# 6

# Imaging Community-Acquired Pneumonia

GERRY SCHALLER AND MARK LOGAN

## Introduction

Community-acquired pneumonia (CAP) is defined as pneumonia* that developed either outside the hospital or within the first 48 hours after admission (Areno et al., 1996). A chest radiograph is often included in the initial work-up of suspected pneumonia. If the imaging facility is hospital-based it is likely that a radiologist will be available to examine the films and provide a timely report. However, often the attending physician must examine the chest radiographs and rely on her or his own interpretation as part of the diagnostic work-up. Thus it is essential that every physician who treats patients with pneumonia know how to interpret a chest radiograph.

This chapter discusses diagnostic imaging as it applies to CAP. The majority of the discussion relates to standard chest radiographs as virtually all patients who require imaging require only chest radiography. It is important to note that the radiologist does not distinguish CAP from hospital-acquired pneumonia on the basis of the chest radiographs. That distinction is made on a clinical and epidemiological basis; the imaging features of pneumonia are similar for both.

*Terminology used in this chapter is in accordance with Fraser, R. G., Paré, J. A. P., Pare, P. D., Fraser, R. S., & Genereux, G. P. (1988) Glossary of words, terms and symbols in chest medicine and roentgenology, In: *Diagnosis of Diseases of the Chest,* 3rd ed. Philadelphia: W. B. Saunders Co., pp. xiii–xxx.

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## The Role of the Chest Radiograph

When the clinical findings suggest that the patient has CAP, a chest radiograph enables the attending physician to confirm the clinical suspicion of pneumonia, assess the extent of the infection, detect concurrent conditions that may complicate diagnosis or management, and expedite empiric therapy decision-making (Torres et al., 1996; Conces, 1994).

In the outpatient setting some patients are mildly ill and are often treated entirely on the basis of a clinical diagnosis of CAP (Areno et al., 1996). It is important to note that the diagnosis of pneumonia based on symptoms and signs is not reliable (Metlay et al., 1997). If CAP is suspected clinically, in most patients a chest radiograph should be obtained at the time of the initial work-up. Generally, the radiographic manifestations of pneumonia will appear within 12 hours of the onset of clinical symptoms (Herold, 1997). In the outpatient setting, patients typically will have been ill for several days before presentation. Therefore, most ambulatory patients with pneumonia will have radiographically visible evidence of their disease. Hospitalized patients will often, however, be imaged more promptly and may not have yet developed radiographic evidence of pneumonia at the time of their chest radiograph. Close follow-up of these patients will be necessary to detect or to exclude pneumonia. The

sensitivity of the chest radiograph will also be lower, especially in the elderly, when there is dehydration or other debilitating illness (Ely & Haponik, 1991) that delays the development of a pulmonary inflammatory response sufficient to cause visible opacities on the films. In these cases, as the concurrent illness is brought under control, imaging should be repeated to detect developing radiographic evidence of clinically suspected pneumonia.

Radiographic evidence of comorbid illness (e.g., carcinoma) may be obscured by an acute pneumonia on a chest radiograph obtained at the initial work-up. If a patient presenting with CAP requires chest radiographic screening for comorbid illness the initial chest radiograph might not be of any benefit in that regard. Under such circumstances a follow-up chest radiograph would be indicated when treatment of the pneumonia can be expected to have reduced the extent of the pneumonia enough to adequately assess the chest x-ray for underlying abnormality.

Patients presenting with symptoms consistent with a lower respiratory tract infection may not have pneumonia. In fact, studies suggest that from 8% to 30% of patients clinically suspected of having CAP actually have another diagnosis (Areno et al., 1996). The clinical diagnosis of pneumonia is best confirmed by a chest radiograph showing new or progressive findings that are consistent with pneumonia and that correlate with the patient's signs and symptoms (Areno et al., 1996; Herold, 1997).

Imaging is useful and necessary to assess the extent of the infection and to detect complications in those patients who appear clinically to be more seriously ill, those who are at risk for complications, those who require hospitalization (Areno et al., 1996), and those suspected of having severe CAP. Severe CAP is defined as a life-threatening pneumonia, acquired in the community by a nonimmunocompromised patient, that requires admission to the intensive care unit (ICU). The American Thoracic Society Guidelines for the classification of CAP as severe include radiographic criteria. In addition to the clinical criteria, a chest radiograph showing bilateral pneumonia or pneumonia involving multiple lobes or an increase in the size of the pneumonia of 50% or greater within the preceding 48 hours justifies defining a case of CAP as severe and requiring ICU admission (Torres et al., 1996). Patients most at risk for severe CAP and for other complications include those with chronic underlying cardiopulmonary disease, e.g., chronic obstructive pulmonary disease [COPD] and those with chronic conditions such as diabetes mellitus, alcoholism, and neuromuscular disorders (Torres et al., 1996).

As will be discussed later, some patterns of pneumonia on the chest radiograph can be associated with certain causative organisms or groups of organisms. However, there is a great deal of overlap between the various infectious and noninfectious causes of pulmonary infiltrates and opacities. Chest radiography is therefore no more able than other noninvasive clinical tests to provide a specific etiologic diagnosis in the majority of cases of pneumonia. In fact, both sputum culture and radiology are able to identify the correct etiology in less than 50% of patients (Areno et al., 1996; Bowton & Bass, 1991; Fein, 1996).

On the other hand, analysis of the radiographic pattern in the context of the patient's clinical and epidemiological circumstances (Conces, 1994; Fein, 1996; Herold, 1997) will often enable the clinician to narrow the list of probable causes and thereby facilitate empirical therapy decisions.

## Technical Considerations

Standing posteroanterior and lateral views of the chest in full inspiration comprise the usual and the best initial radiologic examination of a patient suspected of having pneumonia (Conces, 1994; Milne & Pistolesi, 1993; Novelline, 1997). Orthogonal views (i.e., views at right angles to each other) are essential (Freundlich & Bragg, 1992) to enable the viewer to examine regions that would be obscured if only a single view had been obtained and to better localize and characterize detected abnormalities. A posteroanterior radiograph places the patient with his or her chest against the film, minimizing the magnification of the heart and the mediastinum on the image, which minimizes the amount of lung obscured by these structures. Similarly, on the lateral view the size of the heart on the image is minimized if the left side is against the film. The *left-lateral* is therefore the preferred position for the

lateral view. The right side will be magnified slightly more than the left. Therefore, on a well-positioned left-lateral the right ribs are larger and project behind the left ribs.

A high-voltage technique (130 kVP-140 kVP) is preferred to the lower voltage techniques (60 kVP to 80 kVP) because there is better penetration of the bones and mediastinum. This results in both a more useful image of the central structures as well as better depiction of the lungs and pulmonary markings due to the lowered contrast and decreased density of the overlying skeletal structures. However, the low-voltage technique can be used when one is examining for bone detail or wants to assess calcific densities (Freundlich & Bragg, 1992).

There are many situations in which the standard views cannot be performed. These occasions are not restricted to the very ill patients. If an otherwise well patient is unable to stand, the examination must be done with the patient seated or lying down. A posteroanterior view is virtually impossible in these patients and one must accept the limitations of the anteroposterior view. Similarly, the lateral view will be less than optimal but nevertheless should be obtained.

In the hospital setting, it may be necessary to bring the equipment to the patient. Portable x-ray units are versatile but cannot produce the quality of stationary units. They use lower voltage and longer exposure times. Equipment limitations are further compounded by patient factors. The patients must be examined while supine or seated on the bed. They are frequently unable to cooperate fully with the technologist. The portable frontal view is therefore virtually always an anteroposterior view with compromised resolution. For the severely ill patient, portable chest radiography, despite its shortcomings, is extremely useful to assess tubes and lines and their complications, to look for complications of the pneumonia, and to assess its response to treatment.

In some cases special views must be obtained (Novelline, 1997). A portable lateral view is technically more difficult to obtain and not routinely obtained in most settings. It is, however, the most sensitive view to detect pleural fluid (Novelline, 1997). Decubitus views with a horizontal beam are useful to detect pleural effusions and to assess their mobility. Decubitus views are frontal views performed with the patient lying on one side. If a high diaphragm is suspected to be due to a subpulmonic pleural effusion the patient lies on the side of the suspected effusion. A mobile subpulmonic effusion will flow along the dependent aspect of the ipsilateral chest wall and thicken the lateral pleural stripe. On the other hand, if there is a thickened lateral pleural stripe consistent with pleural effusion and loculation needs to be ruled out, a decubitus view should be obtained with the abnormal side up. If the fluid is mobile it will flow down to the now dependent medial aspect of the ipsilateral chest wall and clear the previously thickened pleural stripe. A decubitus view is also sometimes helpful to detect air fluid levels. Traditionally inspiration/expiration frontal views have been used to detect or exclude a pneumothorax. It is now generally accepted that expiration frontal views do not significantly add to the diagnostic information afforded by the inspiration views and need not be done as an initial examination for that purpose (Seow et al., 1996). When the patient suspected of having a pneumothorax is unable to stand or sit, a decubitus view, with the abnormal side up, or a supine horizontal beam lateral view may be helpful (Novelline, 1997). Lordotic views afford a clear view of the lung apex by elevating the overlying clavicle. Oblique views are sometimes helpful to determine the location of an opacity.

## Systematic Approach to the Chest Radiograph

Initially, radiographs should be assessed for technical quality. The majority of technically suboptimal chest radiographs are a result of rotation and poor exposure technique. Rotation my be assessed by locating the spinous process at the level of the clavicular heads and measuring the distance from the process to the medial end of the clavicle on either side.

Studies have shown that it is better to first do a routine systematic search of the chest radiograph before doing a specific examination for the suspected abnormality. A routine undirected search has been shown to yield fewer false-positive findings, with no detrimental effect on sensitivity (Swennson et al., 1985). Radiologists are taught,

when shown a film, to do a routine, systematic, and undirected search with no history before examining the film specifically to answer the clinician's question.

Whenever possible previous films should be obtained for comparison. Both the current and the previous chest radiographs should be critically evaluated for positioning and technique before attempting to evaluate the thorax. This allows one to take these factors into account as possible reasons for new or altered findings and thereby improve specificity. For example, an increased size or prominence of a feature compared to the previous study may be due to patient rotation. A change in density may be due, for example, to a difference in the kVP. To the extent that it is possible and practical one should ensure that follow-up films match the previous examinations in positioning and technique.

The film reader should then perform a systematic search of the chest radiograph. Experienced chest radiologists recommend a search based on the body systems (Freundlich & Bragg, 1992). A mnemonic can be used to guide the search. For example, the acronym LAMPS denotes Lungs, Airways, Arteries (pulmonary vessels and mediastinal cardiovascular structures), Adenopathy (examine the hila), Mediastinum, Pleural margins and surfaces, Soft tissues of the abdomen, chest, shoulders and neck, and Skeletal structures. A separate and complete perusal of the chest radiograph should be carried out for each of these headings so that one is examining only the structures specified by the heading with each pass. When a significant abnormality has been found, the search should not be stopped immediately since there may be other possibly very significant abnormalities elsewhere on the film.

## Radiographic Appearance of Pneumonia

On the normal chest radiograph, air spaces and most airways are not visible; one sees only well-defined pulmonary blood vessels, major and minor fissures, and the walls of the larger bronchi. A radiographic diagnosis of pneumonia is possible only when the pathophysiologic response to the infection renders the disease visible by altering the appearance of normal structures or by the addition of new, abnormal opacities. The radiographic images of pneumonia are composed of patterns of air space or interstitial opacification alone or in combination.

The terms air space opacification, air space disease, and alveolar consolidation are virtually synonymous and are used to describe the presence of a purulent exudate or other material of similar density that fills alveolar spaces and displaces alveolar air to cause homogeneous opacification, usually with ill-defined margins, in the affected region or regions (Webb et al., 1996; Freundlich, 1992). Pneumonia can therefore be mimicked when edema fluid, hemorrhage, inflammation, neoplastic infiltration, pulmonary alveolar proteinosis, or fluid aspiration displaces and replaces alveolar air.

Interstitial lung disease refers to conditions causing fluid and inflammation to enter and enlarge the interstitial compartments. Together the axial, subpleural, and intralobular interstitium form a continuous fiber skeleton that supports the lung (Webb et al., 1996). Internally the axial interstitium is composed of the central peribronchovascular interstitium which merges peripherally with the centrilobular peribronchovascular interstitium. Externally the subpleural interstitium courses beneath the visceral pleura and envelops the lung in a continuous fibrous sac. Fibrous septa project from the subpleural interstitium into the lung parenchyma between secondary lobules. These projections are known as interlobular septa and define the boundaries between the secondary lobules. Within the secondary lobules there is a system of very fine fibers supporting the walls of the alveoli. These alveolar septal fibers collectively form the intralobular interstitium which links the axial interstitial compartment to the subpleural interstitial compartment. On a chest radiograph the lung interstitium is normally invisible. Interstitial lung disease will be seen on the radiograph when it enlarges and opacifies the interstitial compartments in the affected region and renders them visible. Whereas air space consolidation (Fig. 1) produces homogeneous opacification due to displacement of air from the alveolar spaces, interstitial disease does not (Fig. 2). Alveolar air remains within the alveolar spaces in the affected area and the interfaces between alveolar air and the interstitial opacities may be seen on the radiograph. As a result, purely interstitial opaci-

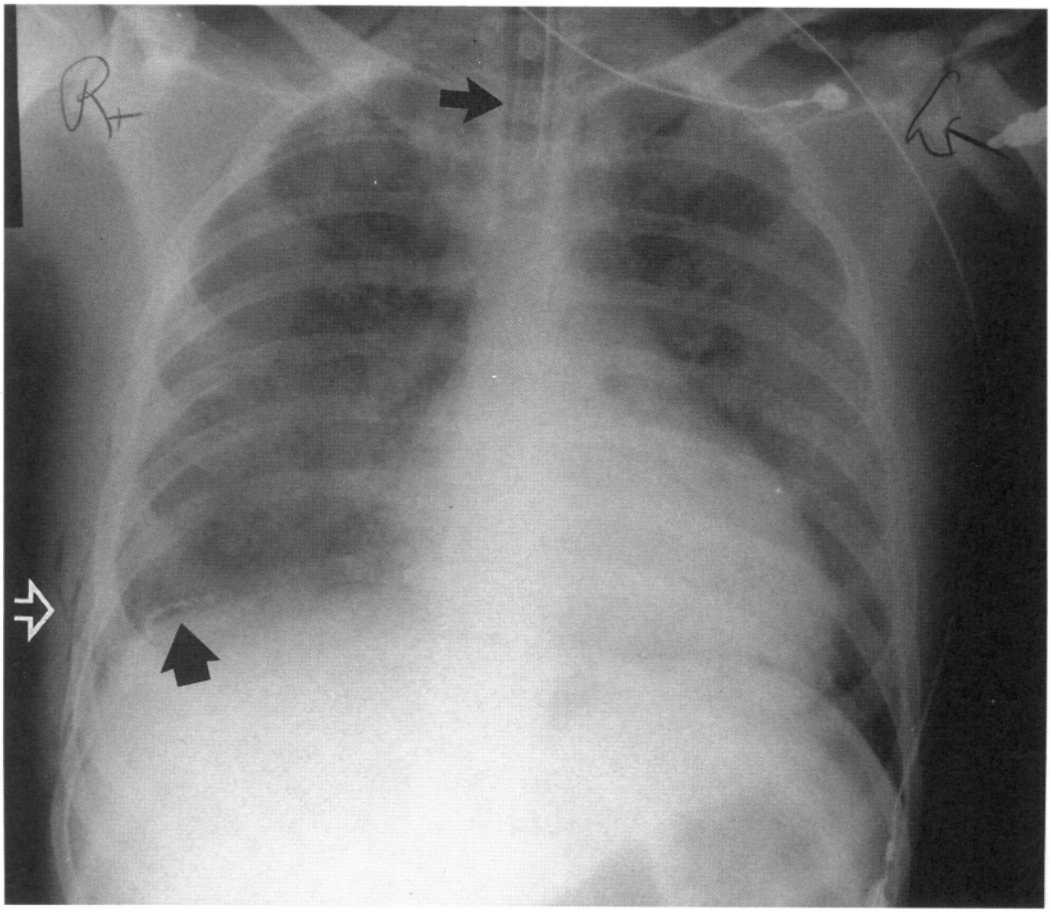


**FIGURE 1.** Diffuse air space opacification in a patient with adult respiratory distress syndrome. The patient has had a recent lung biopsy as indicated by the surgical clips (wide arrow) and the small collection of subcutaneous air (open white arrow). Note the presence of the endotracheal tube (thin arrow).

ties are relatively discrete with lack of confluence and preservation of lung margins. For practical purposes, the only acute interstitial lung disease that mimics acute interstitial pneumonia is pulmonary edema, usually due to congestive heart failure or fluid overload (Müller, 1992). Subacute conditions mimicking interstitial pneumonia include pulmonary lymphangitic carcinomatosis and interstitial pneumonitis due to drug or treatment reactions. Chronic interstitial lung diseases may have a similar appearance on chest radiograph.

Pneumonia occurs when a sufficiently large inoculum of infected material gains access to the lower airways and alveoli and is not promptly neutralized and cleared. For most patients with CAP the initiating event is aspiration of pathogens that have colonized the upper respiratory mucosa. Aspiration may occur more readily when there is a neuromuscular disorder or when there is alteration or depression of consciousness, as for example, with alcohol use. The pathogens will have entered the nose or mouth either by direct introduction of infected material or by the inhalation of an infected aerosol. Direct inhalation of a contaminated aerosol into the lower tract is a much less common cause of CAP. It is associated particularly with viruses, fungi, tuberculosis (Arunabh & Niederman, 1996), and *Legionella* and the other atypical organisms (Mason & Nelson, 1996).

When airborne aerosolized infected material is

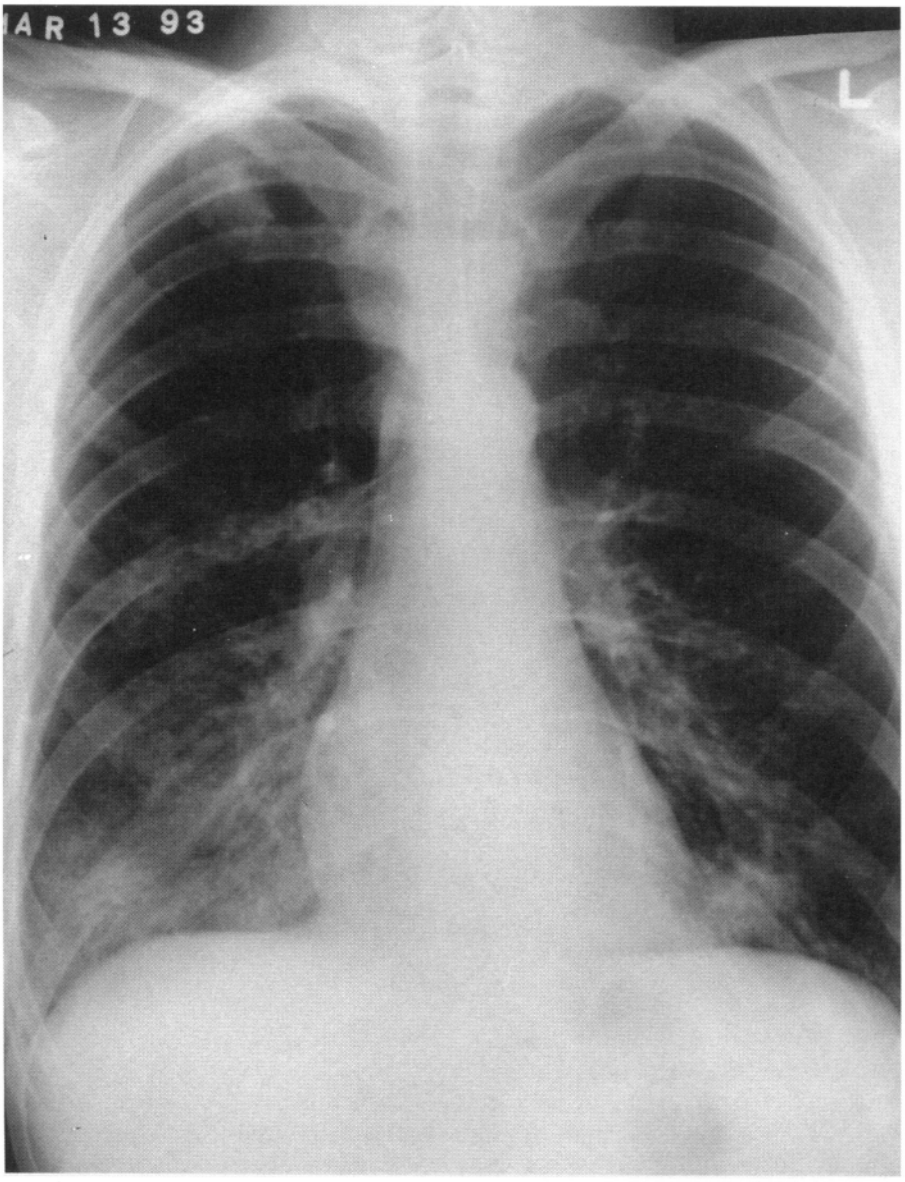
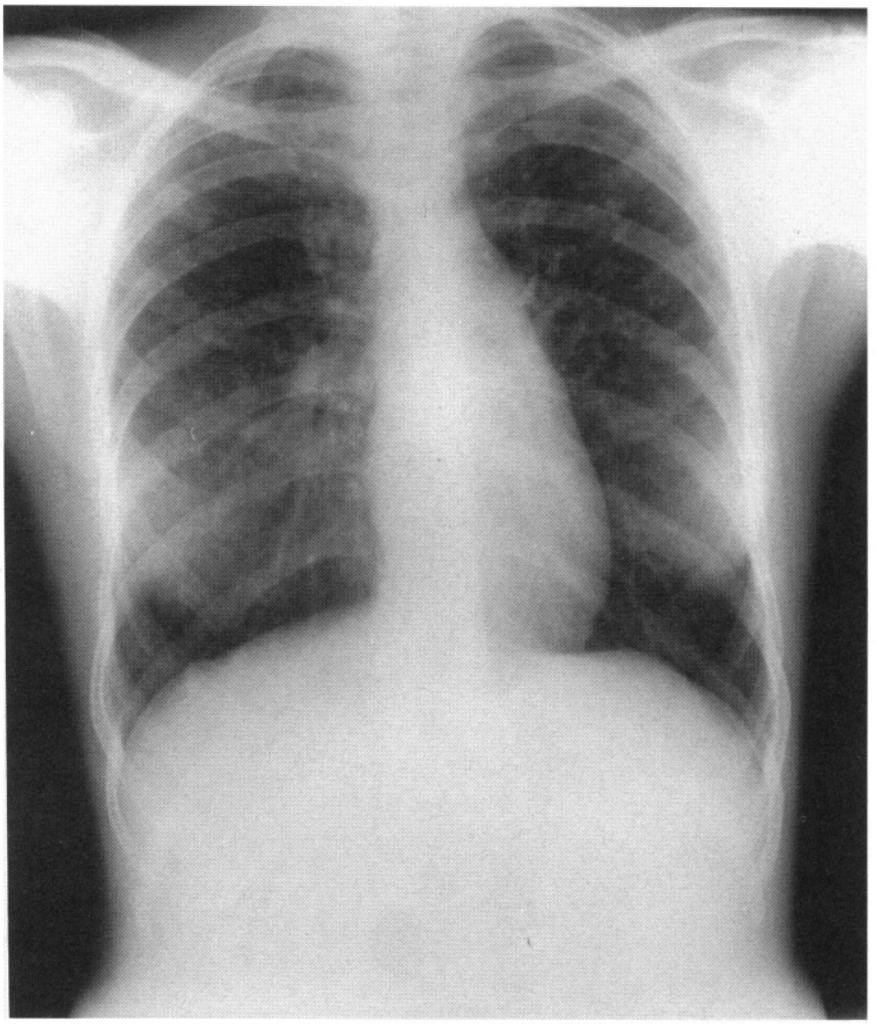



**FIGURE 2.** Example of predominantly interstitial opacification in a patient with *Pneumocystis carinii* pneumonia.

inhaled, heavier droplets and particles are deposited earlier in the larger, more proximal airways and may not reach the lower airways. Lighter ones are carried further into the lower respiratory tract and may reach the bronchi and more peripheral bronchioles. Only the smallest airborne droplets or particles, less than 5 μm in diameter (McCloud, 1992), will actually reach the alveoli. Once deposited on the walls of the lower airways or alveoli, the pathogens are either cleared or cause infection.

Pathogens may also reach the lung via the hematogenous route (e.g., septic emboli in drug addicts) or by direct extension. These sources are uncommon etiologies of CAP (Mason & Nelson, 1996; Woodridge, 1992).

Wherever the pathogen becomes established



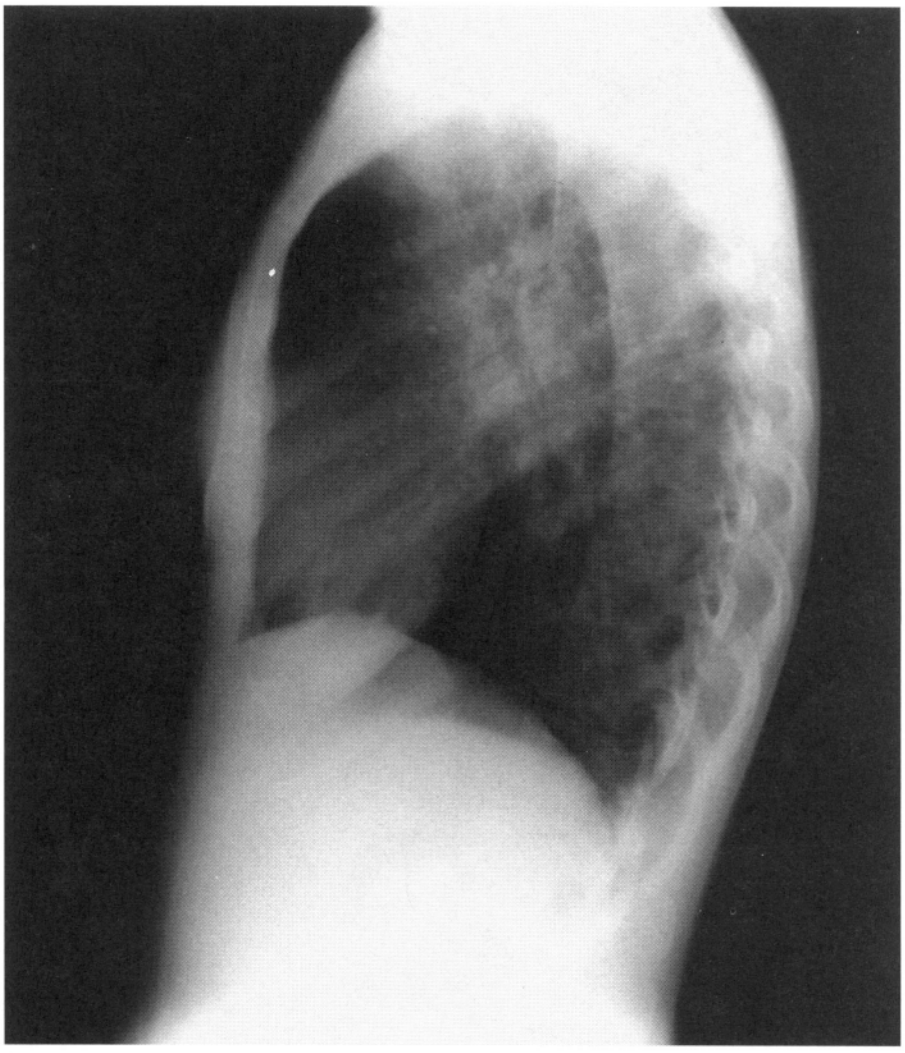
A

**FIGURE 3.** Diffusely distributed bilateral mid- and upper-lung nodular opacities and prominent peribronchial markings in a patient with bronchopneumonia due to tuberculosis. There is right hilar prominence on a posteroanterior view (A) and hilar density on the lateral view (B) consistent with adenopathy. (*Continued*)

the normal host inflammatory response causes cellular infiltration and edema of the local and adjacent tissue. If the infection progresses the combination of the infective process and the host response will cause an outpouring of purulent, hemorrhagic fluid into adjacent airway lumens and alveolar spaces.

If the pathogen is deposited onto the alveolar epithelium and not cleared promptly the inflammatory response in combination with the infective process results in edema and infiltration of the alveolar wall and an outpouring of fluid into the alveolar spaces, resulting in various patterns of air space opacification on the chest radiograph. If only the centrilobular (peribronchiolar) alveoli of noncontiguous secondary lobules in the region are involved, the pneumonia will be visible as ill-defined micronodules on the chest radiograph (Woodridge, 1992) (Fig. 3). This pattern can be seen in other causes of air space disease such as edema and bronchioloalveolar cell carcinoma. The features may also be consistent with causes of poorly defined pulmonary nodules such as metastases.

If the air space disease spreads, the alveolar micronodules will become confluent and larger

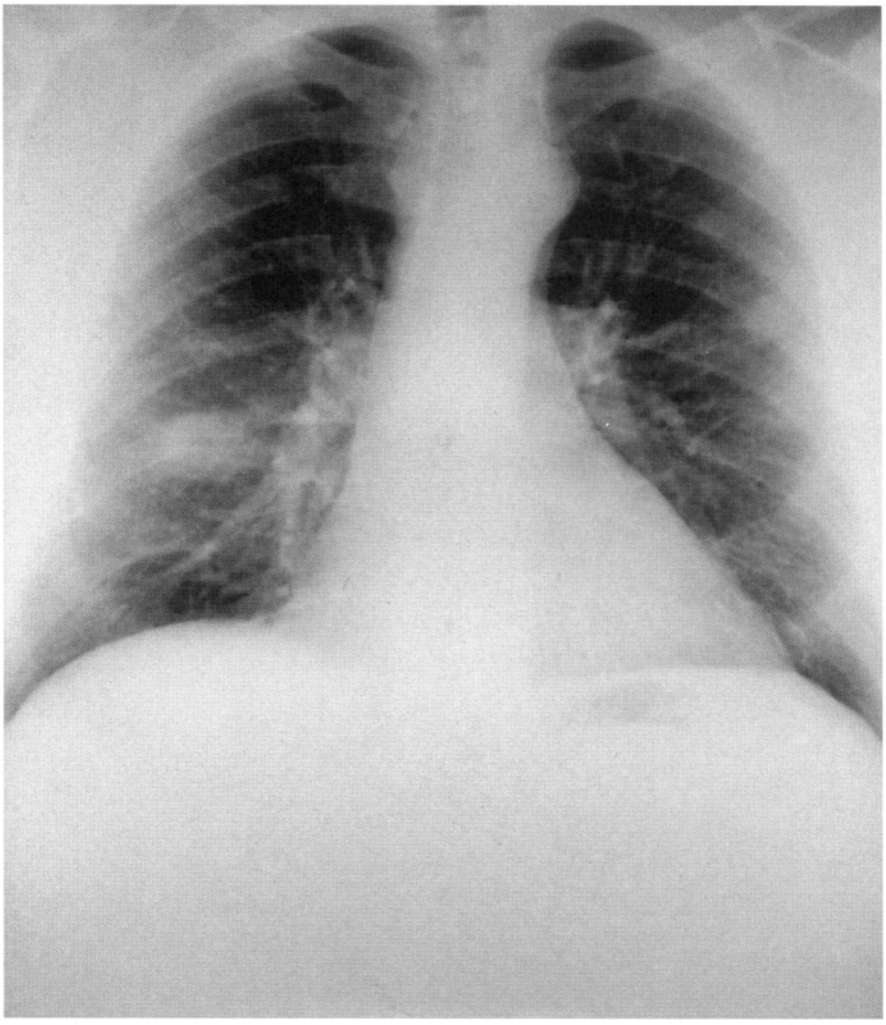


B

FIGURE 3. (*Continued*)

areas of homogeneous opacification may ensue. When only one localized and well-defined opacity ensues it is called round pneumonia (Fig. 4A,B), mimicking a mass. If the infection continues to spread via the pores of Kohn and terminal airways an entire segment or lobe may become opacified to produce the typical lobar pneumonia. Classically the lobe is homogeneously opacified. Radiolucent air bronchograms caused by patent airways within the infected region may be present and will confirm to the reader of the radiograph that the opacification is indeed predominantly pulmonary air space consolidation (Fig 5).

Where air space consolidation abuts the mediastinum, diaphragm, or pleura, the normal margin of these structures is rendered invisible on the radiograph due to loss of the air-soft tissue interface (the silhouette sign) (Fig. 6). It can be a clue to the presence of subtle air space consolidation. Correlating the lobar anatomy with the structure whose silhouette is lost is a major clue to identify which lobe is involved. When air space consolidation is

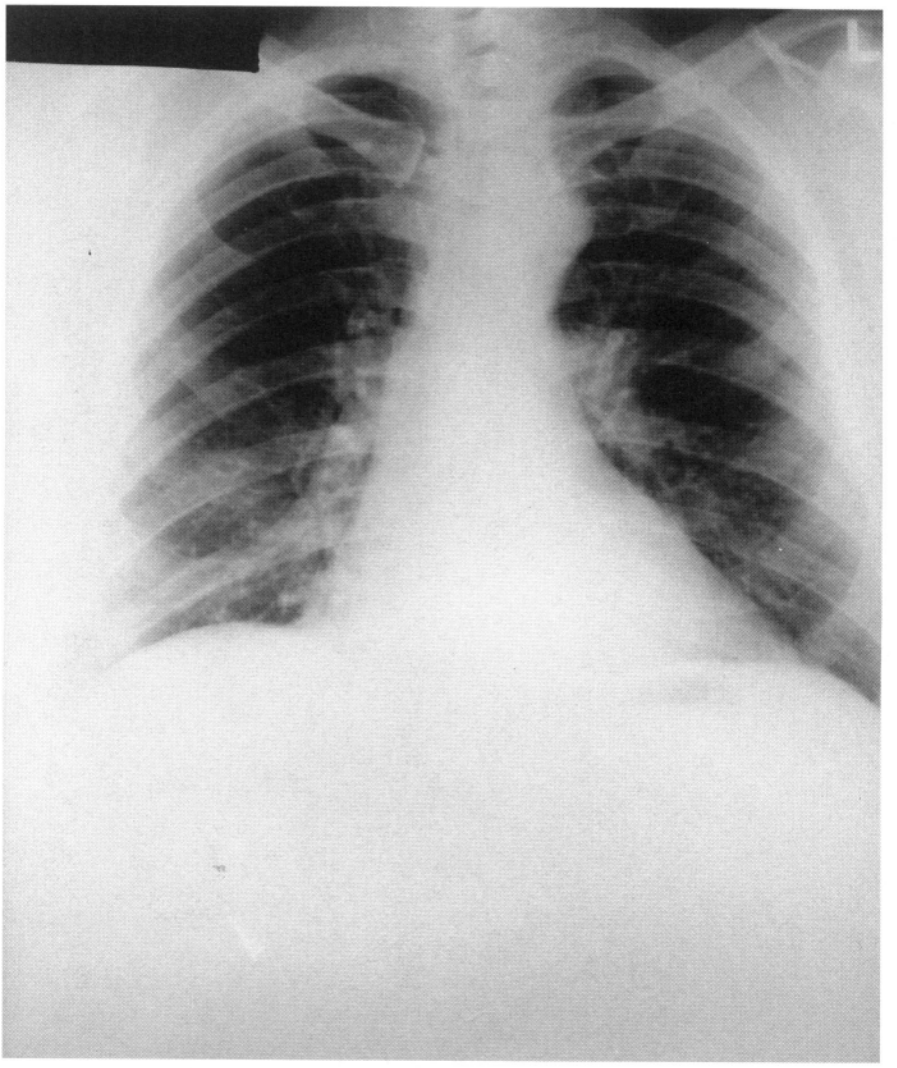


A

**FIGURE 4.** (A) Round pneumonia in the right lower lung which is resolving on a follow-up examination several weeks later (B). (*Continued*)

behind or in front of a structure its density is added to that of the structure and it can be a clue to its presence. This is called summation (Figs. 6–8). For example, one should see pulmonary markings through the heart. Loss of these markings and an increase in the heart density on the frontal view suggests consolidation in the left lower lobe where it lies posterior to the heart. Another region where summation is often helpful is over the lower thoracic spine on the lateral view. The opacification of the thoracic spine should gradually decrease toward the diaphragm. A lower lobe consolidation increases the density in this region.

Lobar opacification may be incomplete and remain inhomogeneous. This may occur, for example, when the natural history has been altered by antibiotic therapy or when the patient has emphysema. Air space opacification can also be caused by edema, hemorrhage, alveolar proteinosis, and bronchioloalveolar cell carcinoma (Fig. 8) (Freundlich, 1992) and mimicked by pulmonary lymphoma or Kaposi's sarcoma (Goodman, 1992).



B

**FIGURE 4.** (*Continued*)

There may be an overlap of interstitial and air space pneumonia. The two components may coexist from the start or one may progress to produce the other. For example, segmental or lobar consolidation may develop in a case of primary interstitial pneumonia when there is progression of the original infection or the patient develops a secondary, usually more aggressive, superinfection.

Bronchopneumonia is an example of mixed interstitial and air space pneumonia. It is usually the result of copious aspiration of oropharyngeal secretions or gastric contents containing pathogens which are deposited onto the walls of the airways and subsequently infect them. If the aspirated material or infection reaches the bronchioles the pneumonia spreads rapidly to involve the peribronchiolar alveoli. The earliest phase seen on the chest radiograph consists of peribronchial thickening, increased bronchovascular markings, and acinar nodules (Woodridge, 1992). It is not commonly seen since the infection has usually progressed further by the time a chest radiograph is obtained. The multiple foci of infection usually progress rapidly to produce multiple, frequently bilateral confluent

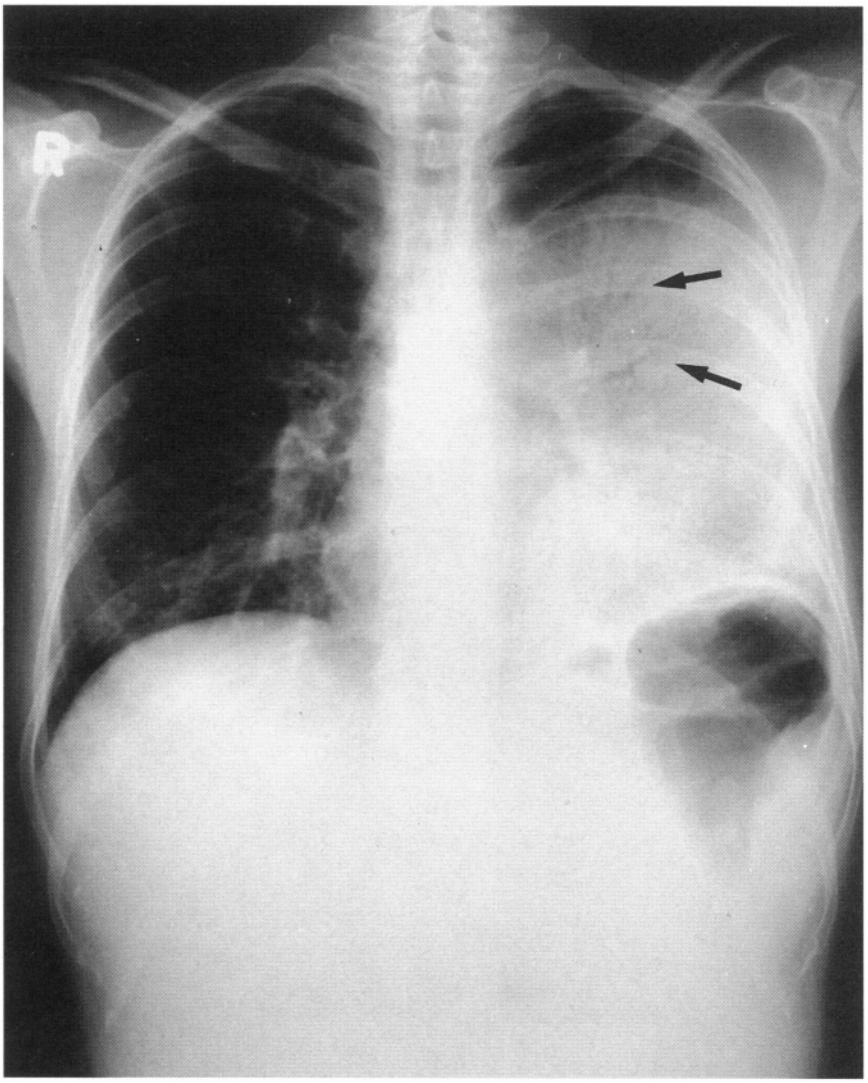
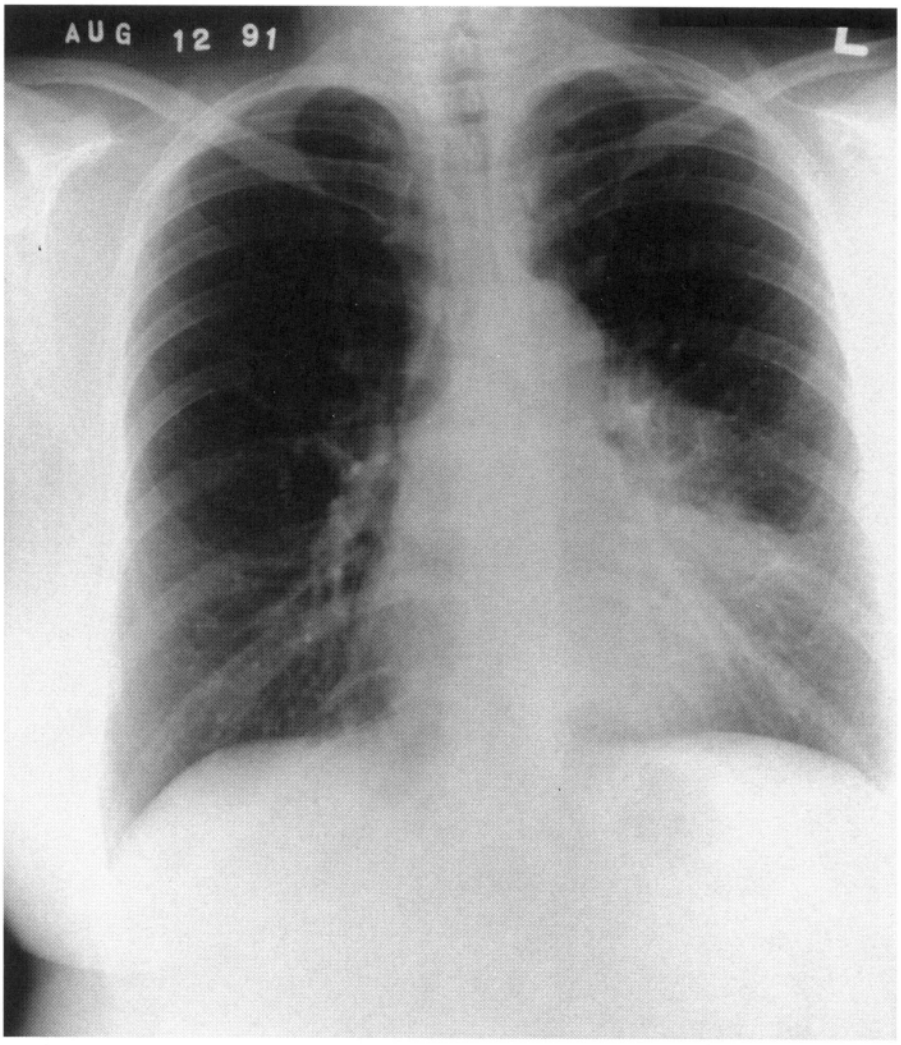


**FIGURE 5.** Homogeneous air space opacification of the left lower lobe (complete lobar consolidation) in a patient with pneumonia due to *Streptococcus pneumoniae* infection. Air bronchograms are present (arrows). Note also complete loss of the heart and left hemidiaphragm margins.

patches of poorly defined opacification overlying and often obscuring the associated peribronchial disease. If the infection continues to spread the patches coalesce to opacify entire segments or lobes. If unchecked, an entire lung or both lungs may opacify (Fig. 9).

An infectious inflammatory process involving the walls of bronchi and larger bronchioles may spread to the peribronchial and peribronchiolar interstitium. The resulting interstitial edema and infiltration will thicken the walls and margins of the airways. The thickened peribronchial interstitium, when seen end on, will be apparent as peribronchial cuffing. The margins of the companion blood vessels will become indistinct. Inflammation of the smaller bronchioles, especially the intralobular bronchioles, may cause a reticulonodular or micronodular pattern of opacification as they are seen variably *en face* and end on. If the interstitial inflammation spreads to the interlobular septa the

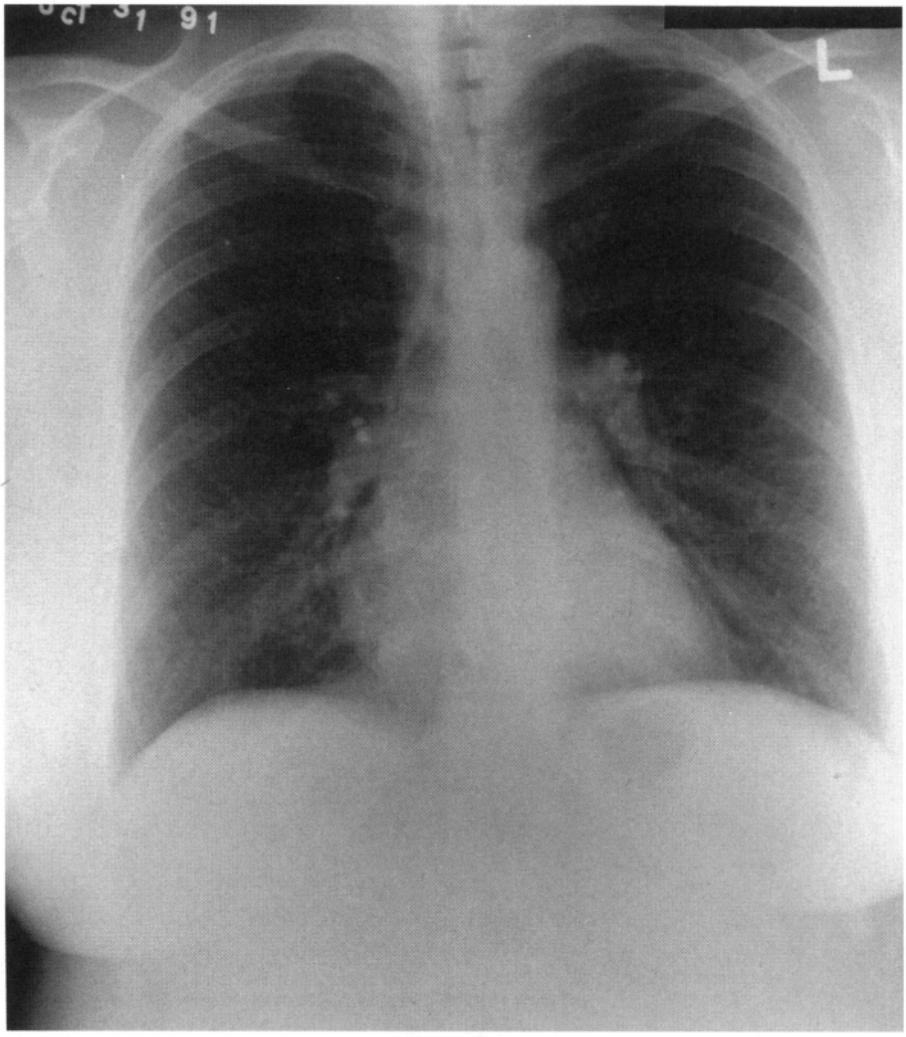



A

**FIGURE 6.** Silhouette sign. Left lower lobe pneumonia causes loss of the left heart border (A). In (B) the pneumonia has resolved with restoration of the left heart border.

thickened septa may be visible as additional reticulonodular opacities or as septal lines (Kerley B lines). Interstitial pneumonia may, therefore, have an appearance similar to noninfectious interstitial pneumonitis, interstitial edema, and chronic interstitial lung disease (Fig. 2). This is the least common radiographic manifestation of CAP.

In the majority of cases of CAP the tracheobronchial tree is the route of access to the lung and a hematogenous source for the pulmonary infection is much less common. It is virtually always caused by multiple septic bacterial emboli. They are usually widely deposited in a "vascular distribution," which favors the periphery of the dependent regions of the lung. Each focal deposit causes localized hemorrhagic edema, and the pneumonia will typically be seen on the chest radiograph as multiple nodules, often pleural, in the lower two thirds of the lung. They may range from large and poorly defined to small and well defined. Cavitation within the nodules is often seen. If the seeding has been massive and is seen early there may be a miliary pattern of tiny nodules. Nodules in a vascular distribution are also typical in metastatic disease.



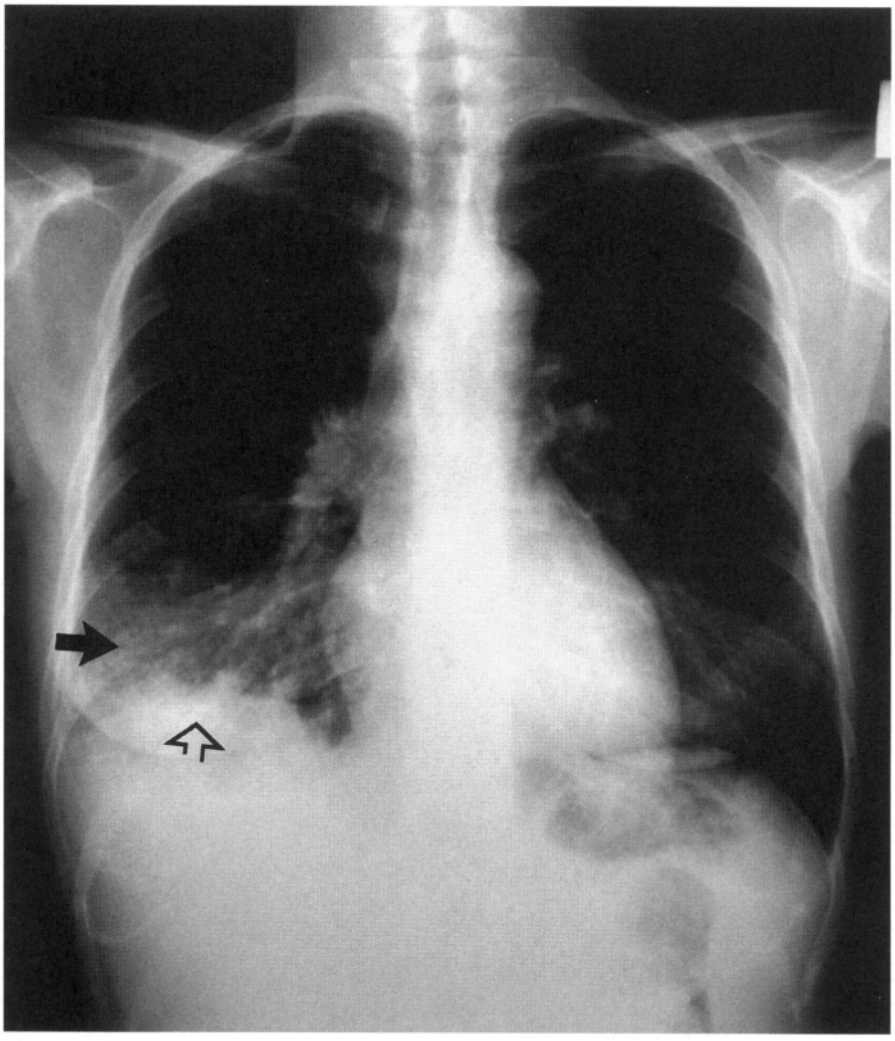

B

FIGURE 6. (*Continued*)

## Pneumonia Pattern Recognition

The sensitivity of the chest radiograph for the detection of pneumonia (and its negative predictive value) depends on the technical quality of the images, the training of the observer, the timing of the examination with respect to the onset of the clinical findings and the degree to which the patient is able to develop an inflammatory response sufficient to render the process radiographically visible. Specificity is primarily dependent on the ability of the observer to minimize false-positive interpretations by recognizing and differentiating the various diseases that produce a radiographic image that can mimic pneumonia.

Notwithstanding the ability of the chest radiograph to detect pneumonia and to aid in assessing its severity (Torres et al., 1996) and response to treatment (Fein, 1996), it is unable to provide a specific etiologic diagnosis. It is, however, no worse than the clinical assessment as both are of limited value in this regard. In fact, when multiple clinical and radiologic criteria are combined in the assessment of CAP, the correct etiologic agent is identi-



A

**FIGURE 7.** Summation sign. Posteroanterior (A) and lateral (B) demonstrate right lower lobe pneumonia. The right lower lobe density (thinner arrow) sums with the heart density to yield a markedly increased density overlying the posterior aspect of the heart on the lateral view (wider arrow) bounded anteriorly by the right major fissure. The retrocardiac density is also greater than normal. There is partial silhouetting of the right hemidiaphragm margin on both views (open arrows).

fied in less than half of the cases (Bowton & Bass, 1991).

Although the chest radiograph cannot specifically diagnose the causative organism, it can nevertheless expedite empiric therapy decision-making by enabling the skilled reader to narrow the list of probable causative organisms through systematic analysis of the radiographic pattern of the pneumonia in conjunction with the clinical and epidemiological circumstances (Bowton & Bass, 1991; Conces, 1993, 1994; Herold, 1997; Freundlich & Bragg, 1992; Torres et al., 1996).

Pneumonia pattern analysis requires that pulmonary parenchymal abnormalities noted on the chest radiograph of the patient suspected of having CAP be analyzed and characterized as air space disease, interstitial disease, or a mixture of the two. If the radiographic pattern is atypical (e.g., a mass),

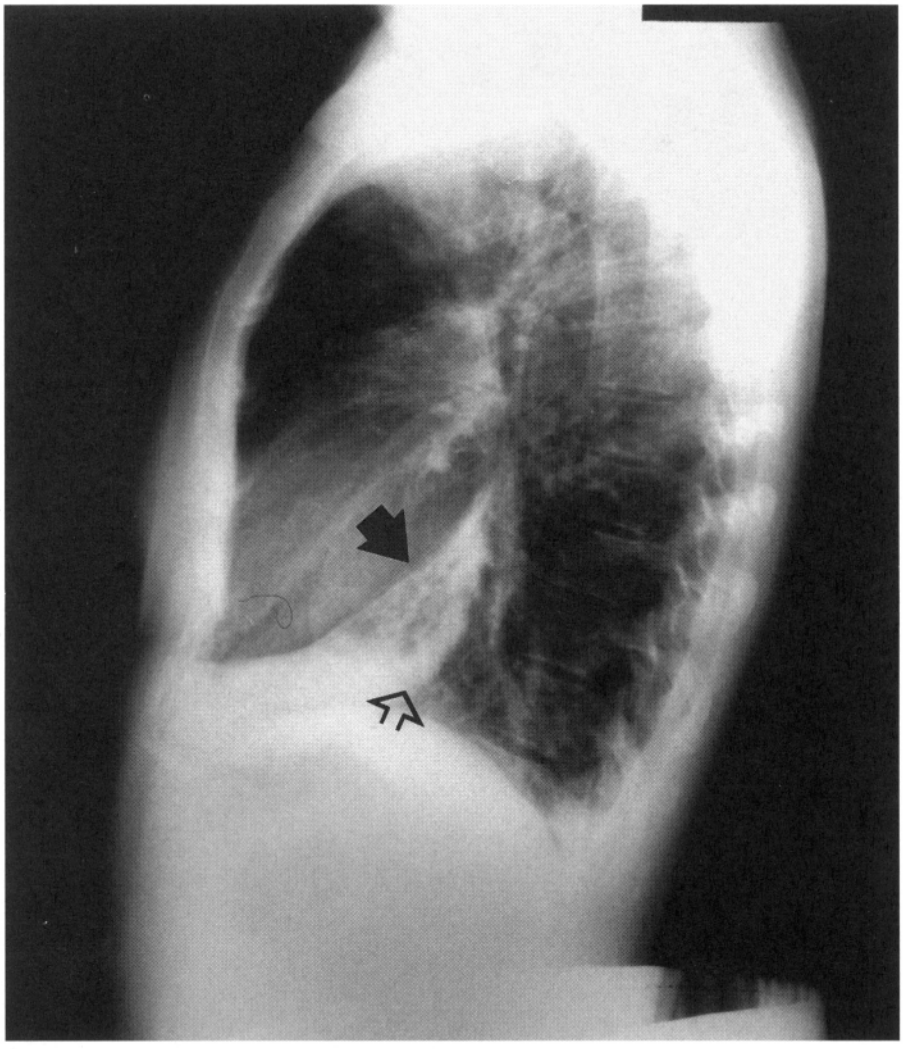
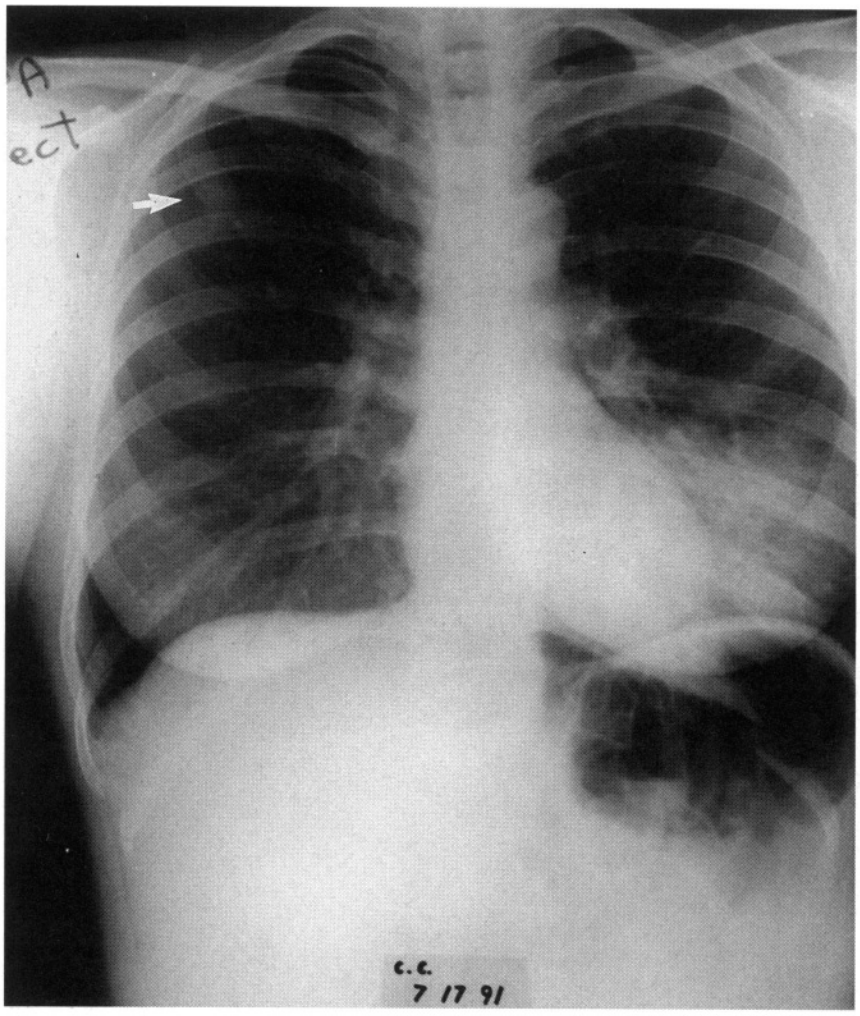



**FIGURE 7.** (*Continued*)

one should include other conditions in the differential diagnosis. Findings consistent with pneumonia should then be classified according to their location. The pneumonia may be focal, segmental, lobar, multilobar, or diffuse. The area of abnormality can be homogeneous, heterogeneous, or confluent. The pneumonia may have a central or peripheral predominance; have an upper, middle, or basal predominance; or simply be scattered.

The mediastinum and hila should be assessed for adenopathy. The presence of pneumothorax, atelectasis, and collapse need to be noted. Pneumatoceles, cavitation, and abscess formation are associated with more virulent organisms, with varying degrees of necrosis. Pleural effusion is the most common complication of pneumonia (Woodridge, 1992) and it may develop into an empyema. Infrequently the infection can extend beyond the pleural space to involve the heart, mediastinum, or chest wall.

Finally, the reader should assess the films specifically for evidence of comorbid conditions that




A

**FIGURE 8.** Summation sign. In (A) there is air space opacification in the left lower lobe causing increased density of the cardiac shadow and loss of the lung markings normally seen through the heart. In (B) the pneumonia has resolved restoring the normal cardiac density and the visibility of the lung markings behind the heart. Note the bronchoalveolar cell carcinoma in the right upper lobe (arrows). It has enlarged in the 3-month interval between the examinations.

may complicate the course of the illness or that may actually be mimicking pneumonia, such as a mass or congenital anomaly.

## Specific Pneumonia Patterns

Pneumonia patterns on the radiograph can be grouped into four categories: lobar pneumonia, bronchopneumonia, nodular patterns of pneumonia, and diffuse patterns of pneumonia (Conces, 1994).

### *Lobar Pneumonia*

Lobar pneumonia is a primary air space pneumonia that starts as a small peripheral subsegmental opacity that, if unchecked, may spread to involve the entire segment and then, via the pores of Kohn and terminal airways, may spread to homogeneously opacity the entire lobe (Woodridge, 1992). Early recognition and treatment of the lobar pneumonia will modify its progression, and the chest

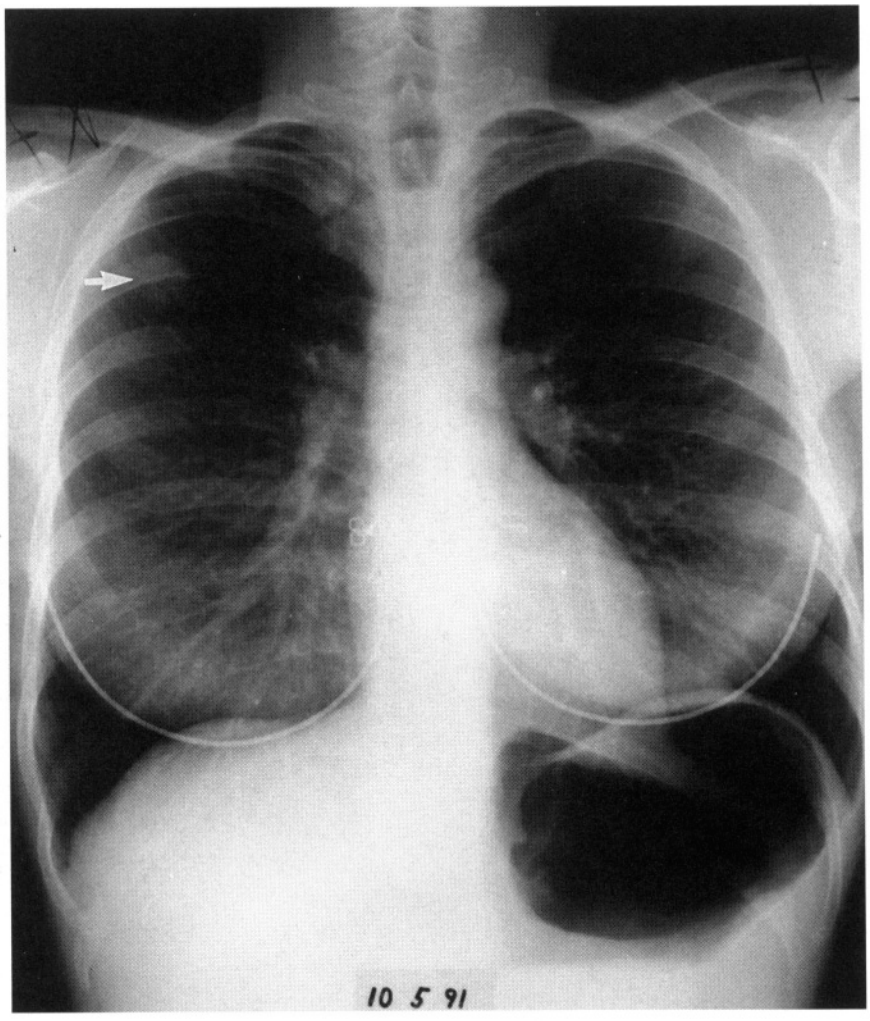



**FIGURE 8.** (*Continued*)

radiograph may never demonstrate more than patchy subsegmental or segmental opacification within the affected lobe. Since lobar pneumonia does not involve the bronchi, they typically remain open and there is no atelectasis. The patent bronchi within the region or regions of air space opacification may be seen as air bronchograms.

Community-acquired lobar pneumonia is most commonly caused by a bacterial infection (Conces, 1993; Miller 1994) with *Streptococcus pneumoniae* being the most common etiologic agent in normal individuals. *Klebsiella* follows as the next most common. *Klebsiella* is noted to be particularly associated with marked edema that fills and expands the infected lobe causing the fissures to bulge. However, lobar pneumonia with lobar enlargement can be seen with other bacterial infections including *S. pneumoniae, Staphylococcus aureus, Legionella pneumophila, Haemophilm influenzae,* and *Mycobacterium tuberculosis* (Woodridge, 1992) (Fig. 10). The round variety of lobar pneumonia is uncommon. However, it is most frequently located in the superior segment of the lower lobes, most commonly in children. The usual pathogen is *S. pneu-*

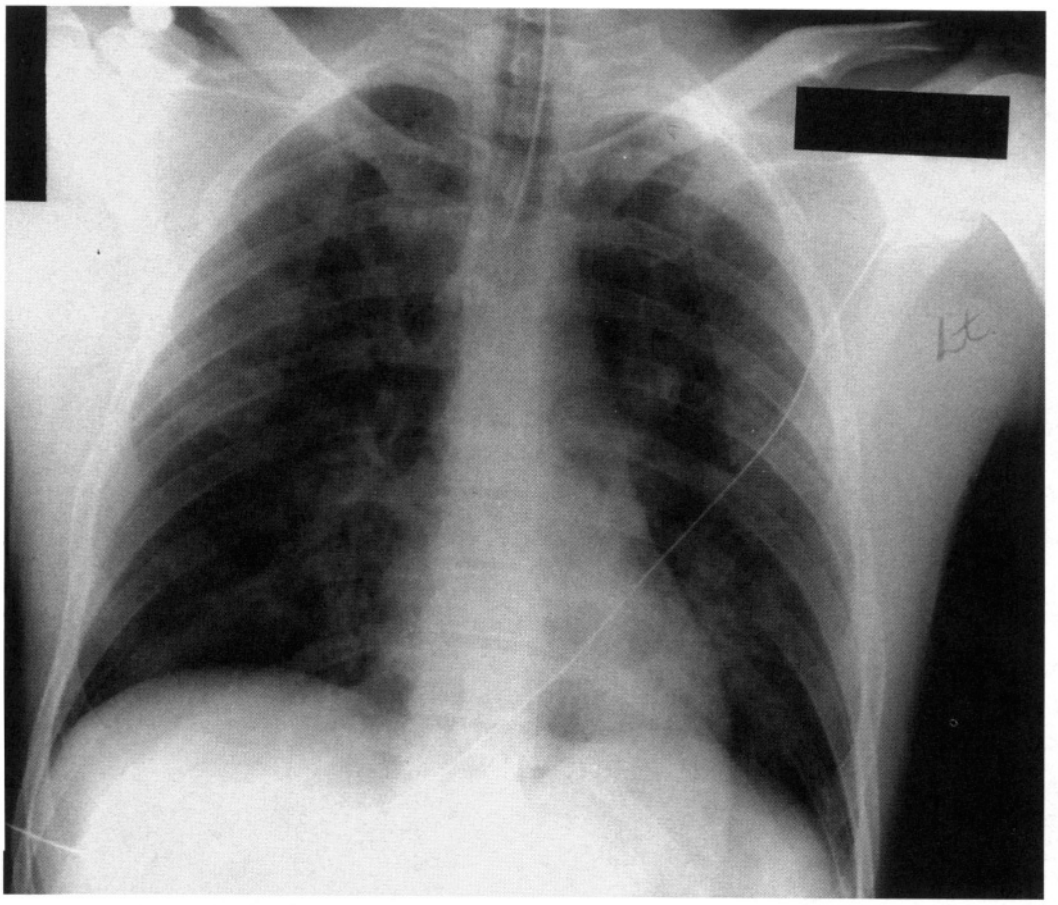
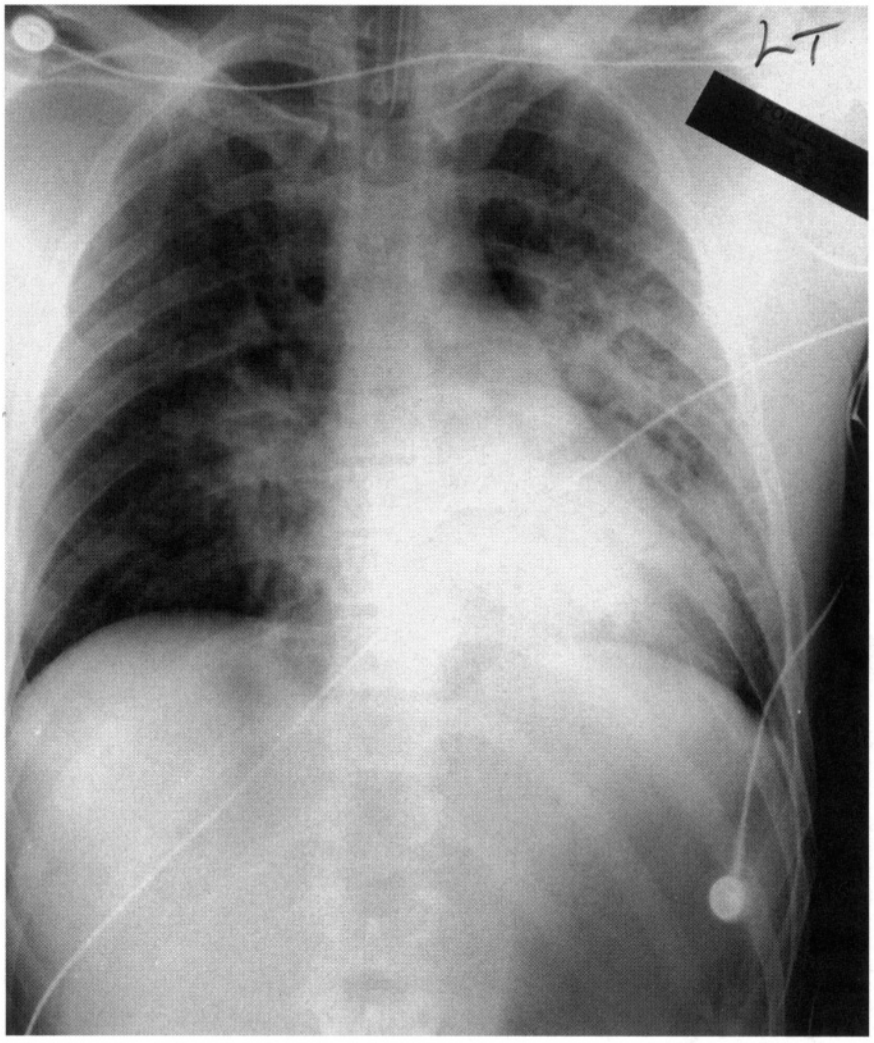



**FIGURE 9.** Progression of bronchopneumonia over a 2-day period from predominantly peribronchial and nodular opacities (A) to regions of confluent air space opacification in both lungs (B).

*moniae* (Woodridge, 1992; Singleton, 1992). Primary pulmonary tuberculosis presenting as a segmental or lobar pneumonia is typically in association with adenopathy and occasionally with pleural effusion (Miller, 1992). *L. pneumophila* pneumonia may present as patchy subsegmental air space opacification that spreads to involve an entire lobe. Other bacteria that may cause lobar pneumonia include aerobic gram-negative bacilli and *Mycoplasma pneumoniae.* On occasion, viral pneumonia may mimic a bacterial lobar pneumonia (Woodridge, 1992). A number of pulmonary fungal infections may involve one lobe but usually in a patchy subsegmental distribution. Fungi most closely associated with focal air space disease include *Histoplasma capsulatum, Coccidioides immitis, Blastomyces dermatitidis, Cryptococcus neoformans,* and invasive *Aspergillus fumigatus.*

If there is bilateral lobar pneumonia or unilateral multilobar pneumonia it should be classified as severe CAP. The organisms most frequently associated with severe CAP are *S. pneumoniae, L. pneumophila,* gram-negative bacilli, *M. pneumoniae, H. influenzae,* and *S. aureus* (Torres et al., 1996).

Cavitation, necrosis, and gangrene (Fig. 11) frequently occur in pneumonia with lobar enlargement (Woodridge, 1992) and cause lucencies within the consolidated lobe. Lobar pneumonia in a patient with emphysema may have a similar appearance. Comparison with previous films will confirm the preexisting lucencies in this case.




B

**FIGURE 9.** (*Continued*)

### *Bronchopneumonia*

Bronchopneumonia initially involves the bronchi and bronchioles but spreads rapidly to involve the peribronchiolar alveoli. In its earliest stage it will be apparent on the chest radiograph as regional, diffuse or bilateral accentuation of the bronchovascular markings and bronchial wall thickening. However, by the time the chest radiograph has been obtained the infection usually has spread to the peribronchiolar alveoli and has begun to opacify multiple noncontiguous secondary lobules and is seen on the film as multiple ill-defined acinar nodules (Fig. 3). As the disease progresses more secondary lobules become involved and the chest radiograph will show multiple regions of patchy air space opacification (Woodridge, 1992; Conces, 1994). The underlying accentuated bronchovascular markings and bronchial wall thickening may be obscured but is typically apparent if sought (Fig. 9). In aggressive bronchopneumonia the patchy air space disease spreads, becoming more confluent, to involve one or more entire lobes and may become indistinguishable from lobar pneumonia. However, atelectasis of the involved lobes is more common in bronchopneumonia due to airway obstruction by

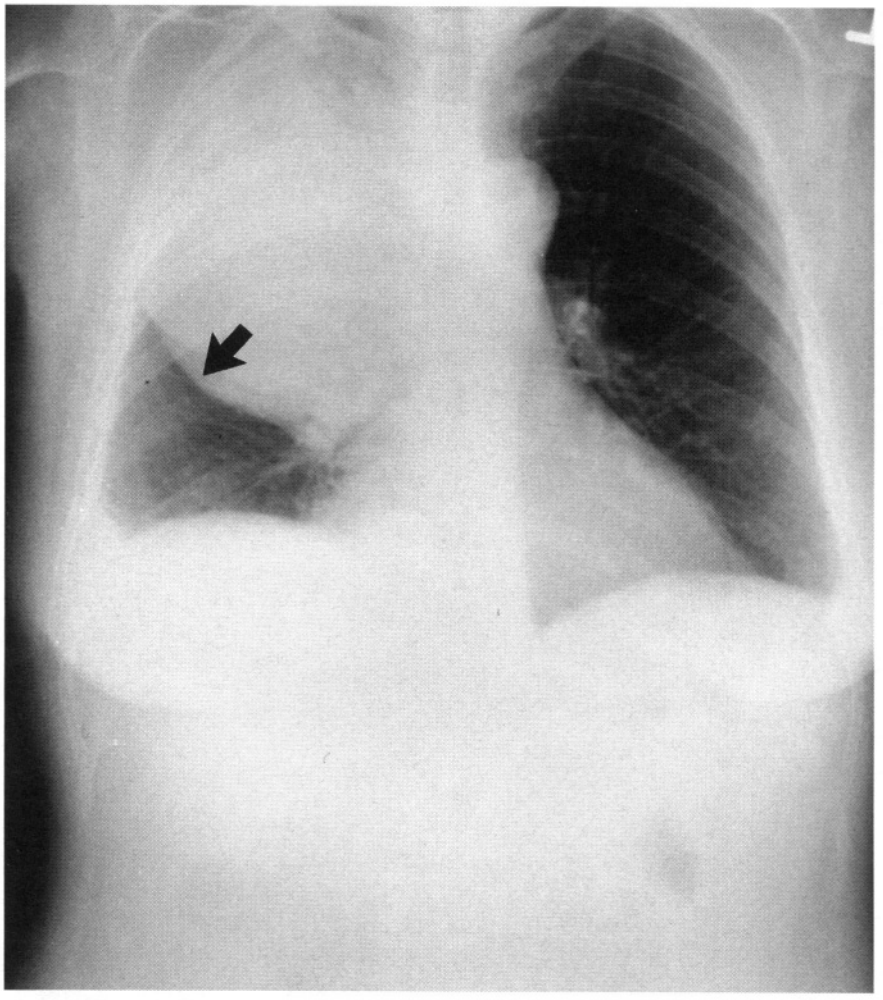


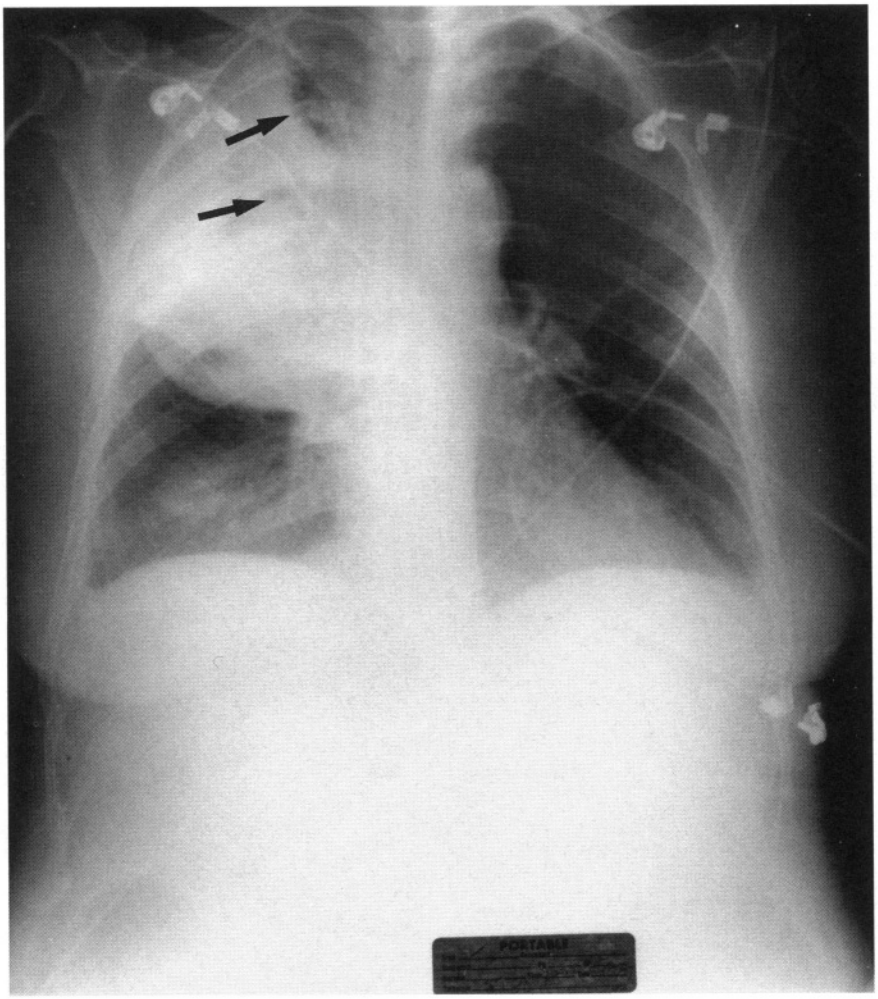
**FIGURE 10.** *Legionella pneumophila* pneumonia in the right upper lobe causing lobar enlargement as indicated by the bulging minor fissure (arrow).

inflammatory debris. In the most severe cases of bronchopneumonia the patient will develop diffuse bilateral consolidation. Necrosis with formation of pneumatoceles, cavities, and abscesses are frequent complications of bronchopneumonia (Woodridge, 1992) (Fig. 12).

Bronchopneumonia is typically caused by bacterial infection (Conces, 1993) and is most commonly due to *S. aureus, L. pneumophila, S. pneumoniae,* gram-negative bacilli *(Escherichia coli, Proteus, Pseudomonas aeruginosa,* and *Serratia)* and/or anaerobic bacteria. Gram-negative bacilli and anaerobic bacteria are frequent causes of bronchopneumonia in patients who aspirate oral or gastric contents due to neuromuscular dysfunction or loss of consciousness. Aggressive viral pneumonia may cause a bronchopneumonia pattern (Woodridge, 1992).

### *Nodular Pneumonia*

Nodular patterns of pneumonia may range from a single focus of round pneumonia to more numerous and widely distributed nodules. Pneumonia nodules are typically poorly defined and range in size from acinar nodules, measuring less




A

**FIGURE 11.** Pulmonary gangrene (necrosis). This is the same patient as in Fig. 10. Lucencies (arrows) have developed in the enlarged right upper lobe consistent with necrosis. (*Continued*)

than 1 cm in diameter, to nodules measuring 10 cm or more that resemble masses (Miller, 1992). The nodular opacities can be due to foci of air space consolidation, septic emboli, or granulomas and may have a regional or diffuse distribution.

*S. aureus, S. pneumoniae, L. pneumophila,* and *Nocardia* and various gram-negative bacilli are foremost among bacterial pathogens that can cause a mass-like round pneumonia. Multiple acinar and larger nodular opacities can be seen in the earliest stages of bronchopneumonia. Reactivation tuberculosis can produce nodular opacities (Fig. 13). Acute fungal infections typically begin as focal areas of air space opacification that may become better defined and take on a nodular or mass-like appearance as granuloma formation develops. Some may cavitate. Fungal infections commonly producing nodular opacities include A. *fumigatus, C. immitis, C. neoformans* and Mucorales. The most common pattern seen with invasive pulmonary aspergilosis (Conces, 1993) is multiple nodules which cavitate due to infarction within the infected regions.

Septic emboli produce focal areas of pneumonitis, predominantly in the dependent regions of the lungs, that are poorly defined and frequently

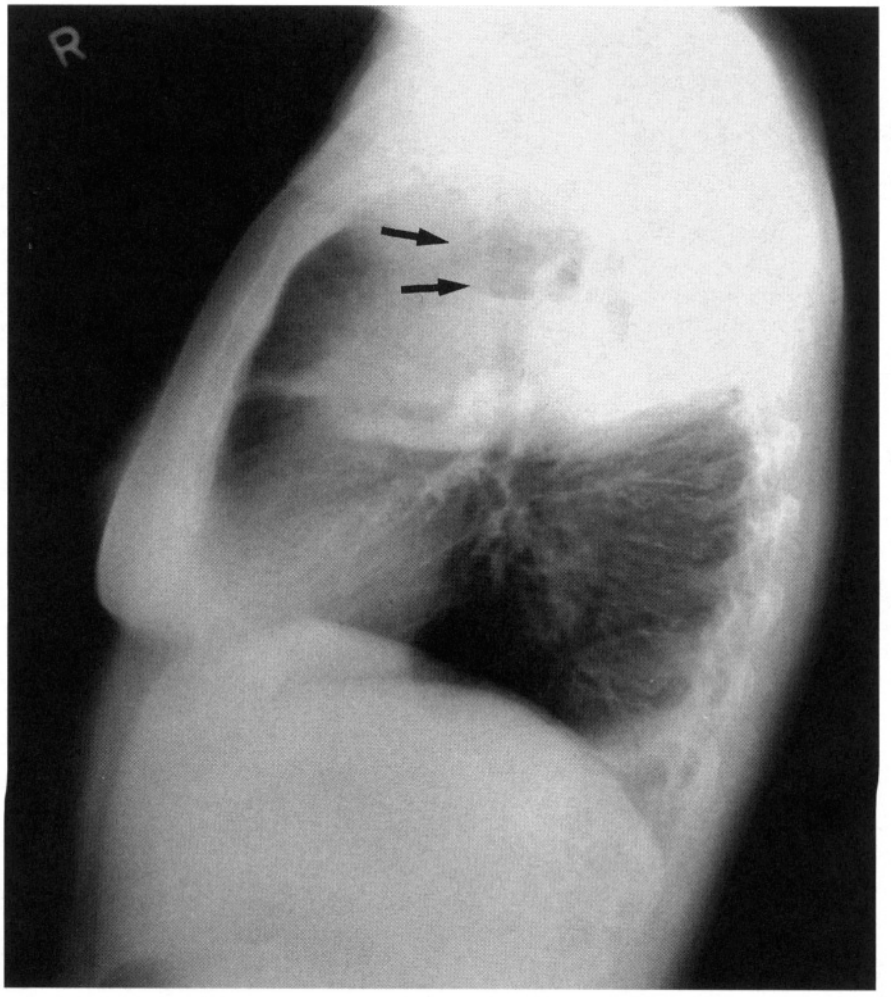



FIGURE 11. (*Continued*)

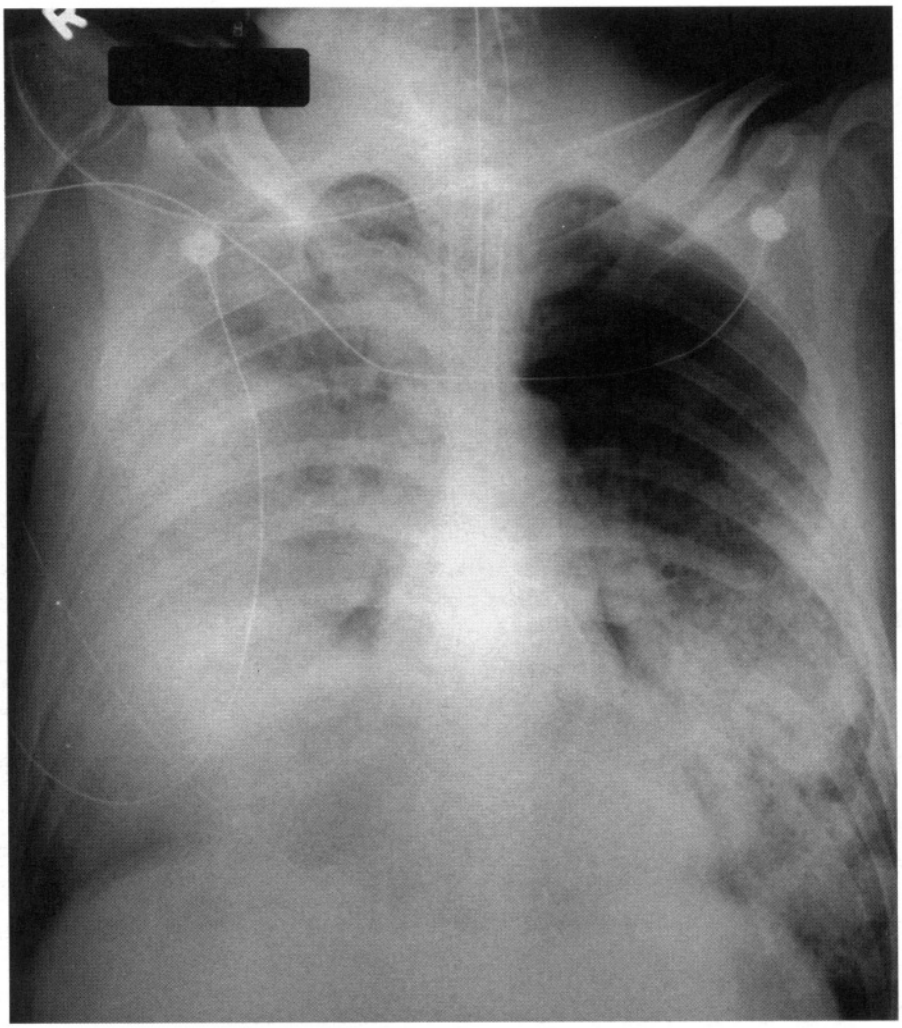
cavitate. The opacities may be pleural and wedge-shaped. In outpatients they occur most typically in intravenous drug abusers, patients with indwelling central venous catheters, and those with endocarditis and infected thrombophlebitis (Conces, 1994). The pathogens are most commonly *S. aureus,* gram-negative bacilli, anaerobes, and streptococci.

Also included in this category are the various causes of a miliary pattern of lung infection (Fig. 14). Hematogenously disseminated mycobacterial or fungal infection may cause numerous small nodules measuring less than 5 mm in diameter. The fungi most associated with this pattern are *H. capsulatum* and C. *immitis.*

*Diffuse Pneumonia*

A diffuse distribution in one or both lungs can be seen with lobar (primary air space) pneumonia, bronchopneumonia, and nodular pneumonia. Lobar pneumonia or bronchopneumonia with a diffuse distribution typically represents an advanced bacterial pneumonia and a seriously ill patient. Diffusely distributed acinar nodules or confluent acinar nodules may be seen in bronchopneumonia or disseminated fungal pneumonia (Conces, 1994).

*M. pneumoniae, C. pneumonia,* and viral pneumonias typically present as an interstitial pneumonia. The chest radiograph demonstrates ac-



A

**FIGURE 12.** (A) diffuse bilateral *Klebsiella* pneumonia with relatively homogeneous opacification of the involved lung regions. Four days later (B) there are numerous lucencies in both lungs consistent with pneumatoceles. (*Continued*)

centuated bronchovascular markings and reticular or reticulonodular opacities. The involved region usually does not extend beyond one lobe (Möller, 1992) but the interstitial pneumonia may be diffusely distributed and may progress to a bronchopneumonia pattern (Woodridge, 1992). An interstitial pattern can be seen in disseminated fungal and mycobacterial infections (Conces, 1993). Other bacterial infections may present as an interstitial pneumonia. This is especially true of *S. pneumoniae* in HIV-infected patients (Yu & Maurer, 1996). *Pneumocystis carinii* pneumonia (PCP) (Fig. 2) primarily involves the alveoli but is usually first seen on the chest radiograph as diffuse bilateral interstitial reticular or reticulonodular opacities. If unchecked the opacities typically progress to regions of air space consolidation that may contain air bronchograms (Conces, 1993). A similar but coarser pattern may be seen with *Toxoplasma gondii.*

## Location of the Pneumonia

There are pneumonia etiologies that favor certain regions of the thorax. Reactivation or postprimary tuberculosis (Miller, 1992) (Fig. 13) in ambulatory patients characteristically involves the apical and posterior segments of the upper lobes and the

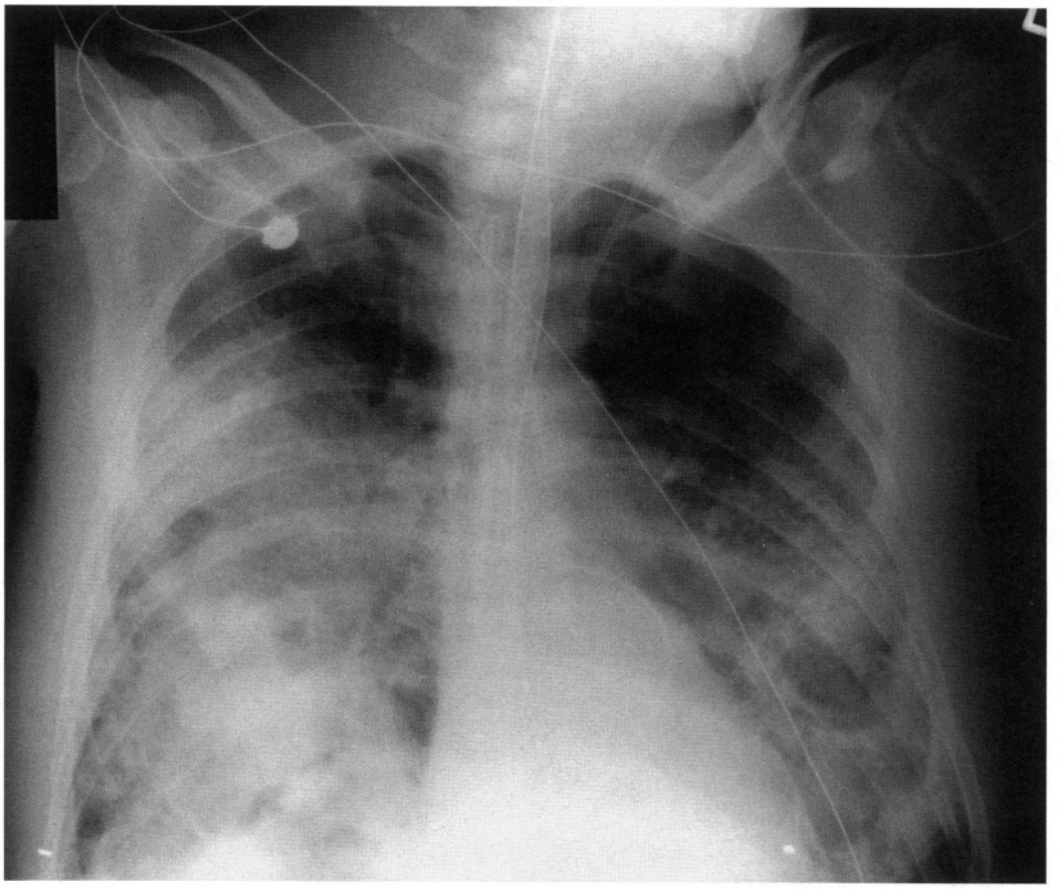



FIGURE 12. (*Continued*)

superior segments of the lower lobes. If fact, tuberculosis confined to another segment is likely to be primary infection. In patients with AIDS, primary tuberculosis usually presents as a mid- or lower-lung air space disease (Miller, 1992). PCP relapse in patients receiving pentamidine prophylaxis is seen typically in the apical regions of the lungs in ambulatory patients.

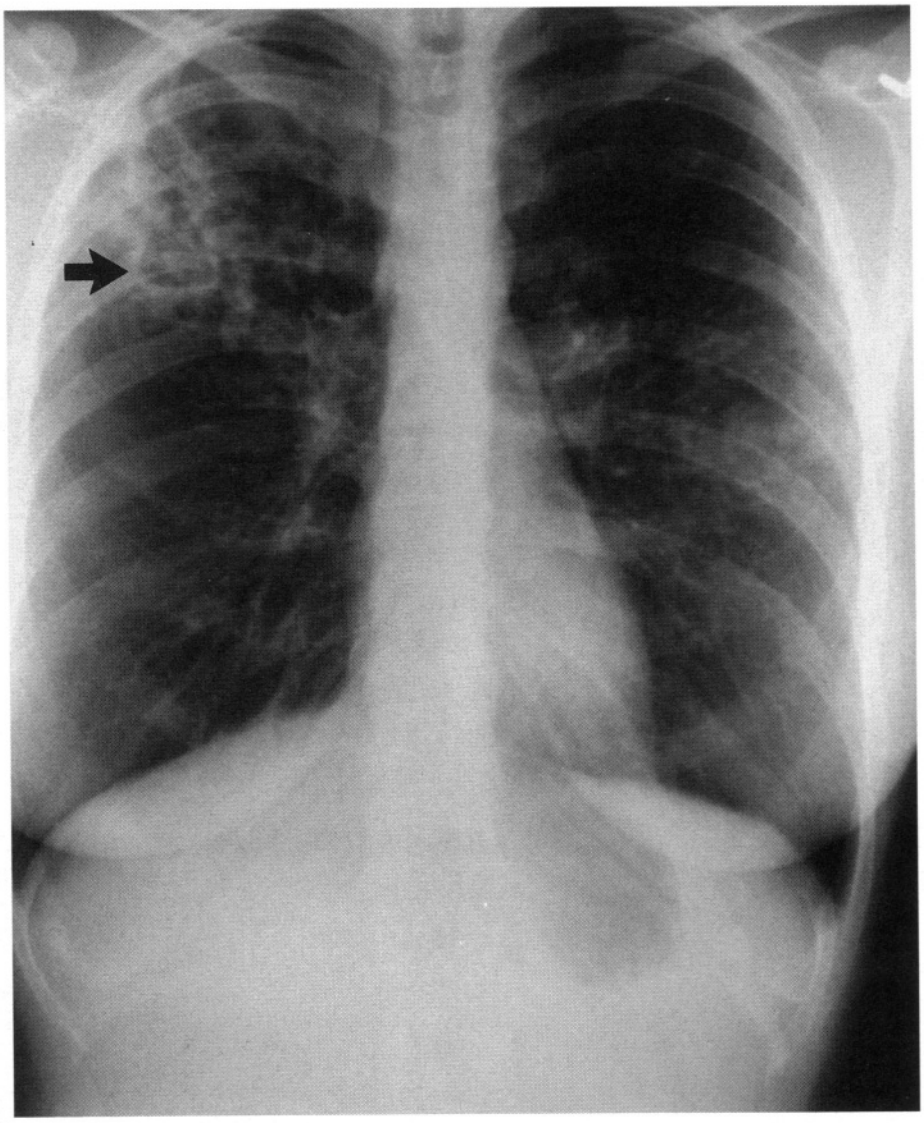
A lower-lung distribution with a peripheral bias is typical of hematogenous pulmonary infections (Woodridge, 1992). Acute miliary varieties of mycobacterial, fungal, and bacterial infections are included in this group. However, chronic miliary tuberculosis and blastomycosis will show an upper-lung preference (Gurney, 1992), likely reflecting different pathophysiology.

Aspirated infected oropharyngeal secretions and gastric contents will most heavily contaminate the dependent regions of the lung and the resulting aspiration pneumonia, typically gram-negative and anaerobic bacterial infections, will be most often found in the dependent regions predicted by the patient's position at the time of the aspiration episode. In the supine patient, the posterior aspect of both lower and upper lobes are the typical locations.

Pneumonia caused by viruses, *P. carinii,* and the atypical organisms may have a bilateral distribution and will (especially *M. pneumoniae)* radiate outward from the hila (Conces, 1993; Miller, 1994).

## Lymphadenopathy

Lymphadenopathy is not a feature of the common acute bacterial pneumonias unless there is a



A

**FIGURE 13.** Reactivation tuberculosis. There is coarse reticulonodular opacification with cavitation (arrows) in the right upper lobe with slightly less prominent predominantly nodular opacification in the left lung consistent with intrabronchial spread of the infection from the right upper lobe likely due to a ruptured cavity. (*Continued*)

complicating lung abscess or the pneumonia is secondary to an obstructing neoplasm. It is most typically associated with granulomatous infections caused by fungi and mycobacteria (Miller, 1994). Adenopathy accompanying pulmonary tuberculosis is strongly associated with primary infection (Miller, 1992). Adenopathy is not associated with reactivation tuberculosis or pulmonary infection with atypical mycobacteria (Woodridge, 1992; Conces, 1994). It is not a feature of pulmonary infection due to *L. pneumoniae* (Freundlich & Bragg, 1992) or *P. carinii* (Goldsmith, 1993; Goodman, 1992). Lymphadenopathy can occasionally be seen in pulmonary infection due to viruses and the atypical organisms *M. pneumoniae* and *C. psittaci* (Woodridge, 1992; Marrie, 1996). Tularemia is also a cause of hilar adenopathy (personal communication, Marrie, 1999).

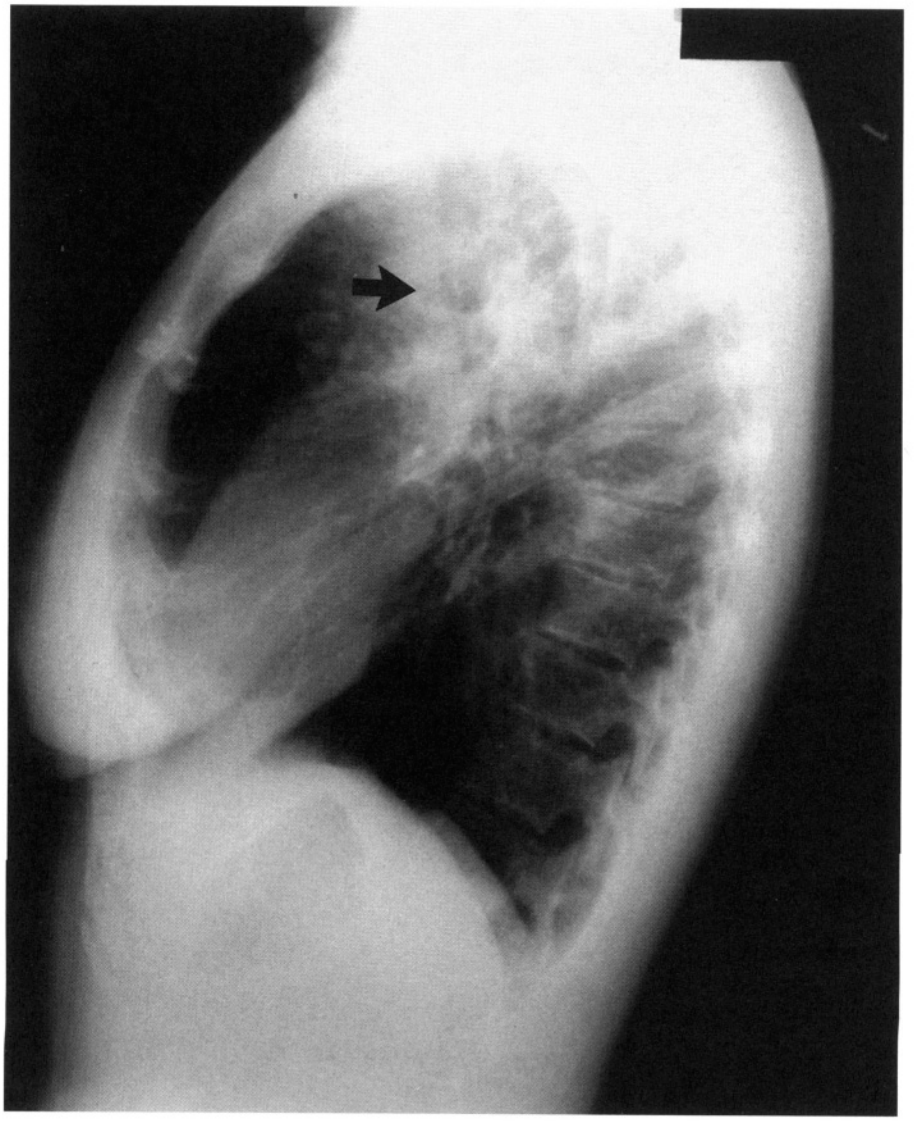


FIGURE 13. (*Continued*)

## Specific Etiologies

*Streptococcus pneumoniae* is the most common cause of CAP (Woodridge, 1992) (Table 1). Classically, lobar pneumonia has been the most typical radiographic pneumonia pattern ascribed to this organism (Freundlich, 1992). However, in the experience of recent and current clinical practice, bronchopneumonia is becoming a more frequent presentation of *S. pneumoniae* pulmonary infection, and lobar pneumonia caused by this organism is becoming considerably less common (personal communication, Marrie, 1999). *S. pneumoniae* is the most common cause of round pneumonia (Fig. 4) and complete lobar consolidation (Fig. 5). *S. pneumoniae* may also cause lobar enlargement. *S. pneumoniae* pneumonia may be complicated by empyema or bronchopleural fistula. Pulmonary

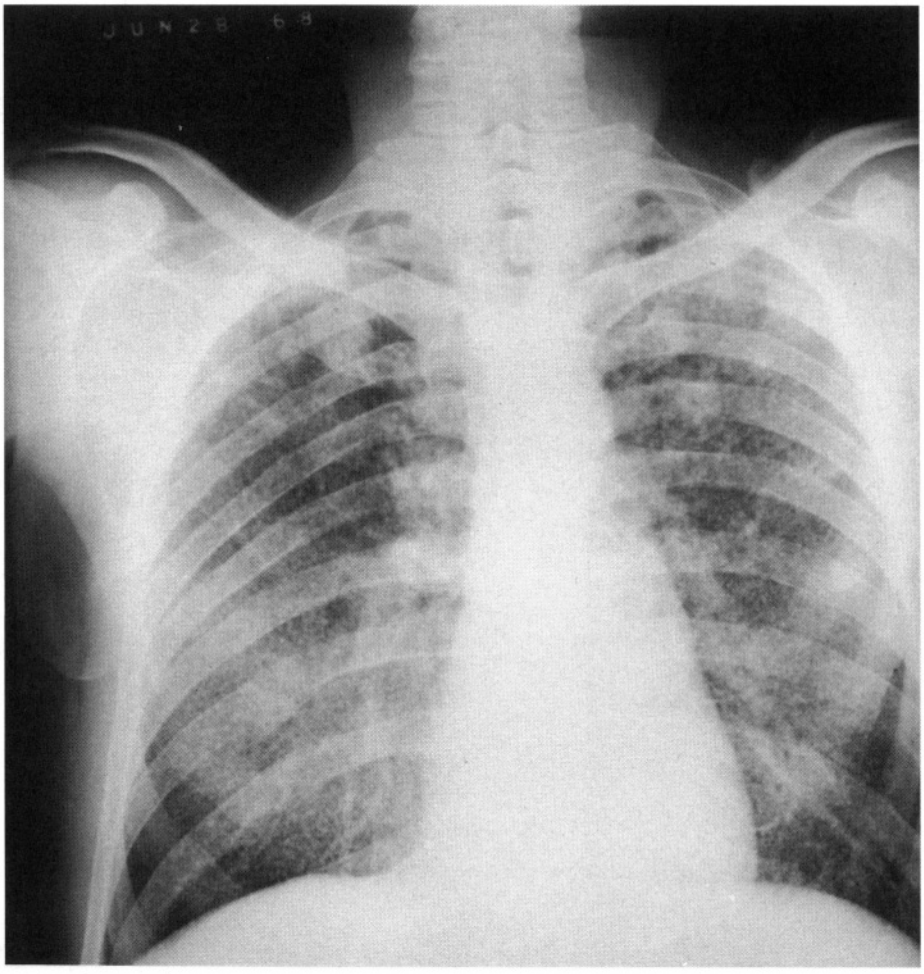


**FIGURE 14.** Miliary tuberculosis.

gangrene can be seen, especially when lobar enlargement has occurred. Cavitation is uncommon and pneumatoceles are rare.

*H. influenzae* pulmonary infection is associated with both lobar pneumonia and, occasionally, interstitial pneumonia patterns (Yu & Maurer, 1996). *H. influenzae* and *S. pneumoniae* cause the majority of air space CAP in children (Condon, 1991) and are the cause of most bacterial pneumonias in patients with AIDS and COPD (Yu & Maurer, 1996). It may occasionally cause lobar enlargement and pulmonary necrosis with pneumatoceles or gangrene. It is one of the most common causes of severe CAP (Marrie, 1996; Torres et al., 1996) and is one of the two organisms which cause the majority of abscesses and empyema in children (Wallenhaupt, 1991). The other is *S. aureus.*

*Klebsiella* is typically associated with lobar pneumonia and is the second most common cause of complete lobar consolidation. It is strongly associated with lobar enlargement and pulmonary gangrene.

*S. aureus* may cause lobar pneumonia, with or without lobar enlargement, or bronchopneumonia with a unilateral or bilateral distribution. *S. aureus* is the most common cause of septic emboli. Complications strongly associated with this infection include cavitation, pneumatoceles, and a fulminant course with adult respiratory distress syndrome (ARDS).

**TABLE 1. Pneumonia Caused by Pyogenic Bacteria**

| Organism | Patterns | Complications | Comments |
|---|---|---|---|
| *S. pneumoniae* (Woodridge, 1992; Yu & Maurer, 1996) | Lobar[a]<br>Bronchopneumonia<br>Interstitial | Lobar enlargement<br>Empyema<br>Bronchopleural fistula<br>Pulmonary gangrene | Most common cause of CAP, round pneumonia, and complete lobar consolidation; cavitation uncommon, pneumatoceles rare |
| *H. influenzae* (Condon, 1991; Ely & Haponik, 1991; Marrie, 1996; Wallenhaupt, 1991; Torres et al., 1996; Yu & Maurer, 1996) | Lobar[a]<br>Interstitial | Lobar enlargement<br>Pulmonary necrosis<br>Pneumatoceles<br>Pulmonary gangrene | *H. influenzae* and *S. pneumoniae* cause most air space CAP in children and most bacterial pneumonia in patients with AIDS and COPD<br>*H. influenzae* causes up to 20% of CAP in adults and is one of the most common causes of severe CAP<br>*H. influenzae* and *S. aureus* cause most abscesses and empyema in children. |
| *Klebsiella* (Woodridge, 1992) | Lobar[a] | Lobar enlargement<br>Pulmonary gangrene | Second most common cause of complete lobar consolidation |
| *S. aureus* (Woodridge, 1992) | Lobar[a]<br>Bronchopneumonia[a]<br>Septic emboli | Lobar enlargement<br>Cavitation<br>Pneumatoceles<br>Fulminant course ARDS | Most commmon cause of septic emboli |
| Gram-negative bacilli (Woodridge, 1992) | Lobar[a]<br>Bronchopneumonia[a] | Empyema<br>Cavitation<br>Pneumatoceles<br>Bronchopleural fistula<br>Pulmonary gangrene | May cause complete lobar consolidation<br>Septic emboli may contain gram-negative bacilli |
| Anaerobic bacteria (Woodridge, 1992) | Bronchopneumonia[a]<br>Septic emboli | Empyema<br>Cavitation<br>Bronchopleural fistula<br>Pulmonary gangrene | |

CAP, community-acquire pneumonia; COPD, chronic obstructive pulmonary disease; ARDS, adult respiratory distress syndrome.
[a]Most common pattern observed.

Gram-negative bacilli are associated with both lobar and bronchopneumonia patterns. In lobar pneumonia there may be complete lobar consolidation. Complications include empyema, cavitation, pneumatoceles, and bronchopleural fistula (Woodridge, 1992).

Anaerobic bacteria are associated with bronchopneumonia patterns. Complications include empyema, cavitation, pulmonary gangrene, and bronchopleural fistula (Woodridge, 1992).

Pulmonary infections due to *M. tuberculosis* (Table 2) can be classified as primary, reactivation, miliary, and progressive primary (Miller, 1992). Primary tuberculosis typically causes pulmonary air space consolidation. Occasionally there may be lobar enlargement which may be complicated by necrosis, pneumatoceles, cavitation, and pulmonary gangrene. These will be manifested by lucencies developing within the consolidated area. Hilar and mediastinal adenopathy is associated with primary tuberculosis. The incidence of hilar adenopathy in primary tuberculosis decreases with age. It ranges from virtually 100% in toddlers to about 40% in young adults to as low as 10% in the older population (Leung, 1999). Pleural effusions are also common; they may be loculated and indolent but nevertheless up to one third will harbor active *M. tuberculosis* organisms (Leung, 1999). Rarely the clinical course may be fulminant and complicated by ARDS. In the normal host, the adenopathy and pneumonia usually resolve over several months. The enlarged lymph nodes progressively shrink and frequently calcify. The parenchymal consolidation clears by contracting until it disappears or forms a nodule that may mimic a neoplasm. The nodule is known as a Ghon's focus. When it is accompanied by calcified central lymph nodes the combination is known as a Ranke's complex. The nodule typically

**TABLE 2. Pneumonia Caused by Granulomatous Bacteria**

| Organism | Patterns | Complications | Comments |
|---|---|---|---|
| *M. tuberculosis* (Miller, 1992) | Primary air space consolidation | Effusion<br>Atelectasis<br>Lobar enlargement<br>Necrosis pneumatoceles<br>Pneumothorax<br>Cavitation<br>Pulmonary gangrene<br>Bronchopleural fistula<br>Fulminant course with ARDS | Hilar and mediastinal adenopathy common<br>Pleural effusion common (may locutate, may contain MTB)<br>Consolidation typically resolves in several months; may clear or shrink to form granuloma (Ghon's focus)<br>Ghon's plus calcified central nodes is a Ranke's complex<br>With time Ghon's disappears or shrinks and calcifies.<br>Immunocompromised hosts may demonstrate progressive primary disease where the infection spreads from one lobe to the other |
| | Reactivation: consolidation (usually upper lung) | Cavitation<br>Opacification may be pleural, with pleural thickening<br>Usually causes fibrocalcific scarring and architectural distortion | To be considered radiographically stable there must be no change for at least 6 months (Miller, 1992) |
| | Miliary TB: Acute; numerous 1-mm nodules with diffuse basal predominance. Chronic; 3- to 5-mm nodules with upper predominance (Gurney, 1992; Miller, 1992) | | Most commonly in patients with compromised immunity (Miller, 1992) |
| Atypical mycobacteria (Miller, 1992) | Solitary or multiple air space opacity[a]<br>Nodules | May become chronic<br>Cavitation | More mid- and lower-lung and less upper lung involvement than MTB<br>Immunocompetent: lymphadenopathy uncommon and cavitation frequent<br>Immunocompromised: cavitation rare and lymphadenopathy frequent<br>Direct inhalation of contaminated soil or water (no human-to-human transmission)<br>Virtually all primary infections, no reactivation form |
| *Nocardia asteroides* (Champter, 1993; Miller, 1992) | Localized[a] or extensive air space consolidation | Cavitation<br>Effusion | May mimic a pulmonary mass<br>Severely immunocompromised may have disseminated disease<br>Ubiquitous in soil; inhalation of spores will cause disease only in immunocompromised or chronic lung disease, especially pulmonary alveolar proteinosis |
| *Actinomyces israeli* (Chambers, 1993; Miller, 1992) | Region of air space opacification | Cavitation<br>Effusion | May mimic a pulmonary mass<br>Adenopathy is rare<br>Where infection abuts chest wall or mediastinum it may become locally invasive<br>Organism is component of normal oropharyngeal flora and pulmonary disease is usually due to aspiration |

MTB, *Mycobacterium tuberculosis*; ARDS, adult respiratory distress syndrome; TB, tuberculosis.
[a]Most common pattern observed.

continues to shrink over several more months until it disappears or becomes a 5- to 10-mm, usually calcified, chronic granuloma.

In cases of reactivation tuberculosis (Stauffer, J. L., 1993), the chest radiograph demonstrates consolidation, alone or with cavitation, usually in the apical and posterior segments of the upper lobe and superior segment of the lower lobes. Other regions may be involved at the same time but active tuberculosis not involving the apical, posterior, or superior segments is probably a primary infection. Reactivation tuberculosis usually leaves fibrocalcific opacification consisting of coarse reticular or reticulonodular opacities with or without associated calcification. The opacification may be pleural, with pleural thickening and architectural distortion due to fibrotic contraction. To be considered radiographically stable there must be no change for at least 6 months (National Tuberculosis and Respiratory Disease Association) (Miller, 1992). However, radiographically stable disease may continue to harbor and shed active *M. tuberculosis,* and clinical correlation is still necessary to rule out active disease (Leung, 1999).

Miliary tuberculosis (Fig. 14) is due to hematogenous dissemination of the organisms. It is seen most commonly in patients with compromised immunity (Miller, 1992). Miliary tuberculosis may be acute or chronic. The acute form has the typical vascular distribution and the chest radiograph demonstrates numerous tiny, approximately 1-mm nodules that are more predominant in the lower lungs. The chronic form consists of larger nodules, 3 to 5 mm, which may have an acinar appearance. They are more predominant in the upper lungs (Gurney, 1992; Miller, 1992).

The immunocompromised patient may also demonstrate progressive primary tuberculosis where disease appearing initially in one lobe spreads to involve multiple ipsilateral and contralateral lobes.

Tuberculosis may be complicated by pleural effusion, empyema, cavitation, bronchiectasis, aspergilloma, hemoptysis, bronchopleural fistula, pneumothorax, and atelectasis. A solitary tuberculous granuloma may mimic a carcinoma.

*Mycobacterium kansasii* and *Mycobacterium avium-intracellulare* are the atypical mycobacteria that most commonly cause pulmonary disease (Miller, 1992). Unlike *M. tuberculosis,* there is no human-to-human transmission of these pathogens. Pulmonary disease is usually caused by direct inhalation of contaminated soil or water. There is no reactivation form of atypical mycobacterial infection. Virtually all are primary infections. Atypical mycobacterial infections may become chronic.

Atypical mycobacterial pulmonary infection typically causes solitary or multiple regions of air space opacification or nodules. Unlike tuberculosis there is more mid- and lower-lung involvement and less upper lobe predominance. In the immunocompetent patient, lymphadenopathy is uncommon and cavitation in the affected regions or nodules is frequent. The cavities are often thin-walled and may simulate bronchiectasis on the chest radiograph. On the other hand, in the immunocompromised patient with AIDS the situation is reversed. Cavitation is rare while lymphadenopathy is common.

*Nocardia asteroides* has in the past been classified as a fungus but it is now classified as a member of the actinomycetes group and recognized to be a higher-level bacterium (Chambers, 1993). It is ubiquitous in the soil. Inhalation of its spores will typically cause infection only in patients who are immunocompromised or who have chronic lung disease, particularly pulmonary alveolar proteinosis. It most often causes a localized region of air space consolidation. The margins of this region may be quite well defined and mimic a pulmonary mass. Cavitation and effusion are common. The air space disease may be extensive. Disseminated disease may occur in the severely immunocompromised.

*Actinomyces Israeli* also used to be classified as a fungus but is now recognized to be a higher-level bacterium (Chambers, 1993). It is a component of the normal oropharyngeal flora. Pulmonary disease is usually due to aspiration of oropharyngeal secretions. On chest radiograph the infection manifests as a region of air space opacification that cannot be differentiated from other causes of pneumonia. The region of consolidation may resemble a pulmonary mass. Cavitation and effusion are common whereas adenopathy is rare. Where the infection abuts the chest wall or mediastinum it may become locally invasive, occasionally causing localized osteomyelitis or a draining sinus (Miller, 1992).

Atypical pneumonia is a term used to desig-

nate a pneumonia syndrome that is marked by a slow onset, an early prominence of systemic rather than respiratory symptoms, a nonproductive cough, and more marked findings on the chest radiograph than would be suggested by the clinical examination. The currently recognized causes of this syndrome include several bacteria and a number of viruses (Marrie, 1996). *M. pneumoniae, L. pneumophila, C. pneumoniae, C. psittaci,* and *Coxiella burnetii* are the bacterial agents (Table 3). The viral agents are Hantavirus and a number of others, including adenovirus, respiratory syncytial virus, papainfluenza virus, influenza A and B, rubeola, varicella, Epstein-Barr, and cytomegalovirus.

*M. pneumoniae* is spread by inhalation of droplets. The most typical initial chest radiographic pattern is interstitial pneumonia with reticular, micronodular, or reticulonodular opacification which may be diffuse but is usually confined to one lobe (Müller, 1992). *M. pneumoniae* is the most common bacterial cause of interstitial pneumonia (Woodridge, 1992). It may progress to segmental or lobar pneumonia or bronchopneumonia. With severe *M. pneumoniae* pulmonary infection, the most frequent complication is respiratory failure.

*L. pneumophila* is an aquatic organism found in natural and manmade water supplies. It is spread by inhalation of contaminated water droplets. Most patients with pulmonary infection caused by this organism have comorbidity due to cardiac or pulmonary disease. In the majority of patients the chest radiograph will demonstrate patchy air space disease with a segmental, lobar, or diffuse distribution. Another frequent radiographic presentation is multiple poorly defined nodular opacities. In some studies there has been a predilection for the lower lobes. There is typically rapid progression of inhomogeneous disease to produce bilateral bronchopneumonia or complete uniform consolidation in the affected lobe or lobes. *L. pneumophila* is the second most common cause of severe CAP and may have a fulminant course with ARDS. Pleural effusions are common complications. Adenopathy and cavitation are uncommon (Woodridge, 1992; Freundlich & Bragg, 1992). *Legionella* pneumonia clears slowly compared to other pneumonias, and delayed resolution should suggest the possibility of this disease (Freundlich & Bragg, 1992).

*C. pneumoniae* pulmonary infection accounts for 10% or more of CAP and up to 10% of severe CAP (Marrie, 1996). *C. psittaci,* a closely related bacterium carried by psitticine birds (e.g., parrots, parakeets, finches, and turkeys), can cause an atypical pneumonia known as psittacosis. Chest radiographic appearances are similar to those found in other cases of atypical pneumonia or atypical pneumonia syndromes.

Pulmonary infection due to inhalation of *C. burnetii* is known as Q fever. The chest radiographic features may be similar to the other causes of CAP. The most common findings (90%) are segmental, lobar, or patchy air space disease. Nodular or mass-like disease is found in about 7% and interstitial pneumonia in about 3% (Gikas et al., 1999). Gikas et al. reported pleural effusion in about 17% in the series of 85 patients, but they did not see any evidence of adenopathy. In their series, resolution was slow and took up to 6 weeks. When the radiographic features are nonspecific, multiple rounded pulmonary nodules following exposure to parturient cats should suggest Q fever (Gordon et al., 1984).

Viral pneumonias account for an estimated 25% of CAP cases. Infections typically result from inhalation of contaminated droplets and initially involve the walls of the airways and may be limited to them. Spread of the infection to the peribronchial interstitium, peribronchiolar interstitium, and interlobular septa causes acute interstitial pneumonia. Further spread to the intralobular interstitium (Webb et al., 1996) and peribronchiolar alveoli will cause either a bronchopneumonia pattern or, if the interstitial opacities are not prominent, nodular (typical of varicella) or patchy air space opacification. The disease may be diffuse and bilateral or confined to one region or lobe. If confined to one lobe it may simulate bacterial lobar pneumonia. Acute fulminant viral pneumonia causes a generalized hemorrhagic pulmonary edema that begins with a central distribution and spreads to involve the whole lung, causing ARDS. This is typical of influenza and Hantavirus infection but may be caused by other viruses especially varicella and cytomegalovirus. Complications of viral pneumonias include adenopathy, small effusions, and ARDS (Woodridge, 1992; Freundlich & Bragg, 1992).

Fungal pneumonias (Miller, 1992) due to *H. capsulatum, C. immitis, Blastomyces dermatitidis.*

**TABLE 3. Organisms Causing an Atypical Pneumonia Syndrome[a]**

| Organism | Patterns | Complications | Comments |
|---|---|---|---|
| *Mycoplasma pneumoniae* (Müller, 1992; Woodridge, 1992) | Interstitial pneumonia[b]<br>Segmental or lobal pneumonia<br>Bronchopneumonia | Respiratory failure | May be diffuse but usually confied to one lobe<br>Most common bacterial cause of interstitial penumonia |
| *Chlamydia pneumoniae* (Marrie, 1996) | Interstitial pneumonia[b]<br>Segmental or lobal pneumonia<br>Bronchopneumonia | Severe CAP | Accounts of 10% of CAP and 10% of severe CAP<br>*Chlamydia psitacci* is closely related, carried by healthy birds and causes a similar atypical pneumonia syndrome known as psittacosis |
| *Coxiella burnetii* (Marrie, 1996; Gikas et al., 1989) | Segmental or lobar pneumonia<br>Bronchopneumonia<br>Multiple rounded opacities<br>Interstitial pneumonia[b] | Respiratory failure | Q fever |
| *Legionella pneumophila* (Freundlich & Bragg, 1992; Woodridge, 1992) | Patchy air space disease with segmental, lobar or diffuse distribution[b]<br>Poorly defined nodular opacities | Pleural effusions<br>Rapid progression to complete lobar consolidation or bilateral bronchopneumonia<br>Fulminant course with ARDS | Aquatic organism found in all water supplies<br>Spread likely by inhalation of contaminated water droplets<br>Second most common cause of severe CAP<br>Predilection for lower lobes<br>Clears slowly compared with other pneumonias<br>Adenopathy and effusion are uncommon<br>Patients with *Legionella* pneumonia usually have comorbid cardiac or pulmonary disease<br>Adenopathy and cavitation are uncommon |
| Viruses (Freundlich & Bragg, 1992; Webb et al., 1995; Woodridge, 1992) | Interstitial pneumonia[b]<br>Bronchopneumonia<br>Nodular pneumonia<br>Patchy air space<br>Pneumonia | Adenopathy<br>Small effusions<br>Fulminant pneumonia with hemorrhagic edema and ARDS (especially influenza, Hantavirus, CMV, and varicella) | Accounts for about 25% of CAP<br>Pulmonary infections caused by inhalation of contaminated droplets<br>Disease may be diffuse and bilateral or confined to one region or lobe<br>If confined to one lobe is may simulate bacterial lobar pneumonia |

CAP, community-acquired pneumonia; ARDS, adult respiratory distress syndrome; CMV, cytomegalovirus.

[a]Atypical pneumonia is a term used to designate a pneumonia syndrome which is marked by a slow onset, an early prominence of systemic rather than respiratory symptoms, a nonproductive cough, a more marked findings on the chest radiograph than would be suggested by the clinical examination. The currently recognized causes of this syndrome include several bacteria (*M. pneumoniae*, *L. pneumophila*, *C. pneumoniae*, *C. psittaci*, *Coxiella burnetii*) and a number of viruses (Hantavirus, adenovirus, respiratory syncytial virus, parainfluenza virus, influenza A and B, rubeola, varicella, Epstein-Barr, and CMV) (Marrie, 1996).

[b]Most common pattern observed.

and *C. neoformans* are virtually always caused by inhalation of contaminated dust and are relatively common where these organisms are found naturally in the soil (Table 4).

On the chest radiograph an acute pneumonia due to histoplasmosis is typically seen as solitary or multiple focal air space opacities frequently accompanied by adenopathy. Multiple nodules throughout both lungs may be seen if there has been a more marked degree of exposure to this fungus. The nodules may be well or poorly defined. Their sizes range from 1 to 5 mm. Pulmonary infection with this fungus may cause a solitary granulomatous nodule measuring up to about 4 cm which may mimic a neoplasm. Chronic pulmonary histoplasmosis may occur in patients with bullous lung disease. Air space opacification with cavitation and architectural distortion due to fibrosis occur and may mimic tuberculosis or carcinoma. Disseminated disease may occur in the immunocompromised patient. Complications of pulmonary infection by *H. capsulatum* include mediastinal adenopathy and fibrosing mediastinitis.

Acute pulmonary infection with *C. immitis* in the immunocompetent patient usually presents as a region of patchy air space consolidation most often involving a lower lobe. Adenopathy occurs in an estimated 20% of cases. Persistent air space consolidation is most commonly due to persistent primary disease. Reactivation pulmonary coccidioidomycosis and chronic pulmonary coccidioidomycosis may cause persistent regions of air space opacification or nodules and may cavitate. An asymptomatic, solitary thin-walled cavity may be found. It is considered to be the classic lesion for this disease and is seen in 10% to 15% of patients with this disease. Disseminated disease, including miliary disease, may occur, especially in the immunocompromised patient (Miller, 1992).

Pulmonary blastomycosis is seen on chest radiograph most often as a region of focal or patchy pneumonia that may be found in any lobe. Solitary or multiple nodules may occur. Adenopathy and cavitation are unusual. Rarely there is diffuse nodular, micronodular, or miliary disease.

The most common chest radiographic finding in pulmonary cryptococcosis is a solitary pleural mass that may measure up to 10 cm in diameter and mimic carcinoma. It may also present as a region of air space opacification. These findings may be multifocal. Cavitation, adenopathy, and effusion are uncommon. Diffuse air space or miliary disease may occur in immunocompromised patients.

*A. fumigatus* and other species of this group are ubiquitous in nature and form part of the normal human oropharyngeal ecology. Aspiration or inhalation of oropharyngeal secretions containing these organisms normally does not cause pulmonary disease unless lung architecture or immunity has been altered or compromised. Pulmonary infections with this fungus are classified as noninvasive, semi-invasive, and invasive aspergillosis and allergic bronchopulmonary aspergillosis.

Noninvasive aspergillosis is a saprophytic colonization of a bronchiectatic airway, bulla or preexisting cavity, or other space by *Aspergillus* species. The colonization may occur in the host with normal immunity when there is a preexisting cavity to which the fungal spores can gain access. The fungus proliferates within the space and fills it with a mass of matted hyphae called a mycetoma (Fig. 15). The mass may be mobile within the space. *Aspergillus* species are not the exclusive causes of mycetomas. Saprophytic colonization may also occur with semi-invasive aspergillosis.

Semi-invasive aspergillosis occurs when there is mild immunosuppression, for example, due to age or debilitating illness. The pulmonary infection begins as a region of air space pneumonia, usually at an apex, that eventually cavitates due to internal necrosis. A mycetoma may form within the cavity, typically in association with apical pleural thickening. An "air crescent sign" can be seen during this process when either the necrotic tissue or the mycetoma separates from the wall of the developing space and forms a radiolucent arc along its margin.

Invasive aspergillosis occurs when there is severe immunocompromise. Most commonly there are multiple bilateral nodular foci of pulmonary infiltration or diffuse patchy air space opacification although in some cases there may be only a solitary region of air space opacification or a solitary nodule. Cavitation typically does not occur while the immune system is at its nadir but, rather, as the immune system is recovering. A crescent sign may be seen as central necrotic tissue separates from the margins of the cavity. Pleural effusion may occur and adenopathy is uncommon.

**TABLE 4. Pneumonia Caused by Fungi**

| Fungus | Patterns | Complications | Comments |
|---|---|---|---|
| *Histoplasma capsulatum*[a] (Miller, 1992) | Solitary or multiple air space opacities[b]<br>Solitary or multiple nodules | Mediastinal adenopathy<br>Cavitation<br>Focal pulmonary fibrosis<br>Fibrosing mediastinitis | Nodules (1–5 mm) may be well or poorly defined<br>Solitary nodule may be up to 4 cm and mimic neoplasm<br>Chronic disease may be seen in bullous lung disease<br>Immunocompromised may have disseminated disease |
| *Coccodioides immitis*[a] (Miller, 1992) | Region of air space opacification, usually lower lung[b] | Adenopathy<br>Solitary thin-walled cavity (classic lesion) | Immunocompromised may have disseminated disease including miliary nodules<br>Persistent air space disease most likely persistent primary<br>Infection but reactivation and chronic forms do occur |
| *Blastomyces dermatitidis*[a] (Miller, 1992) | Focal or patchy air space disease in any lobe[b]<br>Solitary or multiple nodules | Rarely, adenopathy and cavitation | Diffuse nodular or miliary disease may occur |
| *Cryptococcus neoformans*[a] (Miller, 1992) | Solitary pleural mass[b]<br>Region of air space opacification; may be multifocal | Uncommonly, cavitation, adenopathy, effusion | Mass may mimic carcinoma |
| *Candida albicans* (Miller, 1992) | Bronchopneumonia[b]<br>Multiple pulmonary nodules | Effusion<br>Seldom, cavitation, adenopathy | Usually only in immunocompromised patients<br>Infection caused by aspiration of oropharyngeal secretions comtaminated by *C. albicans* |
| *Aspergillus fumigatus* (Miller, 1992) | | | Ubiquitous in nature; normal human oropharyngeal flora<br>Aspiration causes disease only if there is prior lung damage or if immunity is altered or compromised<br>Infections classified as noninvasive, semi-invasive, invasive, and ABPA |
| Noninvasive | Mycetoma | | Colonizes and proliferates within a cavity, bulla, or bronchiectactic airway<br>Mycetoma may be mobile within the space |
| Semi-invasive (mildly immunocompromised) | Region of air space opacification usually at apex | Eventually cavitates due to necrosis<br>May form mycetoma | Air cresent may be seen as necrotic tissue separates from margin of cavity |
| Invasive (severely immunocompromised) | Multiple bilateral nodular opacities or diffuse patchy air space opacification (may be solitary) | Cavitation not seen until immune system begins to recover<br>May have pleural effusion | Air crescents may be seen as cavities form<br>Adenopathy is uncommon |
| ABPA (seen in asthmatics) | Finger-in-glove mucous casts of opacified airways<br>Segmental or lobar atelectasis<br>Post-obstruction pneumonia | Rarely cavitates with mycetoma formation | Bronchial walls are hyperreactive to fungal spores and marked mucus production leads to masses of hyphae wihtin inspissated mucus within the affected airways |

ABPA, allergic bronchopulmonary aspergillosis.
[a]Found in the soil; pulmonary infection usually caused by inhalation of contaminated dust.
[b]Most common pattern observed.

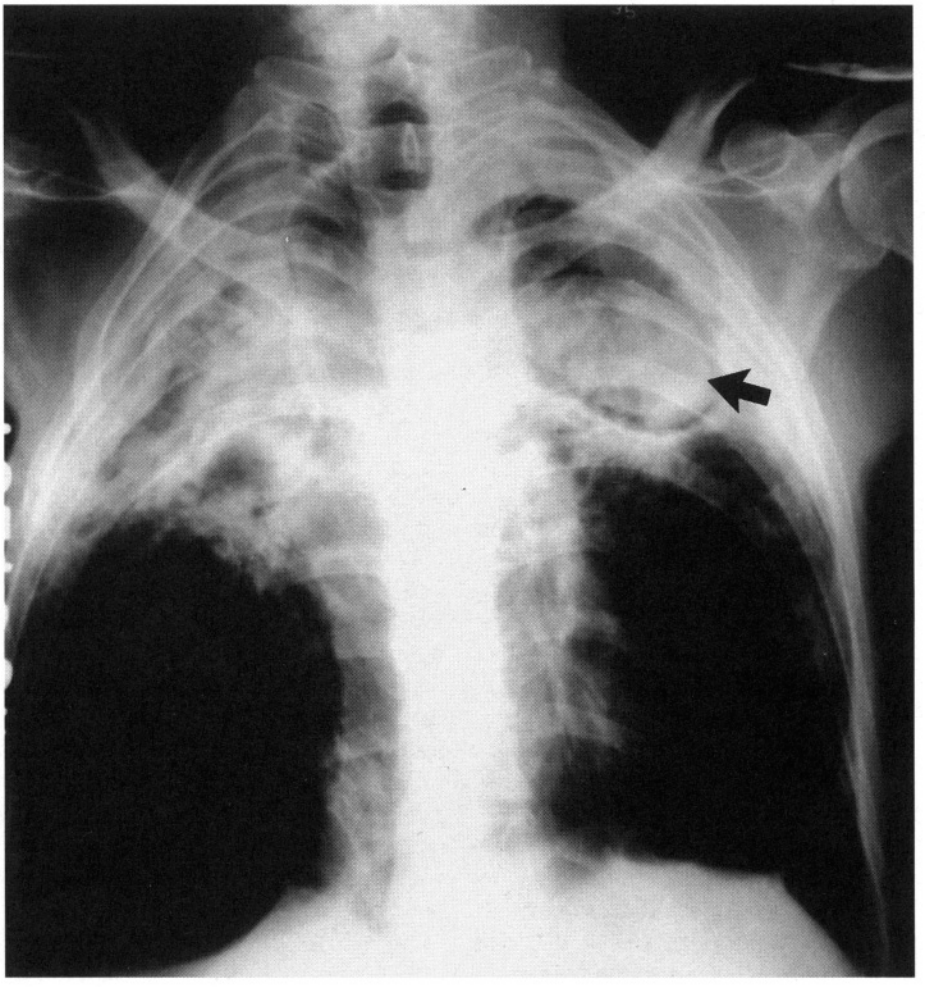


**FIGURE 15.** An aspergilloma (arrow) within a left upper lobe cavity.

Allergic bronchopulmonary aspergillosis (ABPA) occurs in asthmatics whose lungs have become hyperreactive to *Aspergillus* spores residing in the airways. The allergic reaction causes marked mucus production in the affected regions. *A. fumigatus* hyphae proliferate in the mucus and generate more spores which then provoke a more widespread and severe reaction, and the cycle repeats itself. Eventually large masses of hyphae suspended in inspissated mucus will fill the involved airways, usually central airways. These mucus plugs can be seen on chest radiograph as typical "finger in glove" opacities which are actually radio-opaque casts of the airways. The mucus plugs will obstruct the affected airways and cause segmental or lobar atelectasis or post-obstruction pneumonia. Rarely cavities may occur and mycetomas may develop within them.

*P. carinii,* an opportunistic pathogen formerly thought to be protozoal but recently recognized as likely to be fungal (Goldsmith, 1993), is the most frequent cause of pulmonary infection in patients with AIDS (Table 5). On chest radiograph PCP is most commonly seen as a diffuse mild to moderate interstitial pneumonia (Goodman, 1992) (Fig. 2). If treatment is delayed it may progress rapidly to cause diffuse bilateral pulmonary opacification and ARDS. In some patients it may present as a focal or

**TABLE 5. *Pneumocystis carinii* Pneumonia**

| Organism | Patterns | Complications | Comments |
|---|---|---|---|
| *Pneumocystis carinii* (Goldsmith, 1993; Goodman, 1992) | Diffuse bilateral interstitial pneumonia (most common)<br>Focal or regional interstitial pneumonia<br>Focal or regional lobar pneumonia<br>One or more noduules (uncommon) | Pneumatoceles<br>Pneumothorax<br>May progress rapidly to diffuse bilateral opacification and adult respiratory distress syndrome | Opportunistic pathogen classically classified as protozoal but recently recognized as probably fungal<br>Most frequent cause of pulmonary infection in AIDS<br>Adenopathy and pleural effusion are rare<br>Patients on pentamidine may have upper-lung disease resembling reactivation tuberculosis |

regional interstitial or lobar pneumonia. Rarely, it presents as one or more pulmonary nodules. An upper-lung pattern resembling reactivation tuberculosis may be seen, especially in patients receiving pentamidine prophylaxis. Complications of this infection include pneumatoceles and pneumothorax and, as mentioned above, ARDS. Adenopathy and pleural effusion are rarely caused by this organism.

## Specific Complications of Pneumonia

A poor response to treatment may be due to treatment failure or to complications of the pneumonia (Fein, 1996). The former may be due to a resistant organism, to an unusual or unanticipated pathogen, or to a noninfectious pneumonia mimic. Complications of the pneumonia include parapneumonic pleural effusion, empyema, bronchopleural fistula, lung abscesses, pulmonary gangrene, pneumatoceles, and ARDS.

A parapneumonic pleural effusion (Hanna et al., 1991) is caused by the pleural response to a nearby pulmonary infection and is the most common complication of pneumonia. It is most commonly seen in bacterial pneumonias, especially those due to *S. aureus, H. influenzae, L. pneumophila, Nocardia,* anaerobic bacteria, gram-negative bacilli, and *S. pneumoniae.* A pleural effusion is usually evident on plain radiographs, especially if an erect film can be obtained. On a supine film, fluid accumulates in the dependent portion of the hemithorax and is best appreciated at the apex (as a crescent of soft tissue density), in the lateral costophrenic angle or as an increased subpulmonic density accompanied by a flattening of the diaphragm contour with a lateral shift of the dome apex.

Parapneumonic pleural effusion is most commonly unilateral and on the same side as the pneumonia (Hanna et al., 1991), with the pneumonia being the dominant feature. If the effusion is the dominant feature and adjacent lung opacities are less conspicuous it is likely that the effusion is a primary process with passive atelectasis causing the lung opacities (Webb et al., 1996). A bilateral parapneumonic effusion is usually larger on the side of the pneumonia. If it is symmetrical it is more likely due to something other than the pneumonia (Hanna et al., 1991).

An infected pleural effusion is known as an empyema. All empyemas begin as a sterile pleural effusion which becomes secondarily infected. The source is usually an adjacent pneumonia or lung abscess. Radiographic evidence of loculation developing within a previously mobile pleural effusion strongly suggests empyema. If loculation is suspected on the routine chest radiograph it may be confirmed or ruled out by right and left lateral decubitus views. More specific radiographic signs of empyema are tension hydrothorax and chest wall edema. Within a few weeks the inflammatory reaction will generate a thick pleural peel which will impair regional lung function. Rarely the infection may spontaneously drain through the chest wall (empyema necessitatis). To avoid these complications empyema must be detected and treated in its earliest stages (Woodridge, 1992; Conces, 1994; Hanna et al., 1991).

Pneumonia complicated by parenchymal necrosis may cause a bronchopleural fistula which is a fistulous communication between the pulmonary airways and the pleural space. A bronchopleural fistula may cause a secondary pneumothorax or, if there is associated parapneumonic effusion or empyema, a secondary hydropneumothorax or pyopneumothorax. The air fluid levels in the pleural fluid are best detected on horizontal beam chest radiographs. The organisms most commonly associated with bronchopleural fistula include *S. aureus,* gram-negative bacilli, anaerobic bacteria, and streptococci, occasionally *S. pneumoniae.*

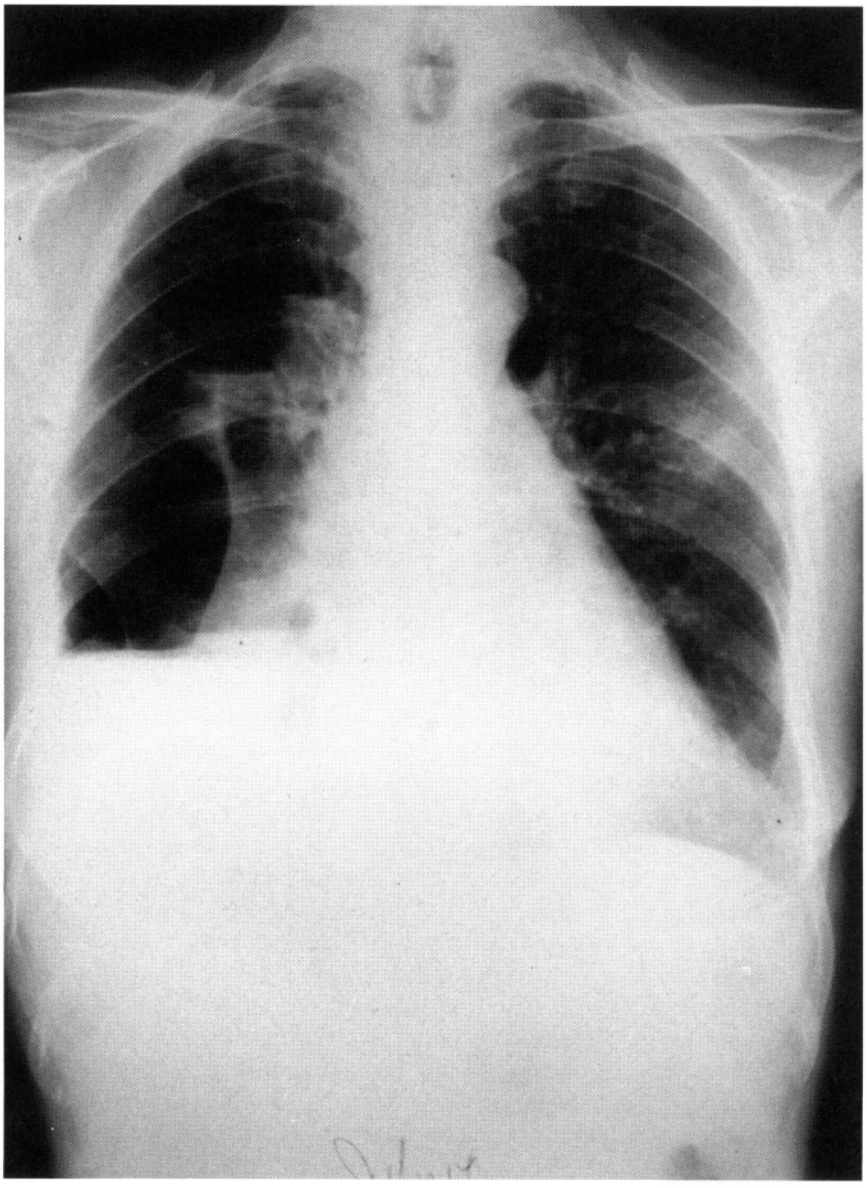
Radiographic differentiation between a bronchopleural fistula with a pleural air fluid level and an air fluid level within a lung abscess can on occasion be difficult. A pleural location for the air collection is suggested when it has relatively thin and uniform walls, when the fluid contour is oblong and conforms to the pleural space with obtuse angles between it and the lung, and when the air fluid levels on orthogonal horizontal beam projections are of unequal length (Fig. 16). A pleural fluid collection may be seen to cross the anatomical location of a pleural fissure. Multiple air fluid levels are more frequently associated with pleural fluid because abscesses typically, but not always, have a single air fluid level (Fig. 17) (Hanna et al., 1991; Woodridge, 1992; Conces, 1994).

Pulmonary infection by particularly virulent bacteria, such as *S. aureus,* streptococci, gram-negative bacilli, and anaerobic bacteria evokes an intense inflammatory response that causes varying degrees of pulmonary parenchymal necrosis that may become radiographically discernible by causing cavitation or pneumatoceles. Cavitation may occur within an abscess, microabscesses, or pulmonary gangrene.

A pyogenic lung abscess most commonly occurs as a complication of a focal pneumonia caused by aspiration of infected oropharyngeal or gastric secretions (Groskin et al., 1991; Hanna et al., 1991; Woodridge, 1992; Conces, 1994; Bragg & Freundlich, 1992). In one study (Groskin et al., 1991) fewer than 2% of cases occurred in the right middle lobe or lingula. A cavity filled with purulent fluid forms following central necrosis. Over a period of 7 to 14 days after the initiating event, the necrosis may spread and penetrate the wall of the abscess and establish free communication between the abscess cavity and adjacent airways. The purulent contents of the abscess may be coughed up as foul-smelling sputum and air can enter the abscess cavity. Chest radiographs obtained during the early phase of a lung abscess, before communication with the airways has become established, will demonstrate simply a nonspecific opacity with ill-defined margins for which the differential diagnosis will include pneumonia and neoplasm. After communication with the airways has become established the intracavitary air may be seen as an air fluid level on chest radiographs performed with a horizontal beam. It may be differentiated from bronchopleural fistula by obtaining orthogonal views. An abscess is roughly spherical and the air fluid levels will have equal lengths on orthogonal horizontal beam views as opposed to the unequal lengths typically seen with intrapleural air fluid levels (Fig. 17). Early radiographic diagnosis of a lung abscess therefore requires that a high index of suspicion be maintained and that horizontal beam views of the chest be obtained to detect an intracavitary air fluid level when a lung abscess is suspected. A vertical beam chest radiograph, such as the typical portable supine or semi-erect frontal examination obtained for very ill patients, will not show an air fluid level, and an abscess that contains air will continue to be imaged as a nonspecific opacity and may be missed. A visibly cavitated lung abscess with or without an air fluid level will have a chest radiographic appearance that overlaps with the radiographic appearance of other cavitated lesions such as nonpyogenic abscess, cavitated granuloma, and cavitated neoplasm. The differential diagnosis cannot be made on the basis of the radiographic features alone and the clinical setting and the clinical findings must be taken into consideration.

When there is a widespread intense inflammation, such as would be seen in lobar pneumonia with lobar enlargement, the entire region of involved lung may become necrotic and separate from the adjacent still viable lung. A large cavity may form, a phenomenon known as pulmonary gangrene (Fig. 18).

Multiple microabscesses may form in a region or regions of intense inflammation and be radiographically visible as multiple lucencies with or without air fluid levels. This is termed necrotizing

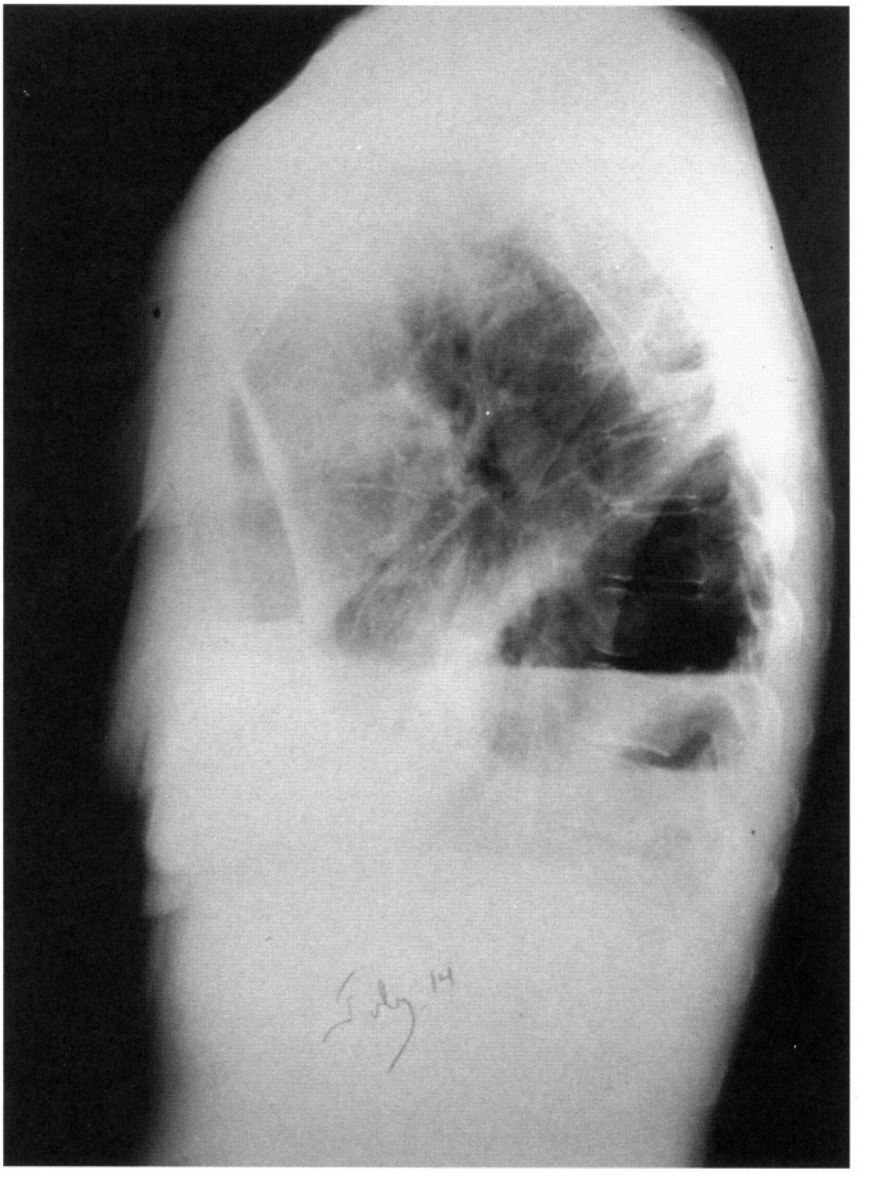


A

**FIGURE 16.** Air and fluid in the pleural space. Note that there are several asymmetric air-fluid levels on the patient's right side.

pneumonia. The microabscesses may become confluent to produce a focal abscess or a region of pulmonary gangrene.

A pneumatocele is thought to form when necrotizing pneumonia causes focal loss of alveolar wall integrity and air is allowed to enter the lung interstitium and form a subpleural collection (Fig. 12). If the point of rupture becomes a check valve the collection can become massive. Pneumatoceles may be multiple. Unlike abscesses, pneumatoceles have thin smooth walls, may form and change rapidly, and are not marked by the sudden production of foul-smelling sputum. Rupture of a pneumatocele may cause a pneumothorax.



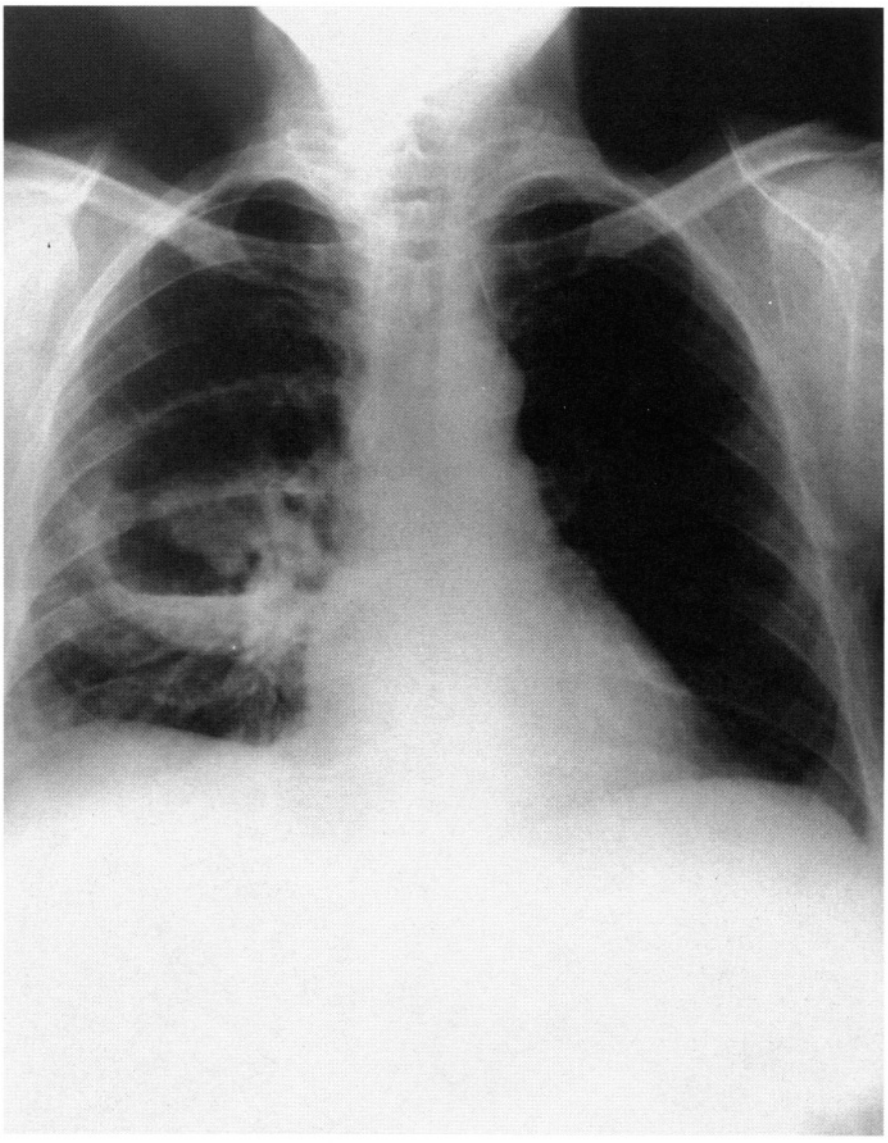
B

FIGURE 16. (*Continued*)

Pneumonia may have a fulminant course culminating in ARDS (Fig. 1). This complication can be secondary to pneumonia of any cause but is most particularly associated with viral, PCP, staphylococcal, streptococcal, and *Legionella* pneumonia and miliary tuberculosis (Woodridge, 1992; Goodman, 1992).

## Additional Imaging—Chest Radiographs

Radiographic evidence of clinically suspected pneumonia will usually appear within 12 to 24 hours (Herold, 1997). If the chest radiograph is negative at presentation it should be repeated in 2 to



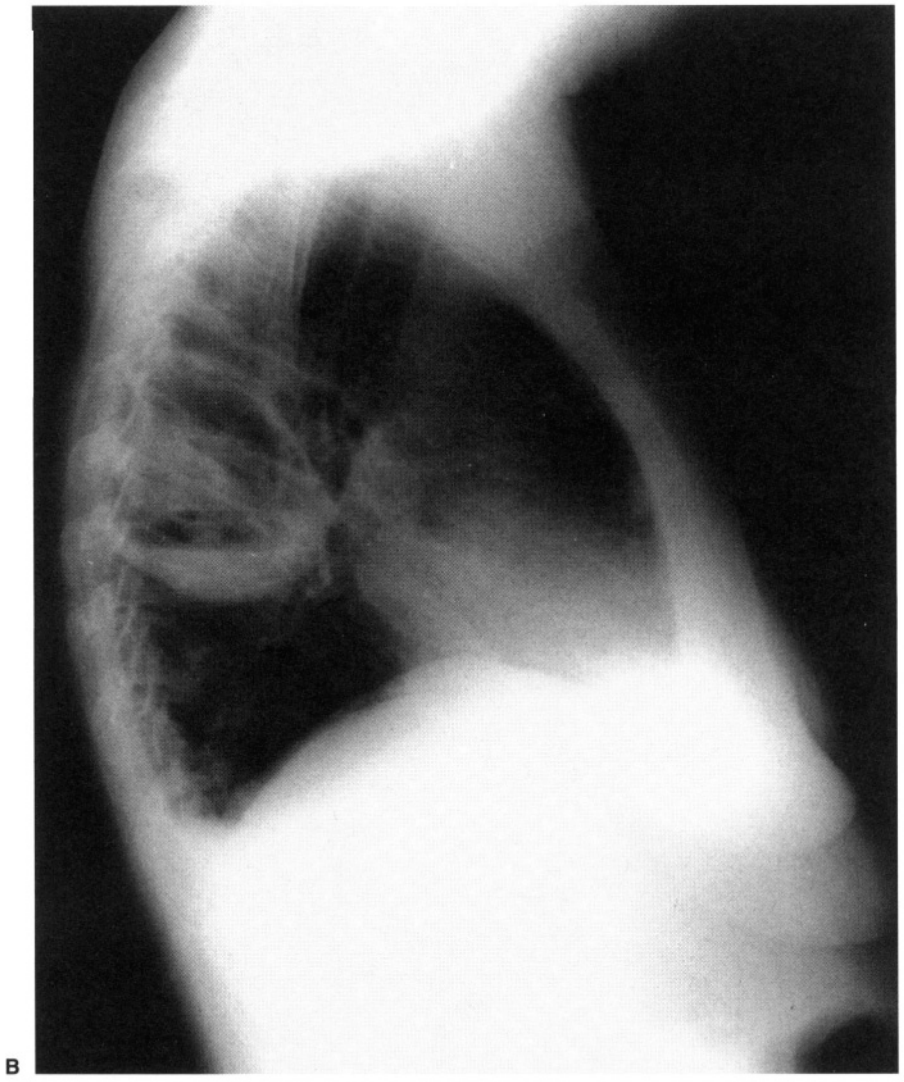
A

**FIGURE 17.** Symmetrical air-fluid levels within an abscess in the superior segment of the right lower lobe on posteroanterior (A) and lateral (B) views.

3 days. If it is still negative the need for additional diagnostic studies should be considered.

Early in the course of uncomplicated CAP frequent imaging is of little value to monitor response to treatment. With effective therapy clinical improvement should be noted within 48 to 72 hours with resolution of fever and leukocytosis by day 4 (Fein, 1996). Chest radiographic improvement, however, will typically lag behind the clinical improvement by several days and may even get worse immediately following the initiation of treatment (Fein, 1996). In otherwise uncomplicated CAP worsening of the chest radiographic findings in the first few days is of no concern if the clinical indicators are improving. On the other hand, it is associated with a poorer prognosis if the patient has severe CAP (Areno et al., 1996).

Radiographic findings are typically slow to

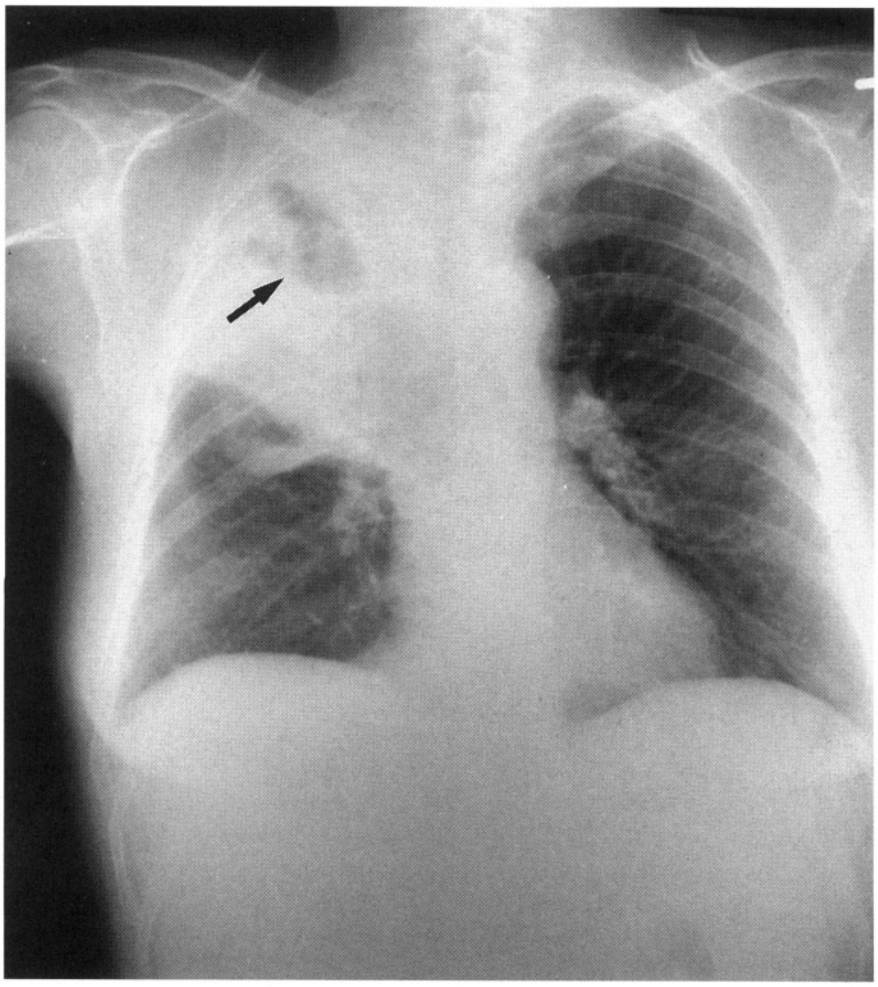


B

FIGURE 17. (*Continued*)

clear in patients with pneumonia. In young and middle-aged patients they usually clear in 2 to 4 weeks but may persist for up to 8 weeks. For the elderly, it will typically take longer, up to about 3 months. It may take even longer for immunocompromised patients and those with debilitating illness such as alcoholism and COPD (Webb et al., 1996; Freundlich & Bragg, 1992; Ely & Haponik, 1991; Herold, 1997). Beyond 2 to 3 days from the start of treatment the chest radiograph should not get worse, but rather show a steady improvement to resolution. It is particularly important to document complete resolution in middle-aged and elderly patients to exclude carcinoma or other structural abnormality, especially in refractory or recurrent pneumonia (Webb et al., 1996). Routinely obtaining frequent chest radiographs to follow these patients is of little benefit. Follow-up chest radiographs should only be obtained when indicated by the patient's clinical course, especially when the clinical response to treatment is not as expected.

Failure to resolve is only occasionally due to bronchogenic carcinoma and is more frequently due to the nature of the disease or to inappropriate



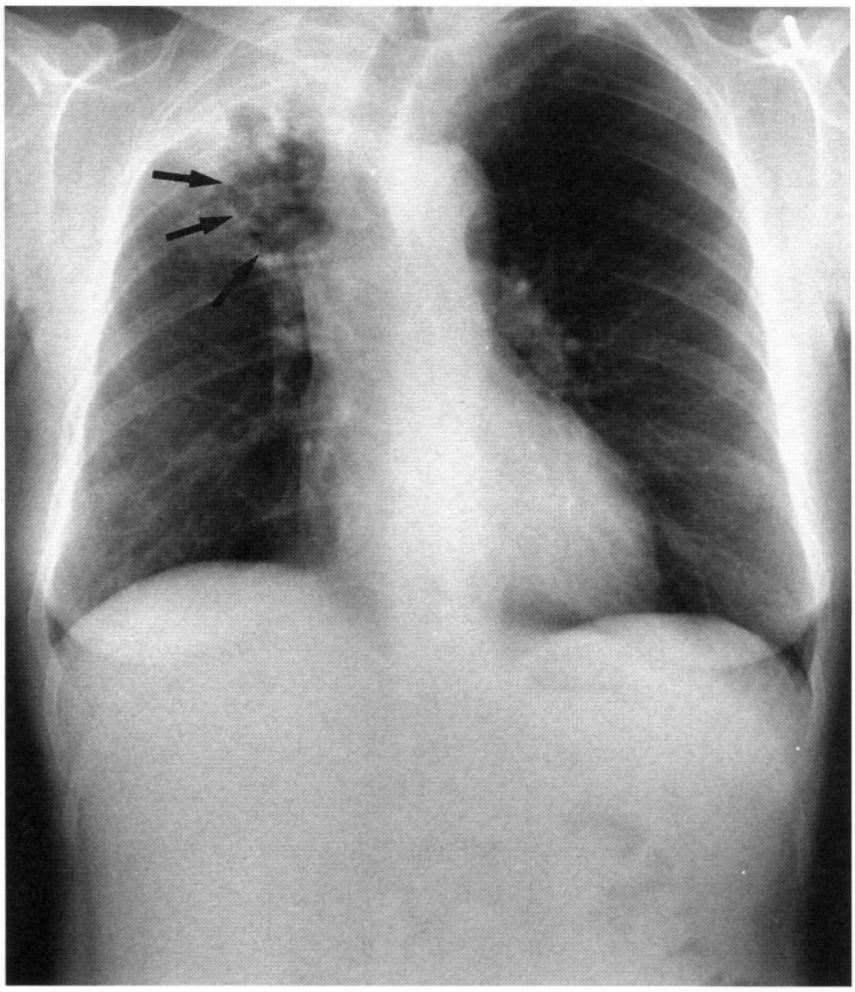
A

**FIGURE 18.** Follow-up examinations of the patient shown in Figures 10 and 11 with *Legionella pneumophila* pneumonia enlarging the right upper lobe. The examinations were obtained at 3 weeks (A) and 3 months (B). A large cavity has been revealed (arrows) indicating the extent of the pulmonary necrosis.

therapy (Woodridge, 1992). This has implications for additional imaging at the time of work-up and indicates that chest radiography is more appropriate for follow-up than other modalities. Over 90% of recurrent pneumonia is associated with predisposing factors (Woodridge, 1992). These include COPD, bronchiectasis, congestive heart failure, extrathoracic disease, or a combination of these. Recurrent pneumonia in one location is most frequently associated with a pulmonary abnormality in that location, most frequently bronchiectasis (Woodridge, 1992). Only about 1% of patients with recurrent pneumonia have bronchogenic carcinoma (Woodridge, 1992). It is therefore recommended that investigation for bronchogenic carcinoma be limited to patients with recurrent pneumonia in the same location (Woodridge, 1992).

## Additional Imaging—Other Modalities

The overall sensitivity of chest radiography for detecting pulmonary disease is approximately 80%. Up to 16% of patients with interstitial lung disease and up to 10% of immunosuppressed patients with acute lung disease will have a normal



B

FIGURE 18. (*Continued*)

chest radiograph (Webb et al., 1996). The probability that a chest radiograph will miss clinically silent pneumonia is about 5% to 7% (Ely & Haponik, 1991).

Conventional computed tomography (CCT) and high-resolution computed tomography (HRCT) are more sensitive and specific than chest radiography for detecting and diagnosing both acute and chronic lung disease.

CCT is the modality of choice for evaluating the state of the lungs and to detect underlying pathology such as tumor, necrotizing pneumonia, abscess, fluid loculations, and empyema (Yu & Maurer, 1996). CCT gives a more precise demonstration of the extent of pulmonary parenchymal involvement than is possible with a chest x-ray. CCT is better able to define the proportion of pleural versus parenchymal disease when both are present and accurately differentiates between effusion and lung opacification (Fu & Maurer, 1996). CCT is better able to demonstrate the presence and location of a pleural fluid collection and to detect loculation of the fluid and the need for thoracenteses. CCT is especially useful where empyema is known but the presence and extent of a pleural peel is not. CCT may be used to guide thoracentesis and chest tube

placement and to later assess the position of the tubes and the adequacy of drainage. Bronchoscopy with BAL may be facilitated by CCT to direct the examination to the specific branch of the airway most likely to yield useful results.

HRCT is a technique designed to assess the lung parenchyma with anatomic detail similar to gross pathology specimens (Webb et al., 1996). HRCT is better than CCT and chest radiographs for the demonstration of localized and diffuse pulmonary parenchymal disease. In selected cases HRCT may detect abnormalities when chest radiographs are normal, confusing, or equivocal (Syrjala et al., 1998). In this regard it plays an especially important role in detecting diffuse pneumonia in immunocompromised patients (Webb et al., 1996). HRCT has a sensitivity of 99% and a specificity of 93% for detecting acute lung disease in patients with AIDS (Webb et al., 1996). HRCT is also indicated for patients presenting with hemoptysis and can be used to assess the need for and the optimal site for lung biopsy (Webb et al., 1996). CCT assessment of diffuse lung disease is better than HRCT only for the detection of small nodules (Webb et al., 1996).

HRCT may suggest a specific diagnosis when pneumonia due to the organism has a distinctive appearance, for example, selected cases of PCP or cytomegalovirus pneumonia (Webb et al., 1996). HRCT may help to avoid confusion with noninfectious causes of diffuse parenchymal lung disease which mimic pneumonia on chest radiograph. A number of them may have a distinctive appearance on HRCT (Webb et al., 1996).

Ultrasound is useful for assessing the amount

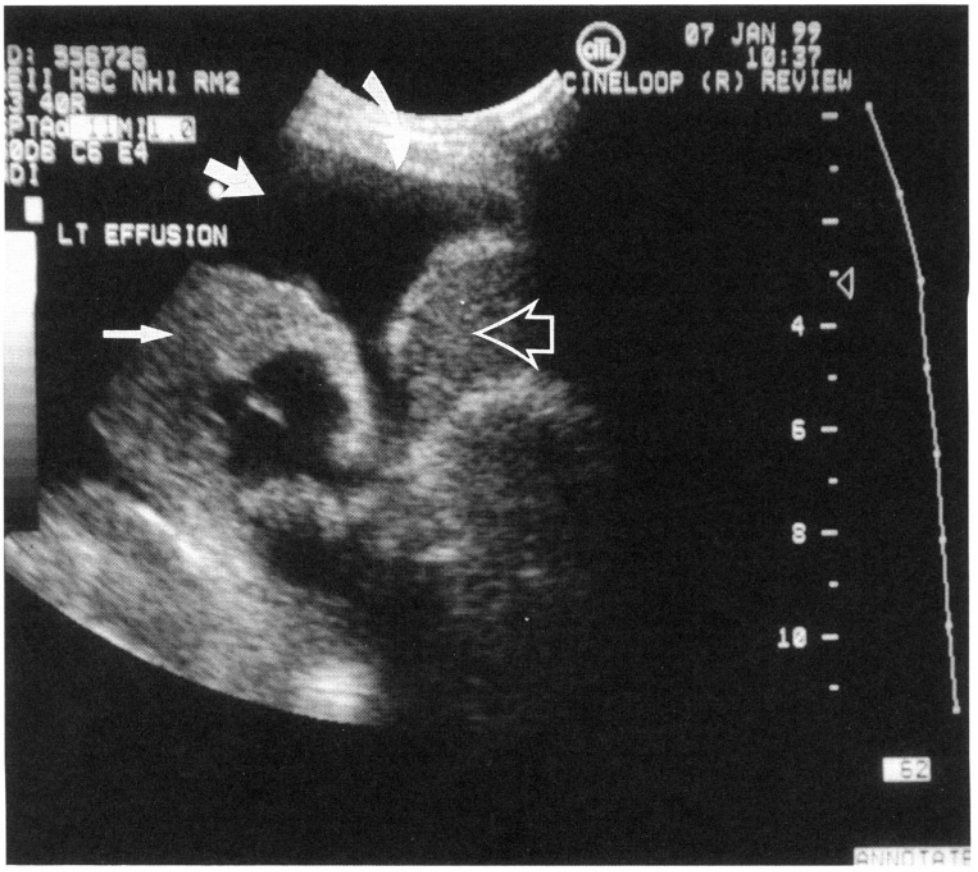



**FIGURE 19.** An ultrasound image of the left upper quadrant showing a pleural effusion (wide arrow) and the consolidated lung base (thin arrow). The open arrow indicates the spleen, the curved arrow marks the normal-thickness parietal pleura.

and location of pleural fluid (Fig. 19), detecting loculation, and for differentiating pleural fluid from pleural organization. It may also be used to guide thoracentesis and closed pleural drainage procedures (Hanna et al., 1991; Wallenhaupt, 1991). Its main advantage is that it can be brought to the bedside of a seriously ill patient when the need is urgent and movement of the patient is contraindicated.

The use of pulmonary scintigraphy in patients suspected of having acute pulmonary infection is likely to be restricted to those who are immunocompromised when chest radiography and CCT are negative or equivocal. Gallium-67 citrate scintigraphy is very sensitive for PCP and is typically positive before the chest radiograph. In PCP there is diffuse homogeneous or heterogeneous uptake of the radionuclide. Focal uptake will suggest bacterial infection and perihilar uptake will suggest cytomegalovirus infection (Conces, 1992). An indium 111-labeled autologous white blood cell scintigram may be able to detect focal purulent pulmonary infection such as an abscess when conventional imaging is unhelpful (Conces, 1992).

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