, and sweat chloride measurements) should be noted. Similarly, childhood illnesses, immunizations, and possible adverse immunization reactions should be documented. If the history is positive for allergic reactions, these have to be confirmed and defined. Previous hospital admissions and their indications should be listed, and the patient's current medications and their efficacy should be documented. If possible, the drug containers and prescriptions should be reviewed. The physician may use the opportunity to discuss the pharmacologic information and the technique of drug administration, particularly with inhaled bronchodilator medications.

One of the most important goals in taking a history is to become more aware of the particular psychological and social situation of the patient. It is impossible to judge current complaints or responses to medical interventions without an individual frame of reference for each patient. The physician should encourage the child and the parents to describe a typical day at home, daycare, kindergarten, or school. This will provide valuable information about the impact of the illness on daily routines, the financial implications, the existing or absent social support structures, and the coping strategies of the family. Compliance with medical treatment is rarely better than 50%, and physicians are generally unable to predict how well their patients follow and adhere to therapeutic regimens. Compliance can improve if the patient and the parents gain a better understanding of the disease and its treatment. It is important to recognize prior experiences that the family may have had with the health care system and to understand individual religious and health beliefs. Particularly in children with chronic respiratory ailments whose symptoms are not being controlled or prevented, the effort and unpleasantness (e.g., of chest physiotherapy) may limit the use of such interventions. The physician should also consider the social stigma associated with visible therapy, especially among peers of the adolescent patient.

A review of organ systems is usually the last part of the history and may actually be completed during the physical examination. Although the emphasis is on the respiratory system, questions about the general status of the child will be about appetite, sleep, level of activity, and prevailing mood. Important findings in the region of the head and neck are nasal obstruction and discharge, ear or sinus infection, conjunctival irritation, sore throat, and swallowing difficulty. The respiratory manifestations of coughing, noisy breathing, wheezing, and cyanosis are discussed in detail at the end of this chapter. Cardiovascular findings may include palpitations and dysrhythmia in hypoxic patients; there may be edema formation and peripheral swelling with cor pulmonale. Effects of respiratory disease on the gastrointestinal tract may appear with cough-induced vomiting and abdominal pain. There may be a direct involvement with diarrhea, cramps, and fatty stools in patients with cystic fibrosis. The physician should ask about hematuria and about skin manifestations, such as eczema or rashes, and about swellings and pain of lymph nodes or joints. Finally, neurologic symptoms, such as headache, lightheadedness, or paresthesia, may be related to respiratory disease and cough paroxysms or hyperventilation.

■ THE PHYSICAL EXAMINATION

Traditionally, the physical examination is divided into inspection, palpation, auscultation, and percussion. The sequence of these steps may be varied depending on the circumstances, particularly in the assessment of the respiratory tract in children. The classic components of the physical examination and some modern aids and additions are discussed in the following sections.

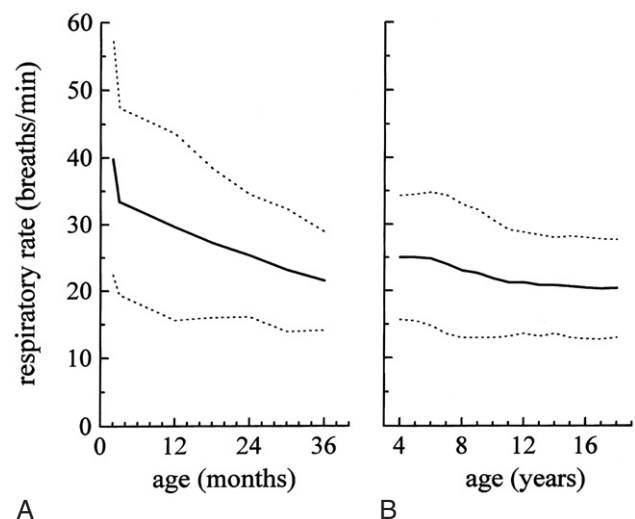
■ INSPECTION

Much can be learned from simple observation, particularly during those precious moments of sleep in the young infant or toddler, who when awake can be a challenge even for the skilled examiner. First, the pattern of breathing should be observed. This includes the respiratory rate, rhythm, and effort. The respiratory rate decreases with age and shows its greatest

variability in newborns and young infants (Fig. 6-1). The rate should be counted over at least 1 minute, ideally several times for the calculation of average values. Because respiratory rates differ among sleep states and become even more variable during wakefulness, a note should be made describing the behavioral state of the patient. Observing abdominal movements or listening to breath sounds with the stethoscope placed before the mouth and nose may help in counting respirations in patients with very shallow thoracic excursions.

Longitudinal documentation of the respiratory rate during rest or sleep is important for the follow-up of patients with chronic lung diseases, even more so for those too young for standard pulmonary function tests. Abnormally high breathing frequencies or tachypnea can be seen in patients with decreased compliance of the respiratory apparatus and in those with metabolic acidosis. Other causes of tachypnea are fever (approximately five to seven breaths per minute increase per degree above 37°C), anemia, exertion, intoxication (salicylates), and anxiety and psychogenic hyperventilation. The opposite, an abnormally slow respiratory rate or bradypnea, can occur in patients with metabolic alkalosis or central nervous system depression. The terms *hyperpnea* and *hypopnea* refer to abnormally deep or shallow respirations. At given respiratory rates, this determination is a subjective clinical judgment and is not easily quantified unless the pattern is obvious, such as the Kussmaul type of breathing in patients with diabetic ketoacidosis.

Significant changes in the rhythm of breathing occur during the first months of life. Respiratory pauses of less than 6 seconds are common in infants under 3 months of age. If these pauses occur in groups of three or more that are separated by less than 20 seconds of respiration, the pattern is referred to as periodic breathing. This pattern is very common in premature infants after the first days of life and may persist until 44 weeks post-conceptual age. In full-term infants, periodic breathing is usually observed between 1 week and 2 months of age and



■ **FIGURE 6-1.** **A** and **B**, Mean values (solid line) \pm 2 SD (dotted lines) of the normal respiratory rate at rest (during sleep in children under 3 years of age). There is no significant difference between the sexes, and the regression lines represent data from both boys and girls. The respiratory rate decreases with age and shows the greatest normal variation during the first 2 years of life. (**A**, Data from Rusconi F, Castagneto M, Gagliardi L, et al: Reference values for respiratory rate in the first 3 years of life. *Pediatrics* 1994;94:350. **B**, Data from Hooker EA, Danzl DF, Brueggmeyer M, Harper E: Respiratory rates in pediatric emergency patients. *J Emerg Med* 1992;10:407.)

is normally absent by 6 months. Apnea with cessation of air flow lasting more than 15 seconds is uncommon and may be accompanied by bradycardia and cyanosis. In preterm infants, a drop in oxygen saturation may be seen up to 7 seconds after a respiratory pause when in room air and up to 9 seconds later when on supplemental oxygen.

Other abnormal patterns include Cheyne-Stokes breathing, which occurs as cycles of increasing and decreasing tidal volumes separated by apnea (e.g., in children with congestive heart failure and increased intracranial pressure). Biot breathing consists of irregular cycles of respiration at variable tidal volumes interrupted by apnea and is an ominous finding in patients with severe brain damage.

After noting the rate and rhythm of breathing, the physician should look for signs of increased respiratory effort. The older child will be able to communicate the subjective experience of difficult breathing, or dyspnea. Objective signs that reflect distressed breathing are chest wall retractions; visible use of accessory muscles and the alae nasi; orthopnea; and paradoxical respiratory movements. The more negative intrapleural pressure during inspiration against a high airway resistance leads to retraction of the pliable portions of the chest wall, including the inter- and subcostal tissues and the supraclavicular and suprasternal fossae. Conversely, bulging of intercostal spaces may be seen when pleural pressure becomes greatly positive during a maximally forced expiration. Retractions are more easily visible in the newborn infant, in whom intercostal tissues are thinner and more compliant than in the older child.

Visible contraction of the sternocleidomastoid muscles and indrawing of supraclavicular fossae during inspiration are among the most reliable clinical signs of airway obstruction. In young infants, these muscular contractions may lead to head bobbing, which is best observed when the child rests with the head supported slightly at the suboccipital area. If no other signs of respiratory distress are present in an infant with head bobbing, however, central nervous system disorders, such as third ventricular cysts, should be considered. Older patients with chronic airway obstruction and extensive use of accessory muscles may appear to have a short neck because of hunched shoulders. Orthopnea exists when the patient is unable to tolerate a recumbent position.

Flaring of the alae nasi is a sensitive sign of respiratory distress and may be present when inspiration is abnormally short (e.g., under conditions of chest pain). Nasal flaring enlarges the anterior nasal passages and reduces upper and total airway resistance. It may also help to stabilize the upper

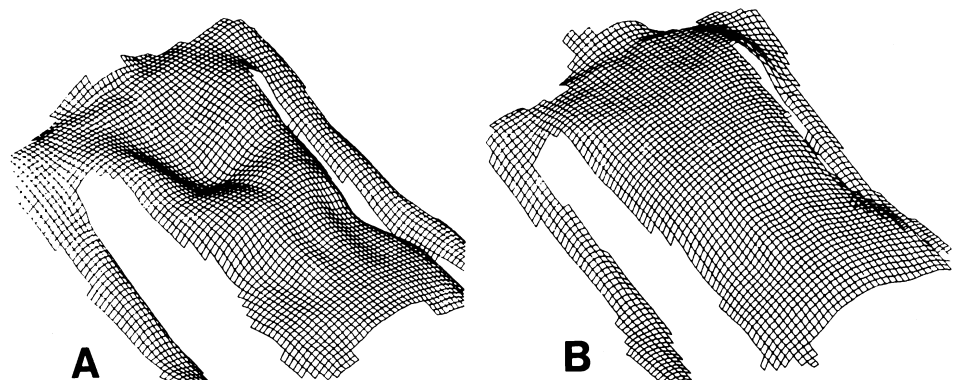
airways by preventing large negative pharyngeal pressures during inspiration.

The normal movement of chest and abdominal walls is directed outward during inspiration. Inward motion of the chest wall during inspiration is called paradoxical breathing. This is seen when the thoracic cage loses its stability and becomes distorted by the action of the diaphragm. Classically, paradoxical breathing with a seesaw type of thoracoabdominal motion is seen in patients with paralysis of the intercostal muscles, but it is also commonly seen in premature and newborn infants who have a very compliant rib cage. Inspiratory indrawing of the lateral chest is known as Hoover's sign and can be observed in patients with obstructive airway disease. Paradoxical breathing also occurs during sleep in patients with upper airway obstruction. The development of paradoxical breathing in an awake, nonparalyzed patient beyond the newborn period usually indicates respiratory muscle fatigue and impending respiratory failure.

Following inspection of the breathing pattern, the examiner should pay attention to the symmetry of respiratory chest excursions. Unilateral diseases affecting lungs, pleura, chest wall, or diaphragm may all result in asymmetric breathing movements. Trauma to the rib cage may cause fractures and a "flail chest" that shows local paradoxical movement. Pain during respiration usually leads to "splinting" with flexion of the trunk toward and decreased respiratory movements of the affected side. The signs of hemidiaphragmatic paralysis may be subtle and are usually more noticeable in the lateral decubitus position with the paralyzed diaphragm placed up. This position tends to accentuate the paradoxical inward epigastric motion on the affected side.

Other methods to augment inspection of chest wall motion use optical markers. In practice, this technique is done by placing both hands on either side of the patient's lateral rib cage with the thumbs along the costal margins. Divergence of the thumbs during expansion of the thorax supposedly aids in the visual perception of the range and symmetry of respiratory movements. This technique is of little use in children. A more accurate method of documenting the vectors of movement at different sites (but one that is not yet practical for bedside evaluation) is to place a grid of optical markers on the chest surface and film their positional changes during respiration relative to a steady reference frame. A similar concept is used in optical studies of chest deformities. Projection of raster lines onto the anterior chest surface allows stereographic measurement of deformities, such as pectus excavatum, and augments the visual image of the surface shape (Fig. 6-2). In practice and without such tools,

■ **FIGURE 6-2.** Optical markers augment the visual perception of chest wall deformities. In this example of rasterstereography, lines are projected onto the anterior thorax, and the surface image is computed as a regular network. The change of the funnel chest deformity before (A) and after surgery (B) is easily appreciated. In practice and at the bedside, the physician should inspect at different angles of illumination to enhance the visual perception of chest wall deformities. (From Hierholzer E, Schier F: Rasterstereography in the measurement and postoperative follow-up of anterior chest wall deformities. *Z Kinderchir* 1986;41: 267-271.)

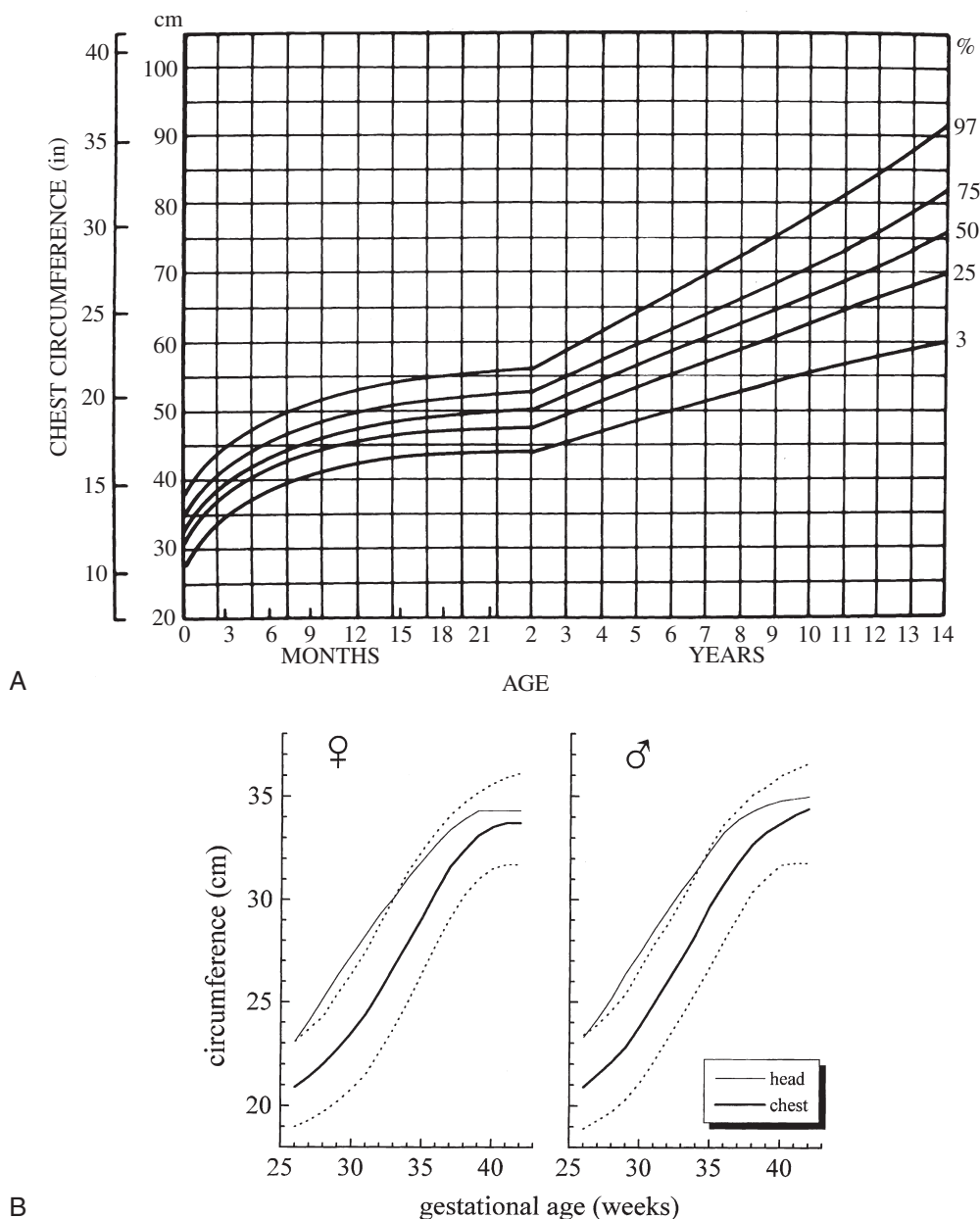


however, the physician should inspect the chest at different angles of illumination to enhance the visual perception of chest wall deformities. Their location, size, symmetry, and change with respiratory or cardiac movements should be noted.

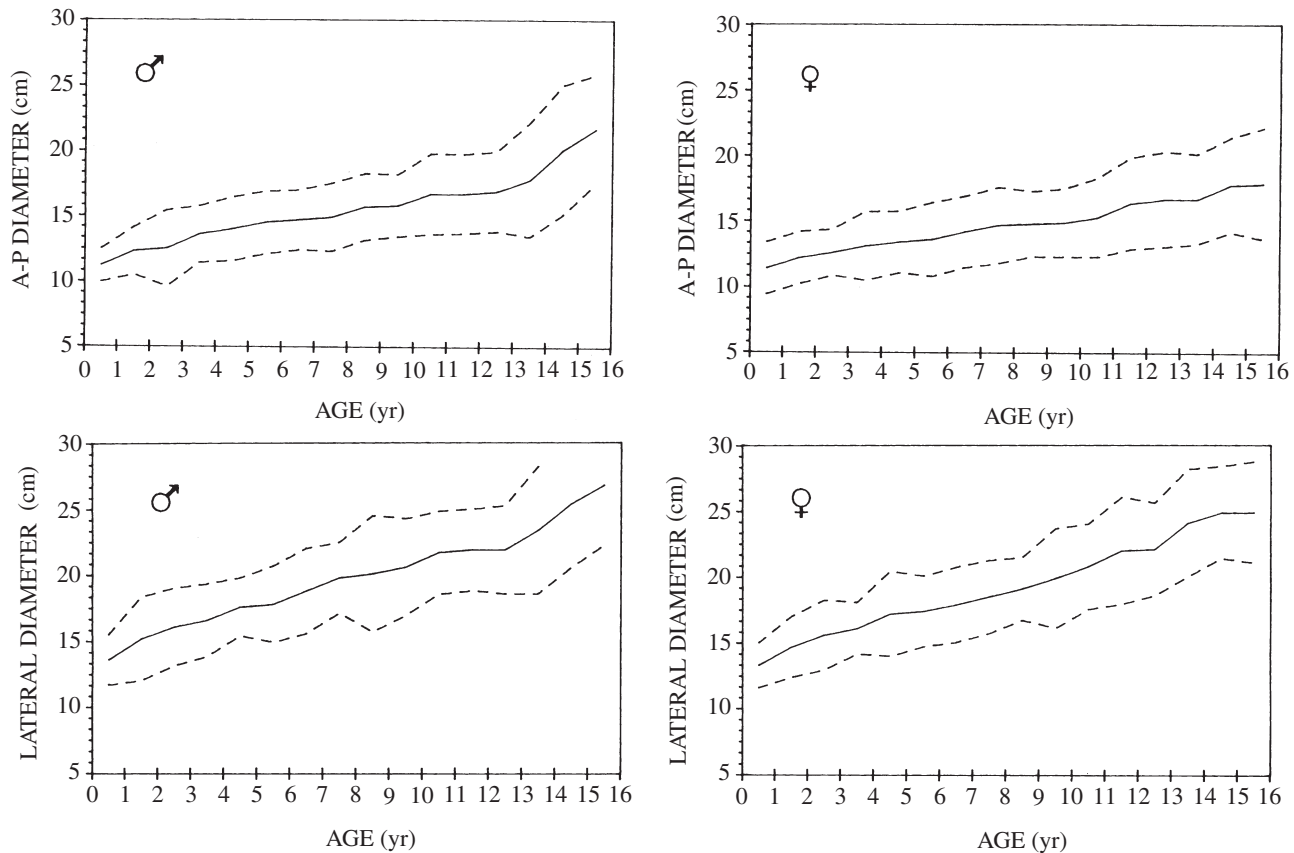
The dimensions of the chest should be measured. Chest size and shape are influenced by ethnic and geographic factors that should be taken into account when measurements are compared with normative data. Andean children who live at high altitudes, for example, have larger chest dimensions relative to stature than children in North America. The chest circumference is usually taken at the mamillary level during midinspiration. In practice, mean readings during inspiration and expiration should be

noted (Fig. 6-3A). Premature infants have a greater head circumference than chest circumference, while these measurements are very similar at term (see Fig. 6-3B). Malnutrition can delay the time at which chest circumference begins to exceed head circumference.

Further objective documentation of the chest configuration may include measurements of thoracic depth (anteroposterior [AP] diameter) and width (transverse diameter). The thoracic index, or the ratio of AP over transverse diameter, is close to unity in infants and decreases during childhood. Measurements should be taken with a caliper at the level of the nipples in upright subjects. Normative values for young children are available but



■ **FIGURE 6-3.** A, Normal distribution of chest circumference from birth to 14 years. Tape measurements are made at the mamillary level during midinspiration. Before plotting the values on the graph, one should add 1 cm for males and subtract 1 cm for females between 2 and 12 years of age. B, Normal distribution of chest circumference from 26 to 42 weeks of gestation. The dotted lines indicate the 10th and 90th percentiles, respectively. Note that chest circumference is close to head circumference at term. (A, From Feingold M, Bossert WH: Normal values for selected physical parameters. An aid to syndrome delineation. Birth Defects 1974;10(13):14. B, Data from Britton JR, Britton HL, Jennett R, et al: Weight, length, head and chest circumference at birth in Phoenix, Arizona. J Reprod Med 1993;38:215.)



■ **FIGURE 6-4.** Mean values (solid line) \pm SD (dashed lines) of the normal distribution of anteroposterior (AP) and lateral chest diameters in boys and girls. Caliper measurements are made at the mamillary level during midinspiration. (Data from Lucas WP, Pryor HB: Range and standard deviations of certain physical measurements in healthy children. *J Pediatr* 1935;6:533-545.)

dated (Fig. 6-4). Most of the configurational change of the chest occurs during the first 2 years and is probably influenced by gravitational forces after the upright position becomes common. Disease-related changes in thoracic dimensions occur either as potential causative factors (e.g., the elongated thorax with a stress distribution that favors spontaneous pneumothorax in lanky adolescents, particularly males who increase their thoracic height vs. width more than females) or as a secondary event (e.g., the barrel-shaped chest in patients with emphysema and chronic hyperinflation of the lung).

Inspection of the patient should also be directed to the extrathoracic regions. Many observations on the examination of the head and neck provide valuable clues to the physical diagnosis. Bluish coloration of the lower eyelid ("allergic shiners"); a bilateral fold of skin just below the lower eyelid (Dennie lines); and a transverse crease from "allergic salutes," running at the junction of the cartilaginous and bony portion of the nose, may all be found in atopic individuals. The nose should always be examined, and bilateral patency should be documented by occluding each side while feeling and listening for airflow through the other nostril. Even without a speculum one can assess the anterior half by raising the nose tip with one thumb and shining a light into the nasal passageways. Color and size of the mucosa should be noted. The frequency of asymptomatic nasal polyps seems to be high. Most polyps arise from the mucosa of the ostia, clefts, and recesses in the ostiomeatal complex. Easily visible nasal polyps are common in patients with

cystic fibrosis. Nasal polyposis may also be familial or associated with allergy, asthma, and aspirin intolerance.

The oropharynx should be inspected for its size and signs of malformation, such as cleft palate, and for signs of obstruction by enlarged tonsils. Evidence of chronic ear infections should be documented, and the areas over frontal and maxillary paranasal sinuses should be tested for tenderness. Inspection of the skin is important and may reveal the eczema of atopy. The finding of a scar that typically develops at the site of a successful bacillus Calmette-Guérin (BCG) vaccination may be relevant. Common physical findings such as cyanosis, clubbing, and the cardiovascular signs of pulmonary disease are discussed in more detail at the end of this chapter.

■ PALPATION

Palpation follows chest inspection to confirm observed abnormalities, such as swellings and deformations; to identify areas of tenderness or lymph nodal enlargement; to document the position of the trachea; to assess respiratory excursions; and to detect changes in the transmission of voice sounds through the chest. Chest palpation may offer the first physical contact with the patient, and it is very important for the physician to perform this procedure with warm hands.

Palpation should be done in an orderly sequence. Commonly, one begins with an examination of the head and neck. Cervical lymphadenopathy and tenderness over paranasal sinuses should

be noted. Palpation of the oropharynx may be indicated to find malformations such as submucosal clefts or to identify causes of upper airway obstruction. The position of the trachea must be documented in every patient. This is a very important part of the physical chest examination because tracheal deviation most often indicates significant intra- or extrathoracic abnormalities.

In the older child, the tracheal position is assessed by placing the index and the ring fingers on both sternal attachments of the sternocleidomastoid muscles. The trachea is then felt between these landmarks with the middle finger on the suprasternal notch. In small children, palpation is done with one index finger sliding gently inward over the suprasternal notch. Looking for asymmetry, the physician should always make sure that the patient is in a straight position, and deformities, such as scoliosis, should be taken into account.

A very slight deviation of the trachea toward the right is normal. Marked deviations may indicate a pulling force toward the side of displacement (e.g., atelectasis) or a pushing force on the contralateral side (e.g., pneumothorax). The physician should note whether the displacement is fixed or whether there is a pendular movement of the trachea during inspiration and expiration that may suggest obstruction of a large bronchus. Posterior displacement of the trachea may occur with anterior mediastinal tumors or barrel chest deformities, whereas an easily palpable anteriorly displaced trachea is sometimes seen with mediastinitis. In patients with airway obstruction and respiratory distress, retractions of the suprasternal fossa may be seen, and a “tracheal tug” may be felt by the examiner.

Placing the hands on both sides of the lateral rib cage, the physician should feel for symmetry of chest expansion during regular and deep breathing maneuvers. Slight compression of the chest in the transverse and anteroposterior directions may help to localize pain from lesions of the bony structures. Voice-generated vibrations are best felt with the palms of both hands just below the base of the fingers placed over corresponding sites on the right and left hemithorax. Asymmetric transmission usually indicates unilateral intrathoracic abnormalities. The patient is asked to produce low-frequency vibrations of sufficient amplitude by saying “ninety-nine” in a loud voice. In young infants, crying may produce the vibrations that are felt as tactile fremitus over the chest wall. This fremitus is decreased if an accumulation of air or fluid in the pleural space reduces transmission. Small consolidations of the underlying lung will not diminish the tactile fremitus as long as the airways remain open, whereas collapse of the airways and atelectasis will reduce the transmission of vibratory energy if larger portions of the lung are affected.

■ AUSCULTATION

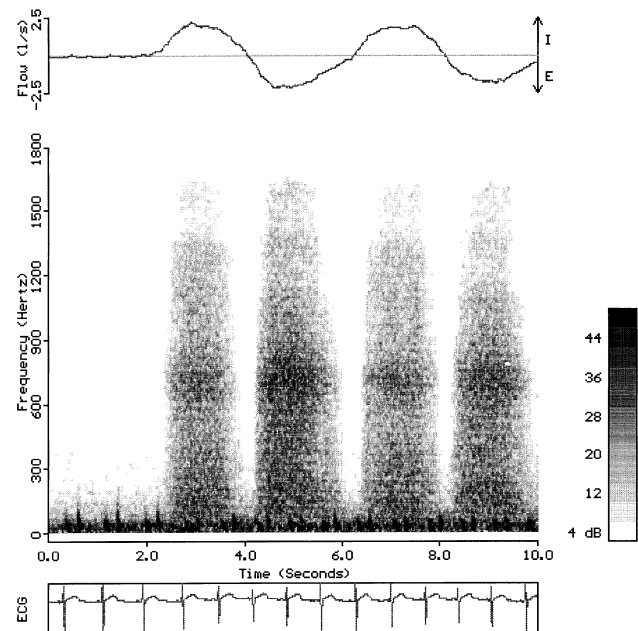
Auscultation is arguably the most important part of the physical chest examination. The subjective perception of respiratory acoustic signs is influenced by the site and mode of sound production; by the modification of sound on its passage through the lung, chest wall, and stethoscope; and finally by the auditory system of the examiner. Knowledge about these factors is necessary to appreciate fully the wealth of information that is contained in the acoustic signs of the thorax.

Thoracic Acoustics

Observations on sound generation in airway models and electronic analyses of respiratory sounds suggest a predominant

origin from complex turbulences within the central airways. The tracheal breath sound heard above the suprasternal notch is a relatively broad-spectrum noise, ranging in frequency from less than 100 Hz to greater than 2000 Hz. Resonances from the trachea and from supraglottic airways “color” the sound (Fig. 6-5). Lengthening of the trachea with growth during childhood causes lower tracheal resonance frequencies. A dominant source of tracheal breath sounds is turbulence from the jet flow at the glottic aperture. However, narrow segments of the supraglottic passages also contribute to sound generation. There is a very close relationship between airflow and tracheal sound intensity, particularly at high frequencies. In the presence of local narrowing (e.g., in children with subglottic stenosis), flow velocity at the stenotic site is increased, and so is the tracheal sound intensity. Relating tracheal sound levels to airflow measured at the mouth can provide information about changes during therapy. Auscultation over the trachea will provide some information under these circumstances, but objective acoustic measurements are required for accurate comparisons.

Basic “normal” lung sounds heard at the chest surface are lower in frequency than tracheal sounds because sound energy is lost during passage through the lungs, particularly at higher frequencies. However, lung sounds extend to frequencies higher than traditionally recognized. New observations on the effects of gas density indicate that lung sounds at frequencies above 400 Hz are mostly generated by flow turbulence. At lower frequencies, other mechanisms that are not directly related to air flow (e.g., muscle noise and thoracic cavity resonances) have

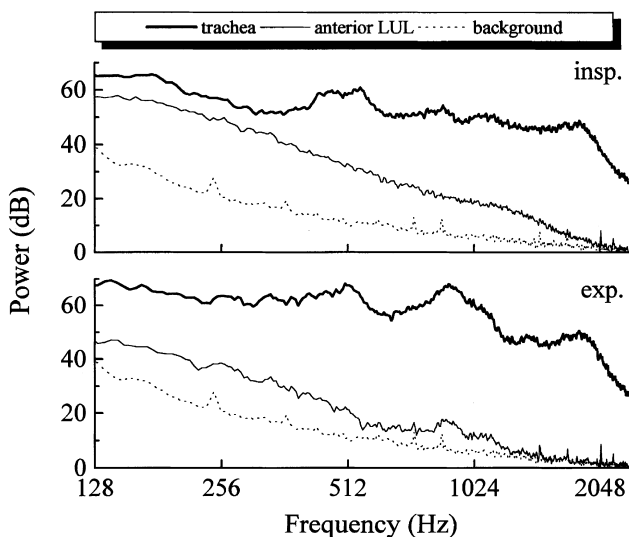


■ **FIGURE 6-5.** Digital respirosoundrogram of sounds recorded over the trachea of a healthy young man. Time is on the horizontal axis, frequency is on the vertical axis, and sound intensity is shown on a scale from black (loud) to white (low). Airflow is plotted at the top, with inspiration above and expiration below the zero line. The sonogram illustrates the broad range of tracheal sounds during both inspiration and expiration. There is a distinct pause between the respiratory phases. Expiration is louder than inspiration, and resonance is apparent around 700 Hz. In this example, the subject was holding his breath at the beginning. During this respiratory pause, heart sounds below 200 Hz are easily identified by their temporal relation to the simultaneously recorded electrocardiogram (ECG).

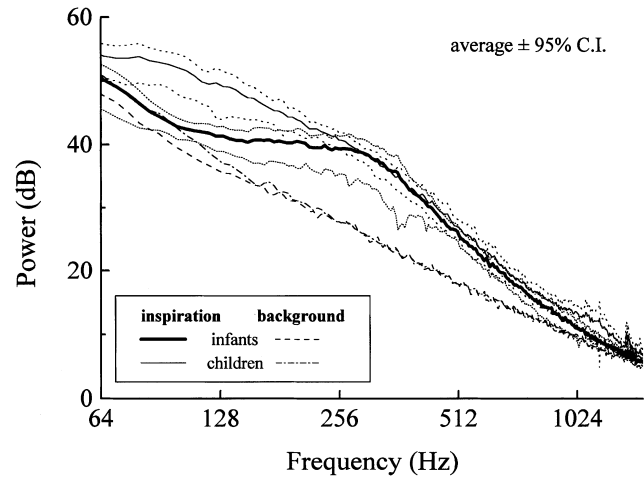
prominent effects on lung sounds and gas density effects are less obvious. Inspiratory lung sounds show little contribution of noise generated at the glottis. Their origin is likely more peripheral (i.e., in the main and segmental bronchi). Expiratory lung sounds appear to have a central origin and are probably affected by flow convergence at airway bifurcations (Fig. 6-6).

Sound at different frequencies takes different pathways on the passage through the lung. Low-frequency sound waves propagate from central airways through the lung parenchyma to the chest wall. At higher frequencies, the airway walls become effectively more rigid and sound travels further down into the airways before it propagates through lung tissue. This information cannot be gathered on subjective auscultation but requires objective acoustic measurements. A trained ear, however, will recognize many of the findings that are related to these mechanisms. For example, lung sounds in healthy children and adults are not necessarily equal at corresponding sites over both lungs. In fact, expiratory sounds are typically louder at the right upper lobe compared with the left side. Similar asymmetry has been recognized when sound is introduced at the mouth and measured at the chest surface. A likely explanation for this asymmetry is the effect on sound propagation by the cardiovascular and mediastinal structures to the left of the trachea. Asymmetry of lung sounds is also noticeable in most healthy subjects during inspiration when one listens over the posterior lower chest. The left side tends to be louder here, probably because of the size and spatial orientation of the larger airways due to the heart.

Objective acoustic measurements have also helped to clarify the difference between lung sounds in newborn infants and in older children. The most obvious divergence occurs in lung sounds at low frequencies where newborn infants have much less intensity. This may be explained by thoracic and airway



■ **FIGURE 6-6.** Average spectra of respiratory sounds at airflow of 1.5 ± 0.3 L/sec, recorded simultaneously at the suprasternal notch (trachea) and at the second intercostal space in the midclavicular line on the left (anterior left upper lobe [LUL]) of a healthy 12-year-old boy. The average sound spectrum during breath holding at resting end expiration (background) is plotted for comparison. Inspiratory lung sounds are louder than expiratory sounds, while the opposite is true for tracheal sounds. Lung sound intensity is clearly above background at frequencies as high as 1000 Hz. Expiratory lung sounds show some of the same spectral peaks that are present in tracheal sounds.



■ **FIGURE 6-7.** Average spectra of inspiratory lung sounds, recorded over the posterior basal segment of the right lung in healthy newborn infants ($n = 10$) and children ($n = 9$) at airflows of 15 mL/s/kg. The spectra of background noise at resting end expiration are plotted for comparison. Dotted lines mark the 95% confidence intervals (C.I.). Note the similarity of spectral slopes in newborn infants and older children at frequencies above 300 Hz and the significantly reduced sound power at lower frequencies in newborns. (Data from Pasterkamp H, Powell RE, Sanchez I: Lung sound spectra at standardized air flow in normal infants, children, and adults. *Am J Respir Crit Care Med* 1996;154:424–430.)

resonances at higher frequencies in newborn infants and perhaps also by their lower muscle mass. Lung sounds at higher frequencies are similar between newborn infants and older children (Fig. 6-7).

Adventitious respiratory sounds usually indicate respiratory disease. Wheezes are musical, continuous (typically longer than 100 msec) sounds that originate from oscillations in narrowed airways. The frequency of the oscillation depends on the mass and elasticity of the airway wall as well as on local airflow. Widespread narrowing of airways in asthma leads to various pitches, or polyphonic wheezing, whereas a fixed obstruction in a larger airway produces a single wheeze, or monophonic wheezing. Expiratory wheezing is related to flow limitation and can be produced by normal subjects during forced expiratory maneuvers. The situation is less clear for wheezing during inspiration, which is common in asthma but cannot be produced by healthy subjects unless it originates from the larynx (e.g., in vocal cord dysfunction). Very brief and localized inspiratory wheezes may be heard over areas of bronchiectasis.

Crackles are nonmusical, discontinuous (less than 20 msec duration) lung sounds. Crackle production requires the presence of air-fluid interfaces and occurs either by air movement through secretions or by sudden equalization of gas pressure. Another mechanism may be the release of tissue tension during sudden opening or closing of airways. Crackles are perceived as fine or coarse, depending on the duration and frequency of the brief and dampened vibrations created by these mechanisms. There may be a musical quality to the sound if a short oscillation occurs at the generation site. This has been called tinkling crackle or squawk and may appear during inspiration, typically in patients with interstitial lung diseases. Fine crackles during late inspiration are common in restrictive lung diseases and in the early stages of congestive heart failure, whereas coarse crackles during early inspiration and during expiration are frequently

heard in chronic obstructive lung disease. Fine crackles are usually inaudible at the mouth, whereas the coarse crackles of widespread airway obstruction can be transmitted through the large airways and may be heard as clicks with the stethoscope held in front of the patient's open mouth. Some crackles over the anterior chest may occur in normal subjects who were breathing at low lung volumes, but they will disappear after a few deep breaths.

Several other abnormal respiratory sounds are not generated in intrathoracic airways. Pleural rubs originate from mechanical stretching of the pleura, which causes vibration of the chest wall and local pulmonary parenchyma. These sounds can occur during both inspiration and expiration. Their character is like that of creaking leather and is similar in some ways to pulmonary crackles. Stridor refers to a more or less musical sound that is produced by oscillations of critically narrowed extrathoracic airways. It is therefore most commonly heard during inspiration. Grunting is an expiratory sound, usually low pitched and with musical qualities. It is produced in the larynx when vocal cord adduction is used to generate positive end-expiratory pressures, such as in premature infants with immature lungs and surfactant deficiency. Snoring originates from the flutter of tissues in the pharynx and has less musical qualities. It may be present during both inspiration and expiration.

There may also be cardiorespiratory sounds. These are believed to occur when cardiac movements cause regional flows of air in the surrounding lung. Because of its synchronicity with the heart beat, this sound may be mistaken for a cardiac murmur. It can be identified by its vesicular sound quality and its exaggeration during inspiration and in different body positions.

At the boundary between different tissues, reflection of sound may occur and sound transmission may decrease, depending on the matching or mismatching of the tissue impedances. Many of the acoustic signs of the chest are explained on the basis of impedance matching alone. The stethoscope is basically an impedance transformer that reduces sound reflection at a mismatched interface, namely, body surface to air. Because it is the only part of the sound transmission pathway that can be kept constant, it is best to always use the same stethoscope. The choice of a bell- or a diaphragm-type stethoscope depends on individual preference. Diaphragm chest pieces can be placed more easily and with less pressure on small chests with narrow intercostal spaces. Compared with bell-type stethoscopes, they tend to deemphasize frequencies below 100 Hz. Both the bell-type and the diaphragm stethoscopes show some attenuation at frequencies above 400 Hz.

Technique of Auscultation

Ideally, auscultation of the chest should be performed in a quiet room; however, with pediatric patients the usual setting may be anything but quiet. Fortunately, the human auditory system allows selective evaluation of acoustic signals even when they are masked by much louder surrounding noises. This psychoacoustic phenomenon, known as the "cocktail party effect," at present cannot be reproduced by modern electronic techniques, which is but one of the reasons for the lasting popularity of the stethoscope.

This instrument, the most widely used in clinical medicine since its introduction almost 200 years ago, carries symbolic value for the health care profession, much like a modern staff of Aesculapius. Every child knows that doctors have stethoscopes.

The physician should use this to advantage when assessing pediatric patients by encouraging children to listen themselves to their heartbeats and breathing sounds. Even infants may be fascinated as long as the stethoscope is shiny. Ice cold chest pieces, on the other hand, scare off most patients.

The patient should be in a straight position during auscultation because incurvature of the trunk may lead to artificial side differences of sound production and transmission. In newborns and young infants, a straight position may be best achieved when they are supine. Infants and toddlers will often be assessed while their parents hold them on their laps. Beginning auscultation on the back of these young patients will provoke less anxiety than a frontal approach. Older children can be examined in the sitting or standing position. The number of sites over the chest that are assessed during auscultation will be determined by the clinical situation. Ideally, all segments of the lung should be listened to, but this may not be possible, particularly in very young children.

Because the intensity of respiratory sounds is related to air-flow, sufficiently deep respirations (with flow >0.5 L/sec) are needed for a good sound signal. An older patient will cooperate and breathe deeply through an open mouth. With infants and young children, however, one may have to rely on sounds made during sighs or deep inspirations in between crying. On the other hand, normal breath sounds can mask the presence of some adventitious sounds (e.g., fine crackles of low intensity). Asking the patient to take very slow, deep breaths with less airflow than is needed to generate normal breath sounds can help to unmask these adventitious sounds.

The physician should make note of the lung sound intensity over different areas of the chest in a qualitative way, keeping in mind that this intensity reflects both local sound generation and sound transmission characteristics of the thorax. It is therefore not correct to speak of local "air entry" when one actually refers to local breath sound intensity. Decreased breath sounds, for example, are common in asthma even when normal blood gases indicate that air entry has to be adequate. Obviously, a qualitative distinction between absence or presence of local breath sounds will be easier than attempts at quantification. Also, when the stethoscope is placed over any given location, it is not known how large an area of the underlying lung is actually being assessed. In adult subjects, moving the chest piece of the stethoscope by 10 cm will position it to receive sound from entirely different lung units, but similar data for children are not available.

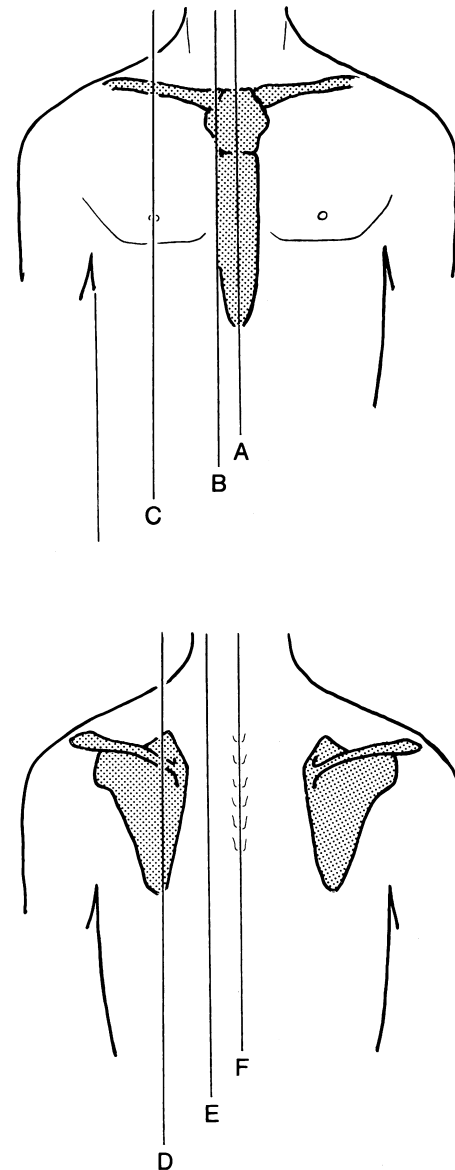
Assessment of regional ventilation by thoracic acoustic signs becomes more meaningful when two sites are compared simultaneously. Differential auscultation with special stethoscopes that employ two chest pieces or a single divided chest piece has not become popular in clinical practice. Comparative auscultation is absolutely essential for airway management in the emergency room and intensive care unit for assessment of endotracheal tube position or for identification of the side of a pneumothorax. Listening simultaneously to two homologous sites over both lungs may also help to detect local abnormalities. Atelectatic areas will transmit sound more slowly than inflated lung tissue, but the resulting phase shift is too small to be detectable on subjective auscultation. With local airway narrowing, however, the maximum sound intensity over the affected side may become sufficiently delayed to be perceived as "phase heterophony." In some cases, breath sounds may still be audible over the affected side after inspiratory efforts have ceased. This "post-effort" breath sound is a sign of incomplete airway obstruction.

There are special circumstances in which only the presence or absence of breath sounds is of interest (e.g., during transportation of critically ill patients in noisy vehicles and during resuscitation in the emergency department). Under these conditions, and when a firm attachment of the chest piece is important, a self-adhering stethoscope, based on negative suction pressure within the bell of the chest piece, may be applied. New techniques of adaptive electronic filtering are soon to be employed in stethoscopes that are optimized for use in very noisy environments.

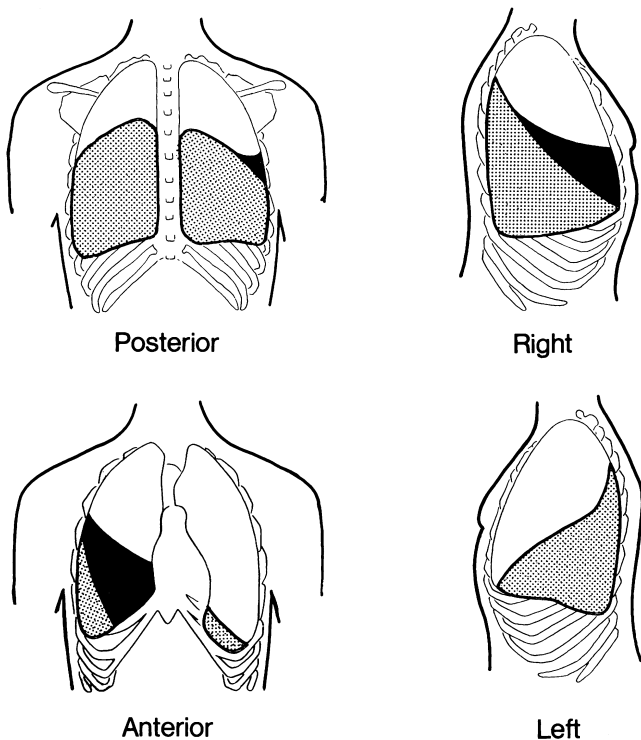
Respiratory sounds should be documented according to their location and character. Normal projections of lobar borders to the surface of the chest are shown in Figure 6-8. These may be distorted by local pulmonary disease, and mapping of respiratory sounds should therefore be done with reference to external anatomic landmarks (Fig. 6-9). The examiner should be familiar with the segmental structure of the underlying lung.

Respiratory sound characteristics include the intensity (amplitude), pitch (predominant frequency), and timing during the respiratory cycle. Also, sounds will have a particular timbre (character) caused by the presence of resonances and overtones. Unfortunately, the terminology in use for the description of respiratory sounds is still confusing and imprecise. During a symposium on lung sounds in Tokyo in 1985, an attempt was made to achieve a global and uniform nomenclature for breath sounds. The resulting recommendations for classification of adventitious lung sounds are presented in Figure 6-10, and Table 6-1 summarizes mechanisms and sites of generation, acoustic characteristics, and clinical relevance of the major categories of respiratory sounds.

A basic grouping into musical, continuous sounds of long duration and nonmusical, discontinuous sounds of short duration



■ **FIGURE 6-9.** Vertical reference lines of the chest. The center line is indicated anteriorly by the suprasternal notch (*A*) and posteriorly by the spinous processes (*F*). The sternal (*B*) and midclavicular (*C*) lines over the front and the scapular (*D*) and paravertebral (*E*) lines over the back provide longitudinal landmarks of the thorax. From a lateral view, the midaxillary line is used for orientation. Horizontal reference points are the supra- and infraclavicular fossae, Ludwig's angle (junction of the second rib at the sternum), the mammillae (normally at the fourth rib), and the epigastric angle. Posteriorly, the prominent spinous process of the seventh cervical vertebra and the supra- and infraspinous fossae of the scapulae provide markers for orientation.



■ **FIGURE 6-8.** Projections of the pulmonary lobes on the chest surface. The upper lobes are white, the right-middle lobe is black, and the lower lobes are dotted.

is made, with the former being referred to as wheezes and the latter as crackles. Furthermore, musical adventitious sounds or wheezes may be classified as high or low pitched. Some use the term *rhonchus* for low-pitched wheezes (<200 Hz), whereas others describe the poorly characterized "secretion sounds," which share musical and nonmusical qualities, as rhonchi. Crackles are subclassified as fine or coarse. Regular breath sounds include tracheal/bronchial, bronchovesicular, and vesicular/normal sounds. Finally, other respiratory sounds should be specified, such as pleural rubs, expiratory grunting, and inspiratory stridor. Historical terms such as *rales* and *crepitations* should

LUNG SOUND NOMENCLATURE

| | English | French | German | Japanese | Portuguese | Spanish |
|---|-----------------|----------------------------------|----------------|----------|--------------------|--------------------|
| Discontinuous | | | | | | |
| Fine | | | | | | |
| (high pitched, low amplitude, short duration) | Fine crackles | Râles crepitants | Feines Rasseln | 捻髪音 | Estertores finos | Estertores finos |
| Coarse | | | | | | |
| (low pitched, high amplitude, long duration) | Coarse crackles | Râles bulleux ou Sous-crepitants | Grobes Rasseln | 水泡音 | Estertores grossos | Estertores gruesos |
| Continuous | | | | | | |
| High pitched | | | | | | |
| | Wheezes | Râles sibilants | Pfeifen | ふえ(様)音 | Sibilos | Sibilancias |
| Low pitched | | | | | | |
| | Rhonchus | Râles ronflants | Brummen | いびき(様)音 | Roncos | Roncus |

■ **FIGURE 6-10.** Recommendation from the 1985 International Symposium on Lung Sounds in Tokyo for a unified nomenclature of adventitious sounds. (Reproduced with permission from Cugell DW: Lung sound nomenclature. *Am Rev Respir Dis* 1987;136:1016.)

be abandoned, and flowery descriptions such as “raspy” or “blowing” breath sounds should not be used because these adjectives are even less well defined than the suggested terms.

Several auscultatory signs are based on the transmission of voice sounds. Speech sounds have a fundamental note of about 130 Hz in men and 230 Hz in women, with overtones from 400 to 3500 Hz. Vowels are produced when particular pairs of overtones or formants are generated. On passage through the lung, the higher frequency formants are filtered, and speech heard over the chest becomes a meaningless mumble. With consolidation and transmission of higher frequency components, however, speech may become intelligible. This occurs with normal speech (bronchophony) and with whispered voice (pectoriloquy). There may be a change in vowels from e to a over areas of lung consolidation. The acoustic basis for these phenomena is the same as for bronchial breath sounds. The American Thoracic

Society and the American College of Chest Physicians recommend the term *egophony* for all of these findings.

■ PERCUSSION

Percussion is used to set tissues into vibration with an impulsive force so that their mechanical and acoustic response can be studied. If the vibrations are undamped and continue for a significant amount of time, the perceived sound will be resonant or “tympanic,” whereas rapid attenuation of the vibrations will lead to a flat or “dull” percussion note. The former occurs when there is a large acoustic mismatch (e.g., tissue overlying an air-filled cavity), whereas the latter occurs when the underlying tissue is similar to the surface tissue and vibratory energy propagates away quickly. Structures that absorb energy when struck by a sound at their natural frequency continue vibrating after the

■ **TABLE 6-1.** Respiratory sounds

| | Mechanisms | Origin | Acoustics | Relevance |
|----------------------------|--|--|---|--|
| Basic Sounds | | | | |
| Lung | Turbulent flow, vortices, other | Central (expiration), lobar to segmental airways (inspiration) | Low pass filtered noise (<100 to >1000 Hz) | Regional ventilation, airway caliber |
| Tracheal | Turbulent flow, flow impinging on airway walls | Pharynx, larynx, trachea, large airways | Noise with resonances (<100 to >3000 Hz) | Upper airway configuration |
| Adventitious Sounds | | | | |
| Wheezes | Airway wall flutter, vortex shedding, other | Central and lower airways | Sinusoidal (<100 Hz to >1000 Hz, duration typically >80 msec) | Airway obstruction, flow limitation |
| Rhonchi | Rupture of fluid films, airway wall vibration | Larger airways | Series of rapidly dampened sinusoids (typically <300 Hz and duration <100 msec) | Secretions, abnormal airway collapsibility |
| Crackles | Airway wall stress-relaxation | Central and lower airways | Rapidly dampened wave deflections (duration typically <20 msec) | Airway closure, secretions |

Modified from Pasterkamp H, Kraman SS, Wodicka GR: State of the art. Respiratory sounds—advances beyond the stethoscope. *Am J Respir Crit Care Med* 1997;156:974–987.

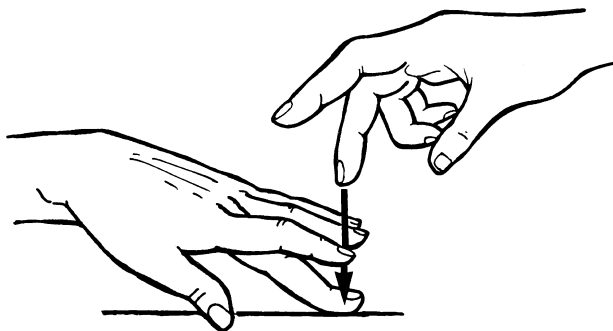
initial sound is gone and are called resonant. The fundamental resonance of the thorax depends on body size and is about 125 Hz for adult males, between 150 and 175 Hz for adult females, and between 300 and 400 Hz for small children.

Chest percussion in children is performed by light tapping with the index or middle finger (the plexor) on the terminal phalanx of the other hand's middle finger (the pleximeter). The pleximeter should be placed firmly but not hard, and care should be taken that other fingers do not touch the chest wall, which may cause artificial damping of the percussion note. Percussion should be gentle, with quick perpendicular movements of the plexor originating from the wrist (Fig. 6-11). The patient should be relaxed during the examination because tension of the chest wall muscles may alter the percussion note. More important, chest deformities and scoliosis in particular will have a significant effect on percussory findings.

Symmetric sites over the anterior, lateral, and posterior surface of the chest should be compared in an orderly fashion. As with chest auscultation, findings should be reported with reference to standard external landmarks (see Fig. 6-9). The ribs and vertebral spinous processes are used for horizontal mapping. The level at which the tympanic lung resonance changes to a dull percussion note should be defined over the posterior chest during maximal inspiration and expiration to delineate the lung borders and their respiratory excursions.

Subjective assessment of percussion note differences includes both acoustic and tactile perception. Tympanic, lower-pitched percussion notes mean less-damped vibrations of longer duration, which are felt by the pleximeter finger. Dull sounds with higher frequencies correspond to vibrations that die away quickly. Dullness replaces the normal chest percussion note when fluid accumulates in the pleural space or when consolidation close to the chest wall occurs in the underlying pulmonary parenchyma. Similar to the vibrations generated by percussion, the vibrations from the patient's voice ("say 'ninety-nine'") will also not be felt under these circumstances. However, the tactile fremitus is equally absent over areas of pneumothorax, whereas the percussion note may have a hyperresonant quality.

Conventional percussion cannot detect small pulmonary lesions located deeply within the thorax. Auscultatory percussion has been proposed to overcome this limitation. This technique combines light percussion of the sternum with simultaneous auscultation over the posterior chest. A decrease in sound intensity



■ **FIGURE 6-11.** Percussion in children should be done with gentle perpendicular movements from the wrist and tapping of the plexor finger (*right*) on the terminal phalanx of the pleximeter finger (*left*). The contact area of the pleximeter on the chest should be small, and other fingers should not touch the surface to avoid damping of the percussory vibrations.

is believed to indicate lung disease. The method is of little value, however, because even large intrathoracic lesions can remain undetected since percussion sounds either may be totally absorbed within the lung or may travel as transverse waves along the thoracic bones. Propagation along the thoracic bones is believed to explain the "coin sound," a sign that is used to detect a pneumothorax. It is elicited by creating a high-pitched sound impulse that propagates into the body, using a coin placed flat on the chest, which is then struck with another coin edge on. On auscultation of the opposite side of the thorax, a distinct click is heard if air separates the ribs from the lung and sound travels along these bones. Under normal circumstances with ribs and parenchyma in contact, however, the sound will be absorbed and no click will be heard.

■ TASTE AND SMELL

A complete physical examination extends beyond the perception of vision, hearing, and touch. Olfactory impressions should also be documented, even if they are subtle. Malodorous breath is easily noticed and may, particularly if chronic, indicate infection within the nasal or oral cavity (e.g., paranasal sinusitis), nasal foreign body, or dental abscess. Bad breath may also originate from intrathoracic infections, such as lung abscess or bronchiectasis, and it may also be noted in patients with gastroesophageal reflux. Nowadays physicians rarely use their tastebuds to make a medical diagnosis. One particular disease of the respiratory tract in children, however, lends itself to gustatory diagnosis. Most often the discovery is made by the mother of a patient with cystic fibrosis who notices that the skin of her child tastes abnormally salty.

■ COMMON SIGNS AND SYMPTOMS OF CHEST DISEASE IN CHILDREN

There are several common complaints and presentations of children with chest diseases that deserve a more detailed description. In particular, cough and sputum production, noisy breathing, wheezing, cyanosis, digital clubbing, cardiovascular signs, and chest pain need to be discussed.

■ COUGH AND SPUTUM PRODUCTION

Cough is not an illness by itself, but it is a cardinal manifestation in many chest diseases. Cough is probably the single most common complaint in children presenting to the physician. The act of coughing is a reflex aimed at removal of mucus and other material from the airways that follows the stimulation of cough or irritant receptors. These receptors are located anywhere between the pharynx and the terminal bronchioles. They send their afferent impulses via branches of the glossopharyngeal and vagus nerves to the cough center in the upper brainstem and pons. The efferent signals travel from the cough center via vagus, phrenic, and spinal motor nerves to the larynx and diaphragm as well as to the muscles of the chest wall, abdomen, and pelvic floor. Cortical influences allow the voluntary initiation or suppression of cough.

There are three phases of coughing: (1) deep inspiration; (2) closure of the glottis, relaxation of the diaphragm, and contraction of expiratory muscles; and (3) sudden opening of the glottis. During the second phase, intrathoracic pressures up to 300 mm Hg can be generated and may be transmitted to the vascular and cerebrospinal spaces. Airflow velocity during the

third phase is highest in the central airways and may reach three fourths the speed of sound. This speed depends on the sudden opening of the glottis and influences the success of expectoration. Patients with glottic dysfunction and those with tracheostomies may therefore have a less effective cough.

Stimuli that cause coughing may originate centrally, such as in psychogenic cough, or they may be pulmonary, located either in the major airways or in the pulmonary parenchyma. Also, cough can be provoked by nonpulmonary causes, such as irritation of pleura, diaphragm, or pericardium and even through stimulation of Arnold's nerve (a branch of the vagus) by wax or foreign bodies in the external ear.

A detailed history should define the nature of the cough, whether it is dry, hacking, or brassy, and whether it is productive by sound and appearance. In young children, expectoration is unusual, but if observed, the quantity and quality of sputum should be noted. In particular, the physician should inquire about the color and odor of the expectorate and about the presence of blood in the sputum. The yellow-green color of purulent sputum results from the cellular breakdown of leukocytes and the liberation of myeloperoxidase from these cells. This finding indicates a retention of secretions and does not necessarily reflect an acute infection.

The timing of coughing is important, and its relationship to daily routines should be sought. Cough during or after feeding occurs with aspiration. Nighttime cough may be related to asthma or to postnasal drip, whereas productive cough early in the morning is typical for bronchiectasis. Cough following exercise or exposure to cold air points toward airway hyperreactivity. Seasonal worsening or coughing on exposure to potential allergens should be documented, as should the association of coughing and wheezing. The physician should ask about active and passive smoking, keeping in mind that, regrettably, there are quite a few children as young as 8 years of age who smoke regularly.

A detailed diary kept by the parents or the patient to note the frequency and timing of cough can be of value. Technology to record, quantify, and characterize cough is being developed. Some acoustic characteristics of cough are quite specific for certain diseases, such as the sound of a barking seal in viral croup or the whooping noise in pertussis. In patients with chronic cough, the physician should weigh the possible causes in view of their prevalence at different ages (Table 6-2). Also, complications of severe coughing paroxysms, such as pneumothorax, cough syncope, or nonsyncopal neurologic manifestations, should be considered. Regarding the last, the physician should

■ **TABLE 6-2.** Causes of chronic cough

| Infant | Preschool | School Age/ Adolescence |
|---------------------------|-----------------|----------------------------|
| Congenital anomalies | Foreign body | Reactive |
| Tracheoesophageal fistula | Infections | Asthma |
| Neurologic impairment | Viral | Postnasal drip |
| Infections | Mycoplasma | Infections |
| Viral (RSV, CMV) | Bacterial | Mycoplasma |
| Chlamydia | Reactive | Irritative |
| Bacterial (pertussis) | Asthma | Smoking |
| Cystic fibrosis | Cystic fibrosis | Air pollution |
| | Irritative | Psychogenic |
| | Passive smoking | |

Data modified from Eigen H: The clinical evaluation of chronic cough. *Pediatr Clin North Am* 1982;29:67.

inquire about lightheadedness, headache, visual disturbance, paresthesia, and tremor.

■ NOISY BREATHING

Quite frequently a child is brought to the physician's attention because of abnormal breathing noises. This noise may be a non-musical hiss, much like the one produced in normal subjects at increased rates of ventilation, or it may have the musical qualities of stridor and snoring. Also, bubbling and crackling noises may be heard, and the tactile perception may contribute to the impression of a "rattly" chest in these patients.

Attention should be focused on the noise-generating structures of the extrathoracic airways that are located at points of anatomic narrowing (e.g., the nasal vestibule, the posterior nasal orifices, and the glottis). The most common cause of noisy breathing in toddlers and young children is nasopharyngeal obstruction; in young infants, laryngomalacia is a leading cause. It is uncertain to what degree sounds from large intrathoracic airways contribute to the noise of breathing. Placing the stethoscope within the airstream in front of the mouth, one hears predominantly those sounds that are produced locally in the mouth and larynx. Noisy breathing is a common finding in patients with asthma and bronchitis and does not necessarily reflect intrathoracic airway pathology because the upper airways are also frequently affected in these patients.

To clarify the causes of noisy breathing, the parents or patient should describe their own perceptions of the noise: does it occur during inspiration, expiration, or both? Is it just an exaggeration of the normal breath sound noise, or does it have musical qualities? Did an episode of choking precede the onset of noisy breathing? Is the abnormal sound more prominent during certain activities, such as exercise? At what times of day or night and in which body positions is it most noticeable? The physician should also inquire about associated cough, sputum production, and dyspnea.

Children may suffer from partial obstruction of the upper airways during sleep; complete obstruction, which is found in adult patients with sleep apnea, is less common. Invariably, these children are heavy snorers at night, whereas normal children's snoring is largely confined to times of upper respiratory tract infection. Usually enlarged adenoids and tonsils cause the breathing disturbance. The physician should inquire about the typical signs and symptoms found in patients with increased work of breathing and abnormal sleep patterns at night (Box 6-1).

In the older child and adolescent, the physician should first inspect the nasal passageways and proceed to an examination of the oropharynx before auscultation of the neck and thorax. The acoustic signs should be checked while the patient breathes first with the mouth open, then closed. In younger children, examination of the nose and mouth is unpopular and often results in agitation and crying. It is better to start with auscultation before inspection in these children. Noisy breathers should be examined when they are sitting or standing upright and when they are lying down because upper airway geometry is position dependent and may influence the respiratory sounds. The examiner should also note abnormal crying or speech in the patient, as this may point to laryngeal disease.

■ WHEEZING

Wheezing is a common respiratory symptom and refers to musical, adventitious lung sounds that are often heard by the patient

BOX 6-1 Clinical Symptoms in Children with Heavy Nocturnal Snoring**Nighttime Manifestations**

Profuse nocturnal sweating
 Restless sleep
 Abnormal movements during sleep
 Special sleeping position
 Enuresis

Problems with Growth and Nutrition

Anorexia
 Weight <3rd percentile
 Nausea with or without vomiting

Behavioral and Learning Problems

Hyperactivity
 Aggression
 Social withdrawal

Minor Motor Problems

Lack of coordination
 Clumsiness

Other Manifestations

Frequent upper airway infections
 Frequent morning headaches
 Excessive daytime somnolence

Data modified from Guillemainault C, Winkle R, Korobkin R, Simmons B: Children and nocturnal snoring. Evaluation of effects of sleep related respiratory resistive load and daytime functioning. *Eur J Pediatr* 1982;139:165.

as well as the physician. Stridor is even more noticeable. Essentially, it is a very loud inspiratory wheeze originating from extrathoracic airways. When asking the patient or the parents about wheezing and stridor, one should keep in mind that the use of lung sound terminology among nonprofessionals is no better standardized than it is among health care providers. Therefore, the physician should inquire about musical, whistling noises during respiration, and, if necessary, demonstrate stridor or the forced expiratory wheeze that can be produced even by healthy individuals.

Most typically, wheezing is associated with hyperreactive airway disease, but any critical narrowing of the airways can produce wheezing. Box 6-2 lists conditions other than asthma that may be associated with wheezing and stridor. The wheezing typical in asthma originates from oscillations of airways at many sites. On auscultation, one hears many different tones simultaneously, which is called polyphonic wheezing. Obstruction of a single airway can produce a single monophonic wheeze or, in the obstruction of extrathoracic airways, stridor. Both inspiratory and expiratory wheezes are present in the majority of asthmatic patients. The audible expiratory phase (expirium) is typically prolonged because of wheezing. Objectively measured expiratory time (expiration), however, is rarely prolonged except in very severe airway obstruction. Under these circumstances, airflow is minimal, and thus wheezing is absent. Respiration becomes ominously silent, and the patient may have carbon dioxide retention and cyanosis. In less severe cases, however, the

proportion of inspiration and expiration occupied by wheezing correlates to some extent with the degree of airflow obstruction. Objective and reproducible wheeze quantification can be achieved by computer-assisted techniques, but in practice the quantification of wheezing severity is made by subjective assessment at the bedside.

Wheezes are often high pitched and will therefore attenuate during their passage through lung tissue, particularly if the lungs are hyperinflated. Auscultation over the neck may give a better impression of respiratory sounds and should be included as a part of the routine physical examination. Tracheal auscultation to determine if and when there is wheezing after methacholine inhalation challenge has been advocated instead of spirometry in young children who are thought to have bronchial hyperreactivity. However, wheezing may be absent even if airways become significantly obstructed during bronchial provocation (Fig. 6-12). In our experience, wheezing heard at the chest but not necessarily at the trachea is very suggestive of airway narrowing and hyperresponsiveness. Listening to respiratory sounds over the neck may help to identify patients who are thought to be asthmatic but

BOX 6-2 Causes of Wheezing and Stridor Other than Asthma**Malformation**

Cardiovascular anomalies (e.g., vascular ring)
 Airway anomalies (e.g., web, cyst, hemangioma, malacia, stenosis)
 Esophageal anomalies (e.g., enteric cyst)

Inflammation

Tracheitis
 Bronchitis
 Bronchiolitis
 Bronchiectasis
 Cystic fibrosis

Compression*Extrinsic*

Esophageal foreign body
 Lymphadenopathy
 Malignancy

Intrinsic

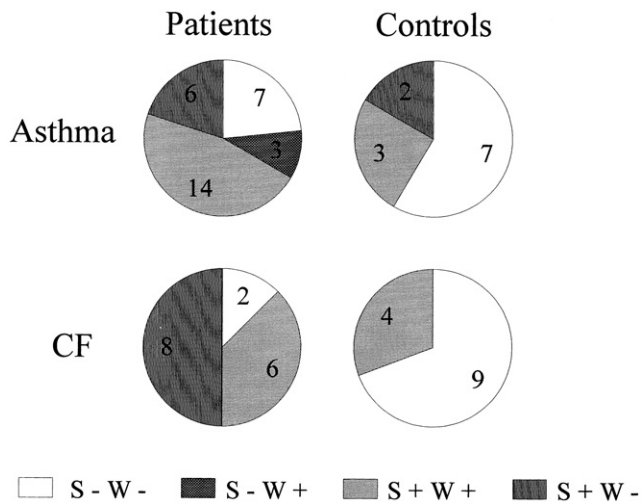
Endobronchial foreign body
 Tumor (rare)

Extrathoracic Disease

Laryngitis
 Epiglottitis
 Vocal cord paralysis
 Retropharyngeal abscess
 Peritonsillar abscess
 Laryngomalacia
 Polyps, adenoids

Other

Metabolic disturbances (e.g., hypocalcemia, hypokalemia)
 Psychosomatic illness (e.g., emotional laryngeal wheezing, factitious asthma)



■ **FIGURE 6-12.** Summary of findings regarding wheeze as an indicator of significant flow obstruction during methacholine bronchial provocation in children. Negative responses, i.e., less than 20% decline in forced expiratory volume in 1 second (FEV) at a dose of 8 mg/mL (spirometry) and no wheeze (acoustic monitoring), are indicated by a minus sign, positive responses by a plus sign. Note the low sensitivity of wheeze as an indicator of airflow obstruction, particularly in children with cystic fibrosis. S, spirometry; W, wheezing. (Data from Sanchez I, Powell RE, Pasterkamp H: Wheezing and airflow obstruction during methacholine challenge in children with cystic fibrosis and in normal children. *Am Rev Respir Dis* 1993;147:705, and Sanchez I, Avital A, Wong I, et al: Acoustic vs. spirometric assessment of bronchial responsiveness to methacholine in children. *Pediatr Pulmonol* 1993;15:28.)

who generate the wheezing noises solely in the larynx. These are usually older children and adolescents who may have emotional problems and vocal cord dysfunction.

■ CYANOSIS

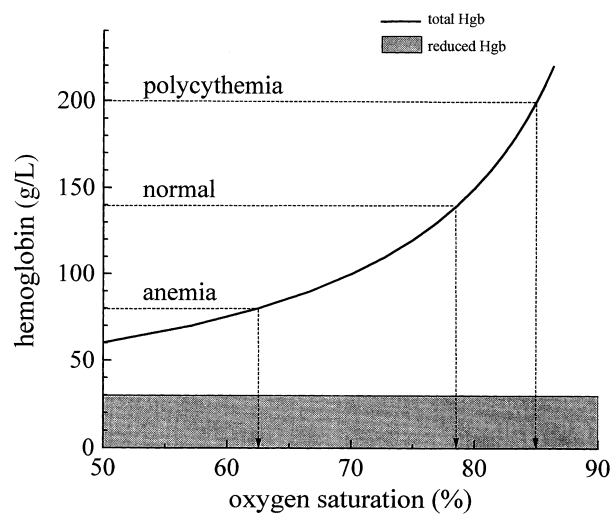
Cyanosis refers to a blue color of the skin and mucous membranes due to excessive concentrations of reduced hemoglobin in capillary blood. The oxygen content of capillary blood is assumed to be midway between that of arterial and that of venous blood. Areas with a high blood flow and a small arteriovenous oxygen difference (e.g., the tongue and mucous membranes) will not become cyanotic as readily as those with a low blood flow and a large arteriovenous oxygen difference (e.g., the skin of cold hands and feet). A distinction is therefore made between peripheral cyanosis (acrocyanosis), which is confined to the skin of the extremities, and central cyanosis, which includes the tongue and mucous membranes. Circumoral cyanosis is not an expression of central cyanosis and is rarely pathological. The absolute concentration of reduced hemoglobin in the capillaries necessary to produce cyanosis is between 4 and 6 g/100 mL of blood. This level is usually present when the concentration of reduced hemoglobin in arterial blood exceeds 3 g/100 mL. Clinical cyanosis will occur at different levels of arterial oxygen saturation, depending on the amount of total hemoglobin (Fig. 6-13).

Physiologically, there are five mechanisms that can cause arterial hemoglobin desaturation in the patient who breathes room air at normal altitude: (1) alveolar hypoventilation, (2) diffusion impairment, (3) right-to-left shunting, (4) mismatch of ventilation and perfusion, and (5) inadequate oxygen transport by hemoglobin. Clinically, diffusion impairment is of little importance as a single cause. Imbalance of ventilation and perfusion is by far the most common mechanism and is

correctable by administration of 100% oxygen. The physician should therefore look for a change in cyanosis while the patient breathes oxygen.

Observer agreement regarding cyanosis was found to range from poor when assessing acrocyanosis to very good in the evaluation of young children with bronchiolitis. To minimize the variability of this finding, cyanosis is best observed under daylight and with the patient resting in a comfortably warm room. The distribution of cyanosis and the state of peripheral perfusion should be noted. Patients with decreased cardiac output and poor peripheral perfusion can be cyanotic despite normal arterial hemoglobin saturation. Some patients may become cyanotic only during exercise, a not uncommon response when restrictive lung disease reduces the pulmonary capillary bed and the transit time of erythrocytes becomes too short for full saturation during episodes of increased cardiac output. Congenital heart disease in infants may lead to differential cyanosis, which affects only the lower part of the body (e.g., in patients with preductal coarctation of the aorta). Less commonly, only the upper part of the body is cyanotic, for example, in patients with transposition of the great arteries, patent ductus arteriosus, or pulmonary hypertension.

The clinical impression of cyanosis is usually confirmed by an arterial blood gas analysis or more commonly by pulse oximetry. Pulse oximetry, however, will not take into account the presence of abnormal hemoglobin. For example, in methemoglobinemia the oxygen-carrying capacity of blood is reduced and patients may appear lavender blue, but pulse oximetry may overestimate oxygen saturation in arterial blood (SaO₂). The blood of newborn infants, conversely, can be well saturated and not cyanotic at lower arterial oxygen tensions because of the different oxygen-binding curve of fetal hemoglobin. In the patient with hypoxemia who does not present with cyanosis (e.g., the anemic patient), the physician has to pay particular attention to other clinical signs and symptoms of hypoxia. These include tachypnea and tachycardia, exertional dyspnea, hypertension,



■ **FIGURE 6-13.** Cyanosis requires at least 30 g/L of reduced hemoglobin (Hgb) in arterial blood or 4 to 6 g/100 mL in capillary blood. The arterial oxygen saturation (SaO₂) at which cyanosis occurs is dependent on the amount of total Hgb. As illustrated, a child with 30 g/L of Hgb plus 110 g/L of oxyhemoglobin has an SaO₂ of 78%. In the anemic patient (i.e., total Hgb of 80 g/L with Hgb of 30 g/L), SaO₂ will drop much lower (to less than 65% in this example) before cyanosis occurs, whereas in the patient with polycythemia (i.e., total Hgb of 200 g/L), cyanosis appears at a higher SaO₂ (85%).

headache, and behavioral changes. With more severe hypoxia there may be visual disturbance, somnolence, hypotension, and ultimately coma. In addition, the patient may have an elevated level of carbon dioxide. Depending on how rapidly and to what extent the level of carbon dioxide has risen, the clinical signs of hypercarbia will largely reflect vascular dilatation. These signs include flushed, hot hands and feet; bounding pulses; confusion or drowsiness; muscular twitching; engorged retinal veins; and, in the most severe cases, papilledema and coma.

■ DIGITAL CLUBBING

Digital clubbing refers to a focal enlargement of the connective tissue in the terminal phalanges of fingers and toes, most noticeably on their dorsal surfaces. This sign was first described by Hippocrates, and the term *Hippocratic fingers* is used by some to denote simple digital clubbing. The pathogenesis of clubbing is still not entirely clear. Vascular endothelial growth factor from the continued impaction of shunted megakaryocytes and platelets in the digital vasculature, potentiated by hypoxia, is considered to drive the cellular and stromal changes in clubbing. Platelet-derived growth factor may contribute to the stromal changes, including the maturation of new microvessels.

Clubbing of the digits may be idiopathic, acquired, or hereditary. Cystic fibrosis, bronchiectasis, and empyema are the most common pulmonary causes of acquired digital clubbing in children. Clubbing is also seen infrequently in extrinsic allergic vasculitis, pulmonary arteriovenous malformations, bronchiolitis obliterans, sarcoidosis, and chronic asthma. Box 6-3 shows a list of nonpulmonary diseases associated with clubbing. A systemic disorder of bones, joints, and soft tissues known as hypertrophic osteoarthropathy (HOA) includes digital clubbing. In the majority of cases, HOA is associated with bronchogenic carcinoma and other intrathoracic neoplasms, but the pediatrician may see HOA in patients with severe cystic fibrosis or chronic empyema and lung abscess. In addition to clubbing, these patients may have periosteal thickening; symmetric arthritis of ankles, knees, wrists, and elbows; neurovascular changes of hands and feet; and increased thickness of subcutaneous soft tissues in the distal portions of arms and legs. The primary idiopathic or hereditary form of HOA—pachydermoperiostosis—appears with prominent furrowing of the forehead and scalp. Approximately half of the reported cases have a positive family history. Genetic studies suggest an autosomal-dominant inheritance with variable expression and a predilection for males.

Digital clubbing is not only an important indicator of pulmonary disease but also may reflect the progression or resolution of the causative process. Pulmonary abscess and empyema may lead to digital clubbing over the course of only a few weeks. In this case, clubbing will resolve if effective treatment is instituted before connective tissue changes become fixed. Interestingly, even long-standing finger clubbing seems to resolve in patients after successful heart and lung transplantation. In patients with cystic fibrosis, progression of finger clubbing suggests a suboptimal control of chest infections. It is therefore useful to quantify the degree of digital clubbing. Measurements have focused on the hyponychial (Lovibond) angle and on the phalangeal depth ratio (Fig. 6-14). Changes of the hyponychial angle are quantified on “shadowgrams”—projections of the finger’s lateral profile onto a magnifying screen—whereas the phalangeal depth ratio is measured from plaster casts. Computerized analysis from digital photographs has provided information on the

BOX 6-3 Nonpulmonary Diseases Associated with Clubbing

Cardiac

- Cyanotic congenital heart disease
- Subacute bacterial endocarditis
- Chronic congestive heart failure

Hematologic

- Thalassemia
- Congenital methemoglobinemia (rare)

Gastrointestinal

- Crohn disease
- Ulcerative colitis
- Chronic dysentery, sprue
- Polyposis coli
- Severe gastrointestinal hemorrhage
- Small bowel lymphoma
- Liver cirrhosis (including α_1 -antitrypsin deficiency)

Other

- Thyroid deficiency (thyroid acropachy)
- Chronic pyelonephritis (rare)
- Toxic (e.g., arsenic, mercury, beryllium)
- Lymphomatoid granulomatosis
- Fabry’s disease
- Raynaud’s disease, scleroderma

Unilateral Clubbing

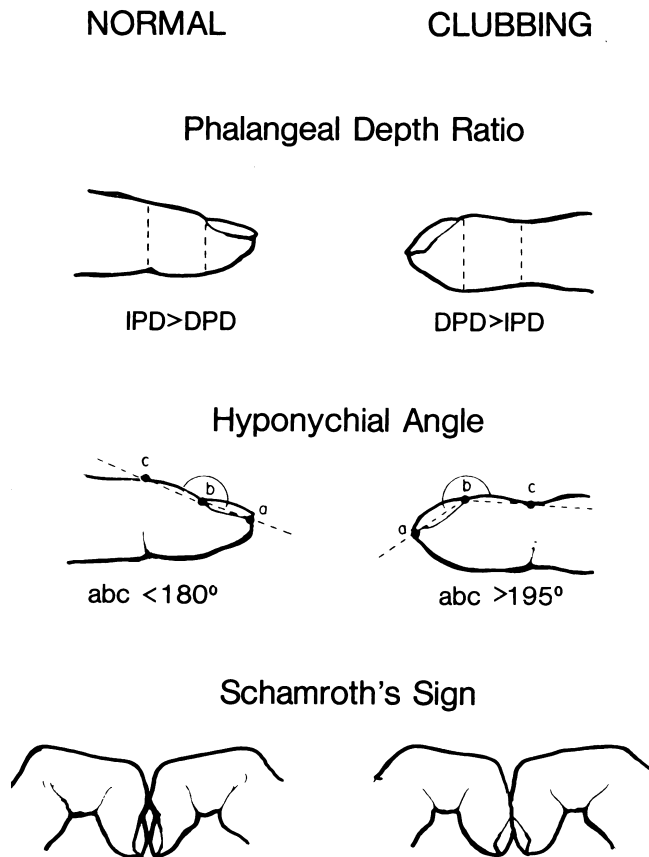
- Vascular disorders (e.g., subclavian arterial aneurysm, brachial arteriovenous fistula)
- Subluxation of shoulder
- Median nerve injury
- Local trauma

distribution of the hyponychial angle in healthy subjects and in patients with various diseases. Almost 80% of adult patients with cystic fibrosis have a hyponychial angle greater than 190 degrees, the upper limit of normal.

For routine clinical practice, the sign described by Schamroth, a cardiologist who himself developed finger clubbing during several attacks of infective endocarditis, is a most useful method of measuring finger clubbing (see Fig. 6-14). Another way is to place a plastic caliper with minimal pressure over the interphalangeal joint. If it is easy to slide the caliper from this joint across the nail fold, the distal phalangeal diameter to interphalangeal diameter ratio must be less than 1, and the patient has no clubbing.

■ CARDIOVASCULAR SIGNS

Pulmonary heart disease, or cor pulmonale, is a consequence of acute or chronic pulmonary hypertension and appears as right ventricular enlargement. The progression of chronic cor pulmonale to ultimate right ventricular failure is accompanied by certain physical signs. Initially, the right ventricular systolic pressure and muscle mass increase as pulmonary artery pressure rises. During this stage, cardiac auscultation may be normal or may reveal an increased pulmonary component of the second heart sound, caused by an increase in diastolic pulmonary



■ **FIGURE 6-14.** Finger clubbing can be measured in different ways. The ratio of the distal phalangeal diameter (DPD) over the interphalangeal diameter (IPD), or the phalangeal depth ratio, is less than 1 in normal subjects but increases to greater than 1 with finger clubbing. The DPD/IPD can be measured with calipers or more accurately with finger casts. The hyponychial angle can be measured from lateral projections of the finger contour on a magnifying screen and is usually less than 180 degrees in normal subjects but greater than 195 degrees in patients with finger clubbing. For bedside clinical assessment, Schamroth's sign is useful. The dorsal surfaces of the terminal phalanges of similar fingers are placed together. With clubbing, the normal diamond-shaped aperture or "window" at the bases of the nail beds disappears, and a prominent distal angle forms between the end of the nails. In normal subjects this angle is minimal or nonexistent.

arterial pressure. The physician should look for a parasternal right ventricular heave. As pulmonary hypertension progresses, there is an increase in right ventricular end-diastolic volume. Dilation of the main pulmonary artery and right ventricular outflow tract lead to systolic pulmonary ejection clicks and murmurs. Diastolic murmurs appear when pulmonary or tricuspid valves or both become insufficient. Third and fourth heart sounds at the left lower sternal border are signs of decreased right ventricular compliance. Most of these right-sided cardiovascular findings are accentuated during inspiration, which augments venous return. Finally, cardiac output falls while end-diastolic pressure and volume increase further in the failing right ventricle.

Clinical findings at this stage include hepatic engorgement, jugular venous distention, and peripheral edema. Occasionally, there may be cyanosis and intracardiac right-to-left shunting through a patent foramen ovale. The physician should exclude the possibility of congenital cardiac defects or acquired left-sided heart disease before making the diagnosis of cor pulmonale.

Hyperinflation of the lungs should be taken into account as a cause of the attenuation of cardiovascular sounds and the lowering of the subcostal liver margin, which may be misinterpreted as hepatic enlargement.

Complete assessment of the cardiovascular system includes a careful auscultation and palpation of the pulse to detect cardiac arrhythmia. This problem is not uncommon in patients with chronic lung disease and may appear as sinus or paroxysmal supraventricular tachycardia, atrial premature contractions, or ventricular ectopic beats. Causes include hypoxemia, acid-base imbalance, and enlargement of the right heart, but effects of common drugs such as aminophylline, beta sympathomimetics, or diuretics should not be overlooked.

During quiet spontaneous respiration there is a phasic variation of arterial blood pressure. The widening of this normal respiratory variation is known as pulsus paradoxus. Increased respiratory resistance may exaggerate the normal inspiratory-expiratory difference in left ventricular stroke volume. This is mediated by effects of intrathoracic pressures on ventricular preload. Clinically, pulsus paradoxus is first assessed by palpation of the radial pulse and then is measured at the brachial artery with a sphygmomanometer as the difference in systolic pressure between inspiration and expiration. The pressure cuff is deflated from above systolic level, and the highest pressure during expiration at which systolic pulse sounds are heard is recorded. Similarly, the highest pressure at which every pulse is just audible throughout inspiration is also noted. In general, a drop of greater than 10 mm Hg during inspiration is taken as clinically significant but only a severe paradox of greater than 24 mm Hg is a reliable indicator of severe asthma. The poor correlation between pulsus paradoxus and objective measurements of airflow obstruction may be explained by other factors that affect pleural pressure swings (e.g., the degree of pulmonary hyperinflation and the airflow rate). Furthermore, the accurate measurement of pulsus paradoxus is a challenge in tachypneic, tachycardic, and uncooperative young children. A work group on behalf of the British Thoracic Society Standards of Care Committee found pulsus paradoxus to be absent in one third of patients with the severest obstruction. In contrast to North American practice guidelines, it was therefore recommended that pulsus paradoxus be abandoned as an indicator of severity of asthma attack. In children, wheeze seems to be the clinical parameter of respiratory severity scores that correlates best with pulsus paradoxus, most likely because wheeze requires critical intrathoracic pressures. Perhaps the application of new plethysmographic methods using pulse oximeters will improve the diagnostic value of this clinical sign.

■ CHEST PAIN

Chest pain is relatively common in older children and adolescents but may also present in younger children. The occurrence rate in an emergency department approaches 2.5 per 1000 patient visits, and chest pain accounts for an estimated 650,000 physician visits of patients between the ages of 10 and 21 years annually in the United States. Chest pain in children is most often benign and self-limited. Typical origins are musculoskeletal problems and idiopathic, dysfunctional, and psychogenic causes (Table 6-3). Younger children more frequently have underlying cardiorespiratory problems, whereas children over the age of 12 years are more likely to have psychogenic pain.

■ **TABLE 6-3.** Causes of chest pain in children

| | |
|---|---|
| Thorax | Cardiovascular System |
| Costochondritis | Structural lesions (e.g., mitral valve prolapse, idiopathic hypertrophic subaortic stenosis [IHSS], coronary disease) |
| Tietze's syndrome | Acquired cardiac disease (e.g., carditis, arteritis, tumor involvement) |
| Muscular disease | Arrhythmia |
| Precordial catch | Esophagus |
| Trauma | Gastroesophageal reflux |
| Connective tissue disorders | Foreign body |
| Xiphoid-cartilage syndrome | Achalasia |
| Rib tip syndrome | Vertebral Column |
| Leukemia | Deformities (e.g., scoliosis) |
| Herpes zoster | Vertebral collapse |
| Breast development or disease (e.g., gynecomastia, mastitis) | Psychogenic Causes |
| Lungs, Pleura, and Diaphragm | Anxiety |
| Asthma | Hyperventilation |
| Cystic fibrosis | Unresolved grief |
| Infection (e.g., bronchitis, pneumonia, epidemic pleurodynia) | Identification with another person suffering chest pain |
| Inhalation of irritants (e.g., chemical pneumonitis, smoking) | |
| Stitch (associated with exercise) | |
| Foreign body | |
| Pneumothorax | |
| Pleural disease (e.g., pleurisy, effusion) | |
| Diaphragmatic irritation (e.g., subphrenic abscess, gastric distention) | |
| Sickle cell anemia | |

The history is most important in the assessment of these patients, who usually have few physical findings and rarely any laboratory data of diagnostic value. The physician should recognize a clinical profile suggestive of psychogenic pain but should also keep in mind that psychogenic and organic causes are not mutually exclusive. A substantial number of patients have a family history of chest pain. Parents of younger children should explain how they know that their child is in pain. It is important to determine whether sleep is affected, because organic pain is more likely than psychogenic pain to awaken the patient or to prevent the child from falling asleep. The duration of symptoms may be an indicator; acute, short-lasting pain is more likely to be organic than pain of many months duration. Localized, sharp, and superficial pains suggest an origin in the chest wall, whereas diffuse, deep, substernal, and epigastric pains are likely to be visceral, originating in the thorax if the pain affects dermatomes T1 to T4 and in the diaphragm or abdomen if it affects dermatomes T5 to T8. The physician should inquire about cough or asthma, recent exercise or trauma, heart disease in the patient and the family, cigarette smoking, and emotional problems.

A close inspection and careful palpation of the chest and abdomen are essential. Common abnormal findings include chest wall tenderness, fever, or both. The physician should use pressure on the stethoscope to elicit local tenderness while the patient is distracted by the auscultation. Cardiac murmurs with or without a midsystolic click may be found in patients with mitral valve prolapse but this condition is rarely associated with chest pain, at least in children. More commonly there are noncardiac causes of chest pain in children with mitral valve prolapse (e.g., orthopedic or gastroesophageal disorders). In general, the presence of systemic signs such as weight loss, anorexia, or syncopal attacks will direct the attention to organic causes of chest pain in children.

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