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SECTION E

DIAGNOSIS

16 HISTORY AND PHYSICAL EXAMINATION

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INTRODUCTION

Taking a careful and complete history and performing a thorough physical examination are hallmarks of the good internist and one of the distinguishing characteristics of a master clinician. The initial visit sets the tone of the immediate and future relationship with the patient and begins the process of diagnosing and managing the illness; it is a dynamic encounter, with each of the patient's responses stimulating further probing and forming of diagnostic hypotheses. The physician must be attentive to the patient's story, piecing together each bit of evidence to form a tentative preliminary diagnosis and differential diagnoses. Nothing should escape the eyes and ears of a watchful diagnostician. History taking is more than information gathering: it affords the opportunity to decipher the patient's body language as the inquiry proceeds. At this stage, no symptom or circumstance should be disregarded. With an understanding of biology and medicine coupled with past experience, the physician tries to connect the salient parts of the patient's story to develop a plausible explanation of the physiologic or pathologic events that lead to illness.

Although striving for a single diagnosis, the physician should realize that more than one disease may be present and that rare diseases are diagnosed only by those who consider them. Nevertheless, the maxim "uncommon presentations of common diseases are more frequent than common presentations of uncommon diseases" is likely to be true. It is important to continue both to gather information and to be open to reforming the diagnostic hypothesis as more information becomes available. Premature judgment or the failure to continue considering reasonable alternatives after an initial diagnosis is made is the single most common diagnostic error.[1,2] In the field of decision science, these failures are postulated to arise from "cognitive dispositions to respond" and include several of the biases in judgment or reasoning defined in Table 16-1.[3] It is hypothesized that a greater awareness of these prejudices among clinicians may facilitate "cognitive debiasing," thereby reducing the frequency of these common errors of reasoning.[4] An alternative or potential complementary approach is to use decision-support software to expand the differential diagnosis and avoid overlooking unusual or severe conditions.[5]

Bayes theorem implies that diagnostic tests will have a higher yield if the prior probability of the diagnosis is high (also called pretest probability). Specific details from the history raise the probability of different diagnoses and direct further tests in a productive manner. Further diagnostic investigations—imaging, blood tests, pulmonary function studies, and even parts of the physical examination—depend on the history. Historical clues raise or lower probabilities, thereby improving the value of subsequent questions and evaluations. Test results plus findings from the history and physical examination may confirm or refute the main and differential diagnoses, setting up either a management plan or the need for an alternative hypothesis.

At the end of the initial evaluation, the assessment and plan should identify problems and a course of action that

Table 16-1 Selected Biases in Judgment or Reasoning

Bias	Definition
Anchoring	Tendency to lock onto salient features in the patient's initial presentation too early in the diagnostic process, and failing to adjust this initial impression in light of later information.
Confirmation bias	Tendency to look for confirming evidence to support a diagnosis rather than to look for evidence to refute it, despite the latter often being more persuasive and definitive.
Framing effect	Tendency to be strongly influenced by how the problem is presented (e.g., perceptions of risk to the patient strongly influenced by whether the possible outcome is expressed in terms of the possibility that the patient might die or might live).
Premature closure	Tendency to accept a diagnosis before it has been fully verified. The consequences of the bias are reflected in the maxim "When the diagnosis is made, the thinking stops."
Search satisfying	Tendency to call off a search once something is found. Comorbidities, second foreign bodies, other fractures, and coingestants in poisoning may all be missed. Also, if the search yields nothing, diagnosticians should satisfy themselves that they have been looking in the right place.
Unpacking principle	Failure to elicit all relevant information (unpacking), which may result in missing significant diagnostic possibilities.

takes into account the patient's concerns and questions. The patient should feel satisfaction that the physician has done a thorough job of exploring his or her complaints, has provided a plausible explanation for them, and has planned a reasonable course of action.

ELECTRONIC DOCUMENTATION

The electronic medical record now allows documents of higher quality than written records, owing to improved organization, increased readability, use of supplementary material, and better comparisons. It eliminates poor handwriting and lost or misplaced information. Additional benefits include cost savings for storage, easy accessibility, and quick transfer to another health care provider.[6] The electronic medical record facilitates a coordinated team approach and reduces duplication of tests. Patients with complicated illnesses often have several different physicians, and electronic records can make it easier for one team to follow what another team is doing. Easy access to a complete record is especially important in emergency situations for physicians who are unfamiliar with the presenting patient. Electronic medication lists and reminders can save time and reduce errors. Electronic prescriptions provide greater patient safety.

Writing directly into the medical record, or initially through a word processing program, makes a cogent summary and chronological story easier to produce. The ability to insert information where it belongs helps maintain a congruous timeline. Spelling and grammar checks and autocorrection of abbreviations should make the finished report an easily readable document.

The disadvantages of electronic medical records include "information overload" and potential loss of privacy. Software downtime can be crippling. Learning how to use specific applications and developing typing skills may require training. Most word processing software used in electronic medical records is less efficient than commonly used commercial word processing software.

The "cut-and-paste" technique, beginning where the last visit left off, both saves time and ensures that ongoing problems are not overlooked. The problem with the cut-and-paste approach is that too much information may get deposited and duplicated in the medical record, including information irrelevant to the purpose of the consultation. Such excess text can at times replace essential information and impair easy understanding and critical reasoning. Cutting and pasting of information from consultants and other involved persons should never substitute for one's own primary history gathering or clinical thought. Cutting and pasting information gathered by someone else implies agreement with the statements. Proofreading is essential, especially of electronic prescriptions, because of the different doses and means of delivery of certain drugs.

Transfer of electronic medical information is not foolproof. Patients and physicians find it easy and convenient to communicate and transfer information by email, but there is a risk that the record of these interchanges may fail to be placed in the patient's permanent medical record, be intentionally or unintentionally intercepted leading to loss of privacy, or otherwise cause misunderstanding. Physicians may send patients electronic copies of their record, but if these are in a word processing format, patients could alter the record for secondary gain. Many of these concerns could have medicolegal consequences.[6]

COMMUNICATION SKILLS

The ability to listen skillfully, and to communicate clearly and empathetically with the patient, is the foundation for the physician-patient relationship. Communicating effectively with patients and peers underlies the success of a physician. The physician's communication should be objective, nonjudgmental, and empathic. Physicians are often better at obtaining medical information than they are at understanding how that information affects the patients.[7] Communication contains both verbal and nonverbal interactions.[8] A calm atmosphere, relaxed setting, and ample time are essential, particularly when disclosing bad news,[9] but even then, a physician with good communication skills should be able to make the patient pleased that she or he saw the physician. This can be accomplished by always stating the truth but by cushioning ominous information with hope. When realistic, the physician might say, for example, that the cancer was caught early, provide reassurance about a probable good outcome, or suggest a new and improved therapy.

The old-fashioned tutorial approach of learning how to be a good physician had many shortcomings; students, residents, and fellows learned medicine as apprentices in "a catch-as-catch-can" manner. More recently, however, scientific testing and social psychological analysis have uncovered egregious flaws in how physicians obtain, sort out, and

evaluate diagnostic information. Sir William Osler used to tell students to "listen to the patient, he (or she) will tell you the diagnosis." Today—instead—as Dr. J. Groopman points out in his excellent book, *How Doctors Think*, physicians interrupt the patient's initial history in just 16 seconds and frequently thereafter, make snap judgments, and fall into cognitive traps that are much more likely than factual ignorance to lead to medical errors.[10] New practice guidelines from evidence-based research help steer a correct diagnostic course, but the presence of overlapping diagnoses, unusual symptoms, and uncommon diseases requires wise and discerning physicians, not inflexible algorithms.

MEDICAL INTERVIEW

There is much more to the medical history than a recitation of questions and recording of answers. Instead, the medical interview has been defined as the entire medium of patient-physician interaction.[11] From this interactive experience, both physicians and patients learn about each other: the knowledge shared and feelings imparted influence subsequent trust, understanding, concern, and adherence to the health plan. Experience is valuable in the acquisition of clinical pattern recognition and in accumulating clinical knowledge. Although interviewing skills can be systematically learned,[12] acquiring the art of adept history taking and physical examination is a lifelong process that is incrementally improved by careful practice.

The main purposes of the medical interview are to (1) gather useful information, (2) develop rapport, (3) respond to concerns, and (4) educate the patient. The ease with which patients can access medical information may lead to a more active role on their part; patients may be well informed or misinformed about their actual or perceived diagnoses. Whatever their knowledge, most patients want to be accurately informed about their condition and to be involved in the deliberations and decision making.[13] At the same time they generally want their physician to direct their health care in a reasoned manner, which entails taking into account the patients' background knowledge, prejudices, and culture in a sensitive manner. This means the physician's plan should take into account the individuality of the patient.

Encouraging the patient to take the lead in expressing his or her symptoms and relationships to these symptoms forms the basis for the patient-centered interview[14] and develops appropriate rapport. Even in this era of reliance on laboratory studies, Platt's original claim[15] that a diagnosis can be obtained by history taking alone in most patients has been reaffirmed by several subsequent investigations.[16-18]

CHIEF COMPLAINT AND PRESENT ILLNESS

The medical history has traditionally been subdivided into the chief complaint; present, past, family, and social histories; and systems review. Because of its relevance and importance in the evaluation of patients with known or suspected pulmonary diseases, the occupational history is included as a separate component of the social history. Travel history, also included in the social history, is helpful in diagnosing certain lung diseases.

Only the chief complaint stands alone as a discrete response to a single question. It is generally recommended that the chief complaint be written in the patient's own words, lest the physician's interpretation be substituted prematurely for the patient's unique concern. Each chief complaint must be explored in detail, and the resulting aggregate of information constitutes the history of the present illness. The various elements of the remainder of the history are sorted into their proper categories after the interview has been completed. The resulting history of present illness is a cogent chronological story that incorporates all the facts and their relationships that support the preliminary diagnosis and differential diagnoses. Although an open-ended and free-flowing encounter, the interview still should be focused and organized. Each new question is often linked to the answer to the previous one. At the end the review of systems is a series of questions designed to cover previously unexamined territory.

Even as the clinician fulfills the roles of information gatherer and detective, a more complex process is occurring in which a patient's verbal and nonverbal responses to symptom queries provide a personal and often explanatory narrative that may encapsulate unique and individual aspects of illness. These may include the experience of illness and its relationship to any and all aspects of the patient's life. The emerging field of narrative medicine highlights the effects such storytelling has on patients and providers, and its ability to enrich the physician-patient relationship and the clinical experience.[19,20]

MAJOR PULMONARY SYMPTOMS

Because dyspnea, cough, and chest pain are among the most common reasons for patients to visit physicians, and because these symptoms may result from serious underlying chest disease, careful questioning is needed to establish their etiology and significance. The anatomic and pathophysiologic basis of these cardinal symptoms is provided in Chapters 29 to 31. To aid the interviewer in obtaining a medical history, a brief overview of these three common presenting symptoms and a related one, hemoptysis, is provided in this section.

Dyspnea

When a healthy person increases his or her level of physical activity sufficiently, an awareness of breathing emerges; if the severity of activity increases even further, the sensation becomes progressively more unpleasant, until it typically compels the individual to slow down or stop.[21] Although dyspnea, shortness of breath, and breathlessness are often used interchangeably, as in Chapter 29, some purists use the term *dyspnea* only when the symptom is abnormal, which implies that the awareness is disproportionate to the stimulus and that the sensation is pathologic. Many patients describe their breathing discomfort as "breathlessness," but many others complain of "tightness," "choking," being "unable to take a deep breath," "suffocating," being "unable get enough air," or occasionally even "tiredness."

The mechanisms that underlie the sensation of dyspnea remain poorly understood and are reviewed in Chapter 29. In contrast to pain and cough, for which specific receptors and neural pathways have been identified, similar detailed

knowledge is lacking for dyspnea, although evidence is mounting that links the symptom with pain.[21,22] Studies of the neurophysiology of dyspnea are further complicated by the lack of objective tools to quantify a subjective sensation with interindividual variation. Rating instruments—such as the Borg scale[23] and questionnaires, such as the British Medical Research Council questionnaire[24] and Pulmonary Functional Status and Dyspnea Questionnaire[25]—have been validated as useful in measuring dyspnea. Self-administered, computerized versions of the Transitional Dyspnea Index and Multidimensional Baseline Dyspnea Index appear to be at least as good as interview questioning for this assessment.[26] Progress, though, is being made: recent studies have clearly shown that dyspnea during exercise in patients with *chronic obstructive pulmonary disease* (COPD) is closely linked to dynamic lung hyperinflation.[27]

Clinical Features. Patients with respiratory, cardiac, hematologic, metabolic, and neuromuscular disorders may all complain of dyspnea. A careful and detailed history is necessary to uncover the cause of the sensation. In addition, it is important to document the impact of the symptom on the patient's daily activities and to be alert to the "decreased activity phenomenon." The latter describes patients who say their dyspnea has not worsened, but only because they now walk more slowly or no longer climb stairs or engage in athletic activities. Sometimes this slowing down is so gradual, patients may be unaware or attribute it to aging. Assessing the activity required to bring about the dyspnea is important. How many stairs can be climbed before stopping? How far can someone walk on level ground at her or his own pace without stopping? Does talking on the phone, getting dressed, or eating cause dyspnea? Is the patient short of breath at rest?

The course over time should be noted. Sudden dyspnea without an obvious provocation suggests pulmonary embolism or pneumothorax, although myocardial ischemia and asthma also may have a rapid onset. Dyspnea caused by cigarette smoke, dusts, molds, perfumes, newly cut grass, cats, and strong odors is characteristic of the increased bronchial reactivity seen in asthma. Associated features, such as wheezing and the presence and type of chest tightness or pain, are important clues. Worsening dyspnea with cough producing increased quantities of purulent sputum over 1 to 3 days characterizes an exacerbation of COPD.

Special types of dyspnea are sufficiently distinctive to warrant separate designations. Episodes of breathlessness that wake persons from a sound sleep, *paroxysmal nocturnal dyspnea,* usually denote left ventricular failure but may also occur in patients with chronic pulmonary diseases because of pooling of secretions, gravity-induced decreases in lung volumes, sleep-induced increases in airflow resistance, or nocturnal aspiration. *Orthopnea,* the onset or worsening of dyspnea on assuming the supine position, like paroxysmal nocturnal dyspnea, is found in patients with heart disease and chronic lung disease. Measurement of amino-terminal pro-B-type natriuretic peptide has proved useful in differentiating between a cardiac and a respiratory origin in patients with dyspnea.[28]

The inability to assume the supine position (*instant orthopnea*) is characteristic of paralysis of both leaves of the diaphragm. Dyspnea soon after assuming the supine position also may be associated with other conditions, such as arteriovenous malformation, bronchiectasis, and lung abscess. *Platypnea,* which denotes dyspnea in the upright position, and *trepopnea,* an even rarer form of dyspnea that develops in either the right or the left lateral decubitus position, suggest lung vascular shunting. Both the terms *hyperpnea,* an increase in minute ventilation, and *hyperventilation,* an increase in alveolar ventilation in excess of carbon dioxide production, indicate that ventilation is abnormally increased. Neither term, however, carries any implication about the presence or absence of dyspnea.

Cough

The quantity of bronchial secretions produced each day by a nonsmoking healthy adult is not precisely known, but it is sufficiently small to be removed by mucociliary action alone: healthy persons seldom cough.[29] As described in Chapter 30, coughing is an essential mechanism that protects the airways from the adverse effects of inhaled noxious substances and defends the lungs by clearing excess secretions.[30] Coughing can be occasional, transient, and unimportant. By contrast, it may indicate the presence of severe intrathoracic disease.

Clinical Features. Most episodes of coughing are associated with short-lived upper respiratory tract infections or allergies, and patients, recognizing this, seldom visit their physicians for this type of cough. Nevertheless, cough is the most common complaint for which patients seek medical attention and the second most common reason for having a general medical examination.[31] Physicians should realize that when patients seek their help for cough, it is often out of concern for something new, different, and alarming about the symptom. The essential first step in evaluating a patient with cough is to obtain a thorough history, paying particular attention to the following aspects: acute or chronic, productive or nonproductive, character, time relationships, type and quantity of sputum, and associated features. It is noteworthy that, of the various components of the workup used by the authors of a systematic anatomic investigation to determine the causes of chronic cough, the medical history alone led to the correct diagnosis in 70% of patients.[31]

Acute coughing is frequently associated with nasopharyngitis, laryngotracheobronchitis, or other, usually virus-induced, upper respiratory tract infections. Less commonly, it may be the chief manifestation heralding the onset of viral or bacterial bronchopulmonary infection or the inhalation of allergenic or irritating substances. The causes of cough that persisted for 3 weeks or longer in 102 patients were postnasal drip (41%), asthma (24%), gastroesophageal reflux (21%), chronic bronchitis (5%), and bronchiectasis (4%).[31] Other important though less common conditions include eosinophilic bronchitis[32] and the use of angiotensin-converting enzyme inhibitors.[33] In 1999 the importance of the "big three"—postnasal drip, asthma, and gastroesophageal reflux—was verified by the results of another survey of the causes of chronic cough.[34] Not everyone agrees, however, and some experts claim that emphasis on the top three conditions is unwarranted and moreover that it stifles interest and research into other important causes and mechanisms of chronic cough.[35]

A careful history of patients with cough lasting at least 3 months revealed that nearly all the patients misdiagnosed as "psychogenic" had one of the conditions listed previously for chronic cough.[36] Even cough that is made worse with psychological stress is often caused by underlying lung disease. Patients with exaggerated cough responses or habitual cough may have a "psychogenic" component; therefore, even when chronic cough has a pulmonary cause, it may respond to behavioral modification.[37]

Most physicians have heard the ancient diagnostic axiom, which is still true, that any change in the character or pattern of a chronic cough in a smoker demands a prompt chest radiographic evaluation for lung cancer. Less well known is that cough may be the sole presenting manifestation of asthma[38] or gastroesophageal reflux disease.[39]

In low-income countries, where the majority of the global population lives, cough, usually productive but not always, of 3 weeks or longer has been the traditional (and reliable) clinical marker of possible pulmonary tuberculosis that should trigger examination of sputum specimens for *Mycobacterium tuberculosis.* Revised recommendations by tuberculosis experts now include cough of "2 or 3 weeks," or longer, as an indication for sputum examination.[40]

Among the many complications of persistent or recurrent cough are tussive syncope; retinal vessel rupture; persistent headache; chest wall and abdominal muscle strains, including the development of abdominal wall hernia[41]; and even rib fractures. Severe chronic cough may create devastating personal distress, causing patients to restrict their social and professional activities.

Hemoptysis

The expectoration of any amount of blood denotes hemoptysis. Every patient with new-onset or appreciable hemoptysis deserves a thorough diagnostic evaluation, which generally includes *computed tomography* (CT) of the thorax and bronchoscopy. For centuries, hemoptysis was considered pathognomonic of pulmonary tuberculosis, a view that is summarized in the Hippocratic aphorism "the spitting of pus follows the spitting of blood, consumption follows the spitting of this, and death follows consumption."[42] The frequency of the different conditions that cause hemoptysis depend to a large extent on the population studied, but bronchitis, lung cancer, tuberculosis, and bronchiectasis are usually the most common causes.[43-45] These are also the leading causes of massive hemoptysis (defined in various series as >200 or >600 mL of blood in 24 hours). Lung cancer and bronchitis usually cause mild to moderate bleeding, whereas patients with bronchiectasis, lung abscess, fungal disease, or a bleeding diathesis are more likely to have severe bleeding.[43] Less common conditions associated with hemoptysis include arteriovenous malformations, broncholithiasis, foreign bodies, aspergilloma, mitral stenosis, trauma, excessive anticoagulation, pulmonary hemorrhage syndromes, heart failure, pneumonia, and granulomatosis with polyangiitis (Wegener granulomatosis).

Clinical Features. Prompt evaluation, beginning with a thorough history, is required in all patients. It is important to determine where the blood is coming from. Surprisingly, patients may not always be able to distinguish hemoptysis from hematemesis and nasopharyngeal bleeding. Vomiting blood may follow a prolonged coughing episode. Patients may swallow or aspirate blood from the upper airway. Some patients report only that the blood "welled up" in their throats. Others will say that it is mixed with sputum. Hematemesis can usually be differentiated from hemoptysis by the presence of symptoms of gastrointestinal involvement, such as nausea and vomiting, a history of peptic ulcer disease, alcoholism, or signs of cirrhosis; when in doubt, esophagoscopy is indicated.

Following physical examination, a chest radiograph and (often) a chest CT are required. Depending on the magnitude of the blood loss and the clinical circumstances, bronchoscopy is indicated to determine the location of the bleeding. Although these studies generally reveal which region of the lungs is the source, the cause of hemoptysis cannot be determined in 20% to 30% of cases.[46] Recent radiologic advances, which enhance identification of the culprit vessel, particularly multidetector computed tomographic angiography, have greatly helped the interventionalist when bronchial artery embolization is required to stop the bleeding.[47]

Chest Pain

Various types of chest pain are extremely common; their mechanisms and clinical patterns are described in Chapter 31. Chest pain is one of the most common symptoms that cause the sufferer to seek medical attention. Because there is no clear relationship between the intensity of the discomfort and the importance of its underlying cause, all complaints of chest pain must be carefully considered. The recent development of dedicated chest pain centers within emergency departments has improved the accuracy and rapidity of diagnosis, the treatment, and the survival of patients with this always troublesome symptom.[48]

Clinical Features. Pleurisy, or acute inflammation of the pleural surfaces, has several distinctive features. Pleuritic pain is usually localized and unilateral—and tends to be distributed along the intercostal nerve zones. Pain from diaphragmatic pleurisy is often referred to the ipsilateral shoulder and side of the neck. The most striking and defining characteristic of pleuritic pain is its clear relationship to respiratory movements. The pain may be variously described as "sharp," "burning," or simply "a catch," but it is typically worsened by taking a deep breath, and coughing or sneezing causes intense distress. Patients with pleurisy frequently also experience dyspnea because the aggravation of their pain during inspiration makes them conscious of every breath.

Acute pleuritic pain is found in patients with spontaneous pneumothorax, pulmonary embolism, and pneumonia, especially pneumococcal pneumonia, whereas a gradual onset over several days is observed in patients with tuberculosis; an even slower development is characteristic of primary or secondary malignancies. Chronic pleuritic pain is characteristic of mesothelioma. It may be difficult to distinguish pleuritic pain from the pain of a rib fracture, although point localization favors the latter. Pericardial pain is typically sharp, retrosternal in location, and relieved by sitting up and leaning forward.

The distribution and the superficial, knifelike quality of the pain of intercostal neuritis or radiculitis may resemble

pleural pain because it is worsened by vigorous respiratory movements but, unlike pleurisy, not by ordinary breathing. A neuritic origin may be suggested by the presence of lancinating or electric shock–like sensations unrelated to movements, and hyperalgesia or anesthesia over the distribution of the affected intercostal nerve provides confirmatory evidence. In many instances of new-onset, neuritic chest wall pain, the diagnosis becomes clear a day or two later when the typical vesicular rash of herpes zoster appears.[49]

Among the most important types of chest pain is myocardial ischemia, which is usually caused by coronary artery atherosclerosis. These attacks, which are provoked by inadequate oxygen delivery to the myocardium, span a continuum of severity from chronic stable angina to classic acute myocardial infarction. Typical anginal pain is induced by exercise, heavy meals, and emotional upsets; the pain is usually described as a substernal "pressure," "constriction," or "squeezing" that, when intense, may radiate to the neck or down the ulnar aspect of one or both arms.[50] Pain from variant or Prinzmetal angina is similar in location and quality to typical anginal pain but is experienced intermittently at rest rather than during exertion.[51] Both typical and variant types of angina are relieved by coronary vasodilator drugs, such as nitroglycerin. Typical angina also decreases with rest or removal of the inciting stress.

By contrast, the pain of acute myocardial infarction, although similar in location and character to anginal pain, is usually of greater intensity and duration, is not alleviated by rest or by nitroglycerin, may require large doses of opiates, and is often accompanied by profuse sweating, nausea, hypotension, and arrhythmias. During attacks of myocardial ischemia and myocardial infarction, patients are often short of breath from associated pulmonary edema, which may be severe, but the pain itself is not related to breathing. Pain similar to that of myocardial ischemia also occurs in patients with aortic valve disease, especially aortic stenosis, and other noncoronary heart disease and extracardiac disorders.

Inflammation of or trauma to the joints, muscles, cartilages, bones, and fasciae of the thoracic cage is a common cause of chest pain.[52] Redness, swelling, and soreness of the costochondral junctions is called Tietze syndrome. All of these disorders are characterized by point tenderness over the affected area.

Most pulmonary thromboemboli are not associated with chest pain; the hallmark of pulmonary infarction, however, is typical pleuritic pain. Both acute and chronic causes of pulmonary hypertension may be associated with episodes of chest pain that resemble the pain of myocardial ischemia in its substernal location and pattern of radiation and in its being described as "crushing" or "constricting."[52] This type of chest pain is believed to result from right ventricular ischemia owing to impaired coronary blood flow secondary to increased right ventricular mass and elevated systolic and diastolic pressures or to compression of the left main coronary artery by the dilated pulmonary artery trunk.

FAMILY HISTORY AND SOCIAL HISTORY

The family history provides important clues to the presence of heritable pulmonary diseases, such as cystic fibrosis, alpha$_1$-antitrypsin deficiency, hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease), immotile cilia syndrome, and immunodeficiency syndromes, among others. Careful history taking also can uncover even more common familial disease associations, which are polygenic or in which the exact mode of genetic transmission has not yet been established. As genomic surveillance is uncovering more and more genetic linkages, the family history assumes an even more important function. The family history should encompass at least three generations to account for sex-linked traits. Family history also can identify exposures such as to tuberculosis or other contagious diseases.

Of course, no evaluation of pulmonary symptoms is complete without a detailed history of smoking habits. The physician should ask, "Have you ever smoked?" A negative answer should prompt a confirmatory response such as, "So you are a lifelong nonsmoker?" and a compliment if the second answer is yes. If the patient has smoked, the next questions should be, "When did you start?" "When did you quit?" and "How much did you smoke while you were at it?" Ask also about different forms of tobacco and exposure at home or workplace to other people's tobacco smoke. A history of exposure to environmental smoke is also important.[53] In many developing countries, smoke from indoor cooking and heating fires is a major cause of lung disease, especially in women. Risk factors for *human immunodeficiency virus* (HIV) infection, such as unprotected sexual activity and injection drug abuse, should be specifically queried.

Medications and Allergies

A complete list of all medications is essential to a thorough history. Ideally, the patient should bring in all his or her medications, and the physician should carefully go through each one, checking that the prescription has been properly written and filled and that the patient understands the benefit and possible side effects of each medicine. It is vital to note whether the patient has ever had an allergic or toxic reaction and what these reactions were. A complete listing of supplements and herbal medications should also be recorded and reviewed for potential interactions with conventional medications. No drug history is complete without assessing whether the patient drinks alcoholic beverages or uses illicit drugs. The amount and frequency of their use should be recorded.

Occupational History

The occupational history, which is often included as part of the social history, is an integral part of a thorough medical interview. Identifying a relevant occupational exposure may provide the only opportunity to remove the patient from the exposure and prevent progressive and irreversible lung damage. Moreover, identifying injurious occupational exposures can facilitate justifiable compensation for the patient and removal of the hazardous materials from the workplace by the industry.

The evaluation of suspected occupational lung disease is discussed in Chapters 64, 72, 73, and 74. Although only a few questions are asked in most initial medical interviews, if occupational illness is seriously being considered, a detailed inquiry about each industry, profession, and job the patient has held needs to be performed.[54,55] Because there are so many environmental agents and different associated

illnesses, the diagnostician should consult online resources such as the National Institute for Occupational Safety and Health, the Environmental Protection Agency, Hazardous Substances Data Bank, the Occupational Safety and Health Administration, or other online resources to learn more about the putative environmental toxin.[56,57]

Travel History

Previous places of residence help diagnose endemic fungal diseases, especially histoplasmosis and coccidioidomycosis. A history of recent travel may help establish the possibility of exposure to infectious diseases that are restricted to specific geographic regions.[58] The physician should inquire into the duration of travel. Long trips by air or car increase the risk for deep venous thrombosis and venous thromboembolism, which are reported in up to 10% of passengers on long-haul flights.[59] It is important to consider events after travel: symptoms of pulmonary thromboembolism and infarction may arise a variable time after the inciting event. The epidemic of *severe acute respiratory syndrome* (SARS) in southeast China in 2002 and its rapid spread throughout the world by airline passengers emphasizes the importance of obtaining a careful travel history.

PAST MEDICAL HISTORY

Previous illnesses may recur (e.g., tuberculosis), and new diseases may complicate old ones (e.g., bronchiectasis as a sequela of necrotizing pneumonia). Information about previous illnesses, operations, intubations, and trauma involving the respiratory system may be essential to understanding the current problem. Although these data may be gathered as part of the past medical history, much of the pertinent information will be absorbed into the chronological sequence of the history of present illness. Prior chest radiographs are an important aid in the evaluation of any abnormal chest radiograph because of the insights they provide into the duration and trajectory of illness. Patients should be asked to bring in previous films, but if they are unavailable, physicians should make every effort to obtain them, because old radiographs may save needless, costly, and sometimes risky interventions.

INFORMATION FROM QUESTIONNAIRES AND OTHER SOURCES

Printed or computer-based questionnaires and histories taken by nurses or allied health professionals are often used to expedite history taking. They can identify problems that can be explored further in the medical interview, and they facilitate a focused yet comprehensive evaluation. Occupational questionnaires have been shown to enhance recognition of occupational illness and correlate well with the findings of an industrial hygienist.[60] Computer-based interviews can gather more information, allow more time to complete the interview, uncover sensitive information, and may be adaptable to the hearing impaired and to persons speaking a language different from that of the physician.[61]

These forms of information gathering should be considered adjuncts and not a substitute for the thorough history taken by the physician. The limitations of the programmed questions are that the patient may not understand or be able to express her or his concerns when confined by a form that does not allow the free exploration of symptoms that the open interview does. Automated data collection, of course, lacks the benefits gained from the patient-physician interaction, such as the establishment of rapport and the ability to observe nonverbal behavior. The interview itself also provides both time and opportunity for the physician to fully comprehend the patient's illness and to contemplate the primary and differential diagnoses.

For monitoring the course of certain disorders such as asthma, daily recording of symptoms, such as wheezing and breathlessness, and objective assessments of severity of disease, such as peak expiratory flow, in a diary are preferable to a single questionnaire because recall of symptoms may be faulty and one measurement may not be representative. The electronic monitoring that now comes as standard equipment with most home noninvasive ventilation devices gives the date and time of the respiratory events and the use of these devices.

PHYSICAL EXAMINATION

Sadly, the declining emphasis on proficiency in physical examination during medical school and residency training and the ever-increasing reliance on technology-based diagnosis have led to a decreased interest in, some say even the "demise" of, the physical examination.[62] However, the old observation that 88% of all diagnoses in primary care were established by taking a thorough medical history and performing a complete physical examination[63] probably still holds today. At the very least, a carefully executed history and physical lead to more intelligent and cost-effective use of diagnostic technology. Plus, a physical examination can be performed virtually anywhere, may provide important information, lends itself to serial observations, and increases patients' confidence in their physicians.

EXAMINATION OF THE CHEST

Physical examination of the chest employs the four classic techniques of inspection, palpation, percussion, and auscultation. Each is described subsequently, as are the constellations of abnormalities that allow the examiner to infer the presence and type of various pulmonary disorders. Apart from inspection, which is not only a visual but also sometimes an olfactory tool and is always a structured cognitive skill, the other three modalities depend on the generation and the perception of sound or tactile sensations and vibrations. As was true of the history, the environment in which the physical examination takes place must be appropriate to the needs of both the examiner and the examined. Privacy, warmth, good light, and quiet are all essential. The best light source is natural sunlight, which should be used if possible. An ill-lit, noisy, or distracting environment will likely result in a physical examination that is flawed or incomplete.

Inspection

The physical examination begins the moment the clinician first sees the patient, even before the introductions and beginning the medical interview. Keen observations, and

the ability to pursue and interpret these observations, are the keys to skilled clinical diagnosis.

Inspection of the chest is carried out after sufficient clothing has been removed and the patient has been suitably draped to permit observation of the entire thorax. Ordinarily, inspection is performed with the patient sitting, but if the patient is too weak or cannot sit unaided, he or she should be supported in this position. Observing the shape and symmetry of the chest allows such abnormalities as kyphoscoliosis, pectus excavatum, pectus carinatum, ankylosing spondylitis, osteoporosis, gynecomastia, and surgical scars or defects to become obvious.

Several classic patterns of ventilation can be readily recognized (Fig. 16-1). Examples are *tachypnea,* which is almost uniform rapid shallow breathing; *Kussmaul breathing,* which is relentless, rapid, and deep breathing (air hunger); *Cheyne-Stokes respirations,* a cyclical waxing and waning of the depth of breathing with regularly recurring periods of apnea; and *Biot breathing,* which is totally irregular breathing, both the size of breaths and the periods of apnea, which are sometimes prolonged. Impending respiratory failure from muscle fatigue can be detected by observing rapid shallow breathing, abdominal paradoxical motion, and alternation between rib cage and abdominal breathing, so-called *respiratory alternans.*[64] The Hoover sign is the paradoxical inward displacement of the costal margin at the end of inspiration or throughout inspiration. Decreased regional ventilation can be detected be seeing a lag in the motion of the affected part of the chest wall during breathing.

Palpation

Palpation of the thorax is a necessary part of the cardiac, breast, and lymph node examinations and often can detect bony abnormalities, such as a cervical rib, and subcutaneous calcinosis seen with systemic sclerosis. It is essential in examining causes of pain to determine point tenderness and thoracic spinal tenderness. It can detect fluctuant areas associated with empyema necessitans and crepitant areas associated with subcutaneous emphysema. Location by palpation of the trachea in the suprasternal notch is a useful way to detect shifts of the mediastinum. A spastic, extrafirm-feeling back muscle recognized by palpation may identify the cause of thoracic pain. A lag in movement of the chest wall, suspected from inspection, can be confirmed by placing the two hands over opposite portions of each hemithorax and both feeling and observing whether or not the thorax moves symmetrically.[65] Symmetry is as important in palpation as it is in inspection.

A palpable vibration felt on the body, usually over the chest, is called *fremitus.* Vocal fremitus is elicited by having the patient speak "one, two, three," while the examiner's two palms or sides of the hands are moved horizontally from top to bottom of the two hemithoraces. Vocal fremitus is increased over regions of lungs through which there is increased transmission of sound, for example, consolidation from pneumonia. Conversely, fremitus is decreased in conditions in which sound transmission is impaired, for example, pleural effusion. Occasionally, fremitus over part of the chest wall can detect the presence of airway secretions (rhonchal fremitus) or an underlying pleural friction rub (friction fremitus).

In examining the heart the examining physician should always search for an apical impulse, heaves and lifts, thrills, and palpable valve closure. In patients with severe COPD, abnormal cardiac movements are often better felt in the subxiphoid region than over the precordium.

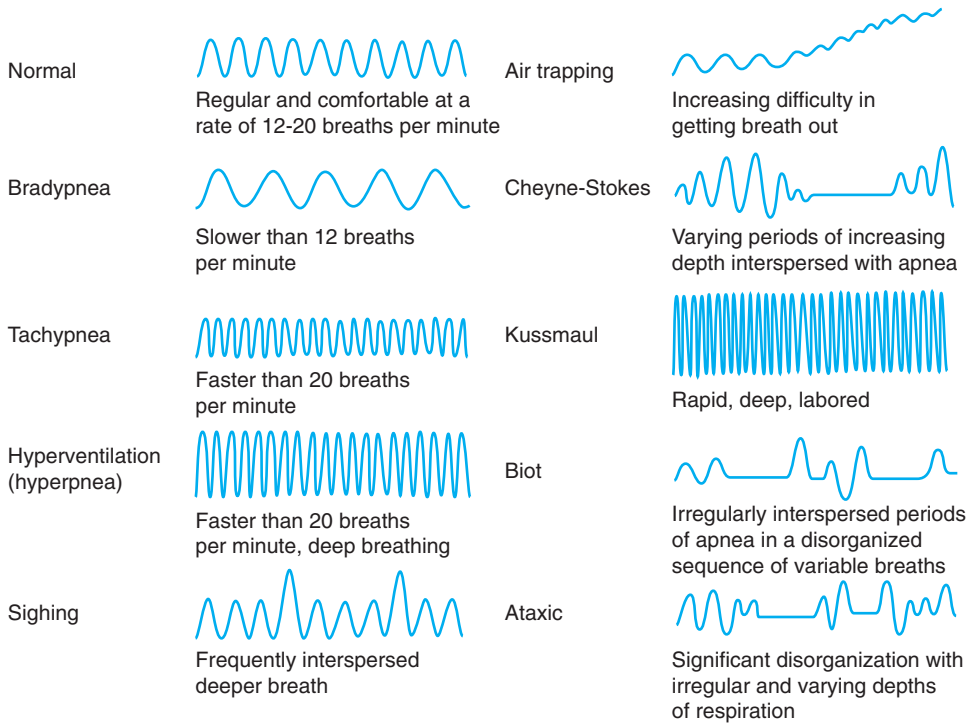

Figure 16-1 Schematic drawing of waveforms in different patterns of breathing. (Adapted from Wilkins RL, Hodgkin J, Lopez B: *Lung and heart sounds online*, St. Louis, 2011, Mosby.)

Percussion

Skillful percussion depends on a uniform free and easy stroke of the striking finger (plexor) on the finger being struck (pleximeter), the ability to sense minor changes in pitch, and a keen sense of vibration—although the percussion note is heard, it is predominantly felt. Percussion of the thorax over normal air-containing lung produces a resonant note.

Sounds and tactile perception from percussion vary depending on the thickness of the skin, subcutaneous layer, breast tissue, and chest wall, as well as on the quality, distribution, and tension of the air under the area percussed. Pathologic processes may impair or enhance the resonating quality of the thorax. For example, the percussion note over a large pneumothorax is hyperresonant and becomes tympanitic when tension is present; in contrast, percussion over a pleural effusion or pneumonia produces dullness, which has been defined as a low-intensity sound of short duration, feeble carrying power, and rather high pitch.[66] Flatness is the unresonating sound obtained by percussing over the liver. Three different tonal zones can thus be detected when percussing large pleural effusions: normal resonance above the fluid, dullness in the middle, and flatness when completely below the fluid; these variations in sound may result from the presence of an internal meniscus or fluid wedge, which points upward into the lung above it. Carrying out a thoracentesis in the dull area offers the best chance of obtaining pleural fluid and avoids puncturing either an abdominal viscus or air-containing lung.

Auscultation

A stethoscope draped around the neck has long been the badge of the medical professional, and it is worn with pride by physicians, nurses, and respiratory therapists, despite predictions such as "it, too, will someday be relegated to a museum shelf."[67] This will not happen for a long time, according to Murphy,[68] who mounts a spirited defense of stethoscopes backed up by analyses of breath sounds obtained by respiratory acoustic recording. Indeed, there is now a body of literature on computer-assisted mapping of breath sounds using both recording and imaging techniques which provides new insights into their origin and clinical significance.[69-71] For example, computerized multisensor breath sound imaging has proved to be a sensitive and specific tool for differentiating pneumonia or pleural effusion from normal lungs.[72] Similarly, signal analysis of heart sounds recorded by digital electronic means has promising clinical applications and is useful for teaching cardiac auscultation.[73] The fundamentals of lung auscultation in physical examination have been reviewed recently.[74]

Stethoscopes are also helpful in picking up wheezes in asthmatics and crackles in patients with interstitial lung disease whose chest radiograph findings are normal. Moreover, patients expect their physicians to listen to their hearts and lungs if they have cardiorespiratory complaints.

Like any piece of medical equipment, there are a number of available choices, and the design and care of the stethoscope may have a substantial impact on its performance. Electronic models promise ambient noise reduction and audio amplification, features that have been shown in randomized trials to provide statistically significant improvements in acoustics, especially in noisy environments.[75,76] However, the magnitude of improvement is small relative to the best acoustic stethoscopes, and electronic stethoscopes have not been shown to improve trainee performance.[77] Sound quality with any stethoscope can be substantially degraded by failure to maintain the integrity of the rubber fittings, and prolonged contact of the tubing with the skin when worn around the neck can lead to hardening of the tubing and decreased performance. In any case, the stethoscope must be kept clean because it is increasingly recognized as a vector of nosocomial infection.[78]

The terminology of breath sounds has been standardized and simplified to enhance understanding and communication (Table 16-2). Although a standardized nomenclature has been proposed by the American Thoracic Society[79] and the Tenth International Conference on Lung Sounds,[80] communication at the bedside often strays from recommended terminology.

The basic technique of auscultation with an ordinary stethoscope is well known to most physicians: the diaphragm detects higher-pitched sounds, and the bell detects lower-pitched sounds, although if the bell is tightly pressed against the body, the taut underlying skin itself may serve

Table 16-2 Classification of Common Lung Sounds

	Acoustic Characteristics	American Thoracic Society Nomenclature	Common Synonyms
Normal	200-600 Hz Decreasing power with increasing Hz	Normal	Vesicular
	75-1600 Hz Flat until sharp decrease in power (900 Hz)	Bronchial	Bronchial Tracheal
Adventitious	—	Adventitious	Abnormal
	Discontinuous, interrupted explosive sounds (loud, low in pitch), early inspiratory or expiratory	Coarse crackles	Coarse crackles
	Discontinuous, interrupted explosive sounds (less loud than above and of shorter duration; higher in pitch than coarse crackles or crackles), mid- to late inspiratory	Fine crackles	Fine crackles, crepitation
	Continuous sounds (>250 msec, high-pitched; dominant frequency of 400 Hz or more, a hissing sound)	Wheezes	Sibilant rhonchus, high-pitched wheeze
	Continuous sounds (>250 msec, low-pitched; dominant frequency < 200 Hz, a snoring sound)	Rhonchi	Sonorous rhonchus, low pitched wheeze

as a "diaphragm" and improve perception of higher pitches. Conversely, the bell should be applied very lightly to hear, for example, the low-pitched rumble of mitral stenosis. Full contact with the skin is necessary for best listening, which may pose a problem in a patient whose intercostal spaces are sunken from weight loss. In addition, the skin or hairs may brush against the diaphragm and produce a sound that resembles a pleural friction rub. As with examiners' hands, a warm stethoscope head is appreciated by patients. The importance of a quiet room and of applying the stethoscope directly to the skin rather than through clothing has recently been reemphasized.[81] At times, especially in the intensive care unit, it is not always possible to sit patients up to listen carefully to their backs, which compromises the completeness of auscultation.

This chapter includes links to audio recordings, some with animations. To hear the recorded lung sounds at their intended pitch and intensity, it is recommended that readers listen through a stethoscope, with the chest piece held 4–5 inches from the audio speaker.

The recommended terminology for the ordinary breathing-associated sounds heard with a stethoscope placed on the chest of a healthy person is *normal lung sounds*, but, as shown in Table 16-2, many physicians prefer the older term *vesicular breath sounds* (Audio 16-1). The usually predominating inspiratory component arises from sounds generated by turbulent airflow within the lobar and segmental bronchi, whereas the weaker expiratory component arises within the larger, more central airways.[79] Sounds are attenuated as they move peripherally along the air passages and are further damped by the large volume of the lungs' air spaces. The intensity of normal breath sounds varies with the magnitude of regional ventilation and, like percussion notes, diminishes with increasing thickness of the tissue overlying the chest wall. There is considerable variation among persons in the quality of breath sounds, which makes it essential to compare breath sounds from one side with those heard over the same location on the opposite hemithorax.

The transmission of normal lung sounds to the chest wall in pathologic conditions may be either attenuated or exaggerated. When the lung parenchyma is consolidated and the airway leading to the involved region is patent, breath sounds are well transmitted to the chest wall and are termed *bronchial breath sounds* (Audio 16-2). Bronchial breath sounds are loud, high-pitched, tubular, or whistling sounds with expiration as loud as or louder than with inspiration. Bronchial breath sounds are similar to *tracheal breath sounds* (Audio 16-3), and their presence is the classic auscultatory sign of pneumonia with consolidation. Similar sounds are heard in patients with other types of consolidation, such as pulmonary edema and hemorrhage. The presence of this sign assumes that the sounds originate centrally and reach the chest wall.[82]

Interposition of a sound barrier between the central airways where sounds originate and the chest wall where they are heard also attenuates or interrupts transmission of normal lung sounds. Accordingly, normal breath sounds are diminished or absent over a pleural effusion, pneumothorax, and peripheral bullae, or distal to an obstructing mass lesion. Conversely, they may be increased if chest wall deformity or bronchial or tracheal derangement allows movement of air to be closer than usual to the stethoscope.

Adventitious Sounds

The major types of adventitious sounds are classified in Table 16-2. Two generic categories of adventitious sounds have been documented by high-speed recording techniques, and each of these has two subdivisions: discontinuous sounds, including fine crackles and coarse crackles, and continuous sounds, including wheezes and rhonchi.[83]

Discontinuous Sounds (Crackles)

Crackles, still often referred to as "rales" in the United States and "crepitations" in Great Britain, consist of a series of short, explosive, nonmusical sounds that punctuate the underlying breath sound; fine crackles (Audio 16-4) are softer, shorter in duration, and higher in pitch than coarse crackles (Audio 16-5). There is general agreement that the brief recurrent detonations that characterize fine crackles are caused by the explosive openings of small airways that had closed owing to the surface forces within them.[79,84] This explains why fine crackles are much more common during inspiration than during expiration and why they are best heard over dependent lung regions—where airways are more likely to close—than over uppermost regions. This is also compatible with the presence of crackles in healthy elderly persons in whom dependent airways close at resting lung volumes. Crackles therefore are best heard during the first deep breaths at the lung bases posteriorly. After several such breaths or intentional coughing, these fine crackles will disappear if the small airways remain open throughout the time the patient is being examined.[84]

The timing of crackles is also important. Nath and Capel[85] have shown that late-inspiratory crackles are more often found in restrictive than obstructive lung disease. In a study by Pürilä and colleagues,[86] the crackles of pulmonary fibrosis began at 45% of inspiration, whereas those of nonfibrotic lung conditions were heard earlier: COPD at 25%, bronchiectasis at 33%, and heart failure at 37% of inspiration.[86] This suggests that more tension is required to open individual airways in fibrosis than in lungs with secretions or edema. As inspiration progresses, radial traction on airway walls increase until suddenly they pop open.[85] Thus crackles heard later in inspiratory time imply that the tension required to open individual airways is greater. Coughing or deep inspiration may change the quality of coarse crackles, such as those associated with underlying alveolar or airway disease, but the crackles rarely disappear entirely. Expiratory crackles are much less frequent than inspiratory crackles and are often seen in obstructive lung disease.[83]

Continuous Sounds (Wheezes)

The American Thoracic Society Committee on Pulmonary Nomenclature defined wheezing as high-pitched (dominant frequency of > 400 Hz) continuous adventitial lung

sounds.[82] Continuous sounds are longer than 250 msec in duration. Wheezes are usually louder than the underlying breath sounds and frequently noted by patients. A leading theory is that wheezes are produced by fluttering of the airway walls and fluid together induced by a critical flow velocity.[87] The pitch of the wheeze is dependent on the mass and elasticity of the airway walls and the flow velocity. The degree of bronchial obstruction is proportional to the amount of the respiratory cycle that it occupies. There is no relationship to the intensity or pitch of the wheeze and pulmonary function. Wheezes are well heard over the trachea, and listening over the trachea may be superior to listening over the lung in most asthmatic patients[87] (Audio 16-6). Wheezing with forced expiration can sometimes be provoked in healthy subjects[79] and does not establish a diagnosis of asthma. More helpful is to elicit wheezing and/or coughing at a full expiration without forced effort. Because several disease states are associated with wheezing, additional information should be obtained to make the correct diagnosis.

Rhonchi are low-pitched continuous sounds with a dominant frequency of approximately 200 Hz or less (Audio 16-7). These sounds are likely to originate from rupture of fluid films and airway wall vibrations.[79] Rhonchi may clear with cough or with suctioning in intubated patients. Some have questioned whether the term rhonchi is needed at all, finding the substitute "low-pitched wheezes" more parsimonious.[88] However, the term retains its place in classification systems and in clinical usage.[79,80,89]

Voice-Generated Sounds

Another way of generating sounds for auscultation is to have a patient speak while the examiner listens to his or her chest. Ordinarily the patient is asked to say in a quiet voice "one, two, one, two," "ninety-nine, ninety-nine," or "E, E." If enhanced responses are heard, the patient repeats the words while whispering. Because sounds of central origin are attenuated as they are transmitted peripherally through normal air-filled lung, voice-generated sounds have a muffled quality and the words are indistinct. In contrast, in the presence of consolidation, the characteristics of the sounds are remarkably different. The term *egophony* (Audio 16-8) indicates sounds that have a high-pitched, bleating quality; a change in sound-filtering properties of consolidated lungs accounts for the presence of egophony, which does not require, as often stated, the presence of an overlying pleural effusion. *Bronchophony* (Audio 16-9) and *pectoriloquy* (Audio 16-10) both mean that spoken sounds are transmitted with increased intensity and pitch; when each syllable of every word, especially when whispered, is distinct and easily recognized, pectoriloquy is the preferred description. An E-to-A sign means that the spoken letter "E" sounds like "A" while listened to over the lungs. Each of these auscultatory findings is a manifestation of the same acoustic property of consolidated lungs and thus has similar diagnostic significance.

Pleural Friction Rub

The small amount of liquid normally present in the pleural space separates the visceral and the parietal pleural layers and allows the lungs to expand and contract freely during breathing. In contrast, when the pleural surfaces are thickened and roughened by an inflammatory or neoplastic process, easy motion is prevented and a pleural friction rub may be produced. These sounds vary in intensity but often have a leathery or creaking quality that may be exaggerated by pressure with the stethoscope (Audio 16-11). Typically rubs are heard during both inspiration and expiration, but they are evanescent and variable and may be heard in only one part of the respiratory cycle. Surprisingly, rubs may still be heard in the presence of a large pleural effusion, which prevents the coarsened pleural surfaces from actually rubbing against each other.

Extrapulmonary Sounds

The presence of air or other gas in the mediastinum may be associated with crunching, crackling sounds that are synchronous with cardiac contraction and are audible when breathing is momentarily stopped. The finding of a mediastinal crunch by auscultation usually signifies mediastinal emphysema, even when the chest radiograph shows no abnormalities. In contrast, a pleural friction rub is usually heard during both inspiration and expiration and has a higher pitch.

Stridor is a high-pitched continuous sound produced by turbulent flow in the extrathoracic airway, which—in contrast to wheezing—is louder and longer during inspiration than expiration (Audio 16-12). Stridor has many causes,[90] some of which are life-threatening and need immediate attention (see Chapter 49).

As previously mentioned, various sounds may originate from the chest wall itself. Some of these have pathologic significance; others do not. Rubbing hairs trapped between the skin and the stethoscope produce intermittent crackling sounds that may be confused with crackles (Audio 16-13). Variable crackles are also produced when the stethoscope is placed over an area of subcutaneous emphysema and is rocked back and forth (Audio 16-14). Contracting chest wall muscles may generate sounds that have a muffled, distant, low-pitched, and rumbling quality. Occasionally it is possible to hear a snapping sound during breathing from motion of a newly fractured rib (Audio 16-15).

Interpretation

When abnormalities are discovered on physical examination of the chest, it is useful to identify them by their anatomic location in the involved lung. This requires knowledge of the surface projections of the underlying bronchopulmonary lobes, which are shown in Figure 16-2. The upper and lower lobes of both lungs are separated by the two oblique fissures, which course from the spinous process of the third thoracic vertebra posteriorly to the level of the 6th rib in the midclavicular line anteriorly. On the right side anteriorly, the upper and middle lobes are separated by the horizontal fissure, which lies at about the level of the fourth costal cartilage. In the presence of either distortions of pulmonary anatomy or the shape of the rib cage, the surface projections of the underlying lung also change.

The classic findings on physical examination of the chest in some common pulmonary disorders are shown in Table 16-3. Ordinarily, consolidation must be within 1 or 2 cm of the costal surface to be reliably detected. Even then, physical examination alone cannot be relied upon to diagnose or exclude pneumonia.[91] Some pneumonias,

Table 16-3 Classic Physical Findings in Some Common Pulmonary Disorders

Disorder	Inspection	Palpation	Percussion	Auscultation
Bronchial asthma (acute attack)	Hyperinflation; use of accessory muscles	Impaired expansion; decreased fremitus	Hyperresonance; low diaphragm	Prolonged expiration; inspiratory and expiratory wheezes
Pneumothorax (complete)	Lag on affected side	Absent fremitus	Hyperresonant or tympanitic	Absent breath sounds
Pleural effusion (large)	Lag on affected side	Decreased fremitus; trachea and heart shifted away from affected side	Dullness or flatness	Absent breath sounds
Atelectasis (lobar obstruction)	Lag on affected side	Decreased fremitus; trachea and heart shifted toward affected side	Dullness or flatness	Absent breath sounds
Consolidation (pneumonia)	Possible lag or splinting	Increased fremitus on affected side	Dullness	Bronchial breath sounds; bronchophony; pectoriloquy; crackles

Modified from Hinshaw HC, Murray JF, editors: *Diseases of the chest*, ed 4, Philadelphia, 1980, WB Saunders, p 23.

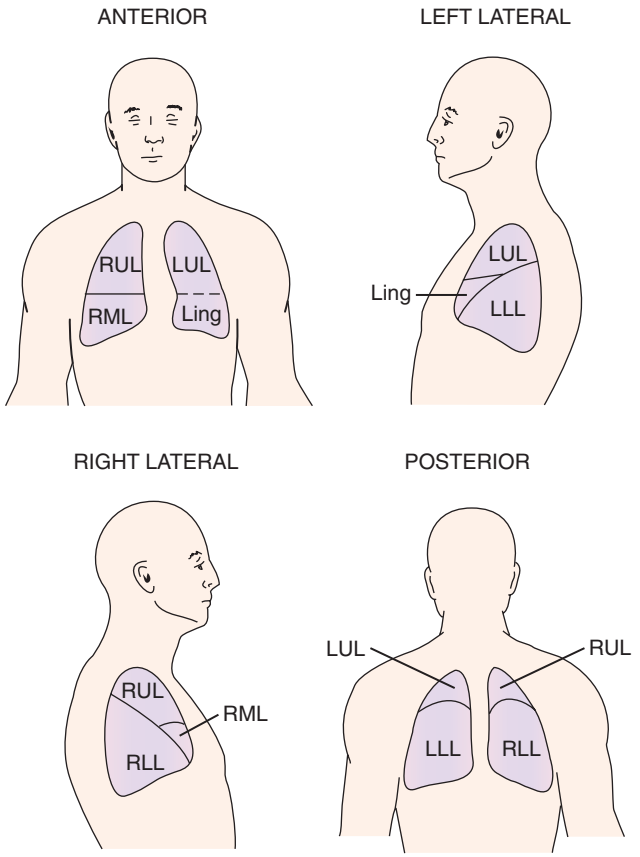

Figure 16-2 Schematic drawing shows surface projections of underlying lobar anatomy of a healthy man. Ling, lingular division of left upper lobe; LLL, left lower lobe; LUL, left upper lobe; RLL, right lower lobe; RML, right middle lobe; RUL, right upper lobe.

such as *Mycoplasma* pneumonia, typically cause surprisingly few physical abnormalities despite extensive radiographic involvement (see eFig. 33-9) but, even in patients with classic lobar pneumonia, the findings may be nonspecific. Although unable to distinguish reliably between new-onset pneumonia and other pulmonary diseases, the findings from physical examination—vital signs, mental confusion, cyanosis, use of accessory muscles, and paradoxical breathing—are extremely important in assessing severity and in deciding whether or not to hospitalize patients with pneumonia.[92]

The distinction between pleural effusion and atelectasis can be made on physical examination by determining whether the heart and mediastinal contents shift toward or away from the abnormal side, a finding that can usually be made only if the effusion is large or the atelectasis involves at least one lobe. When these full-blown manifestations are present, the presence of the causative disorder can be inferred with reasonable certainty. However, the absence of these findings does not exclude an abnormality, and a chest radiograph must always be taken as part of the complete pulmonary workup.

EXTRAPULMONARY MANIFESTATIONS

The examination of the lungs and pleura unlock only some of the clues to the presence of lung disease. Looking for extrapulmonary signs can often point toward a specific pulmonary disease or toward systemic diseases such as lupus erythematosus or toward diseases arising elsewhere in the body that secondarily involve the lung. Certain extrapulmonary manifestations are particularly useful.

Clubbing

The association of clubbing of the fingers or toes with disease has caught the attention of physicians since the time of Hippocrates. Clubbing is easy to recognize when it is severe (Fig. 16-3), but subtle changes are more common and less reliable. The hallmarks of clubbing are (1) a softening and periungual erythema of the nail beds, which causes the nails to seem to float rather than to be firmly attached, (2) an increase of the normal 165-degree angle that the nail makes with its cuticle, (3) an enlargement or bulging of the distal phalanx, which may be warm and erythematous, and (4) a curvature of the nails themselves. Of these features, the straightening of the nail cuticle angle appears to be the most sensitive measurement.[93]

Patients with clubbing may also have *hypertrophic osteoarthropathy,* a condition characterized by subperiosteal formation of new cancellous bone at the distal ends of

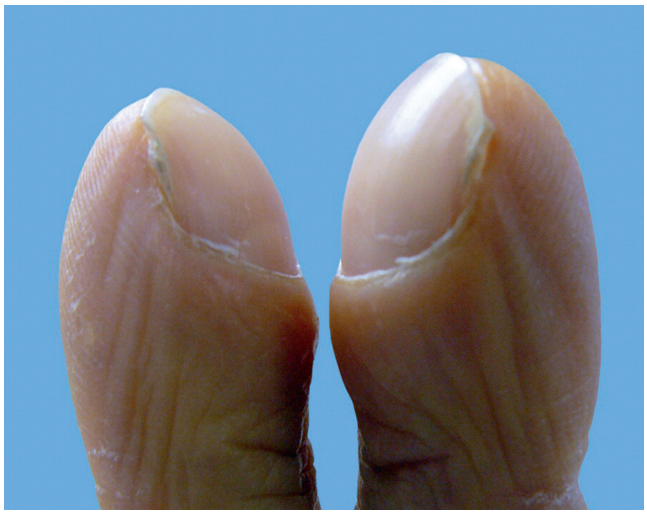

Figure 16-3 Clubbing of the digits as seen in severe diffuse interstitial pulmonary fibrosis. (From Cashman MW, Sloan SB: Nutrition and nail disease. *Clin Dermatol* 28: 420–425, 2010, Figure 2.)

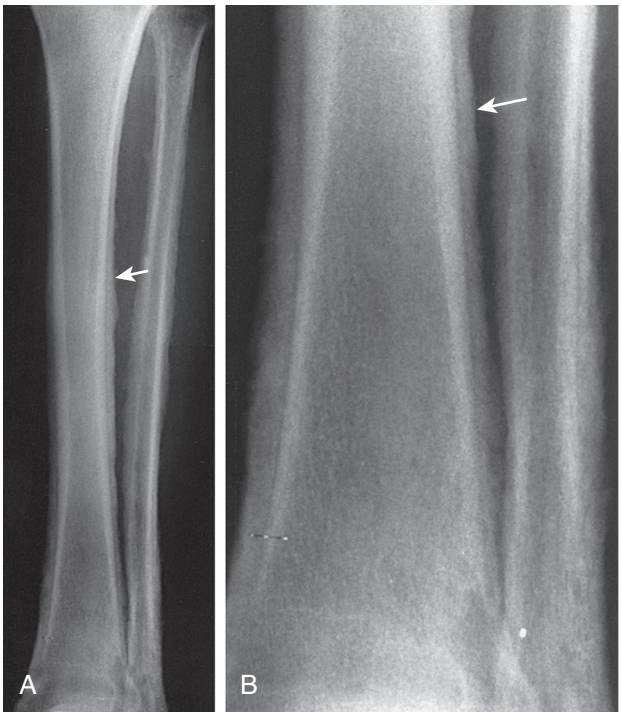

Figure 16-4 Radiographs of the leg show marked subperiosteal new bone formation (arrows) that is diagnostic of hypertrophic osteoarthropathy. A, Most of tibia and fibula. **B,** Detailed view near the ankle.

long bones, especially the radius and ulna and the tibia and fibula. Hypertrophic osteoarthropathy (Fig. 16-4) is almost always associated with clubbing, particularly in patients with bronchogenic carcinoma, other intrathoracic malignancies, and cystic fibrosis. It occasionally develops in patients with bronchiectasis, empyema, and lung abscess but is rare in patients with most of the other conditions in which clubbing has been observed.[94] One of the striking features of clubbing is the speed with which it can develop, about 2 weeks in patients with new-onset empyema, and with which it can reverse, also about 2 weeks in patients after corrective cardiac surgery. The presence of clubbing, which was found in 1% of all admissions to an internal medicine department, was associated with "serious disease" in 40% of afflicted patients[95]; therefore new-onset clubbing always warrants a chest radiograph, and if no abnormality is found, a CT scan to look for a pulmonary neoplasm or other lesion, which may still be localized and curable.

Clubbing has been found in many diverse conditions, such as children with HIV,[96] hepatopulmonary syndrome,[97] and benign asbestos pleural disease[98] (Table 16-4). Both clubbing and hypertrophic osteoarthropathy can be idiopathic or familial; the familial form is often transmitted as a dominant trait. The hereditary form of hypertrophic osteoarthropathy is also called *pachydermoperiostosis,* a condition in which bone and joint involvement is often mild but furrowing of the skin of the face and scalp is usually marked.

The main pathologic finding in clubbing is increased capillary density. The most potent stimulus to new capillary growth is hypoxia, which causes an intense production of vascular growth factors, such as vascular endothelial growth factor. With histochemical staining, Atkinson and Fox[99] showed increases in vascular endothelial growth factor, platelet-derived growth factor, hypoxia-inducible factor-1α, and hypoxia-inducible factor-2α along with increased microvessel density in the stroma of clubbed digits. The second common characteristic of patients with digital clubbing is shunting of blood past the capillary bed of either the lung or the liver, which suggests that lack of metabolism of angiogenic factors that bypass a critical organ may be involved. Several of the conditions associated with clubbing have inflammation and shunting, such as bronchiectasis and liver cirrhosis.

Other Extrapulmonary Associations

Besides clubbing, thoracic neoplasms may cause other extrathoracic abnormalities that may become evident on physical examination, including anemia, Cushing syndrome, gynecomastia, and other paraneoplastic syndromes (Table 16-5). Other common extrathoracic manifestations that provide clues to the presence or state of an underlying malignancy are wasting, hoarseness, adenopathy (especially supraclavicular), and hepatomegaly. When evaluating patients with dyspnea, a thorough examination of the neck veins for evidence of increased central venous pressure and careful cardiac auscultation for the presence of a third heart sound or distinctive murmurs should be performed to exclude heart failure.[100] The extremities should also be examined for evidence of peripheral edema, venous thrombosis, chronic venous stasis, and scars that suggest injection drug abuse.

The association of abnormalities in other organ systems with underlying lung disease can be very helpful in making a diagnosis.

Important lesions associated with various primary lung disorders are listed in tables available in the electronic version of the text at ExpertConsult.

A wide variety of cutaneous or subcutaneous lesions (eTable 16-1)[99] and ocular lesions (eTable 16-2)[100] has been associated with various primary lung disorders. Likewise, a combination of lung and kidney disease (eTable 16-3); lung and bone, joint, muscle, or nerve lesions (eTable 16-4); and gastrointestinal and hepatic involvement (eTable 16-5) may suggest a unifying disease process that can be detected by physical examination.

eTable 16-1 Skin and Subcutaneous Lesions Associated with Lung Disease

SKIN LESIONS

Diffuse pigment change
- Acanthosis nigricans—lung neoplasm
- Albinism—Hermansky-Pudlak syndrome
- Bronze pigmentation—hemosiderosis
- Gray-brown—Whipple disease

Cutaneous draining sinus
- Fungal infections (especially histoplasmosis)
- Mycobacterial infections (especially tuberculosis)
- Necrotizing vasculitis
- Neoplasms (especially mesothelial tumors)
- Other bacterial infections (especially actinomycosis)

Cutaneous ulcers
- Beryllium disease
- Chronic venous insufficiency
- Fungal infections (especially histoplasmosis)
- Mycobacterial disease
- Necrotizing vasculitis
- Parasitic disease
- Polycythemia
- Sickle cell disease
- Tularemia

Cutaneous vasculitis
- Behçet syndrome
- Collagen vascular disease
- Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
- Granulomatosis with polyangiitis (Wegener granulomatosis)
- Sarcoidosis

Erythema multiforme
- Drug reactions
- Fungi (especially coccidiomycosis)
- *Mycoplasma* and other infectious agents
- Neoplasms

Exfoliative dermatitis
- Adverse drug reactions
- Chemotherapy
- Disseminated malignancy
- Graft-versus-host disease
- Radiation therapy

Flushing
- Bronchial carcinoid, pheochromocytoma, other neoplasms
- Carbon dioxide, cyanide, and other toxins
- Drugs
- Foods and vasodilatory substances
- Hormones
- Mastocytosis
- Metabolic states (e.g., hyperthyroidism, fever)

Macular rash
- Anti-glomerular basement membrane disease
- Café-au-lait spots (neurofibromatosis)
- Coal miner's scars
- Collagen vascular disease
- Idiopathic pulmonary fibrosis
- Rose spots (psittacosis)
- Sarcoidosis
- Syphilis
- Viral pneumonia

Maculopapular rash
- Amyloidosis
- Drug-induced lung disease
- Collagen vascular disease
- Gaucher disease
- Kaposi sarcoma
- Lung neoplasm
- Lymphoma
- Lymphomatoid granulomatosis
- Parasites
- Sarcoidosis
- Syphilis
- Vasculitis
- Viral pneumonia

Sicca syndrome (dry mouth and eyes)
- Gaucher disease
- Lymphocytic interstitial pneumonia
- Sjögren syndrome

Telangiectasia
- Arteriovenous malformation
- Ataxia-telangiectasia
- Carcinoid syndrome
- Cushing disease
- Hepatopulmonary syndrome and other chronic liver diseases
- Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu)
- Mastocytosis
- Systemic sclerosis and other collagen vascular diseases

Urticaria
- Asthma
- Drug reactions
- Cystic fibrosis
- Exercise-induced urticaria
- Food allergy
- Hereditary angioneurotic edema
- Infectious agents, such as *Mycoplasma* and *Helicobacter*
- Inhaled antigens
- Insect bites and stings
- Mastocytosis
- Occupational sensitization
- Parasites
- Vasculitis

NAIL CHANGES WITH LUNG DISEASE

Color changes
- Cigarette smoking discoloration
- Splinter hemorrhages
- Yellow nail syndrome

Beau lines (any severe illness)
- Dermatomyositis
- Sarcoidosis
- Seronegative arthropathies
- Systemic sclerosis

LUNG DISEASE WITH SUBCUTANEOUS INVOLVEMENT

Adenopathy
- Environmental mycobacteria
- Fungal infections
- HIV infections
- Metastatic neoplasm
- Leukemia
- Lymphoma
- Sarcoidosis
- Tuberculosis

Calcinosis
- Dermatomyositis
- Metastatic osteosarcoma
- Mixed connective tissue disease
- Scleroderma
- Tuberculosis
- Uremic metastatic calcification

Erythema induratum (Bazin disease)
- Aortic stenosis
- Cryoglobulinemia
- Nodular vasculitis
- Panniculitis
- Peripheral neuropathy
- Streptococcus infection
- Takayasu disease
- Tuberculosis and other mycobacterial disease
- Weber-Christian disease

Erythema nodosa
- Neoplasm
- Other infectious and inflammatory diseases
- Primary coccidiomycosis, histoplasmosis
- Primary tuberculosis
- Psittacosis
- Sarcoidosis

Subcutaneous nodules
- Amyloidosis
- Neoplasm
- Neurofibromatosis
- Rheumatoid arthritis
- Tuberous sclerosis (angiofibromas)
- von Recklinghausen disease
- Weber-Christian disease

LUNG DISEASE WITH SALIVARY GLAND ENLARGEMENT

Bulimia and aspiration
Gaucher disease
Lymphatic carcinoma
Lymphoid interstitial pneumonitis
Lymphoma
Other causes of lymphadenopathy
Sarcoidosis
Sjögren disease

eTable 16-2 Eye Involvement in Lung Disease

BLINDNESS

Amaurosis fugax
- Antiphospholipid syndrome
- Aspergillosis
- Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
- Granulomatosis with polyangiitis (Wegener granulomatosis)
- Temporal arteritis

Giant cell arteritis
Late stage of many diseases
Sarcoidosis

CHOROID

Histoplasmosis
Lupus erythematosus
Toxoplasmosis

CONJUNCTIVA

Allergic reaction
Chlamydia
Granulomatosis with polyangiitis
Herpes
Kaposi sarcoma
Sarcoidosis

CORNEA

Chlamydia
Granulomatosis with polyangiitis
Herpes
Syphilis

IRIS

Neoplasm
Neurofibromatosis

LENS

Cataracts
- Steroid use
- Tobacco smoking

Marfan syndrome (dislocated)

LIDS

Proptosis
- Graves disease
- Leukemia
- Neoplasm

Ptosis
- Myasthenia gravis
- Muscular dystrophy

OPTIC NERVE

Cryptococcosis
Granulomatosis with polyangiitis
Graves disease
Leukemia
Neurofibromatosis
Sarcoidosis
Syphilis

RETINA

Antiphospholipid syndrome
Behçet disease
Candidiasis
Cytomegalovirus, herpes, and other viruses
Diabetes
Disseminated intravascular coagulation
Dysproteinemia
Ehlers-Danlos syndrome
Embolic disease
Endocarditis
Fat emboli
Fungemia
Genetic metabolic deficiencies
Granulomatosis with polyangiitis
HIV disease
Leukemia, lymphoma
Lupus erythematosus
Macroglobulinemia
Marfan syndrome
Neoplasm (especially melanoma)
Polycythemia
Sarcoidosis
Sickle cell disease
Subacute bacterial endocarditis, sepsis (Roth spots)
Syphilis
Temporal arteritis
Toxoplasmosis
Trauma
Tuberous sclerosis

SCLERA AND EPISCLERA

Granulomatosis with polyangiitis
Inflammatory bowel disease
Rheumatoid arthritis
Sarcoidosis
Scleroderma
Systemic lupus
Systemic vasculitis

SICCA

Graft-versus-host disease
Rheumatoid arthritis
Sjögren syndrome

UVEA

Ankylosing spondylitis
Behçet disease
Crohn disease
Granulomatosis with polyangiitis
Herpes zoster
Inflammatory bowel disease
Reactive arthritis
Rheumatoid arthritis
Sarcoidosis
Syphilis

eTable 16-3 Renal Involvement in Lung Disease

GLOMERULONEPHRITIS

Anti-glomerular basement membrane disease
Sarcoidosis
Collagen vascular disease
Systemic vasculitis

NEPHROTIC SYNDROME

Amyloidosis
Disseminated Langerhans cell histiocytosis
Drug-induced lung disease
Paraneoplastic syndrome
Post transplantation
Pulmonary hydatid disease
Systemic lupus erythematosus
Vasculitis
Venous thrombosis

RENAL MASS

Granulomatosis with polyangiitis (Wegener's granulomatosis)
Lymphangioleiomyomatosis
Metastatic neoplasm
Renal carcinoid
Tuberous sclerosis

NEPHROLITHIASIS

Alveolar proteinosis
Cystic fibrosis
Hypercalcemic syndromes
Osteolysis from mycobacteria or fungi
Sarcoidosis

SYSTEMIC HYPERTENSION

Collagen vascular disease
Diffuse alveolar hemorrhage
Neurofibromatosis
Pulmonary-renal syndromes
Sleep apnea

eTable 16-4 Joint, Bone, Muscle, and Neurologic Involvement in Lung Disease

ARTHRITIS

Ankylosing spondylitis
Collagen vascular diseases
Reactive arthritis
Sarcoidosis
Systemic vasculitis
Tuberculosis

BONE LESIONS

Ankylosing spondylitis
Blastomycosis and other fungal disease
Collagen vascular diseases
Eosinophilic granulomatosis
Fibrous histiocytoma
Gaucher disease
Neoplasm
Sarcoidosis
Tuberculosis

MUSCLE DISEASE

Collagen vascular disease
Diabetes insipidus
Eosinophilic granulomatosis
L-Tryptophan
Polymyositis
Sarcoidosis

NEUROLOGIC DISEASE

Acute inflammatory polyneuropathy
Amyotrophic lateral sclerosis
Aspiration
Botulism
Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
Granulomatosis with polyangiitis (Wegener granulomatosis)
Lambert-Eaton syndrome
Myasthenia gravis
Organophosphate poisoning
Polio and postpolio syndrome
Sarcoidosis

eTable 16-5 Gastrointestinal and Hepatic Involvement in Lung Disease

ESOPHAGEAL REFLUX
Aspiration pneumonia
Asthma
Bronchiectasis
Bronchitis
Cough
Mycobacterial disease
Pulmonary fibrosis
Scleroderma
INFLAMMATORY BOWEL DISEASE
Adverse reactions to drug treatments
Bronchiectasis
Bronchiolitis
Bronchitis
COLOBRONCHIAL FISTULA
Desquamative interstitial lung disease
Eosinophilic lung disease
Interstitial lung disease
Necrobiotic nodules
Obstructive lung disease
Organizing pneumonia
Sarcoidosis
Serositis affecting pleura or pericarditis
Tracheal stenosis
LIVER
$Alpha_1$-antitrypsin deficiency
Chronic active hepatitis
Hepatopulmonary syndrome
Portopulmonary hypertension
Primary biliary cirrhosis
Hepatosplenomegaly
Amyloidosis
Collagen vascular disease
Eosinophilic granulomatosis
Lymphatic interstitial pneumonia
Sarcoidosis

Table 16-4 Causes of Clubbing (Partial Listing)

NOT ASSOCIATED WITH OVERT DISEASE
Hereditary clubbing
Sporadic clubbing
Pachydermoperiostosis

THORACIC NEOPLASMS
Lung cancer especially fibrous tumors (accounts for most clubbing)
Benign and malignant pleural tumors
Other thoracic neoplasms, including esophageal cancer, and lymphoma

HEART AND VASCULAR DISEASE
Cyanotic congenital heart disease
Subacute bacterial endocarditis
Infected aortic graft
Aortic surgery
Takayasu arteritis
Behçet syndrome

PULMONARY AV SHUNTING
Cyanotic congenital heart disease
Acquired heart disease
Pulmonary AV fistula
Hereditary hemorrhagic telangiectasias

INTERSTITIAL LUNG DISEASE
Asbestosis
Idiopathic pulmonary fibrosis
Collagen vascular disease
Langerhans cell histiocytosis
Lipoid pneumonia

CHRONIC INFECTIONS
Bronchiectasis
Bronchiectasis from sarcoidosis or tuberculosis
Lung abscess
Empyema
Cystic fibrosis

GASTROINTESTINAL AND LIVER DISEASE
Inflammatory bowel disease
- Crohn disease
- Ulcerative colitis
- Polyposis coli

Amebic colitis
- Bacillary dysentery

Liver disease
- Hepatoma
- Hepatopulmonary syndrome
- Biliary cirrhosis

Esophageal stricture

HEMOGLOBINOPATHY
Hemoglobinopathies
Congenital methemoglobinemia

OTHER
Thyroid acropathy
Secondary hyperparathyroidism
HIV-related
- Lymphoid interstitial pneumonia
- Other infections

Prostaglandin infusion
Fabry disease
Toxic exposure to arsenic, mercury, or beryllium

UNILATERAL CLUBBING
Vascular disorders
- Subclavian artery aneurysm
- Brachial AV fistula

Subluxation of the shoulder
Median nerve injury
Local trauma
Hemiplegia

AV, arteriovenous.

Table 16-5 Paraneoplastic Syndromes (Partial Listing)

PARANEOPLASTIC SYNDROMES
Acanthosis nigricans
Clubbing
Hypertrophic osteoarthropathy
Intravascular thrombosis
Muscle weakness
Neuropathy
Pemphigoid
Polymyositis-dermatomyositis
Raynaud phenomenon

ENDOCRINE SYNDROMES ASSOCIATED WITH LUNG NEOPLASMS
Acromegaly (growth hormone)
Diarrhea (vasoactive intestinal peptide)
Hypercalcemia (parathyroid-like substance)
Hyponatremia (inappropriate antidiuretic hormone)
Carcinoid syndrome (serotonin)
Cushing syndrome (ACTH)
Gynecomastia (gonadotropins) (prolactin)
Hyperglycemia and hypoglycemia (insulin)
Skin pigmentation (melanocyte-stimulating hormone, ACTH)

ACTH, adrenocorticotropic hormone.

Key Points

- Taking a careful history and performing a thorough physical examination are essential first steps in formulating a preliminary differential diagnosis of a patient's complaints.
- After the clinician arrives at a tentative diagnosis, selected radiographic, laboratory, and other tests are ordered for further and confirmatory evaluation.
- The electronic medical record provides documents of higher quality than written records, owing to improved organization, increased readability, use of supplementary material, and better comparisons.
- Because dyspnea, cough with or without hemoptysis, and chest pain are among the most common reasons for patients to visit physicians and because these symptoms may result from serious underlying chest disease, careful questioning and workup is mandatory.
- Physical examination can be performed virtually anywhere, provides important information, lends itself to serial observations, and increases patients' confidence in their physicians.
- Stridor, a high-pitched continuous sound which, in contrast to wheezing, is louder and longer during inspiration than expiration, can indicate a life-threatening upper airway obstruction and requires immediate attention.
- New-onset clubbing of the digits warrants detection and investigation owing to its frequent association with serious underlying disease.

Complete reference list available at *ExpertConsult*.

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