بسم الله الرحمن الرحيم

OCCUPATIONAL LUNG DISEASE

DR.SHAFIEPOUR INTERNIST/PULMONOLOGIT

Occupational lung diseases include a wide spectrum of respiratory disorder with symptoms, signs, and diagnostic test results that often present with features similar to nonoccupational diseases

When evaluating any respiratory disease, the clinician should consider the possibility of an occupational cause or contribution

The onset of disease after an occupational exposure may occur with a short latency period, as for an acute toxic inhalation injury, or over a period of months to years, as for occupational asthma or hypersensitivity pneumonitis

interventions in the workplace may reduce or prevent disease in other workers

EPIDEMIOLOGY

No reliable figures exist for the total incidence or prevalence of occupational lung diseases, and regional variation in occupations and exposures is substantial

Work-Related Asthma

Work-related asthma includes both occupational asthma that is caused by work and asthma that is not caused by work but is exacerbated by work exposures.

SENSITIZER-INDUCED OCCUPATIONAL ASTHMA

Occupational asthma is most commonly associated with a specific immune

In most studies, higher levels of exposure are associated with higher rates of sensitization in the exposed populations, but there is no clear threshold exposure below which all workers are protected response to a high- or low-molecular-weight sensitizer from the risk of sensitization.

PATHOBIOLOGY

Genetic factors increase the risk of sensitization, but the risks appear to be polygenic and may differ for different allergens and sensitizers.

the high-molecular-weight allergens, and smoking has been reported as a risk factor for sensitization to complex platinum salts.

CLINICAL MANIFESTATIONS

Sensitizer-induced occupational asthma has a latency period ranging weeks to several years before it develops

Pulmonary function and histologic changes are similar to those in nonoccupational asthma

Sensitizer-induced occupational asthma

from a high-molecular-weight agent sensitizer typically causes a prompt asthmatic response within minutes after exposure with or without a late asthmatic response starting 4 to 6 hours after exposure. By comparison, responses to low-molecular-weight sensitizers typically start 4 to 6 hours after exposure.

DIAGNOSIS

The diagnosis of sensitizer-induced occupation asthma is clinically suspected by history and should be considered in all cases of new-onset asthma in patients who work.

In patients who are exposed to high-molecular-weight sensitizers, allergic rhinitis or conjunctivitis associated with work frequently appears before the development of asthma

Allergy skin-prick tests, blood relevant sensitizer if feasible. Serial monitoring of peak expiratory flow rates, symptom diaries, or use of rescue inhalers can provide supportive information.

end of a typical work week can help when compared with results after 10 days or more without exposure.

A comparison of eosinophil counts in induced sputum at work and after a period higher levels when exposed, provides supportive diagnostic information away from exposure

Table 72-1 Principal Agents Causing Occupational Asthma

Agent		Occupation/Industry
HIGH-MOLECULAR-WEIGHT AGENTS		
Cereals, flour	Wheat, rye, barley, buckwheat	Flour mills, bakers, pastry makers
Latex	Proteins from the Hevea tree	Health care workers, laboratory technicians
Animals	Mice, rats, cows, seafood	Laboratory workers, farmers, seafood processing
Enzymes	α-Amylase, maxatase, alcalase, papain, bromelain, pancreatin	Baking product production, bakers, detergent production, pharmaceutical industry, food industry
LOW-MOLECULAR-WEIGHT AGENTS		
Isocyanates	Toluene diisocyanate (TDI), methylene diphenyl-diisocyanate (MDI), hexamethylene diisocyanate (HDI)	Polyurethane production, plastic industry, insulation, molding, spray painting
Metals	Chromium, nickel, cobalt, platinum	Metal refinery, metal alloy production, electroplating, welding
Biocides	Formaldehyde, glutaraldehyde, quaternary ammonium compounds	Health care workers, cleaners
Persulfate salts	Hair bleach	Hairdressers
Acrylates	Cyanoacrylates, methacrylates, di- and tri-acrylates	Adhesives, dental and orthopedic materials, sculptured fingernails, printing inks, paints and coatings
Acid anhydrides	Phthalic, trimellitic, maleic, tetrachlorophthalic anhydrides	Epoxy resin workers
Reactive dyes	Reactive black 5, pyrazolone derivatives, vinyl sulphones, carmine,	Textile workers, food industry workers
Woods	Red cedar, iroko, obeche, oak, and others	Sawmill workers, carpenters, cabinet and furniture makers

TABLE 93-1 EXAMPLES OF OCCUPATIONAL RESPIRATORY DISEASES THAT COULD BE MISDIAGNOSED AS COMMON NONOCCUPATIONAL RESPIRATORY DISEASE

DISEASE THAT IS MIMICKED	POSSIBLE OCCUPATIONAL DISEASE	EXAMPLES OF SUGGESTIVE FEATURES LEADING TO A CORRECT DIAGNOSIS
Asthma	Occupational asthma from a work sensitizer	Asthma symptoms begin and are worse during a working period, with some improvement on days or weeks off work. Exposure to a high- or low-molecular-weight workplace sensitizer
	Occupational asthma—irritant-induced, including reactive airways dysfunction syndrome	Asthma begins within days after a high-level (accidental) workplace exposure
	Work-exacerbated asthma	Asthma usually began before starting the job or exposure, but severity is worse on days of work, or work exposures to expected asthma triggers or common allergens at work
COPD	Occupational COPD	Prolonged exposure at work to dusts, fumes, or gases
Pneumonia	Acute hypersensitivity pneumonitis	Symptoms typically resolve within days and recur on re-exposure to the same work trigger (e.g., metal-working fluid, moldy hay, humidifiers)
Acute viral respiratory illness or pneumonia	Humidifier fever, organic dust toxic syndrome, metal fume fever, polymer fume fever, cotton dust fever	Exposure triggers the episodes
Sarcoidosis	Chronic beryllium disease	History of exposure to beryllium dust or fumes up to 30 years or more before onset of disease
	Silicosis	History of exposure; typical radiographic findings of rounded opacities with upper lobe predominance and progressive massive fibrosis, biopsy

Idiopathic pulmonary fibrosis	Asbestosis	History of moderate or high previous asbestos exposure and appropriate latency period, often with other markers of asbestos exposure, such as radiographic evidence of pleural plaques
	Chronic hypersensitivity pneumonitis	± Work exposure to a known trigger, ± improvement during periods away from exposure
	Flock-worker's lung	Lymphocytic bronchiolitis and interstitial lung disease from nylon/synthetic textile microfibers
Idiopathic pulmonary fibrosis or hypersensitivity pneumonitis	Hard metal disease	History of exposure to hard metal (tungsten, cobalt), and histologic findings of giant cell pneumonitis on lung biopsy
Chest infections	Occupational causes of chest infections, e.g., SARS or TB in health care workers, histoplasmosis in construction workers, anthrax in wool workers or farmers	History of occupation and exposures
Pleural effusion	Asbestos-related benign pleural effusion	Previous asbestos exposure with appropriate latency; pleural plaques commonly present
Incidental pulmonary nodule	Rounded atelectasis from asbestos	Previous asbestos exposure with appropriate latency; pleural plaques commonly present
Multiple nodules	Silicosis or pneumoconiosis	History of exposure, distribution of nodules, presence of progressive massive fibrosis
Lung cancer	Occupational lung cancer	History of exposure to carcinogens at work, with an appropriate latency period (e.g., asbestos, radon, chromium)
Bronchiolitis obliterans	Popcorn lung	History of working with microwave popcorn or flavorings

COPD = chronic obstructive pulmonary disease; SARS = severe acute respiratory syndrome; TB = tuberculosis.

TABLE 93-2 COMMON CAUSES OF SENSITIZER-INDUCED OCCUPATIONAL ASTHMA

OCCUPATION	ALLERGEN BY SETTING
Bakers	Wheat, rye, fungal amylase in flour
Laboratory workers	Animal allergens, e.g., proteins in rat urine, mouse or rabbit dander
Detergent-making, medical instrument cleaning, pharmaceuticals or laboratory workers	Enzymes: e.g., *Bacillus subtilis*, pancreatic enzymes
Farmers	Grains, plant, and animal allergens; mites
Greenhouse workers and florists	Pollen, fungi, mites
Food workers	Airborne food allergens, e.g., powdered milk or eggs and vegetables
Some office workers	Fungal allergens in moldy or "sick" buildings
Health care workers	Latex allergens from gloves, glutaraldehyde, orthophthaldehyde, aerosolized medications
Factory or other industrial workers	Chemicals in spray paints, glues, polyurethane, coatings and spray insulation, adhesives
Electronic workers	Soldering flux with colophony

TREATMENT AND PREVENTION

PROGNOSIS

Outcome is best if an early diagnosis results in removal from further exposure while asthma is relatively mild. Improvement may continue to occur up to 10 years after removal from exposure, but asthma does not completely resolve in most patients.

Work-Exacerbated Asthma

EPIDEMIOLOGY AND CLINICAL MANIFESTATIONS

Work-exacerbated asthma is defined as asthma that is not caused by work but is aggravated or exacerbated by work conditions.

DIAGNOSIS AND TREATMENT

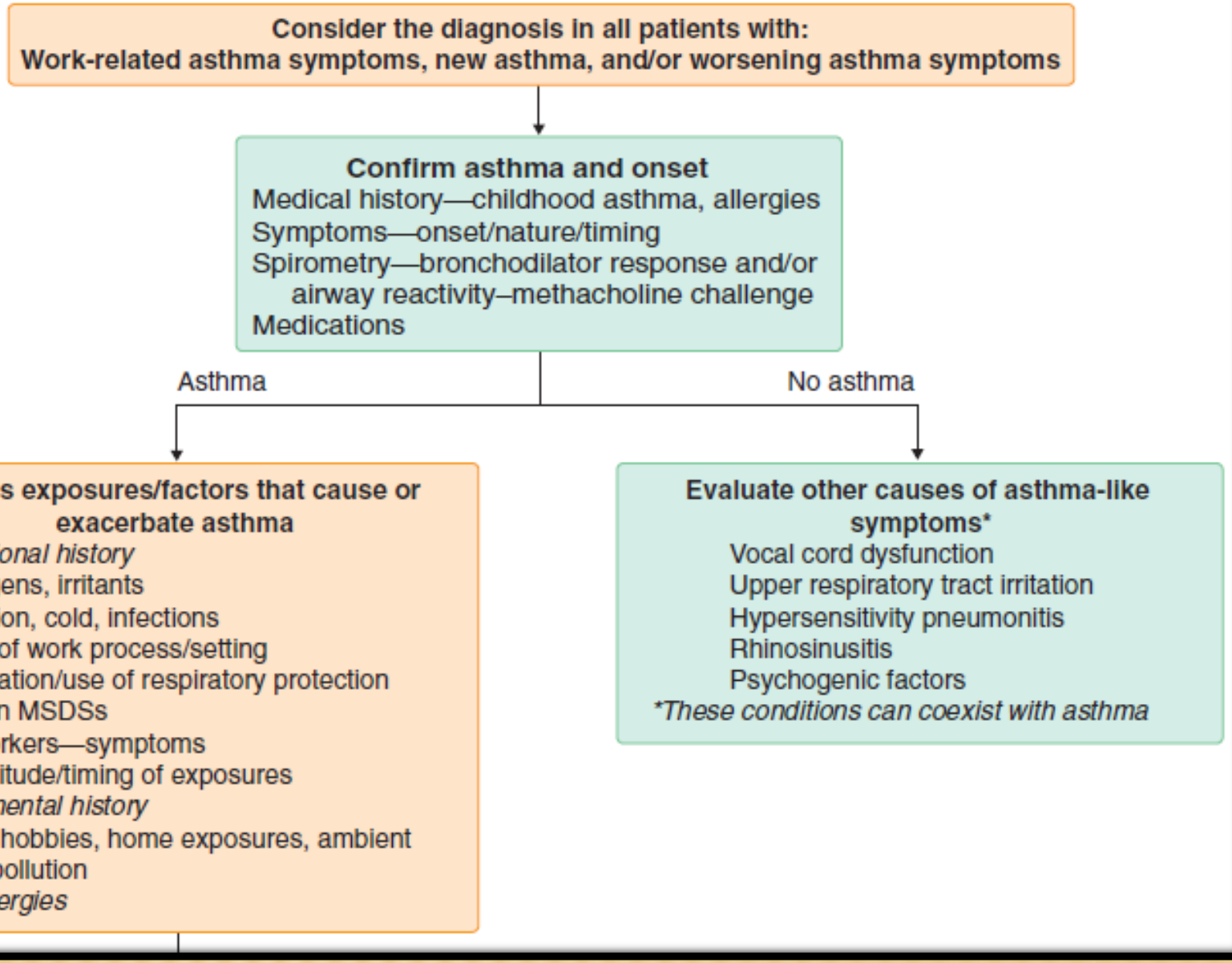
Transient work-exacerbated asthma is commonly diagnosed on the basis of the history of work exposures and the associated increase in asthmatic symptoms,medication requirements, or unscheduled physician visits.

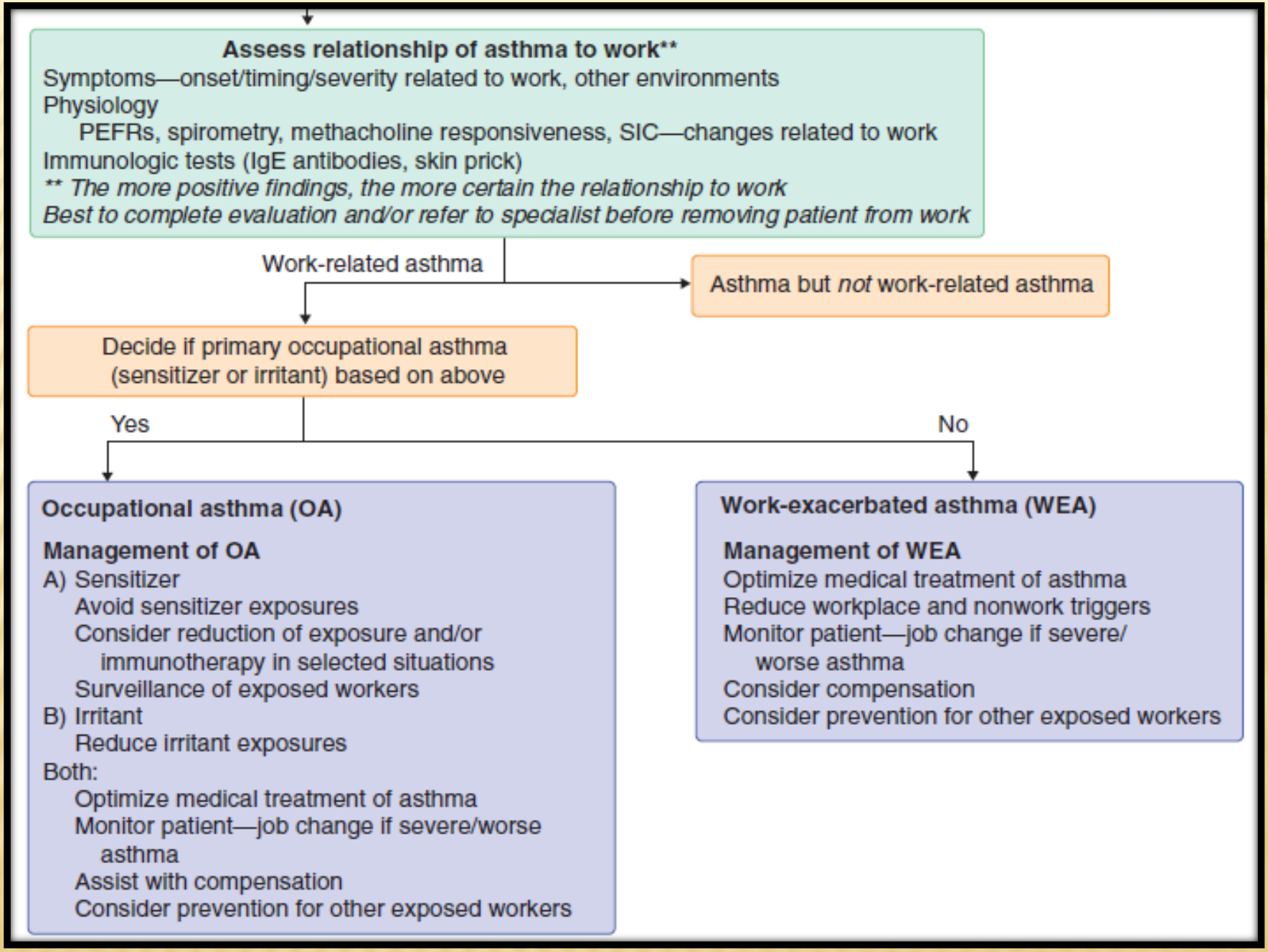
IRRITANT EXPOSURE AND REACTIVE AIRWAYS DYSFUNCTION SYNDROME

A high level of usually accidental exposure to an irritant agent can cause asthma

The most definitive criteria for this condition are those applied to the term *reactive airways dysfunction syndrome*: the onset of asthma symptoms within 24 hours of the exposure, generally severe enough to lead to an unscheduled physician visit; exposure to a single high-level irritant; asthma symptoms that persist for at least 3 months; pulmonary function testing that confirms asthma with a significant beneficial response to bronchodilators or a bronchoconstrictor response to a methacholine challenge; and the lack of preexisting lung disease or other conditions to explain the symptoms

When these criteria are not completely met (e.g., symptoms start later than 24 hours after exposure or resolve within weeks after exposure), the term *irritant-induced asthma* is commonly applied


Consider the diagnosis in all patients with:
Work-related asthma symptoms, new asthma, and/or worsening asthma symptoms
Confirm asthma and onset
Medical history—childhood asthma, allergies
Symptoms—onset/nature/timing
Spirometry—bronchodilator response and/or airway reactivity–methacholine challenge
Medications
Asthma
No asthma
Assess exposures/factors that cause or exacerbate asthma
Occupational history
Allergens, irritants
Exertion, cold, infections
Type of work process/setting
Ventilation/use of respiratory protection
Obtain MSDSs
Coworkers—symptoms
Magnitude/timing of exposures
Environmental history
Pets, hobbies, home exposures, ambient air pollution
Atopy/allergies
Evaluate other causes of asthma-like symptoms*
Vocal cord dysfunction
Upper respiratory tract irritation
Hypersensitivity pneumonitis
Rhinosinusitis
Psychogenic factors
*These conditions can coexist with asthma


Assess relationship of asthma to work**
Symptoms—onset/timing/severity related to work, other environments
Physiology
PEFRs, spirometry, methacholine responsiveness, SIC—changes related to work
Immunologic tests (IgE antibodies, skin prick)
** The more positive findings, the more certain the relationship to work
Best to complete evaluation and/or refer to specialist before removing patient from work
Work-related asthma
Asthma but not work-related asthma
Decide if primary occupational asthma (sensitizer or irritant) based on above
Yes
No
Occupational asthma (OA)
Management of OA
A) Sensitizer
Avoid sensitizer exposures
Consider reduction of exposure and/or immunotherapy in selected situations
Surveillance of exposed workers
B) Irritant
Reduce irritant exposures
Both:
Optimize medical treatment of asthma
Monitor patient—job change if severe/worse asthma
Assist with compensation
Consider prevention for other exposed workers
Work-exacerbated asthma (WEA)
Management of WEA
Optimize medical treatment of asthma
Reduce workplace and nonwork triggers
Monitor patient—job change if severe/worse asthma
Consider compensation
Consider prevention for other exposed workers

OCCUPATIONAL CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic exposure to dusts, fumes, and gases can cause occupationally induced COPD, with pathophysiologic changes essentially identical to those seen in COPD that is related to smoking

Causes include mineral dusts such as silica and organic dust exposures such as those of farmers and woodworkers; particulate matter in diesel exhaust fumes; and nitrogen oxides, ozone, and ultrafine particles in welding fumes.

HYPERSENSITIVITY PNEUMONITIS

Many exposures that lead to hypersensitivity pneumonitis occur in the workplace, and several bear the name of the occupation or job

Most antigenic exposures that lead to HP are organic, especially thermophilic actinomycetes , fungi, atypical mycobacteria , and protozoa. Other common antigens include avian and rat proteins. Less commonly, HP can be induced by low molecular-weight chemical antigens, such as penicillin or methylene diphenyl diisocyanate (MDI), which is used as a sealant or binder. Small particles, commonly 3 to 5 μm in mass median aerodynamic diameter, reach the small airways and alveoli, where the immune response leads to HP. This immune response is associated with specific IgG antibodies and T lymphocytes, and it recurs with repeated exposures.

acute form

cough,
Dyspnea
Chills
malaise

typically 4 to 8 hours after exposure and clearing by 12 to 24 hours.

On examination

febrile and tachypneic, with reduced chest expansion and basal crackles.

Neutrophilia is common

CXR:acute infiltrates.

PFT:may show a restrictive pattern, with a reduced
diffusing capacity,

ABG:hypoxemia owing to ventilation-perfusion mismatch.

Chronic HP

repeat acute episodes or start de novo.
chronic dry cough, progressive dyspnea, and often significant weight loss.

PH.EXAM

reduced chest expansion and basal crackles.

PFT and CXR may be similar to nonspecific idiopathic pulmonary fibrosis , and ground-glass opacities are often seen on a computed tomography (CT) scan of the chest.

Bronchoalveolar fluid typically shows an increase in the lymphocyte count, and there may be a predominance of CD8 T lymphocytes .

D.DX: IPF

although clubbing is less common in hypersensitivity pneumonitis. Radiographic and pulmonary function test findings may also mimic idiopathic pulmonary fibrosis, but adistinguishing finding is often a bronchoalveolar lavage that shows lymphocytes as high as 60 to 80% of the cells, usually with a predominance of CD8+ T lymphocytes, but sometimes with CD4+ cells in chronic forms of disease.

Laboratory investigations include determining the presence of serum IgG antibodies to the suspected antigen.
Some patients can safely undergo “work challenge”

Lung biopsy, if performed, may show granulomas and foreign body giant cells. If other findings are supportive of hypersensitivity pneumonitis, however, open biopsy and challenges usually are not needed.

Removal from exposure but if acute episodes are severe, they may need supportive measures, including cortico-steroids (e.g., 20 to 60 mg of prednisone orally per day), supplemental oxygen, and intensive care

CHP

oral corticosteroid treatment (e.g., 5 to 10 mg of prednisone orally per day)

severe end-stage fibrosis may lead to need for lung transplantation.

CHRONIC BERYLLIUM DISEASE

Chronic beryllium disease is a hypersensitivity disease with a strong genetic association with HLA-DPB1 gene variants that code for Glu69 and that have been identified in 83 to 97% of patients with disease. However, this gene variant occurs in 30 to 48% of the general population and, as a result, is not useful as a screening test.

TABLE 93-4 POTENTIAL EXPOSURES TO BERYLLIUM

OCCUPATIONAL EXPOSURES
Metal and alloy production (alloys of aluminum, copper, and nickel; recently includes golf clubs and metal pen clips)
Ceramic manufacturing
Metal casting, including dental technicians (crowns, bridges)
Electronics, including computer components, transistors, microwave and x-ray windows, heat sinks, telecommunications
Aerospace and atomic engineering (rocket fuels, heat shields, nose cones, and metal parts)
Aircraft manufacture and repair
Nuclear reactors, nuclear weapons and defense industry
Coating of cathode ray tubes for radar and similar installations
Laboratories
Extraction from ore
Metal reclamation and recycling
NONOCCUPATIONAL EXPOSURES
Family members exposed to dust from workers' clothing
Breakage of old fluorescent lamps (made before 1950 in North America)
Downwind exposure from industrial accidents (e.g., from a nuclear processing plant in Kazakhstan, in the former Soviet Union in 1990)

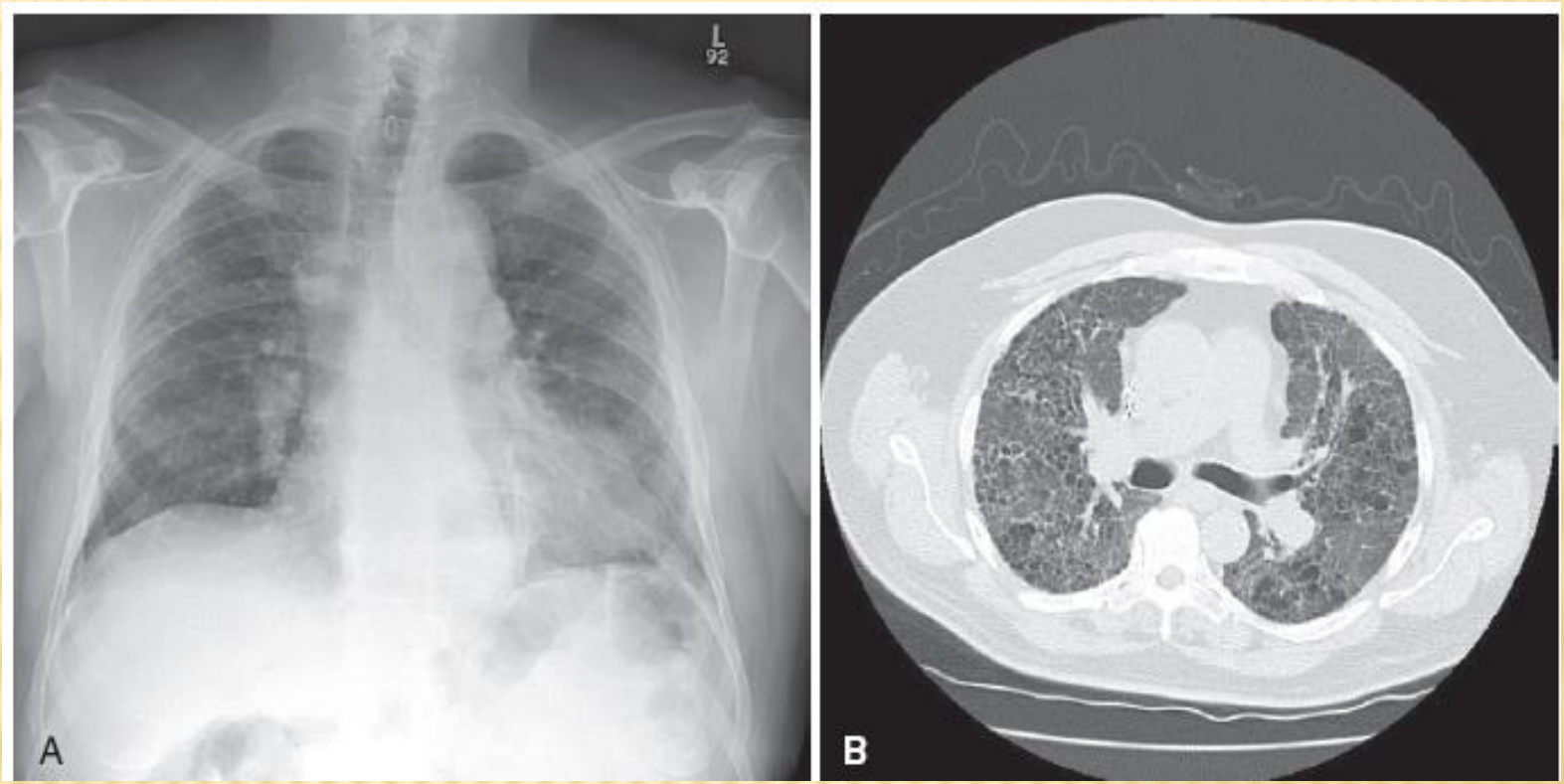
The pulmonary clinical features of chronic beryllium disease are similar to those of sarcoidosis ,ranging from asymptomatic histologic or radiographic findings, to potential progression, to severe granulomatous restrictive lung disease. Onset can occur up to 20 years or more after exposure to beryllium, even if the patient no longer is exposed. The clinical history in all patients with apparent sarcoidosis must include inquiry about possible beryllium exposure, even many years ago.

The pulmonary clinical features of chronic beryllium disease are similar to those of sarcoidosis ,ranging from asymptomatic histologic or radiographic findings, to potential progression, to severe granulomatous restrictive lung disease. Onset can occur up to 20 years or more after exposure to beryllium, even if the patient no longer is exposed. The clinical history in all patients with apparent sarcoidosis must include inquiry about possible beryllium exposure, even many years ago.

DX, TREATMENT

The CXR shows changes that appear identical to sarcoidosis with enlarged hilar or mediastinal lymph nodes or multiple lung nodules, or both .Sensitization to beryllium can be detected by a beryllium lymphocyte proliferation test that demonstrates the presence of sensitized lymphocytes in blood or bronchoalveolar lavage fluid. This test also can detect sensitization to beryllium among asymptomatic exposed workers, who can then be evaluated to assess possible chronic beryllium disease and provided with advice for reducing or eliminating further work exposures.

After disease develops, removal from exposure is advised, but the disease may still worsen. Progressive deterioration in lung function is treated similarly to sarcoidosis , with oral corticosteroids and supportive measures.


L
92
A
B

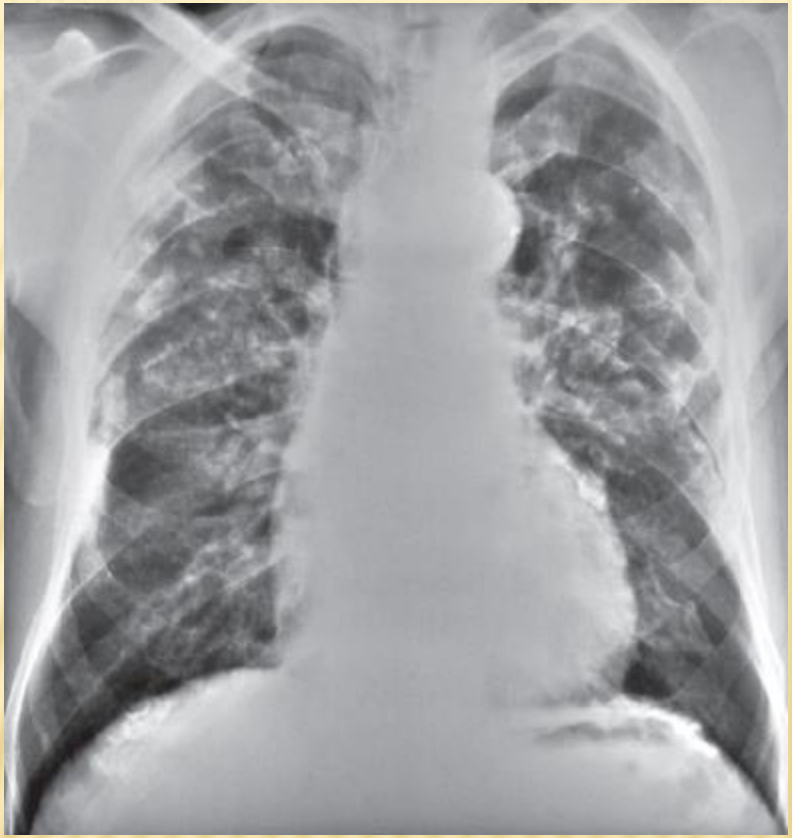
ASBESTOS-RELATED DISEASES

Although the use of asbestos has declined, and better protective equipment has been mandated, asbestos-related disease has continued to occur owing to the long latency between exposure and disease.

Effects of exposure include benign and malignant disease.

Benign asbestos disease is often asymptomatic and identified on chest imaging. Pleural thickening and pleural plaques, commonly with calcification, can occur 20 to 30 years after first exposure and may initially appear on the chest radiograph as calcified linear opacities over the hemidiaphragms and cardiac border with significant asbestos exposure.

They generally do not cause significant changes in lung function, except diffuse pleural thickening may result in exertional dyspnea and extrapulmonary restrictive lung disease.



Pleural thickening may cause rounded atelectasis when encasement of a portion of the peripheral lung tissue by thickened pleura causes an apparent lung nodule, typically with a “comet sign” showing the thickened pleura.

Benign pleural effusion can develop, typically about 10 to 15 years after asbestos exposure. It requires further investigation because the differential diagnosis includes malignant pleural effusion .

Asbestosis is the term for interstitial lung disease caused by asbestos.
The clinical presentation is usually with dry cough and dyspnea on exertion.
PH.EXAM:clubbing and basal crackles
CXR:basal interstitial lung disease, with or without additional pleural changes
PFT:shows restrictive lung disease

histologic findings are the same as in usual interstitial pneumonia .
Findings supporting the diagnosis of asbestosis rather than usual interstitial pneumonia include a significant duration and level of exposure to asbestos, an appropriate latency of usually 20 to 40 years after first exposure, and the finding of ferruginous asbestos bodies in sputum or lung tissue .

Unfortunately, pharmacologic treatment is not effective, and the lung disease may progress to end-stage fibrosis. Management is supportive, including supplemental

Mesothelioma

a malignant tumor of the pleura, peritoneum, or both, is the one complication of asbestos exposure that can occur after even relatively minor exposure, such as second-hand exposure from dust on clothing in the families of those working with exposure.

It typically occurs 30 to 40 years after exposure to asbestos and may present incidentally on chest imaging or with chest pain or weight loss.

Radiographs show pleural thickening, and a pleural effusion may be present.

Mesothelioma often is difficult to distinguish from benign pleural thickening without a biopsy. No treatment has proved effective so routine screening to detect mesothelioma in exposed persons is not currently recommended.
The risk of lung cancer increases after significant exposure to asbestos, with a usual latency period of 20 to 30 years. Smoking and asbestos exposure have multiplicative effects on the risk of lung cancer.

SILICOSIS AND OTHER PNEUMOCONIOSES

There is an association between silicosis and the development of collagen-vascular disease, especially rheumatoid arthritis. Patients with pneumoconiosis and rheumatoid arthritis may be at higher risk of developing rheumatoid nodules in the lung, so-called Caplan's syndrome, and mycobacterial infections. Patients may initially be identified incidentally during a medical surveillance program or by a chest radiograph that shows multiple small lung nodules, often with enlarged mediastinal lymph nodes that can mimic sarcoidosis

Nodules can coalesce and lead to progressive massive fibrosis, especially in the upper lungs, with compensatory emphysema in the lower lung fields. On chest imaging, mediastinal lymph nodes may have a characteristic “eggshell” calcification in silicosis.

Treatment is supportive.
Patients with exposure to silica or coal dust may develop COPD from the dust exposure. Patients who develop end-stage lung disease may be considered for lung transplantation.

TABLE 93-5 JOBS THAT CAN LEAD TO SILICOSIS

Mining: surface or underground mining (tunneling)
Milling: ground silica for abrasives and filler
Quarrying
Sandblasting: e.g., of buildings, preparing steel for painting
Pottery; ceramic or clay work
Grinding, polishing using silica wheels
Stone work
Foundry work: grinding, molding, chipping
Refractory brick work
Glass making: to polish and as an abrasive
Boiler work: cleaning boilers
Manufacture of abrasives

Coal Worker's Pneumoconiosis (CWP)

Occupational exposure to *coal dust* can lead to CWP, which has enormous social, economic, and medical significance in every nation in which coal mining is an important industry. Simple radiographically identified CWP is seen in ~10% of all coal miners and in as many as 50% of anthracite miners with more than 20 years' work on the coal face. The prevalence of disease is lower in workers in bituminous coal mines.

With prolonged exposure to coal dust (i.e., 15–20 years), small, rounded opacities similar to those of silicosis may develop. As in silicosis, the presence of these nodules (*simple CWP*) is not usually associated with pulmonary impairment. Much of the symptomatology associated with simple CWP appears to be due to the effects of coal dust on the development of chronic bronchitis and COPD .The effects of coal dust are additive to those of cigarette smoking.

Complicated CWP is manifested by the appearance on the chest radiograph of nodules ranging from 1 cm in diameter to the size of an entire lobe, generally confined to the upper half of the lungs. As in silicosis, this condition can progress to PMF which is accompanied by severe lung function deficits and associated with premature mortality. *Caplan's syndrome* ,first described in coal miners but subsequently found in patients with silicosis, includes seropositive rheumatoid arthritis with characteristic pneumoconiotic nodules. Silica has immunoadjuvant properties and is often present in anthracitic coal dust.

ACUTE FEBRILE SYNDROMES

A variety of occupational exposures can cause acute febrile respiratory syndromes that may mimic acute viral respiratory illnesses.The mechanism of these syndromes is incompletely understood, but they are associated with systemic neutrophilia and cytokine activation, often with increased interleukin-6 (IL-6) and IL-8.

Typically, chills, fever, malaise, dry cough, and chest tightness start about 6 to 8 hours after onset of an exposure at work and generally resolve by the next day. Occasionally, shortness of breath and other respiratory symptoms are severe enough for patients to seek emergency medical attention. Infiltrates on the chest radiograph can occur with neutrophilia and hypoxemia that can mimic acute pneumonia or acute hypersensitivity pneumonitis.

Symptoms and signs generally resolve in 24 to 48 hours without antibiotics and recur with further exposures, although the clinical manifestations generally become milder with repeated daily exposures (e.g., Monday morning fever in cotton mill workers). Workers are often familiar with the syndrome because it commonly affects up to 30% of exposed workers. If the diagnosis is not provided by the patient, however, careful elicitation of potential work exposures is needed.

TABLE 93-6 OCCUPATIONAL CAUSES OF AN ACUTE FEBRILE SYNDROME

SYNDROME	CAUSE
Polymer fume fever or Teflon fever	Polytetrafluoroethylene and other fluorocarbon polymer fumes
Metal fume fever	Zinc fumes from welding of galvanized steel, less commonly other metal fumes
Cotton mill fever	Dust and endotoxins from bacterial contamination of unprocessed cotton, flax, and hemp
Humidifier fever	Microorganisms found in reservoirs, e.g., humidifiers, air conditioners, aquariums
Organic dust toxic syndrome	Grain dust, moldy wood chips

Treatment is supportive.

OCCUPATIONAL LUNG CANCER

A significant duration and level of exposure to a recognized carcinogen such as asbestos, hexavalent chromium (as in chromate production and the pigment industry), soluble radon compounds or radon gas, polycyclic aromatic hydrocarbons, chloromethyl ethers, arsenic, or silica can increase the risk of lung cancer

Organic Dusts

Cotton Dust (Byssinosis)
Grain Dust
Farmer's Lung

Byssinosis is characterized clinically as occasional (early stage) and then regular (late stage) chest tightness toward the end of the first day of the workweek ("Monday chest tightness"). In epidemiologic studies, depending on the level of exposure via the carding room air, up to 80% of employees may show a significant drop in their FEV_1 over the course of a Monday shift.

Initially the symptoms do not recur on subsequent days of the week. However, in 10–25% of workers, the disease may be progressive, with chest tightness recurring or persisting throughout the workweek. After >10 years of exposure, workers with recurrent symptoms are more likely to have an obstructive pattern on pulmonary function testing. The highest grades of impairment are generally seen in smokers.

Carbon Monoxide Poisoning

Carbon monoxide is a colorless, odorless gas produced by the combustion of carbon-based fuels. Because of the ubiquity of these substances, carbon monoxide inhalation is often coincident with smoke inhalation in fires or may occur accidentally in association with malfunctioning equipment or improper venting of emissions from heaters, stoves, combustion motors, or other similar devices. In addition, intentional inhalation of carbon monoxide is a method commonly used in suicide attempts . Carbon monoxide inhalation is the leading cause of death from poisoning worldwide.

Pathobiology :Carbon monoxide readily diffuses across the alveolar-capillary interface and binds to hemoglobin with extremely high affinity. When the resulting carboxyhemoglobin molecule undergoes an allosteric change at oxygen-binding sites, the ability of bound oxygen to dissociate and to be delivered to peripheral tissues is greatly reduced. This tissue hypoxia can cause severe functional impairment and ischemic injury of oxygen-sensitive tissues, particularly in the brain and heart.

Clinical manifestation:

Mild carbon monoxide intoxication may go unrecognized because the symptoms are nonspecific and may include headache, nausea, malaise, fatigue, and dizziness. With more severe intoxications, neuropsychiatric symptoms may range from minor disturbances in attention and cognition to agitation, confusion, hallucination, or, in the worst intoxications, seizures or frank coma.

Physical findings, which are generally nonspecific, can include tachycardia or hyperthermia. The classic cherry-red skin thought to be associated with carbon monoxide intoxication is rarely seen. Other manifestations of severe intoxications may include lactic acidosis, cardiac dysfunction with arrhythmia or ischemia, pulmonary edema, and rhabdomyolysis.

DX:

A high index of suspicion is required for diagnosis because clinical findings are nonspecific. All patients known to have been involved in fires, suicide attempts, or other scenarios compatible with exposures should have arterial carboxyhemoglobin levels checked by co-oximetry. Although levels do not correlate well with clinical findings or risk for complications, symptoms generally occur at carboxyhemoglobin concentrations of 10% or higher.

TREATMENT:

All patients should be treated with 100% supplemental oxygen, which competes with carbon monoxide for hemoglobin-binding sites and gradually eliminates it from the blood. If patients require mechanical ventilation

Toxic Chemicals

Exposure to toxic chemicals affecting the lung generally involves gases and vapors. A common accident is one in which the victim is trapped in a confined space where the chemicals have accumulated to toxic levels. In addition to the specific toxic effects of the chemical, the victim will often sustain considerable anoxia, which can play a dominant role in determining whether the individual survives.

World Trade Center Disaster

Possibly because of the high alkalinity of WTC dust significant cough, wheeze, and phlegm production occurred among firefighters and clean-up crews. New cough and wheeze syndromes also occurred among local residents

Outdoor Air Pollution

Primary standards regulated by the Environmental Protection Agency (EPA) designed to protect the public health with an adequate margin of safety exist for sulfur dioxide, particulates matter, nitrogen dioxide, ozone, lead, and carbon monoxide. Standards for each of these pollutants are updated regularly through an extensive review process conducted by the EPA. (For details on current standards—*http://www.epa.gov/air/criteria.html*.)

For example, reducing agents, such as sulfur dioxide and particulate matter from a power plant stack, may react in air to produce acid sulfates and aerosols, which can be transported long distances in the atmosphere. Oxidizing substances, such as oxides of nitrogen and oxidants from automobile exhaust, may react with sunlight to produce ozone. In addition, cohort studies comparing cities that have relatively high levels of particulate exposures with less polluted communities suggest excess morbidity and mortality from cardiorespiratory conditions in long-term residents of the former.

Indoor Exposures

Environmental tobacco smoke ,radon gas, wood smoke, and other biologic agents generated indoors need to be considered. Several studies have shown that the respirable particulate load in any household is directly proportional to the number of cigarette smokers living in the home.

Other indoor exposures associated with an increased risk of atopy and asthma include those to such specific recognized putative biologic agents as cockroach antigen, dust mites, and pet danders. Other indoor chemical agents include formaldehyde, perfumes, and latex particles.
Nonspecific responses associated with "tight-building syndrome," in which no particular agent has been implicated, have included a wide variety of complaints, among them respiratory symptoms that are relieved only by avoiding exposure in the building in question.

TABLE 311-2 SELECTED COMMON TOXIC CHEMICAL AGENTS THAT AFFECT THE LUNG

Agent(s)	Selected Exposures	Acute Effects from High or Accidental Exposure	Chronic Effects from Relatively Low Exposure
Acid anhydrides	Manufacture of resin esters, polyester resins, thermoactivated adhesives	Nasal irritation, cough	Asthma, chronic bronchitis, hypersensitivity pneumonitis
Acid fumes: H_2SO_4, HNO_3	Manufacture of fertilizers, chlorinated organic compounds, dyes, explosives, rubber products, metal etching, plastics	Mucous membrane irritation, followed by chemical pneumonitis 2–3 days later	Bronchitis and suggestion of mildly reduced pulmonary function in children with lifelong residential exposure to high levels
Acrolein and other aldehydes	By-product of burning plastics, woods, tobacco smoke	Mucous membrane irritant, decrease in lung function	Upper respiratory tract irritation
Ammonia	Refrigeration; petroleum refining; manufacture of fertilizers, explosives, plastics, and other chemicals	Same as for acid fumes, but bronchiectasis also has been reported	Upper respiratory tract irritation, chronic bronchitis
Cadmium fumes	Smelting, soldering, battery production	Mucous membrane irritant, acute respiratory distress syndrome (ARDS)	Chronic obstructive pulmonary disease (COPD)
Formaldehyde	Manufacture of resins, leathers, rubber, metals, and woods; laboratory workers, embalmers; emission from urethane foam insulation	Same as for acid fumes	Nasopharyngeal cancer
Halides and acid salts (Cl, Br, F)	Bleaching in pulp, paper, textile industry; manufacture of chemical compounds; synthetic rubber, plastics, disinfectant, rocket fuel, gasoline	Mucous membrane irritation, pulmonary edema; possible reduced forced vital capacity (FVC) 1–2 years after exposure	Upper respiratory tract irritation, epistaxis, tracheobronchitis

Hydrogen sulfide	By-product of many industrial processes, oil, other petroleum processes and storage	Increase in respiratory rate followed by respiratory arrest, lactic acidosis, pulmonary edema, death	Conjunctival irritation, chronic bronchitis, recurrent pneumonitis
Isocyanates (TDI, HDI, MDI)	Production of polyurethane foams, plastics, adhesives, surface coatings	Mucous membrane irritation, dyspnea, cough, wheeze, pulmonary edema	Upper respiratory tract irritation, cough, asthma, hypersensitivity pneumonitis, reduced lung function
Nitrogen dioxide	Silage, metal etching, explosives, rocket fuels, welding, by-product of burning fossil fuels	Cough, dyspnea, pulmonary edema may be delayed 4–12 h; possible result from acute exposure: bronchiolitis obliterans in 2–6 weeks	Emphysema in animals, ? chronic bronchitis, associated with reduced lung function in children with lifelong residential exposure
Ozone	Arc welding, flour bleaching, deodorizing, emissions from copying equipment, photochemical air pollutant	Mucous membrane irritant, pulmonary hemorrhage and edema, reduced pulmonary function transiently in children and adults, and increased hospitalization with exposure to summer haze	Excess cardiopulmonary mortality rates
Phosgene	Organic compound, metallurgy, volatilization of chlorine-containing compounds	Delayed onset of bronchiolitis and pulmonary edema	Chronic bronchitis
Sulfur dioxide	Manufacture of sulfuric acid, bleaches, coating of nonferrous metals, food processing, refrigerant, burning of fossil fuels, wood pulp industry	Mucous membrane irritant, epistaxis, bronchospasm (especially in people with asthma)	Chronic bronchitis

Abbreviations: HDI, hexamethylene diisocyanate; MDI, methylene diphenyl diisocyanate; TDI, toluene diisocyanate

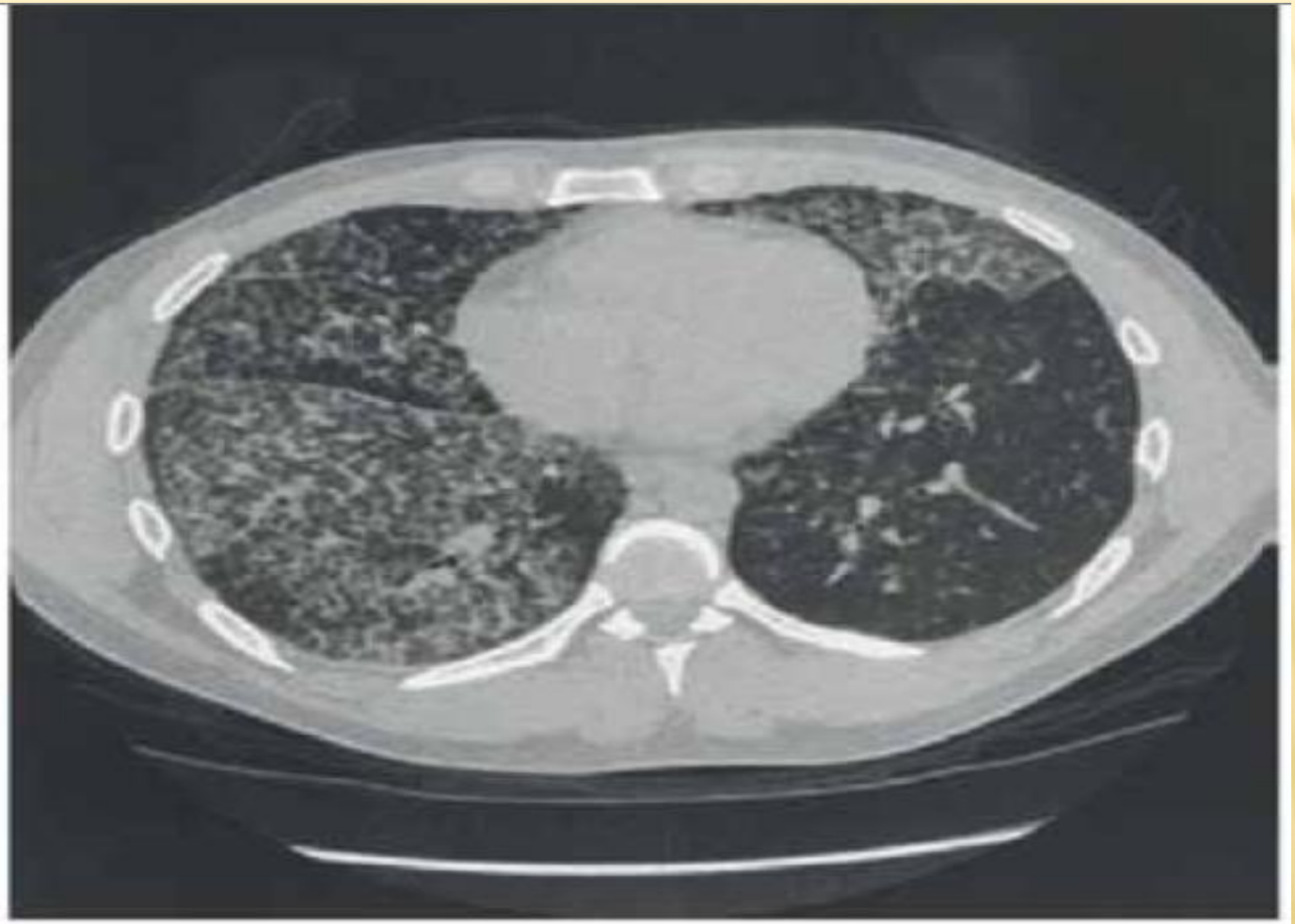


FIGURE 311-2 Acute silicosis. This high-resolution computed tomography scan shows multiple small nodules consistent with silicosis but also diffuse ground-glass densities with thickened intralobular and interlobular septa producing polygonal shapes. This has been referred to as "crazy paving."

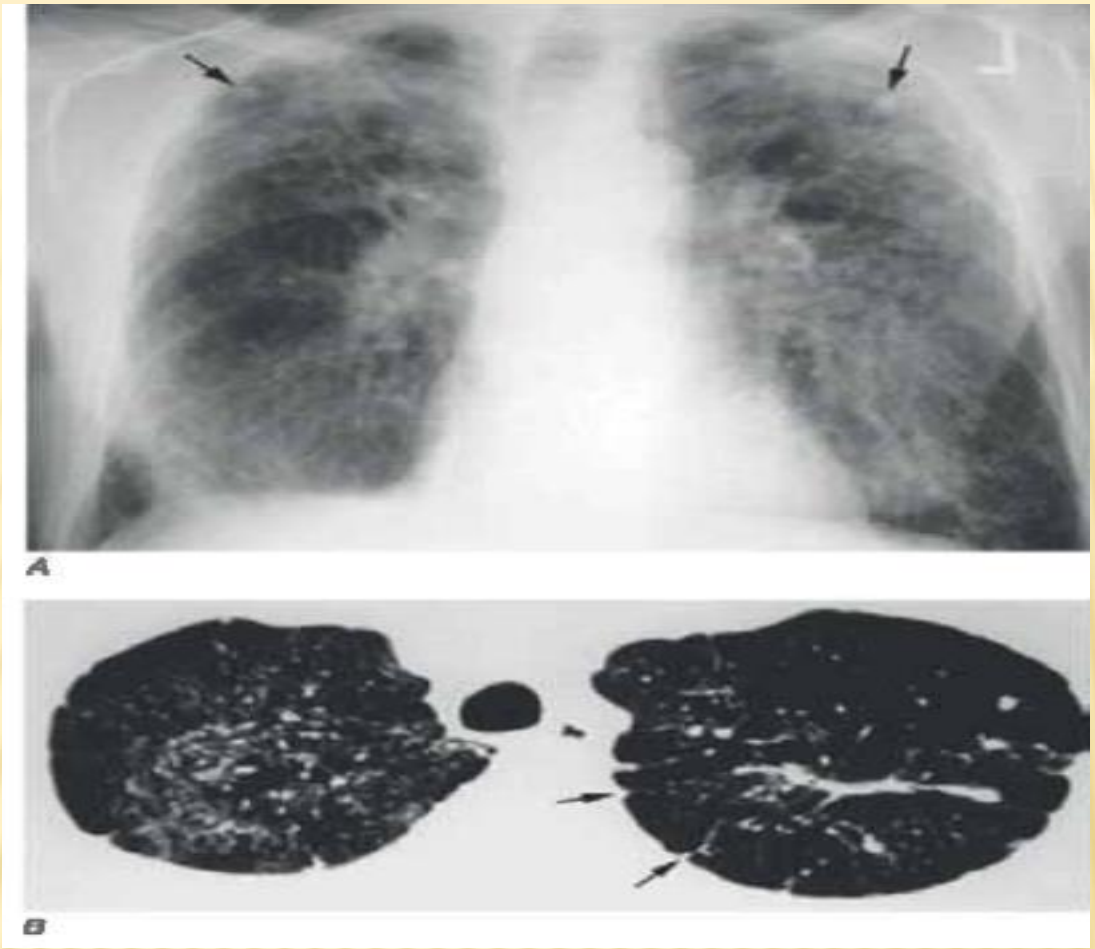


FIGURE 311-3 **Chronic silicosis. *A.*** Frontal chest radiograph in a patient with silicosis shows variably sized, poorly defined nodules (*arrows*) predominating in the upper lobes. ***B.*** Axial thoracic computed tomography image through the lung apices shows numerous small nodules, more pronounced in the right upper lobe. A number of the nodules are subpleural in location (*arrows*).