42 Past Pulmonary Disease

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Definition

The objective is to obtain a history of, or information pertaining to, past pulmonary diseases, especially those that are chronic, progressive, recurrent, or that may have an impact on present or future conditions.

Technique

History

Begin questioning with general statements, such as: "Have you ever had any problems with your lungs?" or "Has any doctor ever told you that you have lung problems?" Having already asked the patient about possible symptoms (dyspnea, wheezing, cough, hemoptysis) and possible predispositions (smoking, environmental inhalation), the goal is to elicit names of past pulmonary diseases, dates and places of treatment and/or hospitalization for them, and the possible existence of additional past medical data, especially places and dates of diagnostic studies such as chest roentgenograms.

An affirmative response to the above questions usually takes one of two forms: (1) a specific diagnosis, such as "emphysema" or "pleurisy" (which is, more frequently than not, mispronounced or misunderstood), for which the patient has been hospitalized and/or treated in the past; or (2) a suspicion by the patient that he or she may have a pulmonary disorder but that no evaluation has ever been made. Occasionally, a patient knows that he or she has been treated for a "lung problem" but does not know or does not remember a specific diagnosis.

If a specific diagnosis is stated, several things must be determined:

- How firm is the diagnosis?
- When did the patient first seek treatment for it?
- What were the dates and places of hospitalizations and/or treatment?
- If the illness is a chronic or recurrent one, what treatments have been followed in the past (as well as the present), what has been the course of the disease, and what has been the impact of the disease and/or treatment on the patient's life?

Regarding the firmness of the diagnosis, any specific disorder offered by the patient should be viewed with some skepticism. Do not convey this skepticism to the patient, however, because it might evoke a defensive reaction that could jeopardize rapport with the patient, or damage the confidence of the patient in his or her personal physician's previous care.

The examiner must be especially wary of diagnoses that are commonly misapplied or misinterpreted. Patients are often told by a physician that they have "emphysema" on the basis of a physical examination, a chest roentgenogram, a cigarette smoking history, or the previous physician's attempt to frighten the patient into stopping smoking. It is important to determine if the patient's symptoms are compatible with the diagnosis, if pulmonary function testing was performed as part of the evaluation, and if the patient had a clinical response to treatment such as bronchodilators. Similarly, a diagnosis of "asthma" (considered in detail in Chapter 37) is often used for patients with any type of "wheezing with dyspnea." A history of "tuberculosis," or "TB," could mean a definite diagnosis of pulmonary tuberculosis, a positive tuberculin skin test only, or a suspicion based on an abnormality on a chest roentgenogram. Patients are often told that they have a "spot on the lung" on a previous chest roentgenogram. This could represent a "pseudo lesion" or transient density no longer present, an unexplained roentgenographic density that needs prompt evaluation, or a stable density, representing benign, self-limited granulomatous infectious disease that needs confirmation by obtaining the previous chest roentgenogram for comparison. Another common past diagnosis is "pleurisy." This diagnosis has been carelessly applied in the past to any and all noncardiac chest pains including musculoskeletal. Patients who state that they have had a "collapsed lung" may have had a spontaneous pneumothorax, atelectasis without a pneumothorax, or neither. Inquiries should include whether or not a thoracostomy tube was inserted and whether or not a bronchoscopy was performed, and the results. A patient will sometimes state that he or she underwent the surgical removal of a "growth," "spot," or "tumor" from the lung. The next question should be, "Did your doctor tell you what it was?" or, more specifically, "Was it malignant?" or "Were you told it was cancer?" Amazingly, patients are often quite uninformed about such surgery; past medical records, once obtained, may tell an entirely different story than the patient relates.

It is totally impractical to present the patient with a long list of possible pulmonary disorders along with the question "Have you ever had. . .?" If the patient responds negatively to the initial question about "lung problems," certain disorders should be mentioned specifically (Table 42.1). For

Table 42.1
History of Specific Diagnoses

The patient should usually be asked about:
Tuberculosis
Asthma
Pneumonia, especially recurrent
Previous chest roentgenogram
The patient may be asked about:
Lung surgery
Lung cancer
Chronic bronchitis/emphysema
Any diagnosis that may seem to fit a specific situation

the possibility of tuberculosis, the following questions should be asked:

- "Have you ever had or been told that you might have had tuberculosis, or TB?" If so, then the date and place of diagnosis, whether the diagnosis was made on the basis of sputum smear and/or culture, the specific drugs and length of treatment, and the success or failure should be determined.
- "Have you ever had a TB skin test?" If so, the dates and the results should be recorded.
- "Have you ever been exposed to anyone with TB?" If so, determine whether this was intimate household contact or whether it was only limited contact with a distant, nonhousehold relative or casual acquaintance.

A past history of pneumonia should also be searched for by specific questioning if not volunteered in the initial general inquiry. Because the term *pneumonia* is well known to most patients, they may be asked simply, "Have you ever had pneumonia?" Patients may use outdated terms such as "walking pneumonia" or "double pneumonia" to describe a past episode. With an affirmative response, it should be determined if the diagnosis was made by a physician based on physical examination and/or chest roentgenogram or a conclusion of the patient based on an unusually severe respiratory illness for which he or she did not seek medical care. If a single episode of pneumonia occurred in the remote past, it is probably not worth additional questions; however, if the pneumonia occurred within the past 1 to 2 years and/or the patient has had multiple episodes of pneumonia, additional information should be obtained. It is important to determine the number of episodes and the dates; the places of treatment where chest roentgenograms and other pertinent information might be obtained; if the pneumonias have been in the same lobe or lobes of one lung or have occurred at random sites in both lungs; whether or not the patient has been well between episodes; and whether or not there has been a pattern of increasing frequency of episodes. The cigarette smoker with dyspnea on exertion and chronic sputum production may be asked, "Has any doctor ever told you that you have chronic bronchitis or emphysema?" If so, the certainty of this diagnosis should be pursued by additional questioning.

Regardless of any past history of specific lung diseases, patients should be asked, "When did you last have a chest x-ray before this present visit?" Names of the physician and/or facility where performed and the date should be obtained. If the patient's current chest roentgenogram is abnormal, suggesting a past or present pulmonary disorder, a previous chest roentgenogram is invaluable. Under these circumstances, the patient's recall may be helped by suggesting possible sources, such as preemployment medical examinations, health department screening examinations (especially if for tuberculosis exposure), military induction and discharge examinations, periodic physical examinations in a physician's office or an employee health clinic, routine chest roentgenograms taken at hospitals prior to elective surgery, as well as chest roentgenograms taken specifically to evaluate or follow pulmonary disorders.

Most outside sources of medical data and chest roentgenograms require an authorization for release of information. Including this with any written request will facilitate access to this data. Sometimes it is more expedient to contact the facility by telephone. If the caller adequately identifies himself or herself as legitimate, a representative of the facility might provide some information verbally. If not, the receipt of information by mail might be expedited by a verbal request.

Basic Science

Tuberculosis

The year 1982 marked the one hundredth anniversary of the announcement by Robert Koch that the tubercle bacillus, now named *Mycobacterium tuberculosis*, was the causative organism of tuberculosis. Once a dreaded disease, it has progressively decreased in incidence and as a cause of death in the United States. There has been a recent increase in the incidence of tuberculosis related to the AIDS epidemic. Transmission of the infection is usually by inhalation of contaminated airborne droplet particles from infected individuals. The organism is most commonly inhaled into the alveoli of the lower lobes where it is ingested by macrophages. It may either be killed by the macrophage or proliferate intracellularly, eventually killing the macrophage. Initial pulmonary infection is usually localized by the immune system; however, organisms often enter the blood stream via the pulmonary lymphatics in what is called a *silent bacillemia*. Organisms may lodge in any tissue, but are most apt to survive and proliferate in the pulmonary apices, the lymphatic system, serosal surfaces, the kidney, adrenal gland, and bone.

Host immunity is entirely cell mediated, with the macrophage as the effector cell and the T-lymphocyte as the immunoresponsive cell. The host response in the tissues is characterized by granuloma formation. With large amounts of antigen, the hypersensitivity reaction may itself cause tissue destruction (caseation or caseous necrosis). Healing occurs by fibrosis of the granuloma, walling off the lesion and hindering spread. Interestingly, the organism may survive for years in "healed" granulomas and may begin multiplying if host immunity weakens. The growth of the organism in the apex of the lung months to years after the initial infection produces the characteristic picture of upper lobe pulmonary tuberculosis. Proliferation at any other site produces extrapulmonary tuberculosis. A host whose immune system is unable to localize and kill the organism adequately may develop diffuse hematogenously borne disease known as *disseminated* or *miliary tuberculosis*.

Epidemiologically, the disease in this country has become largely one of the inner city; high rates are seen in overcrowded slum areas. The derelict alcoholic of the inner city commonly becomes infected. Whether there are racial differences in susceptibility is unclear and controversial.

Pneumonia

With the exception of hematogenously borne organisms that lodge in the lung, microbial organisms that cause pneumonia are either inhaled directly into the lung or reach the lung via upper airway secretions draining into the lower airway (aspiration). Whether an acute pneumonia results depends on the number of organisms reaching the lung and the lung's defense mechanisms. A large number of organisms can produce pneumonia in spite of normal defense mechanisms, whereas a much smaller number may be all that is required in the presence of impaired host defenses.

Almost all individuals aspirate small amounts of oropharyngeal secretions into the tracheobronchial tree, especially during sleep. Organisms present in this material include the normal oropharyngeal flora as well as bacteria that have colonized the upper respiratory tract. This colonization seems to depend on a phenomenon called *bacterial adherence*. Certain organisms are capable of attaching to upper respiratory squamous epithelial cells and thus become a transient or permanent flora. Over 50% of severely ill patients may develop oropharyngeal colonization with aerobic gram-negative bacteria, whereas only 3 to 6% of normal individuals are colonized. This colonization correlates highly with subsequent nosocomial pneumonia from these organisms.

The normal lung defense mechanisms are quite adequate to maintain the sterility of the tracheobronchial tree despite the daily inhalation and aspiration of organisms. Physical barriers include the filtering effect of the nose, sneezing, coughing, the tracheobronchial mucous blanket, and the mucociliary escalator. Cellular mechanisms are very important, with phagocytosis by the alveolar macrophage being the most important. Polymorphonuclear leukocytes are also important and may be attracted to the lung by neutrophil chemotaxins. Specific immunity by both B and T lymphocytes may be present or evoked, and there are also nonspecific components of tracheobronchial secretions that inactivate or kill organisms. These include secretory IgA, lysozyme, interferon, lactoferrin, complement, and granulocyte proteases. Any pathologic state that interferes with one or more of these defense mechanisms increases the risk of pulmonary infection.

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is probably the most commonly used term in this country to refer to patients who have chronic bronchitis and/or emphysema. The accepted clinical definition of *chronic bronchitis* is sputum production on most days of the week for at least 3 months of the year during 2 or more consecutive years. Thus, the basis of this definition is chronic mucus hypersecretion by the bronchial tree related to hypertrophy and hyperplasia of the bronchial mucous glands and an increase in the number of goblet cells. *Emphysema* is defined as a condition of the lung characterized by increase beyond normal in the size of air spaces distal to the terminal bronchioles either from dilation or destruction of their walls.

In most instances, the cause of chronic bronchitis and/or emphysema is cigarette smoking, although there are hereditary forms of emphysema (e.g., alpha-1 antitrypsin deficiency). Functional impairment (decreased exercise tolerance) relates to chronic airflow obstruction (CAO) as measured spirometrically, ventilation–perfusion mismatching, and, with emphysema, a reduction in both alveolar surface area and the pulmonary capillary bed.

Only about 4 to 5% of cigarette smokers will develop disabling CAO by age 65. It is not completely clear why some individuals develop CAO and others do not. In the case of chronic bronchitis, current data suggest that chronic mucus hypersecretion and CAO are independent processes. Experimental emphysema can be produced by aerosolizing human neutrophil elastase into animals' lungs. The protease–antiprotease theory of emphysema proposes that emphysema results whenever more neutrophil elastase is liberated in the lung than the antiproteases are able to inactivate. There are also data to suggest that cigarette smoking may result in neutrophil accumulation in the lung, and that cigarette smoke may interfere with the action of antiproteases such as alpha-1 antitrypsin.

The clinical picture of relatively pure chronic bronchitis is characterized by wheezing, cough and sputum production, hypoxemia, and intermittent hypercapnia, cor pulmonale, and acute respiratory failure. Response to medications such as bronchodilators in early states is substantial; with progression, however, medications become less and less effective. More pure emphysema is characterized almost exclusively by a progressive decrease in exercise tolerance. Toward the end of its course, the patient becomes exhausted with such minor activities as shaving, bathing, and eating. Hypercapnia, cor pulmonale, and acute respiratory failure are near-terminal occurrences and seldom reversible.

Pleurisy and Pneumothorax

The pleural space is a potential space. Parietal and visceral pleura are in constant contact, and the two surfaces glide smoothly across each other during respiratory motion. Any disease state that produces inflammation of the pleural surfaces causes them to become roughened with fibrinous exudate. This can result in the symptom of pleuritic chest pain mediated by the pain fibers in the parietal pleura. A pleural effusion and pleural friction rub may or may not occur.

Air may be present in the pleural space from a perforation of the visceral pleura, perforation of the chest wall and parietal pleura, or from gas-forming bacteria in the pleural space. A spontaneous pneumothorax, or "collapsed lung," occurs by the former mechanism.

Idiopathic spontaneous pneumothorax occurs predominately in males between the ages of 20 and 40. The rupture of small, subpleural blebs are thought to be the cause. Taller individuals seem to be more predisposed, presumably because the greater height of the lungs creates a greater pleural pressure differential from top to bottom, resulting in more negative pressure at the apex of the lungs where the blebs are thought to rupture.

In patients over the age of 40, COPD predisposes to spontaneous pneumothorax. Pneumothorax may be a complication of metastatic pulmonary malignancies, especially sarcomas. Catamenial pneumothorax is thought to be related to pleural endometriosis.

Chest Roentgenogram

Despite the emergence of complex diagnostic techniques such as computerized axial tomography and nuclear magnetic resonance, the standard posteroanterior and lateral chest roentgenograms remain inexpensive and valuable tools. Round densities in the lung must usually be greater than .35 cm in diameter to be visible unless they are calcified. A carcinoma of the lung may have been present for several years before reaching a size at which it can be seen by chest roentgenogram.

Many of the previous widespread screening uses of the chest roentgenogram have been abandoned. Mass chest x-ray screening for tuberculosis has not been cost effective for the past 20 years. Screening for lung cancer is controversial. Screening x-rays in routine hospital admissions have been challenged. Screening chest x-rays in patients under

age 40 prior to elective surgery also seems to be of little value.

The chest x-ray is, however, valuable in following the resolution of pneumonia and the increase in size of undiagnosed pulmonary lesions, as well as in assessing suspected pulmonary disease.

Clinical Significance

Tuberculosis

The official American Thoracic Society classification of tuberculosis is shown in Table 42.2. Certain patients who fall into class 2 should receive isoniazid 300 mg daily for 9 to 12 months. These include: (1) recent tuberculin skin test converters; (2) household contacts of known cases; (3) persons under age 35; (4) persons with increased susceptibility to tuberculosis, including patients on high doses of corticosteroids and/or immunosuppressive drugs, hematologic and reticuloendothelial diseases that suppress cell-mediated immunity, silicosis, diabetes mellitus, and the post-gastrectomy patient. Patients who fall into class 4 should receive similar treatment if they have never received adequate antituberculous chemotherapy. Such patients have reactivation rates that range from .5 to 5% per year.

A patient with active pulmonary tuberculosis whose past history includes previous treatment for tuberculosis in the early days of chemotherapy with isoniazid, streptomycin, and/or para-amino salicylic acid has approximately a 40% chance of having organisms resistant to one or more of these drugs, especially isoniazid. Until susceptibility testing results are known, coverage should include isoniazid, rifampin, ethambutol, and/or pyrazinamide. Other patients who might harbor isoniazid-resistant organisms include immigrants from Asia, Latin America, or Africa; patients who acquired disease from a patient with isoniazid resistance; and patients who previously received isoniazid chemoprophylaxis.

Bear in mind that other granulomatous pulmonary infections, such as histoplasmosis, or other pulmonary fungal infections, as well as necrotizing bacterial pneumonias may produce roentgenographic infiltrates similar to tuberculosis.

Pneumonia

Although pneumonia is a common disease, it requires a major breakdown in host defenses and is therefore somewhat unusual in an otherwise healthy person. In such a person, depression of defense mechanisms may be tran-

Table 42.2
American Thoracic Society Classification of Tuberculosis

0 No tuberculosis exposure, not infected (no history of exposure, insignificant tuberculin skin test reaction).

1 Tuberculosis exposure, no evidence of infection (history of exposure, insignificant tuberculin skin test reaction).

2 Tuberculous infection, no disease (significant tuberculin skin test, negative bacteriologic studies if done, no symptoms or roentgenographic evidence of tuberculosis).

- Chemotherapy status (preventive):
  - None
  - On chemotherapy since (date)
  - Chemotherapy terminated (date)
    - Complete (prescribed course of therapy)
    - Incomplete

3 Tuberculosis: current disease (*M. tuberculosis* cultured, or both a significant tuberculin skin test reaction and clinical and/or roentgenographic evidence of current disease).

- Location of disease (predominant site; other sites if important):
  - Pulmonary
  - Pleural
  - Lymphatic
  - Bone and/or joint
  - Genitourinary
  - Disseminated (miliary)
  - Meningeal
  - Peritoneal
  - Other
- Bacteriologic status:
  - Positive by
    - Microscopy only (date)
    - Culture only (date)
    - Microscopy and culture (date)
  - Negative (date)
  - Not done
- Chemotherapy status:
  - On chemotherapy since (date)
  - Chemotherapy terminated (date)
    - Complete (prescribed course of therapy)
    - Incomplete

The following data are necessary in certain circumstances:

- Roentgenogram findings:
  - Normal
  - Abnormal
    - Cavitary or noncavitary
    - Stable, worsening, or improving
- Tuberculin skin test:
  - Significant
  - Not significant

4 Tuberculosis: no current disease (history of previous episode(s) of tuberculosis, or abnormal stable roentgenographic findings in a person with a significant reaction to tuberculin skin test, negative bacteriologic studies, no clinical or roentgenographic evidence of current disease).

- Chemotherapy status:
  - None
  - On chemotherapy since (date)
  - Chemotherapy terminated (date)
    - Complete
    - Incomplete

5 Tuberculosis suspect (diagnosis pending)

- Chemotherapy status:
  - None
  - On chemotherapy since (date)

sient. Viral respiratory infections, especially influenza, impair lung bacterial clearance and predispose to secondary bacterial pneumonia, reaching a peak at 7 to 10 days from the onset.

Recurrent pneumonias tend to occur in individuals who have chronic alterations in lung defense mechanisms. Recurrent pneumonias can generally be categorized into those that have no predilection for any particular area in either lung and those that tend to occur in the same lobe or lobes (Table 42.3).

Recurrent pneumonias in the same lobe or lobes suggest a focal problem with mucociliary clearance. Partial or complete obstruction of a bronchus by a malignant or benign neoplasm or by an aspirated foreign body can cause either recurrent or persistent pneumonia in one area of a lung. Localized bronchial disease such as bronchiectasis also can predispose to distal pneumonia. Localized bronchial narrowing from extrinsic compression, as in the right middle lobe syndrome, can also cause recurrent distal pneumonias. Recurrent aspiration of gastric contents can produce recurrent pneumonias in gravity-dependent segments of the lungs that include the superior and posterior basilar segments of the lower lobes and the posterior segments of the upper lobes.

Recurrent pneumonias that have no tendency to occur in any one particular area can occur in a number of clinical settings. These include: (1) general debilitating illnesses such as alcoholism, diabetes mellitus, congestive heart failure, and carcinomatosis; (2) situations of impaired humoral and/or cellular immunity including lymphoma, leukemia, multiple myeloma, immunoglobulin deficiency, the acquired immune deficiency syndrome (AIDS), and treatment with corticosteroids and/or immunosuppressive drugs; (3) diffuse airway diseases including asthma (especially in children), chronic bronchitis and emphysema, cystic fibrosis, and the immotile-cilia syndrome; and (4) ineffective ventilation and cough as can occur with a depressed level of consciousness from any etiology or neuromuscular weakness, as with myasthenia gravis or muscular dystrophy.

In approaching patients with recurrent pneumonia or persistent pneumonia in the same site(s), diagnostic measures should be aimed at detecting bronchial obstructions with bronchoscopy and/or radiographic techniques such as computerized axial tomography. Patients who have recurrent pneumonias at random sites who do not have obvious predisposing conditions should be considered for study of serum immunoglobulin determinations, bone marrow aspirations, and urine and/or serum protein electrophoresis. Patients with recurrent pneumonias due to *Streptococcus pneumoniae* may benefit from pneumococcal vaccine. Patients with bronchiectasis sometimes benefit from suppressive antibiotic therapy as well as postural drainage.

Table 42.3
Recurrent Pneumonia

Pneumonia occurring in same site(s)
Narrowing or obstruction of proximal bronchus
Focal bronchiectasis
Recurrent aspiration
Pneumonia occurring in random sites
General debilitating illnesses
Impaired humoral and/or cellular immunity
Diffuse airway diseases
Ineffective ventilation and cough

Chronic Obstructive Pulmonary Disease

A history of COPD is important from several standpoints. First is prognosis. Patients with COPD lose about 6 to 8% (average of 80 ml) of their forced expiratory volume in 1 second (FEV_1) each year, although this varies considerably from patient to patient. Data suggest that the cessation of cigarette smoking results in a less rapid decline in function; therefore a major effort should be launched to get the patient to quit smoking. This should be done in a very sympathetic, understanding manner and *not* with the approach of "Can't you see what you are doing to yourself [you idiot]?" The use of nicotine gum plus counseling can be quite helpful.

Second, a diagnosis of COPD should bring to mind several predispositions. These patients are more likely to contract lobar or bronchopneumonia. If they undergo major abdominal or thoracic surgery, they are more apt to develop postoperative atelectasis, pneumonia, and respiratory failure than the normal patient. Patients with COPD also have a higher incidence of pulmonary thromboembolism and duodenal ulcer.

The patient with COPD may not be on an optimum medical regimen. The addition of aerosolized and oral bronchodilators in most patients (and corticosteroids in selected patients) can improve symptoms and functional capacity considerably. Additionally, data now suggest that patients with chronic hypoxemia ($Pao_2 < 55$ mm Hg) and/or evidence of hypoxic organ dysfunction (cor pulmonale, secondary polycythemia) have a lower 3- to 5-year mortality when given oxygen sufficient to raise the Pao_2 to 60 to 80 mm Hg at home for at least 16 hours each day.

Lastly, a very young patient with emphysema may have alpha-1 antitrypsin deficiency. If so, other members of the family are at risk, and genetic counseling may be in order.

Pleurisy

If a history of "pleurisy" is elicited, a number of possible etiologies exist. A single past episode in an otherwise healthy individual was most likely viral or idiopathic pleurisy or perhaps related to pneumonia. It is probably of little clinical importance.

On the contrary, other causes of pleurisy may have greater significance. Patients who develop tuberculous pleurisy and are not treated have a very high incidence of pulmonary or extrapulmonary tuberculosis within 1 to 2 years of the episode. Previous exposure to tuberculosis and a positive tuberculin skin test reaction should raise a high index of suspicion. Acute pulmonary embolism is a serious cause of pleuritic chest pain. Patients with predisposing factors for, or a past history of, venous thrombosis should be scrutinized carefully because pulmonary thromboembolism tends to be recurrent and can be fatal. Pleuritic chest pain and a pleural effusion can also occur with polyserosal disorders such as systemic lupus erythematosis. Any disorder capable of causing an exudative pleural effusion can cause pleuritic chest pain.

Pneumothorax

A history of a previous spontaneous pneumothorax is important because the recurrence rate of idiopathic spontaneous pneumothorax can be as high as 60%. Multiple recurrences may warrant consideration for some type of pleural ablative procedure, usually surgical.

A young woman who develops recurrent spontaneous pneumothoraces during her menses is suffering from the rare condition of catamenial pneumothorax. Both hormonal therapy and pleural ablation have been used to prevent recurrences.

Chest Roentgenogram

Obtaining a previous chest x-ray for comparison in the patient with a solitary pulmonary nodule can save the patient the expense, discomfort, and possible morbidity of invasive diagnostic procedures if it can be shown that the lesion has been stable for several years. Patients with stable benign pulmonary lesions can be given a copy of their chest x-rays to keep at home. One of the most frustrating circumstances is to find a lesion on a current chest x-ray that was probably present on a previous x-ray, but the previous x-ray has been misplaced or destroyed, and the stability of the lesion cannot be proved. Previous chest roentgenograms help to evaluate the progression, stability, or resolution of numerous diffuse and localized pulmonary diseases.

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