28 Environmental Lung Disorders Secondary to Inhalation of Toxic Gases, Fumes, and Aerosols

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Inhalation lung injuries comprise fewer than 1% of total occupational injuries (1,2) and among chemically caused lung injuries they occupy only second place. Inhalation lung injuries are, however, of primary importance where morbidity, mortality, and cost of treatment are concerned (3).

There are several classifications of toxic gases, fumes, and aerosols (4–6). Noxious gases, vapors, and aerosols may be classified as simple asphyxiants, chemical asphyxiants, irritants, organometabolic gases, and vapors. A practical classification into lipid-soluble and water-soluble gases, vapors, and aerosols is summarized in the literature (3,4,7,8).

Intoxication with lipid-soluble gases is caused by interaction of two factors: (a) primary damage of the alveolar and capillary endothelium with consecutive increase of the capillary permeability and accumulation of exudate in the alveoli, and (b) blockade of enzymatic systems of the lung with secondary damage of the capillary endothelium and pathologically increased capillary permeability. The combination of these two pathologic processes leads to an increase in the lung water content, and to early radiographic signs of lung edema (5,7–9). Water-soluble gases, vapors, and aerosols, in contact with the moisturized mucosa of the respiratory tract, form acids and alkali compounds that produce erosions and ulcerations. There are also mixtures of different toxic gases, vapors, and aerosols (e.g., smoke) that produce damage to the upper airways, tracheobronchial tree, lung alveoli, and capillaries.

Mechanisms of Injury

Toxic gases, fumes, and aerosols cause injury through asphyxiation, systemic toxicity, immunologic mechanisms or direct mucosal and alveolar damage (4,6).

Asphyxiation is caused by displacement of oxygen by other inhaled gases (e.g., carbon monoxide or nitrogen dioxide). Tissue asphyxia results from altered oxygen-carrying capacity of blood (e.g., carboxyhemoglobinemia after carbon monoxide inhalation, methemoglobinemia after carbon monoxide inhalation, methemoglobinemia following oxides of nitrogen poisoning, and sulfhemoglobinemia due to hydrogen sulfide intoxication). Another cause of tissue asphyxia is the poisoning of cellular respiratory enzyme systems, for example after inhalation of phosgene or hydrogen sulfide.

Systemic toxicity results from the inhalation of minute particles of oxides of metals, particularly zinc, cadmium, and nickel.

Immunologic mechanisms are induced by a variety of inhaled gases and fumes, which act not only as respiratory irritants, but also as sensitizers.

Direct injury to the mucosa of the respiratory tract is the most common mode of injury in the survivors of acute inhalation poisoning. Pathologic changes may occur at all levels of the respiratory tract. The localization of toxic injury is determined primarily by (a) the physical and chemical properties of the inhaled gas, (b) the concentration of the agent, (c) the duration of exposure, and (d) the rate and pattern of breathing of the exposed person. The highly water-soluble agents (e.g., ammonia) tend to produce maximal injury in the upper airways. Less water-soluble gases (e.g., phosgene, oxides of nitrogen) cause injury to bronchioles and alveoli (4,5). Acute exposure to high concentration of almost any gas leads to severe injury at all levels of the respiratory tract (10–12).

Lipid-Soluble Inhalation Agents Causing Primary Damage to the Lung Parenchyma

Phosgene

Phosgene, or carbonyl chloride ($COCl_2$) is a colorless gas, 100 times more toxic than carbon monoxide and about

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800 times more toxic than the equivalent amount of hydrochloric acid. Phosgene was commonly used in chemical warfare, and more than 80% of gas fatalities in the First World War were caused by phosgene. At the present time, phosgene is used in the chemical industry as an agent for direct chlorination. Cases of phosgene poisoning occur as a result of accidental leakage of containers, pipes, or valves.

Inhalation of phosgene in low concentrations causes only mild irritation of the upper respiratory tract, so that a dangerous amount may be inhaled before its presence is recognized. As phosgene reaches the lung parenchyma before it begins to act, workers proceed to work and inhale more of the noxious gas. Injuries caused by phosgene are significantly increased by physical exertion.

Phosgene inhalation is potentially lethal because of the resulting severe pulmonary edema with a decrease in the plasma volume and severe hypoxemia (13,14). There is a dose–effect relationship. Depending on the concentration of phosgene, alveolar edema may become histologically manifest only a few minutes following inhalation. Lymph drainage from the lung increases substantially (13,15). When a sufficiently large amount of fluid has collected in the lung, edema becomes apparent. At this stage, gas exchange becomes insufficient, with edema fluid gradually rising from the alveoli into the proximal segments of the respiratory tract (14).

At very high doses (e.g., 200 ppm) phosgene passes through the blood–air barrier, reaches the lung capillaries, and reacts with blood constituents, causing hemolysis that results in hematin formation in the pulmonary capillaries. Capillary circulation is stopped by obturation with erythrocyte fragments. Death occurs within a few minutes from acute cor pulmonale (14,16).

Radiologic Findings

The first radiologic signs in phosgene poisoning are recognized as a result of the increase of lung water by 30–80%. It is strongly recommended that a chest radiograph be made immediately on admission of the patient in order to provide a basis for subsequent comparison and early detection of initial signs of toxic edema (Fig. 28.1). The duration of the radiographic latent period is inversely proportional to the inhaled phosgen dose (4,7,8,17). If the suspected inhalation dose is low, a follow-up chest radiograph should be made about 8 h after inhalation. In cases with high or moderate inhalation doses, a follow-up radiograph should be made much earlier. A high kilovoltage technique does not seem to be suitable for the detection of minimal radiographic changes in these cases, and the recommended range for a chest radiograph is 50–80 kV (13,14).

Complications and Prognosis

Complications of phosgene poisoning are mostly bronchopneumonia and bronchiolitis obliterans. Rarely, mediastinal and subcutaneous emphysema as well as thromboembolic pneumonia are observed. There is a general consensus that the vast majority of phosgene poisoning cases have a good prognosis. Diller (18) demonstrated in a well-documented study that nearly all patients complain of exertional dyspnea and reduced physical fitness for several months to years after exposure to phosgene. Lung function parameters remain pathologic for several years. The impairment of pulmonary function appears to depend on preexisting chronic bronchitis and smoking habits. Also chronic inflammatory changes such as bronchiolitis, bronchiectasis, emphysema, and lung abscesses may be observed. Asthma bronchiale is a rare late complication (18).

Oxides of Nitrogen

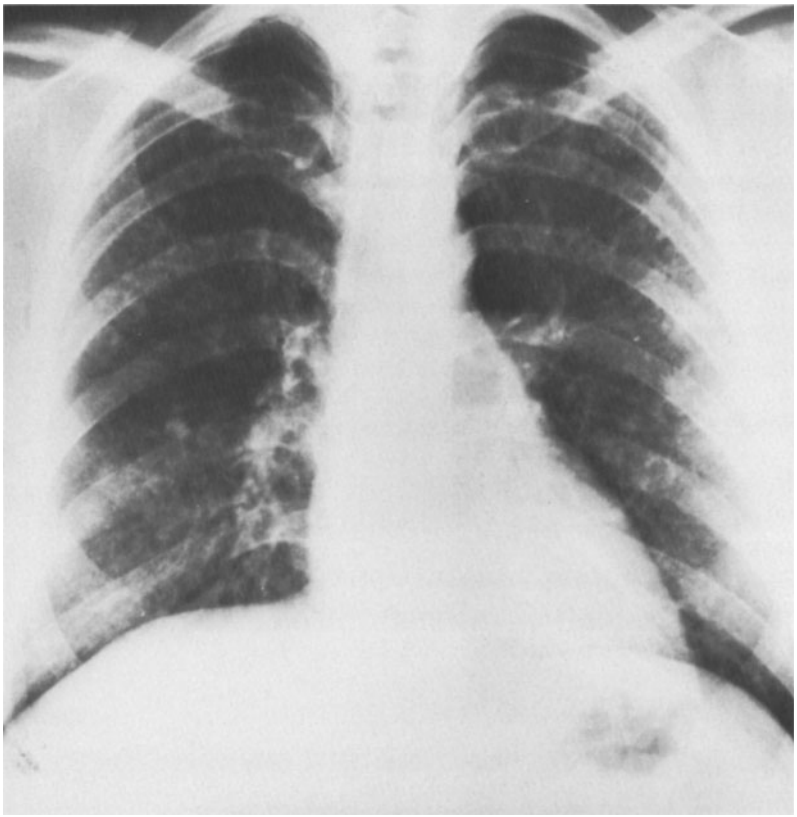
Nitrogen dioxide is one of the best-studied noxious gases (19–21). It is a poorly water-soluble, reddish-brown gas with a pungent odor. Because of its low water solubility, nitrogen dioxide is more likely to penetrate to the distal airways and alveoli (22). The low water solubility also explains the time lag from the initial exposure to the onset of significant clinical and radiologic abnormalities (19,21,23).

Acute poisoning occurs accidentally during the manufacturing and use of nitric acid (22), in the manufacturing of dyes and lacquers (24), in agriculture as a result of exposure to fresh silage (silo filler's disease) (20,21), in fire fighters as a result of combustion of nitrogenous compounds (25), and even in astronauts (26).

Sudden death may occur after inhalation of high concentrations of NO_2 because of bronchiolar spasm, laryngospasm, reflex respiratory arrest, or simple asphyxiation (27). Nitrogen dioxide in contact with moist mucous membranes forms a mixture of nitric and nitrous acid, which are very irritating and corrosive to the epithelial layer of mucosal membranes. Nitric acid dissociates in the lungs into nitrates and nitrites, which react with hemoglobin, resulting in formation of methemoglobin. Methemoglobinemia was observed following exposure to high concentration of nitrogen dioxide (27).

Clinical Symptoms and Signs

The toxic disease that follows moderate to marked exposure to oxides of nitrogen is basically triphasic. The first phase is characterized by severe breathlessness with central cyanosis and sinus tachycardia. Death may occur in this phase



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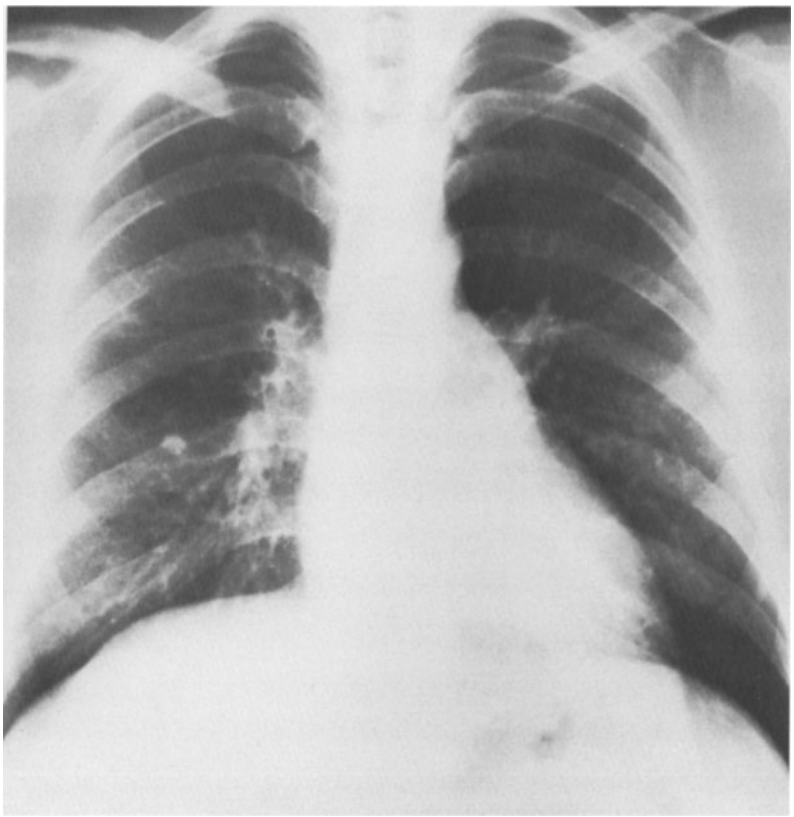

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Figure 28.1. Phosgene low dose inhalation poisoning. **a** Following a latent phase of 48 h a chest radiograph reveals hazy perihilar shadows with patchy alveolar consolidations. **b** Toxic pulmonary changes resolved mainly in the peripheral regions in 72 h.

because of acute pulmonary edema. However, the onset of pulmonary edema may be delayed for up to 36 h from the time of exposure. During the second phase, lasting for 2–5 weeks, patients have minimal dyspnea or no symptoms. In the third phase, pyrexia, recurrence of cough, breathlessness, and cyanosis are observed. Reduced carbon monoxide transfer factor and hypocapnia are also found (19). Follow-up studies in patients developing lung edema demonstrated in all cases persistent pulmonary dysfunction (27).

Radiologic Findings

Following exposure to high concentrations of oxides of nitrogen, acute pulmonary edema may occur in a short period of time or up to 24 h later, with the typical radiographic signs of perihilar haziness in the early stages and diffuse alveolar shadowing in the more severe cases (7,8) (Fig. 28.2).

The radiologic lung findings following accidental poisoning in industrial settings do not differ from lung changes in silo filler's disease (20,21,23). If there is no secondary bacterial infection, acute pulmonary edema usually resolves within a few days to a week (7,8).

Dimethyl Sulfate

Dimethyl sulfate is an organic ester. It is used as a solvent and as a methylating agent in the chemical and pharmaceutic industries. Dimethyl sulfate is highly toxic as a liquid and as a vapor. In the presence of water or moisture, dimethyl sulfate hydrolyzes readily to sulfuric acid and methyl alcohol. The toxic effects of dimethyl sulfate are comparable to those of phosgene in causing severe damage to small airways and alveoli, resulting in pulmonary edema (1,3).

The radiographic picture is mainly that of pulmonary edema, similar to that caused by poisoning with phosgene or oxides of nitrogen. Initial roentgenologic signs are perihilar shadows and fine patchy consolidation of the lungs. Alveolar pulmonary edema follows. Roentgenologic diagnosis and differential diagnosis is identical to inhalation poisoning with phosgene or oxides of nitrogen (9,10).

Water-Soluble Inhalation Agents Causing Damage to the Upper Respiratory Tract and the Tracheobronchial Tree

Chlorine Gas

Chlorine is a highly toxic irritant gas. Its characteristic sharp odor can be detected in the air at 0.1 ppm. However, irritation of mucous membranes occurs only at 1.0 ppm. The potential risk for exposure to chlorine gas is widespread, since chlorine is used or generated during many industrial processes, which include the manufacture of plastics and the

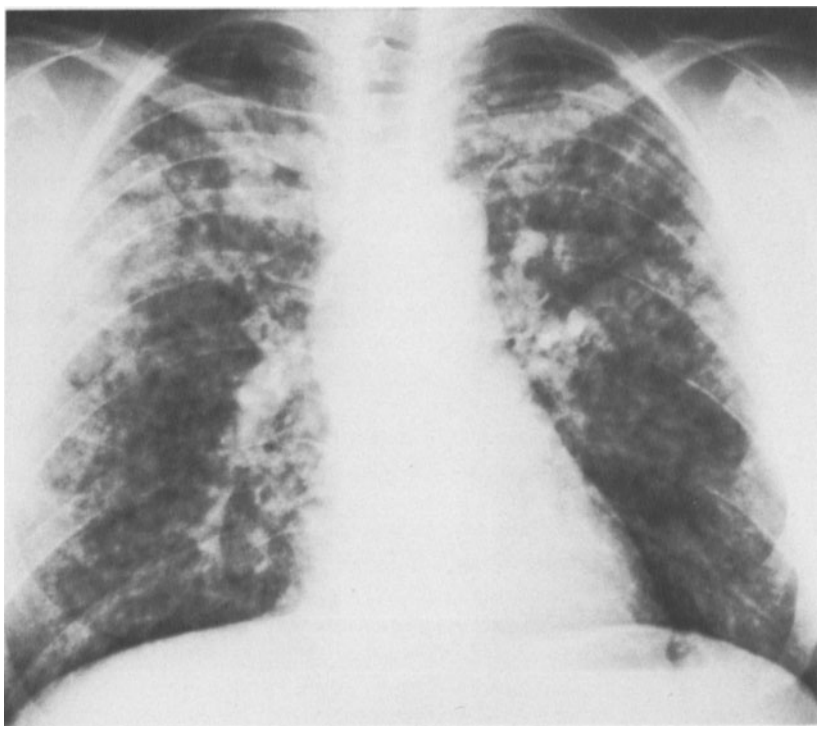


Figure 28.2. Acute pulmonary edema following inhalation of nitrogen dioxide in high concentration. Extensive involvement of both lungs by diffuse alveolar pulmonary edema 5 h after inhalation.

production of hydrochloric acid, alkali compounds, and bleaching powders. One of the major uses of chlorine has been in water purification (18,19). Exposure to chlorine gas may occur during the manufacturing process, from leakage of pipes or tanks, from accidental spillage during transportation, from mixing chlorine bleach with an acid cleaner, or as a swimming pool hazard (7,28–30).

Pathogenesis

The toxic action of chlorine gas on respiratory mucous membranes upon exposure is due to its potent oxidative properties, which lead to liberation of nascent oxygen, a protoplasmic poison, and formation of hydrochloric acid. The nascent oxygen as well as the hydrochloric acid possess severe toxic properties. Chlorine is nearly 20 times as toxic as hydrochloric acid (31).

Clinical Symptoms and Signs

Immediate symptoms of chlorine inhalation are a choking, suffocating sensation associated with acute anxiety. There is retrosternal burning pain, with burning of the nose and mouth and increased salivation.

Radiologic Findings

Radiologic findings in the initial stages of poisoning with chlorine resemble those found in "status asthmaticus": hyperinflation of the lung fields with lowered diaphragm. A preedema stage may follow when fine interstitial changes can be seen in the lung fields. Pulmonary edema usually resolves within a few days; however, severe chlorine gas poisoning with lung edema is often complicated by the development of bronchopneumonia, lung abscesses, and emphysema (32).

Chest radiology plays an important role not only in the early detection of the first signs of incipient lung edema caused by chlorine gas inhalation, but also in the demonstration of preexisting lung disease (e.g., emphysema and fibrosis), which determines the lung response to noxious gas, the duration of the toxic disease, and development of complications, as well as the prognosis (32,33).

Ammonia

Ammonia is a colorless, highly water-soluble, extremely irritant alkaline gas (34,35). Ammonia gas is widely used in the manufacturing of fertilizers, plastics, and synthetic fibers, in the production of nitric acid and explosives, in oil refining processes, and in refrigeration plants. Exposure to ammonia gas is usually due to an industrial accident. In most cases, poisoning occurs as a result of rupture or leakage of containers, fractured pipes, or valve failures. The general public may also be affected by occasional accidents involving transportation of ammonia in containers (34,36).

Ammonia gas per se is not poisonous, but, because of its high solubility in water, the toxic action takes place in the moist mucous membranes of the airways (34). Ammonia as an alkaline compound produces liquefaction of the tissue with which it comes into contact. Tissue liquefaction allows deeper penetration of the toxic agent with increased damage. Therefore ammonia burns are very often severe and may be fatal (37).

Clinical Symptoms and Signs

Ammonia gas has an immediate irritating effect on the eyes, mouth, throat, and upper respiratory tract. This primary irritation usually prompts the exposed person to escape from the dangerous zone, avoiding inhalation of higher doses of noxious gas. Symptoms include acute pharyngitis and tracheitis, hoarseness, dysphagia, respiratory distress, cyanosis, pulmonary edema, and shock (35).

Radiographic Findings

The inhaled ammonia gas directly irritates and erodes bronchial mucosa almost at all levels. Thus the chest roentgenogram initially shows slightly enlarged pulmonary markings at the hila. Also, perihilar vascular and bronchial wall prominence is found. At this stage the main lung changes are therefore interstitial. In cases of severe poisoning, alveolar edema and segmental atelectasis can develop (38).

Sulfur Dioxide

Sulfur dioxide is an intense respiratory irritant and a commonly encountered toxic gas. It is used extensively in the chemical and paper industries and in bleaching, fumigation, and preserving. It has also been widely used in the liquefied state as a refrigerant. It occurs in combustion of sulfur and in burning of coal containing sulfur.

On contact with moist mucosal surfaces sulfur dioxide is hydrated and subsequently oxidized, forming sulfuric acid, which causes severe injuries to the tracheobronchial mucosa. Acute exposure to a high concentration of sulfur dioxide results in injuries of eyes, nasopharynx, and respiratory tract. Accidental exposure to high concentrations occurs in pulp and paper factories and in refrigeration plants.

Pulmonary changes documented on chest radiographs are similar to inhalation injuries caused by other irritant gases such as chlorine or ammonia. Follow-up examinations in patients who inhaled sulfur dioxide may show signs of extensive fibrotic bronchitis and generalized bronchiectasis (39).

Hydrogen Sulfide

Hydrogen sulfide (H_2S) is a colorless gas with a powerful odor of rotten eggs. It is somewhat heavier than air and it therefore tends to accumulate in tunnels, caissons, vats, and cellars. Hydrogen sulfide is an asphyxiant as well as an irritant. It is encountered in the production of many chemicals and dyes, in the tannery and rubber industries, in petroleum refining, in mines with sulfide ores, in sewers, and in the fishing and fish meal industries (28).

Accidental exposure to concentrations of hydrogen sulfide greater than about 700 ppm causes death from respiratory failure due to depression of medullary centers before the irritant effects in the lung have time to develop (28). More prolonged exposure to concentrations of 300–600 ppm results in irritation of the mucosa of the nose, throat, and chest with cough, headache, and dizziness. Pulmonary edema follows, while absorption of hydrogen sulfide in the lungs results in respiratory paralysis due to the blockade of intracellular cytochrome oxidases.

Radiographic Manifestations

Severe intoxication with hydrogen sulfide causes diffuse pulmonary edema. The most frequent complication is bronchopneumonia (7,8).

Metal Vapors and Aerosols

Nickel Carbonyl

Nickel carbonyl ($Ni(CO)_4$) is a heavy, colorless, unstable liquid that is vaporized at room temperature. The vapor is highly toxic. The toxicity of nickel carbonyl is at least five times as great as that of carbon monoxide. Accidental exposure to nickel carbonyl vapors can occur during the nickel refining process in nickel factories. Nickel carbonyl is used as a catalyst. Nickel is also used extensively in the production of nickel-based alloys in the manufacture of steel, glass, enamels, and ceramics (28).

Nickel carbonyl causes severe toxic damage to bronchial mucosa and alveoli. The lungs are the main target of the toxic action (28,40). In the respiratory tract, nickel carbonyl breaks down, depositing nickel as a slightly soluble compound in a very fine state of subdivision over the immense respiratory surface of the lungs. This causes irriation, congestion, and edema.

An acute disorder caused by inhalation of nickel carbonyl vapors has two phases. The immediate phase consists of dizziness, slight dyspnea, nausea, vomiting, and severe headache. These symptoms disappear quickly when the exposed person reaches open air. The delayed phase is characterized by paroxysmal coughing, breathlessness, chest tightness, substernal pain, and extreme weakness. The onset of this intoxication phase may vary from 10 to 36 h, or occasionally up to 8 days (41). In fatal cases, delirium develops with death on the 4th to 12th day. The cause of death is usually edema of the lungs (41,42).

Radiographic Findings

Early radiologic signs in nickel carbonyl poisoning are widening and blurring of hilar structures and perihilar hazy shadows. In about 60% of patients patchy bilateral consolidation due to a toxic pneumonitis develops (Fig. 28.3**a**). This usually is followed by extensive pulmonary edema. As a sequela of nickel carbonyl poisoning, lung fibrosis may also develop in some patients (Fig. 28.3**b**) (7).

Zinc Chloride

Zinc chloride is employed in oil refining, in the production of dry batteries, and in galvanizing iron. It is also used in smoke bombs. Exposure to zinc chloride smoke in confined spaces may be lethal.

Zinc chloride is extremely caustic to mucous membranes because of its hygroscopic and astringent nature, leading to protein denaturation. Inhalation of zinc chloride fumes causes severe acute tracheobronchitis and a rapidly developing diffuse interstitial fibrosis, resulting in lung induration (43,44).

Chest radiography demonstrates initially a diffuse reticulonodular pattern that changes to more patchy irregular consolidation as a result of a chemical pneumonitis. This is followed by bilateral diffuse consolidation due to toxic pulmonary edema. Acute lung edema and toxic pneumonia may progress within a few weeks to diffuse interstitial pulmonary fibrosis. Among the complications, subpleural emphysematous blebs are common in zinc chloride inhalation, and may result in a pneumothorax. Also, the development of lung abscesses and generalized lung fibrosis have been reported (7,43,44).

Cadmium

Cadmium oxide and chloride dusts are soluble in body fluids. Damage to the mucous membranes of the respiratory tract causes "chemical pneumonia" with edema.

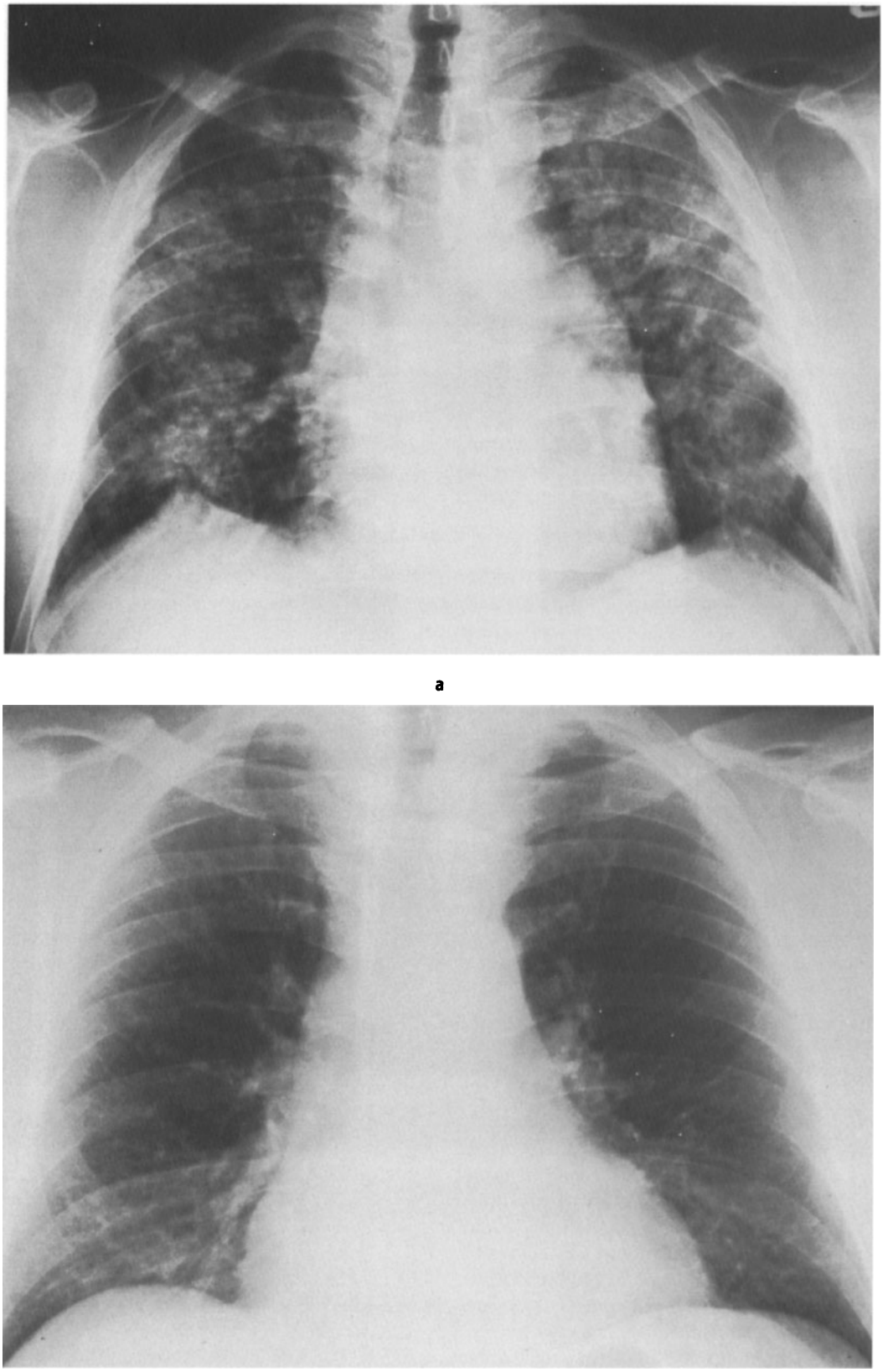


Figure 28.3. Nickel tetracarbonyl inhalation poisoning. **a** Thirteen days after inhalation, extensive patchy consolidations appear in both lung fields. **b** Signs of lung fibrosis are present 7 years after exposure.

Exposure to cadmium is especially dangerous, because concentrations of cadmium or fume dust sufficient to cause severe illness or death usually do not give rise to early warning symptoms (28,45,46).

The main radiographic feature in cadmium poisoning is pulmonary edema, which usually resolves in a week or two, but may not disappear completely for 2 or 3 months.

Cobalt

Cobalt is a silvery blue-white metal with magnetic properties. Cobalt is used for a variety of purposes in industry, medicine, and nuclear weapon production. Inhalation of particulate cobalt metal is strikingly toxic to the lungs. Poisoning results in hemorrhagic edema and obliterative bronchiolitis.

Acute pulmonary edema encountered in severe cases of cobalt inhalation shows the same typical radiographic pattern as described for toxic inhalations of other vapors. In the subacute type of poisoning, linear or ill-defined rounded opacities in the lung parenchyma with prominent hilar shadows develop within a year or less (28).

Organophosphates

Organophosphates are used as insecticides and have dozens of derivates. The most commonly used organophosphate compounds are parathion, malathion, and meoinphos. The most toxic organophosphate compounds have been stockpiled as "nerve gases" for possible use in chemical warfare and are available as powder, concentrate, and aerosol. Exposure to organophosphates is most common in agricultural workers during or shortly after spraying crops, and less common in industrial workers during manufacturing and transportation. Respiratory injuries are caused by (a) inhalation of organophosphate insecticides during spraying, (b) absorption through skin and mucous membranes, and (c) ingestion for suicide, homicide, or by mistake (acute poisoning in children).

The primary toxic effect of an organophosphate is the inhibition of the enzyme cholinesterase (47,48). As a result, a large amount of acetylcholine accumulates at nerve endings. The pulmonary edema observed in organophosphate poisoning has been attributed to the muscarinic effect of the organophosphates (48). Hypoxia may also play a role in the production of pulmonary edema by causing pulmonary capillary permeability (48).

Acute pulmonary edema induced by organophosphate poisoning can present either as diffuse pulmonary densities, basically of peripheral distribution with an enlarged cardiac shadow, or, a mixed pattern of alveolar and interstitial edema (Fig. 28.4). Pleural effusion can also be observed.

Smoke and Carbon Monoxide Inhalation

Smoke Inhalation Injury

Respiratory tract injury from smoke inhalation is one of the major causes of death in fire victims (49–52). There is a synergistic lethal relationship between body surface burns and the inhalation injury. The incidence of pulmonary complications in fire victims varies between 15% and 24%, with a mortality rate of 71–89%. The acute respiratory distress in burn patients is caused by smoke inhalation, carbon monoxide poisoning, and airway obstruction. To date, it is not possible to separate the respiratory effects of inhalation injury from the ventilatory effects of shock, massive fluid therapy, burn wound toxins, and sepsis (53).

The radiographic diagnosis of pulmonary edema in burn patients may be an early indicator of lung parenchymal injury and impending pulmonary insufficiency. Therefore the early diagnosis of pulmonary edema is crucial for the management of patients suffering from smoke inhalation (7). The initial radiologic findings appear in the first 24 h (54). Lung changes developing after 24 h are usually related to aspiration pneumonia, bacterial pneumonia, or a hemodynamic abnormality (55). The radiologic diagnosis of inhalation injury may be made at a time when results from other diagnostic tests are still equivocal or mildly abnormal, thus alerting the clinician to impending pulmonary failure. A single negative chest roentgenogram does not rule out respiratory tract damage. It is recommended therefore, to repeat chest examinations in patients with second- or third-degree flame burns of the face.

Early signs of alveolar and bronchial plugging and incipient alveolar edema are the appearance of patchy pulmonary densities. Atelectasis is suggested by the demonstration of linear densities, vascular crowding, elevation of the diaphragm, or displacement of a hilus. Atelectasis may shift from lobe to lobe or disappear according to the localized trapping of air caused by bronchial plugs. Widening of the vascular pedicle is a sign of increased circulating blood volume. Therefore the enlargement of the vascular pedicle is associated with early burn-related pulmonary edema and provides a useful radiographic predictor of this complication (56). Pulmonary edema may often be seen as a very faint haze or merely as lack of definition of the hilar and perihilar structures (54).

In general, chest radiologic findings in burn patients can be divided into three phases. In the acute intoxication phase during the first 24 h, patchy areas of chemical pneumonitis are often accompanied by diffuse pulmonary edema. In the subacute phase in the following 2–5 days, atelectasis, pulmonary microembolism, and adult respiratory distress syndrome (ARDS) develop. The relatively

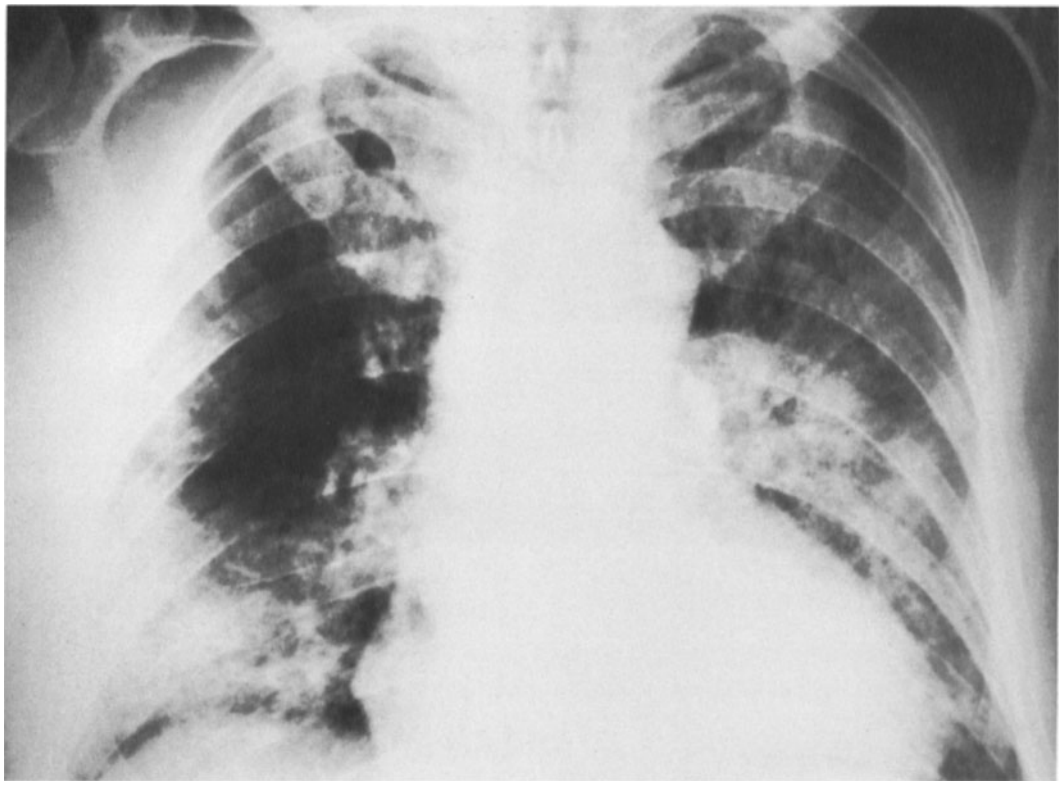


Figure 28.4. Lung changes following organophosphate poisoning with insecticide. A chest radiograph taken after hospital admission demonstrates a combination of alveolar and interstitial pulmonary edema. The heart shadow is markedly enlarged.

rapid change of size and location of pulmonary infiltrates strongly suggests the development of lung hemorrhage secondary to microembolism. In ARDS, moderate alveolar opacifications extending to the periphery of lungs without regard to lobar anatomic boundaries are demonstrated. This radiographic appearance of ARDS closely resembles diffuse pulmonary edema. However, it ordinarily does not spare the periphery of the lungs and is associated with volume loss leading to diminution of the vertical lung diameter. The third phase of radiologic findings is characterized by delayed complications such as pulmonary thromboembolism and pneumonia. Airborne pneumonia may also show alveolar densities that involve the basal areas of the lungs. Bronchiolitis obliterans, bronchostenosis, and bronchiectasis have been described as sequelae of smoke intoxication (57).

Carbon Monoxide Inhalation Injury

Smoke and carbon monoxide inhalation injuries are very often combined, but carbon monoxide intoxication has some specific characteristics. Carbon monoxide (CO) is a colorless, tasteless, and odorless gas with a specific gravity 0.97 times that of air. Carbon monoxide originates from the incomplete combustion of carbonaceous material (e.g., exhaust from automobiles, faulty heaters, mine explosives). Its toxic effects are caused by tissue hypoxia. The affinity of hemoglobin for carbon monoxide is 218 times greater than for oxygen. In addition, carbon monoxide may also have a direct toxic effect on the lung parenchyma when inhaled in high concentrations (58).

The most common finding on a chest radiograph of a patient with carbon monoxide inhalation is an increase in interstitial markings or a ground-glass appearance in both lung fields (Fig. 28.5). This pattern is attributed to interstitial edema, probably caused by a combination of tissue hypoxia and a direct toxic effect of carbon monoxide on the alveolocapillary membrane. Other findings include perihilar haze and perivascular and perihilar cuffing. Cardiac enlargement is usually encountered, suggesting myocardial damage secondary to carbon monoxide toxicity. Alveolar edema is another manifestation (58).

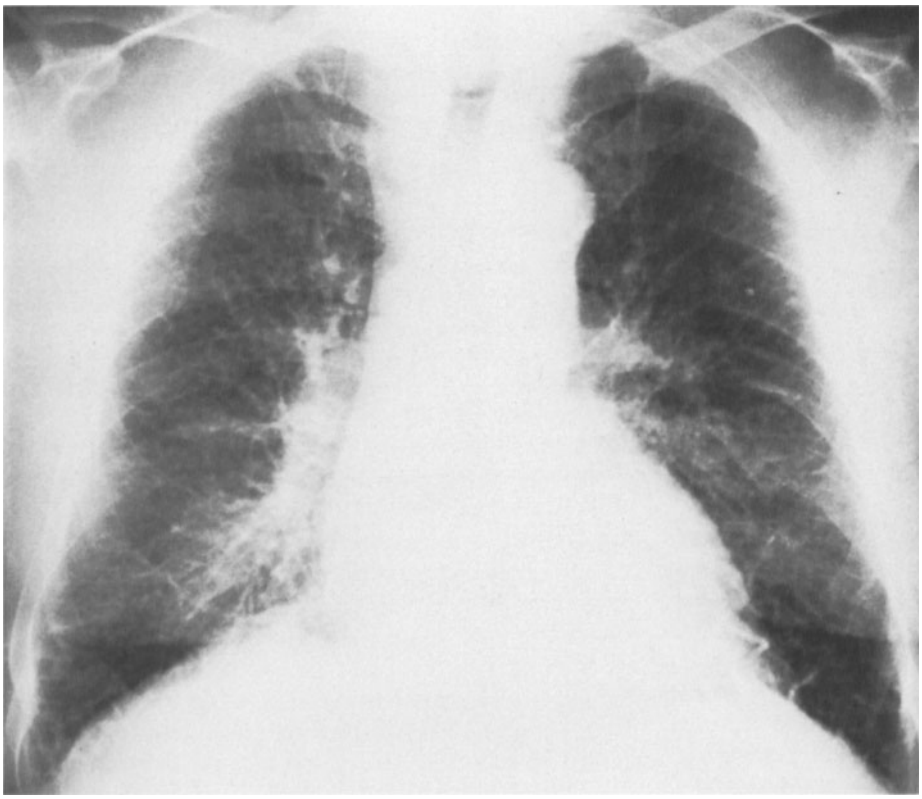


Figure 28.5. Combined inhalation intoxication with smoke, sulfur dioxide, and carbon monoxide. Bilateral increase in the interstitial markings is a sign of incipient pulmonary edema.

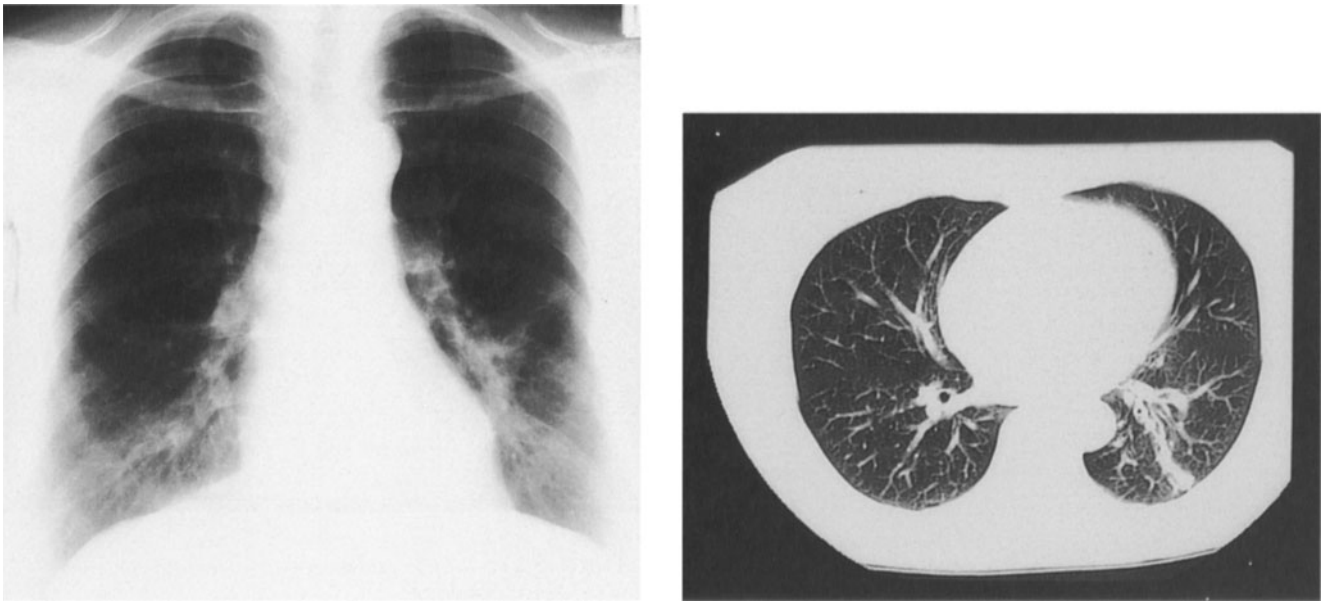
Lung Intoxication in Heavy Cigarettes Smokers ("Smoker's Lung")

Cigarette smoking is the most frequent source of indoor pollution affecting millions of people. Chest radiography remains an inexpensive method of evaluating pulmonary changes resulting from cigarette smoking (59–63). Computed tomography (CT), and especially HRCT (64–117), as well as CT with thin-slab maximum intensity projection (97) have increased the ability to identify specific abnormalities, especially in long-term, multiple packs day smokers. Most studies in these patients demonstrated proliferative lung changes, such as peribronchial thickening, interlobar thickening and ground-glass attenuation, as well as rarefication of lung vasculature and the presence of low attenuation areas (84,89,90,93,97–99,103,104) (Figs 28.6–28.9)

Conclusions

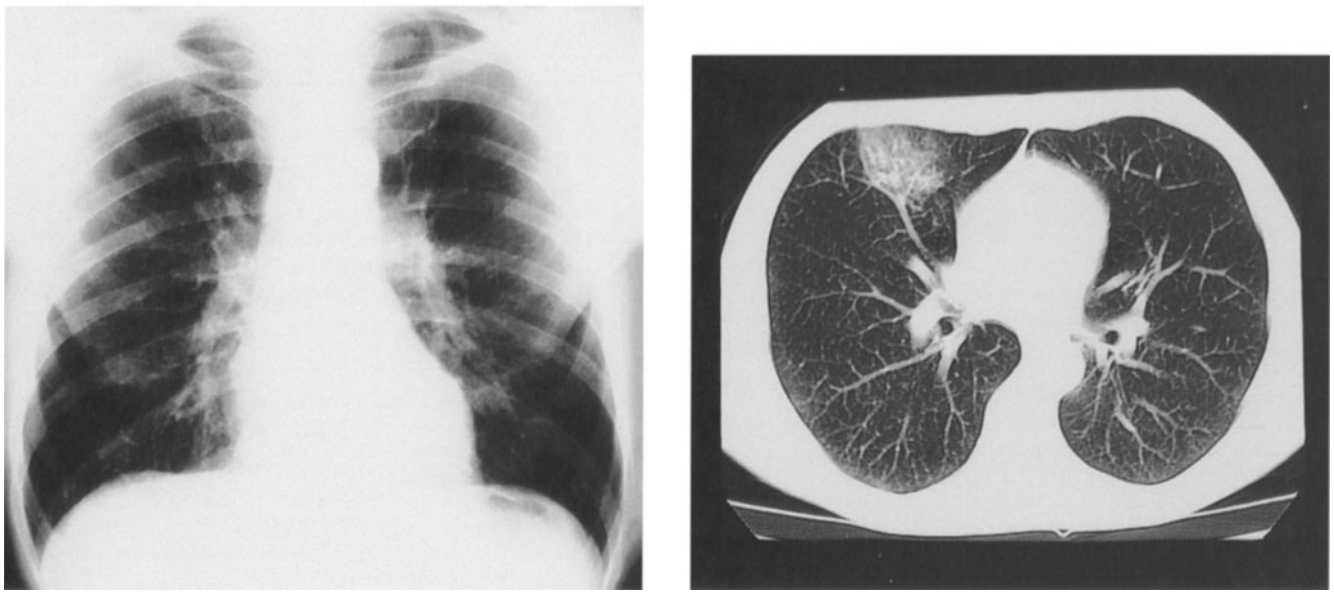
In general the radiologic input in the diagnosis and treatment of inhalation lung injuries can be summarized as follows:

1. Radiologic signs of pulmonary edema precede clinical manifestations for hours. The radiologic latent phase is inversely proportional to the dose and duration of exposure to a toxic gas. Recognition and exact interpretation of early radiographic signs of lung injury are vital in the timing of intensive care treatment.
2. Negative findings on the initial chest radiograph do not exclude respiratory injury. Repetition of chest examinations at short time intervals is therefore highly recommended. A chest radiograph should be performed also in asymptomatic patients. The lung response to an inhalation injury depends on preexisting lung disease. Complications and inhalation injury sequelae are also determined by the pulmonary status.
3. The most characteristic pattern of inhalation lung injury is pulmonary edema. Interstitial and alveolar edema, and very often a combination of both, are observed.
4. In the follow-up of patients with inhalation injuries of the lung, mostly chronic proliferative changes and lung fibrosis are observed. In such patients CT examination of the lung is the method of choice.



a b

Figure 28.6. A 60 year old female smoking 20 cigarettes per day for 20 years. She became a nonsmoker for the 10 years prior to this radiograph. Chest radiograph (**a**) and CT (**b**) show heavy peribronchial and interlobar thickening.



a b

Figure 28.7. A 59 year old male patient smoking 20 cigarettes per day for 40 years. Radiograph (**a**), and CT (**b**), show the predominance of a triad of signs such as peribronchial thickening, interlobar thickening, and ground-glass attenuation.

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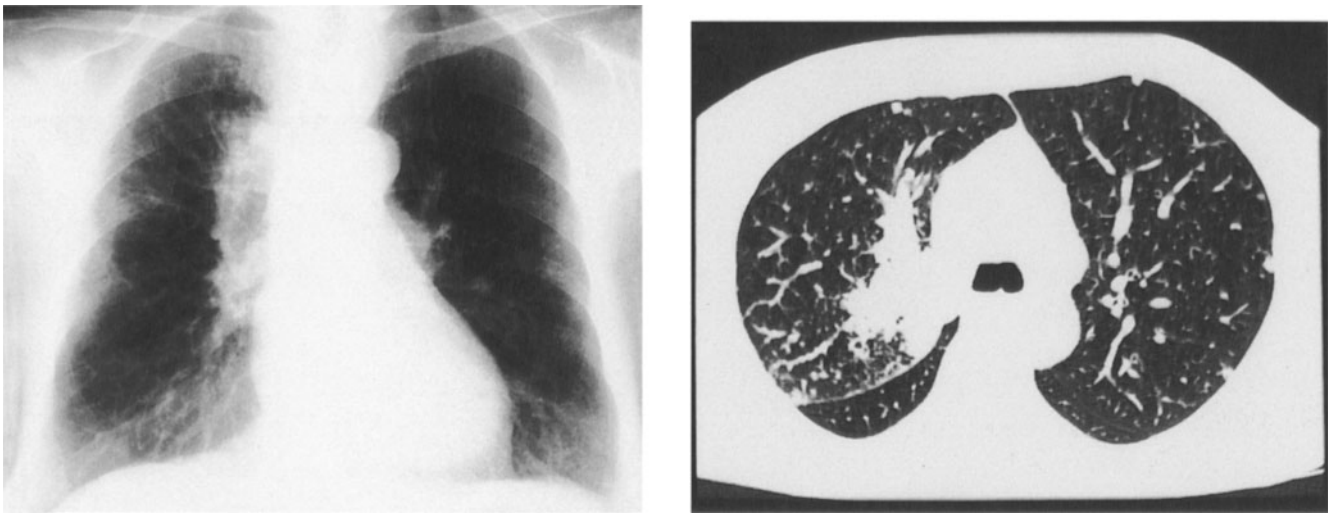

a b

Figure 28.8. A 57 year old female smoking 15–20 cigarettes per day for 27 years. Radiograph (**a**), and CT (**b**), demonstrate the proliferative lung changes classified as a cluster of CT findings: peribronchial thickening, interlobular thickening, ground-glass attenuation, and bronchial carcinoma of the right lung.

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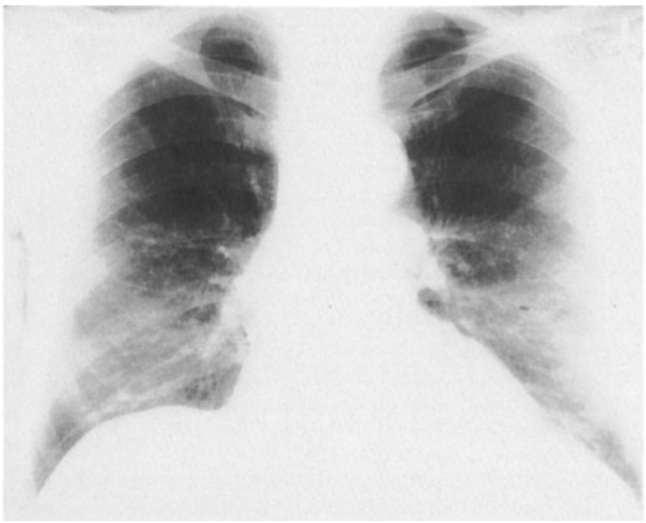
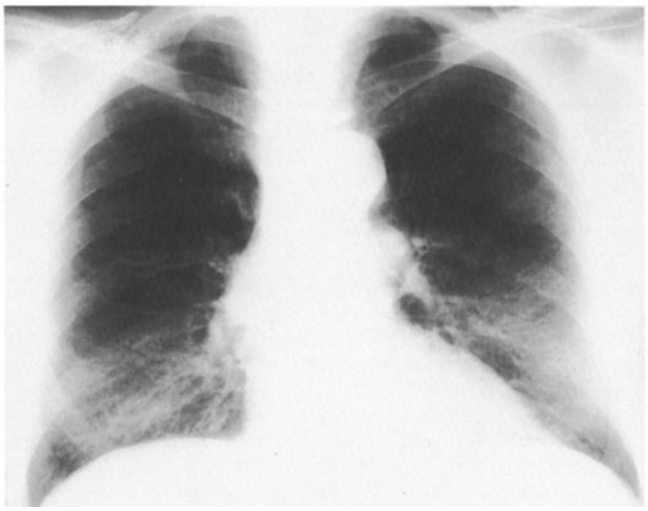
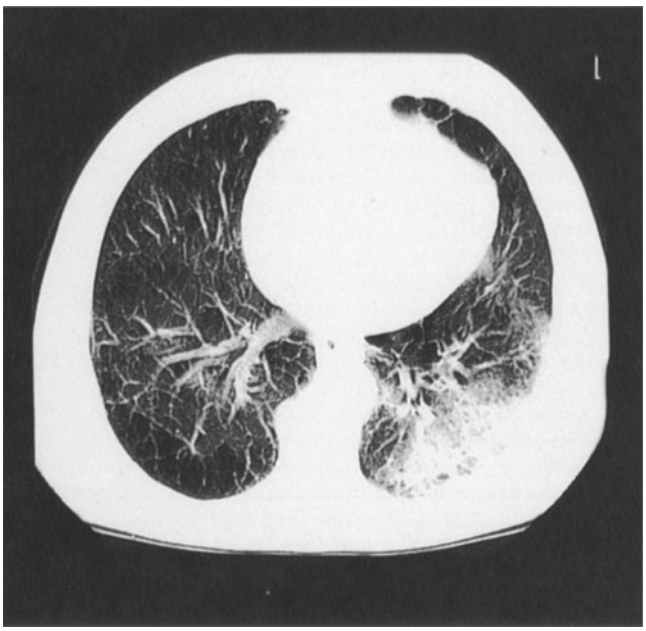


a



b



c

Figure 28.9. A 72 year old man smoking 20 cigarettes per day for 50 years. The radiograph (**a**) taken in 1988, and radiograph (**b**) taken 3 years later demonstrate the progression of proliferative lung changes. The CT (**c**) was performed 1998. It shows the further progression of proliferative lung changes as well as a bronchial carcinoma of the left lung.

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