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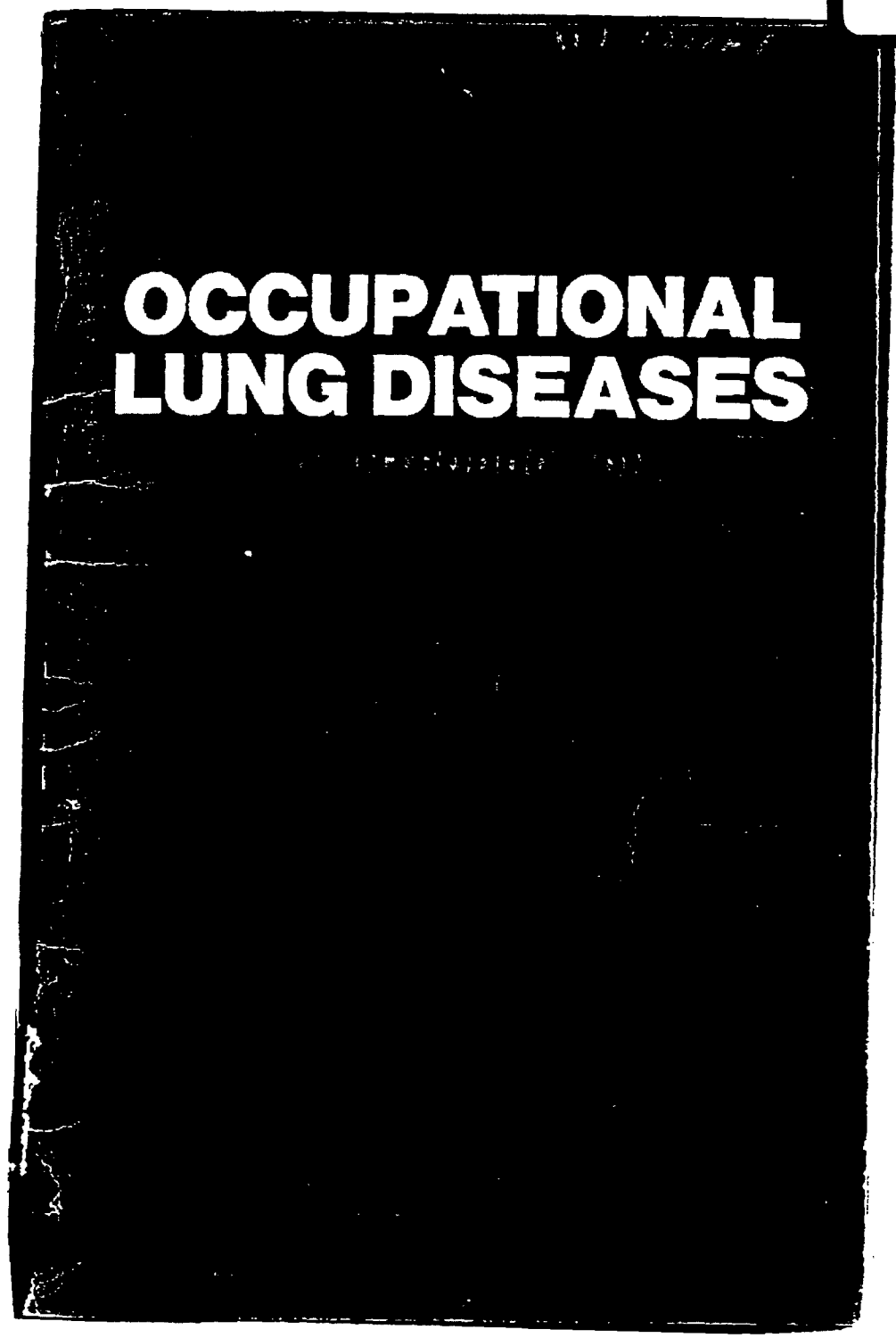
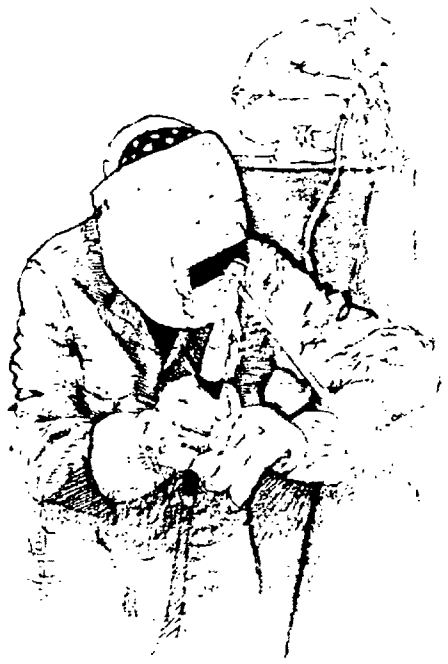


OCCUPATIONAL LUNG DISEASES

OCCUPATIONAL LUNG DISEASES

AN INTRODUCTION



AMERICAN LUNG ASSOCIATION
The Christmas Seal People

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In spite of the expert advice and guidance received, if errors of fact or differences of interpretation in developing areas of knowledge are found in the pages of this volume, the responsibility is ALA's alone.

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Chapter 1

INTRODUCTION TO OCCUPATIONAL LUNG DISEASES

Occupational lung diseases are diseases that people develop because the air in their workplaces is not clean. Coal dust, ammonia gas, solvent vapors, cereal dusts, welding fumes, and degreasing sprays are just a few of the thousands of pollutants found in America's workplaces. The medical profession calls these pollutants occupational lung hazards and calls the resulting diseases occupational lung diseases.

Occupational diseases affect all types of people in many walks of life—laborers, crafts workers, business people, and professionals. But the diseases are commoner in blue-collar workers than in white-collar workers, commoner in industrialized areas than in rural areas, and commoner in small and medium-sized businesses than in large concerns.

Some occupational lung diseases are well-known, such as the asbestosis that afflicts asbestos workers. It's easy to label this disease as occupational, because the worker's lungs contain asbestos that clearly comes from working with this substance. Many other occupational lung diseases are also easily recognized as job-related, either because a workplace substance accumulates in the lungs, such as silica in silicosis, or because the disease pattern is found only in a certain working group, such as the fume fever that afflicts smelters. These readily attributable diseases are truly occupation-specific diseases.

But many victims of occupational lung disease don't have a clearly defined, occupation-specific disease. Instead, they have bronchitis, asthma, emphysema, or cancer—conditions that can be caused or aggravated by many different occupations and by such nonoccupational factors as smoking and pollution of the general atmosphere. For example, workers in mines, foundries, and rubber and fertilizer industries suffer an above-normal prevalence of chronic bronchitis, which is also a common disease in

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cigarette smokers. So chronic bronchitis can be an occupational disease, but it is not an occupation-specific disease. Nonspecific occupational disease may, in fact, afflict more people than do the better-defined occupation-specific diseases.

Most occupational lung diseases worsen if occupational exposure continues. They cause increasing damage to the lungs—sometimes quickly, sometimes slowly. Only when discovered in the very early stages can the damage be reversed. Sometimes a disease can be stopped in its tracks, and further lung damage can be avoided. But, all too often, the most that can be done is to slow down the relentless course of the disease. The result can be a person eking out a living on a disability income, wheezing, coughing, wracked by chest pains, dying prematurely. Occupational lung disease can be as crippling as paraplegia—in some ways more so.

THE AMERICAN LUNG ASSOCIATION AND OCCUPATIONAL LUNG DISEASES

The American Lung Association is concerned about occupational lung diseases because a great many people are losing their lives and their health to these diseases—diseases that are preventable.

And because, all too often, neither the victim nor the physician realizes the link between job and illness.

And because efforts by government, management, and labor to prevent these diseases are insufficient.

And, most of all, because the American Lung Association believes that, if more people understood the facts about occupational lung diseases, fewer people would be suffering from them.

NUMBER OF PEOPLE AFFECTED

Millions of Americans are probably suffering from job-related diseases. Exact statistics don't exist.

The Department of Health, Education, and Welfare (HEW) has estimated that every year 400,000 people develop a disease caused by their jobs. Few working populations escape unscathed. Business executives get hypertension, plastics workers are plagued by skin rashes, machine operators go deaf, farmers suffer from farmer's lung, dentists have a relatively high rate of leukemia.

HEW also estimates that 100,000 deaths each year are caused by occupational diseases—more than twice the number killed in the annual carnage on the nation's highways. The National Heart, Lung, and Blood Institute, which is part of HEW, stated in a 1977 report on respiratory

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diseases that lung diseases probably account for more than half these deaths, adding that there is good reason to believe the problem of occupational lung diseases is underestimated.

The same report estimates the number of people likely to be exposed to each of 22 separate workplace lung hazards, ranging from sulfur dioxide and phosgene gases to asbestos, chromium, and arsenic. The conclusion: Some 15.5 million people risk exposure to these hazards alone. The report does not attempt to estimate the number of people exposed to such hazardous natural products as sawdust, detergent enzymes, fungi in moldy crops, and cereal dusts. But many people do work with these substances, and their health may suffer.

The World Health Organization estimates that more than 75 per cent of all human cancers are caused or aided by environmental factors, including diet, living habits, stress, chemicals, smoking. A number of environmental factors are manufactured in workplaces where laborers, supervisors, engineers, and managers may be exposed to such substances at relatively high levels.

Economists also try to estimate the cost of occupational diseases. Some $5 billion a year is paid out in workers' compensation for illnesses and injuries recognized as job-related. The National Safety Council estimates the cost of occupational illnesses and injuries to be at least 1 per cent of the gross national product.

High though these figures are, new evidence seems to reinforce the view that the prevalence of occupational diseases, including lung diseases, is underestimated. The National Institute for Occupational Safety and Health found in a survey of a group of manufacturing industries that 28 per cent of the 908 people examined had some occupational disease. Yet less than 10 per cent of these diseases had been recorded in official documents, as required by law. Put another way, more than 90 per cent of the diseases were unrecognized officially.

Behind all these cold and conservative figures lies a world of human suffering—pain, disablement, lost earnings, family strain, and medical bills. Much of this suffering can be prevented.

PREVENTION OF JOB-RELATED LUNG DISEASES

Occupational lung diseases are preventable. Workplace air doesn't have to damage the lungs—or the kidneys, the liver, or any other part of the body.

Whether it's a mining dust, a welding fume, fungal spores, wheat dust, a paint spray, a radioactive gas, or a mixture of emissions from a coke oven, a hazard is usually in the air because whoever initiated the operation

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Bakers are among those who may be threatened on the job by organic dusts to which they may have allergic or asthmatic reactions. Farmers, cheesemakers, and woodworkers are among the others who may be affected in this way.

(sometimes decades ago) just didn't know the health dangers involved—or chose to ignore them.

As a result, many workplaces contain badly designed or malfunctioning equipment and poorly planned areas and procedures. Well-designed, effective ventilation systems and other measures to control hazardous emissions are frequently lacking.

It isn't by chance that a lot of occupational diseases are found in small concerns. These businesses have less access to technical information and expert advice than large businesses do. The more borderline a company's finances are, the less likely it is to worry about health issues.

Keeping hazards out of the lungs of the people in the workplace is not necessarily a highly expensive proposition. There are many different ways of approaching the problem, such as changing to safer ingredients, isolating processes or operators, installing or improving local or general ventilation systems. These approaches are discussed in more detail in Chapter 5.

Many occupational health specialists have commented that the people who are most closely involved with a process can often suggest the most practical ways to reduce the generation of hazards. Yet, paradoxically, these people are frequently unaware that their health is in jeopardy.

Clean workplace air benefits more than just the lungs. Most occupationally encountered substances that are toxic to the human body—whether to the lungs, the brain, the blood, or the bone marrow—get into the system

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through the lungs. So preventing these noxious substances from entering the lungs should reduce not only occupational lung diseases but other occupational diseases as well.

However, some occupational lung hazards cannot realistically be avoided without changing jobs. The poultry farmer whose asthma is caused by chicken feathers can either change his or her job or business, pay someone else to work around the feathered crop, or accept the probability of disabling disease and early death. Most people, once they truly appreciate the health danger involved, consider the difficulties and disruption of a job change preferable to permanently damaged health.

WHY THE JOB-DISEASE LINK IS FREQUENTLY UNRECOGNIZED

The relationship between job and disease often goes unrecognized by both the victim and the physician for many reasons. One reason is that many occupational diseases develop so gradually and progress so slowly over months and years that most patients aren't conscious of becoming ill. Accustomed to diseases that the medical profession calls acute diseases—such as flu, colds, mumps, and measles—most people expect illnesses to hit hard and fast with fevers, rashes, aches and pains, and other symptoms and then to resolve just as quickly.

But many occupational diseases are chronic diseases. Their onset is slow and insidious, characterized by lingering colds, occasional complicating infections, slight breathlessness on exertion, an increasing need to cough up mucus. Nothing sudden or noticeable drives the worker to a physician. Yet, underneath and unseen, the worker's lungs are steadily becoming inflamed, obstructed, stiffened, or overdistended. By the time things get serious enough to send the victim to a doctor, it's often too late to do more than slow down the disease.

And even a physician may have trouble recognizing that the disease is job-related. Most physicians in practice today have had very little, if any, training in occupational medicine. The disease picture, as they see it, often looks very similar to that caused by heavy smoking, living in polluted areas, repeated bouts of infection, and other nonoccupational factors. Some physicians think that their role is limited to treating the disease and relieving individual symptoms as they occur, not establishing the exact cause of the disease.

As a whole, the medical profession has not encouraged its members to provide leadership against occupational disease, as it did with such infectious diseases as diphtheria and polio. Such epidemics were controlled by educational and immunization programs in which the medical profession

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was heavily involved. If the same energies could be brought to bear on occupational diseases, which now kill more people than many of the infectious diseases of yesteryear, the results could be equally striking.

WHY GOVERNMENT ACTION HAS BEEN SLOW

Government action has had only a limited effect, partly because of the complex nature and the size of the occupational disease problem, partly because legislation is often a cumbersome way to tackle a problem, and partly because of political pressures.

Three major pieces of federal legislation now attempt to ensure safe and healthy working conditions: the Federal Mine Safety and Health Act of 1977, the Occupational Safety and Health Act of 1970, and the Toxic Substances Control Act of 1976.

But the United States has an estimated 80 million working people, scattered throughout more than 4 million workplaces and facing 12,000 or more separate health hazards. Expecting government action alone to solve the problem is rather like trying to prevent traffic accidents by finding and correcting every stretch of road where an accident may occur.

The existence of legislation has undoubtedly led, directly and indirectly, to many improvements in working conditions and will continue to do so. But it has been a slow process—uneven, frequently controversial, and criticized by both management and labor. A great deal more can and must be done.

Just as traffic accidents can be reduced by alert, careful, and defensive driving, so occupational health hazards can be reduced, eliminated, or avoided by those who work around them—provided these people are aware of the dangers and how to deal with them. That's why the American Lung Association is publishing this handbook.

WHAT IS NEEDED

The prime need is for more awareness and understanding of the relationship between polluted workplace air and lung diseases. Many people who could play a part in reducing occupational lung diseases simply don't appreciate the extent of the problem. They include not only employers and employees but also the engineers and planners who design the operations; the buyers and purchasers who select the ingredients, cleaning agents, and equipment; and the physicians who see the resulting diseases.

It is essential that relevant technical and medical information on occupational hazards and diseases be made generally available in an understandable form. The working population must be aware of potential dangers and able to find out what hazards are involved in an operation,

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how dangerous they are, and how to combat or minimize their effects.

The second need is for a general commitment to reducing, eliminating, or avoiding the pollution of workplace air. The possible health effects should be fully considered when operations are planned and initiated, and conditions should be improved whenever possible. If the possible effects of hazards and ways to control them were automatically reviewed every time changes are made in a workplace—when machinery is replaced, processes changed, or new operations added—major benefits could be reaped in a number of small stages.

A third and crucial need is for the elimination of the most prevalent and notorious lung hazard: cigarette smoke. The combined effects of cigarette smoke and occupational dusts, gases, fumes, and vapors cannot be overstated. Smokers get occupational lung diseases more often than do nonsmokers, and smokers' lungs succumb faster than do nonsmokers' lungs to the effects of disease. The risk of chronic bronchitis, emphysema, lung cancer, and heart disease is multiplied when the worker smokes.

UNAVOIDABLE OBSTACLES

Any effort to improve workplace conditions is bound to come up against resistance of various kinds. The nation's occupational health problem didn't happen overnight. It began long ago, smoldered and gathered strength during the industrial revolution, diversified and spread further during the technological explosion after World War II. Along the way, deeply rooted attitudes developed toward work and workplace conditions. These attitudes have substantially affected the legislation passed to attack the problem, and they sometimes conflict with other efforts to improve conditions. Some aspects of the country's economic system, which developed while occupational disease was unheeded, also tend to militate against improved conditions.

This handbook attempts to sketch out the overall occupational lung health problems: how hazards damage the lungs, the diverse nature of lung hazards in a technological nation supported by a vast food-supply industry, approaches to hazard control, and some of the problems inherent in the subject.

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Chapter 2

HEALTHY LUNGS AND THEIR REACTION TO DIRTY AIR

Send any home owner to clean out a long-forgotten corner of the attic, and he or she will sneeze, cough, and probably still be clearing his or her throat an hour or two after finishing the job. Tell someone with a bad head cold to get a good night's rest, and chances are he or she will lie awake coughing miserably. Why? Because the mechanisms designed to protect the lungs from harm are doing their job.

The lung-protecting mechanisms normally work well. Everyday air is full of potentially lung-damaging hazards—house and roadside dust, automobile emissions, smoking fireplaces, aerosol sprays, bacteria, and viruses. Healthy lungs manage to do their job in the midst of all this. They are capable of handling such pollutants—provided the pollutants are limited in quantity and occur occasionally, rather than continuously.

Watch a longtime heavy smoker coughing persistently, struggling for breath, and spitting out mucus (often called "phlegm"). Or a foundry worker, breathless, weak, and complaining of chest pains. Or a farmer who's losing weight, feels breathless, and has a dry cough. All three show what happens when the lung-protecting mechanisms fail. Disease develops.

The smoker's lungs are damaged by tobacco smoke; chronic bronchitis and emphysema have developed, and there is a possibility that lung cancer has already developed or will develop. The foundry worker has inhaled a fine, sandy dust for years, and has developed silicosis; the lungs are increasingly filled with a stiffened, useless tissue provoked by the dust, and the heart is weakening because it is overworking in an attempt to compensate for the diseased lungs. The farmer's lungs have similar patches of stiff, nonfunctional tissue, but the cause is different; the lungs

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are overreacting to something in moldy hay, and the disease—farmer's lung—results from many attacks of allergic alveolitis.

Trouble can begin when the lungs' defensive mechanisms function poorly for some reason, when a hazard is concentrated or especially toxic, when the defensive mechanisms are constantly challenged—or for a combination of those reasons. And this is where the workplace comes into the picture. The workplace may be full of lung hazards—mineral or agricultural dusts, gases, fumes, solvent vapors, and even radiation. People are exposed to these hazards eight or more hours a day, five days a week, year in and year out. Many workers also smoke cigarettes. Lung disease begins and progresses.

This chapter explains briefly how the lungs and their protective mechanisms normally work, the effect of cigarette smoke, how occupational lung hazards can overwhelm the lungs' defenses and lead to disease, and other factors that can interact with and affect the disease process.

LUNG STRUCTURE AND FUNCTION

The task of the lungs is to supply the oxygen needed by the body and to remove the carbon dioxide (a waste product) produced as body cells use oxygen—a process called gas exchange.

Gas exchange occurs in microscopic air sacs (alveoli) deep within the lungs. Here inhaled air is separated from the bloodstream only by a very thin, delicate membrane. In normal health, both oxygen and carbon dioxide are able to pass readily through this membrane.

The lungs are only part of the overall system supplying oxygen to the body. The bloodstream carries oxygen from the lungs to the rest of the body, where it picks up carbon dioxide to be returned to the lungs. It's a transportation system powered by the heart. Lungs, bloodstream, and heart are closely interrelated elements of the entire oxygen-supply system. Disease in any one can cause changes or disease in the other two. Occupational lung disease, like the foundry worker's silicosis mentioned earlier, can lead to heart disease.

Air travels from the nose and the mouth to the air sacs through a network of repeatedly branching tubes. The largest tube, the trachea, leads from the throat into the chest cavity, where it divides into two smaller tubes, the main bronchi. Each bronchus divides into smaller bronchi, which then divide repeatedly into bronchioles, with the smallest bronchioles ending at the air sacs. After each division, the tubes are more numerous but smaller in diameter.

The trachea, main bronchi, and first 12 or so divisions of the smaller bronchi all have rings of cartilage in their walls. This resilient, stiff tissue

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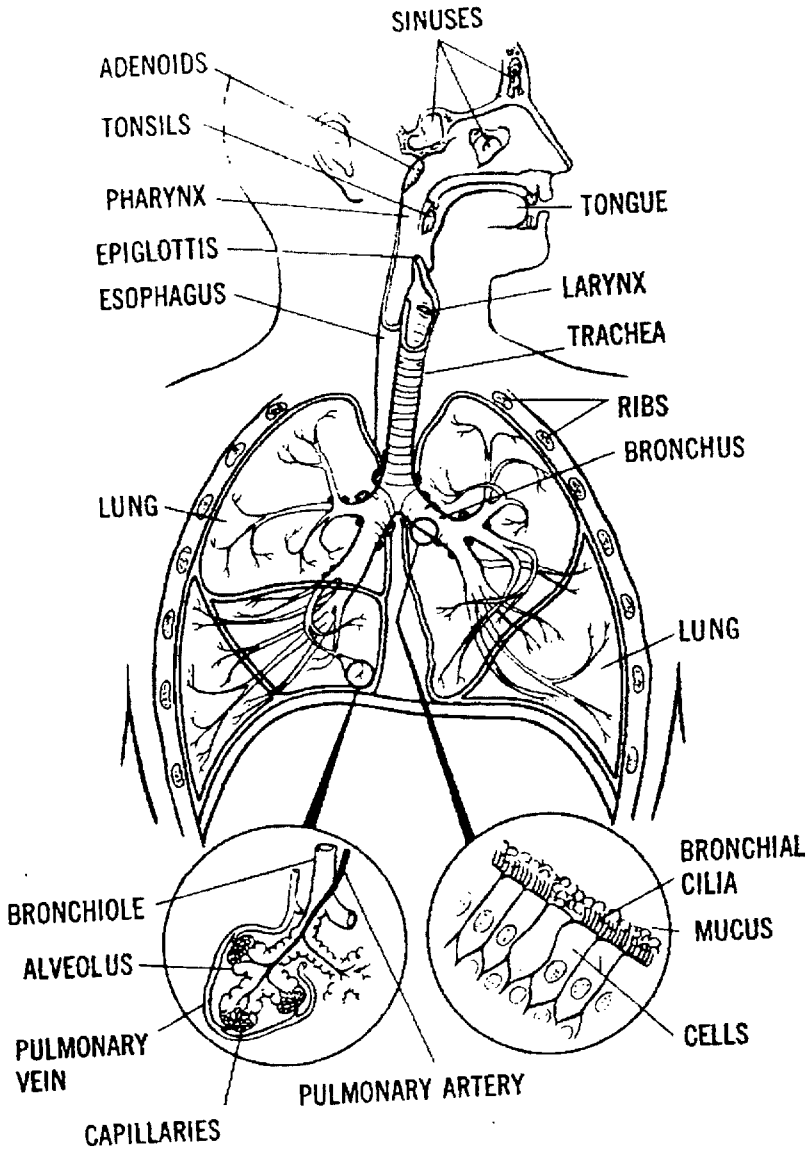
THE RESPIRATORY SYSTEM



helps to prevent these airways from collapsing and blocking the flow of air. Beyond these first dozen or so divisions, the later 10 or 11 divisions of smaller and smaller airways (bronchioles) have no cartilage in their walls. There are literally millions of these bronchioles in each lung.

The alveoli are the gas-exchange areas. There are about 300 million alveoli in each lung, with walls as thin as the walls of a soap bubble just before it bursts.

The elasticity of the alveoli is essential to the breathing process. These areas respond to pressure changes within the chest cavity as the chest expands and contracts. Anything that affects this elasticity, as some occupational lung hazards do, adversely affects breathing.

A large blood vessel leaves the heart and divides into two pulmonary arteries, one to each lung. Within the lungs, the pulmonary arteries divide repeatedly into many smaller arteries. The tiniest branches, called capillaries, form a network around the air sacs and bring blood cells to the membrane, where the exchange of carbon dioxide for oxygen takes place.

LUNG-PROTECTING MECHANISMS

Efficient functioning of the lungs can be interfered with in several ways: constricted or blocked air passages, reduced elasticity of lung tissue, thickened or destroyed membranes. Air pollutants, both inside and outside the workplace, can produce all these adverse effects. Lung-protecting mechanisms are designed to keep such hazards out of the lungs or to remove them as quickly as possible once they gain entry.

Nose

The nose is the first line of defense. It filters large particles of dusts and fibers out of the air, preventing them from reaching the lungs. Unfortunately, this filtering mechanism is so efficient that it permits only a limited flow of air to reach the lungs. As soon as a person exercises or works strenuously, he or she has to resort to breathing through the mouth to get enough air, thereby bypassing the nasal filtering system completely.

Coughing

Coughing is a protective reflex that expels foreign bodies from the trachea and main bronchi. Whenever dust particles or irritant materials hit the walls of these airways, the cough reflex is triggered. The chest and the lungs contract rapidly, forcing air out of the lungs quickly and usually blowing the foreign body or irritant out at the same time. This reflex frequently keeps the head-cold sufferer awake at night. Secretions from the nose and the throat drip down into the trachea and trigger coughing.

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Mucociliary Escalator

The mucociliary escalator is a continuous cleaning mechanism that serves the nose, the trachea, the bronchi, and the large bronchioles. Special secretory cells keep the lining of these passages coated with a thin layer of watery mucus, which traps dust particles and microorganisms that penetrate into these passages. These airways are also lined with cells that have moving, hairlike extensions (called cilia) that stroke upward, slowly but continually sweeping the mucus along the airways and toward the throat. In healthy lungs, particles trapped in the mucus get swept out of the lungs. When the mucus reaches the trachea, it is swallowed, usually subconsciously, or spat out.

Macrophages

Alveolar macrophages are highly specialized mobile cells that engulf and digest bacteria and viruses. In the process the macrophages themselves may be destroyed; the resulting debris is swept away by the mucociliary escalator.

Macrophages also engulf and attempt to digest such lung hazards as dusts. When toxic dust is involved, the macrophages are often destroyed, and the dust remains in the lungs.

Other Mechanisms

An incompletely understood cleaning mechanism serves the small bronchioles and the alveoli, where both cilia and mucus-secreting cells are few or absent. Macrophages are thought to play an important role, transporting engulfed particles and microorganisms either outward toward the mucociliary escalator or into specialized drainage channels (lymphatics). Experimental evidence shows that the overall clearing of the innermost areas is slower than that of the larger airways of the lung, usually taking days, rather than hours.

NORMAL REACTIONS TO HAZARDS

In pure, clean air the lung-protecting mechanisms work at a minimum level. Mucus secretion is just enough to keep the lining of the respiratory passage moist.

Whenever such hazards as dust, fumes, and smoke are inhaled, the lungs react by increasing the amount of mucus secreted in an effort to clear the lungs of the offending substance. Infection of the lungs or the upper respiratory tract also increases mucus secretion and increases both the number and the activity of the macrophages. When the pollutant has been removed or the infection has been eliminated, mucus secretion and

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macrophage activity fall back to normal levels.

Normal levels of mucus secretion vary. Some people produce more mucus than others do. Some people have a more marked response to irritants than do others. There is a similar variation in macrophage numbers and activity. Therefore, the overall efficiency of the lung-clearing mechanisms varies from one person to another. Two people can work together under identical conditions for many years and have different degrees of lung disease. In any one person the ability to handle hazards and to resist disease varies with age, smoking habits, nutritional status, and other less-well-understood factors, such as stress.

CIGARETTE SMOKE

Cigarette smoke isn't an occupational lung hazard, but it is inhaled many times during the waking hours, and it has a marked effect on the lung-protecting mechanisms. It irritates the airways, increases the amount of mucus secreted, slows down the rhythmic beat of the cilia, and decreases the activity of the macrophages. Cigarette smoke causes lung disease by itself, without any help from other lung hazards. But when smokers are also exposed to occupational lung hazards, their protective mechanisms are already handicapped, and disease develops more rapidly and progresses more quickly than in nonsmokers.

Cigarette smoke is carcinogenic; it can induce cancer. It is also a powerful booster of other carcinogens, such as asbestos, arsenic, chromium compounds, and other substances found in the workplace. Smokers exposed to these substances increase their chances of getting lung cancer.

HOW OCCUPATIONAL LUNG DISEASES DEVELOP

A coal miner doesn't contract black lung the first day he or she goes underground and inhales some coal dust. A farm worker's lungs aren't permanently damaged the first time he or she shakes out a bale of moldy hay. An asbestos worker doesn't get lung cancer in his or her first week on the job. Most occupational lung diseases take months or years to develop. Some diseases take decades.

Some occupationally encountered lung hazards, such as phosgene gas, are capable of seriously damaging the lungs instantly or within a few hours. The results are usually medical emergencies and are life-threatening situations. Such occupational exposures, usually classed as accidents or poisonings, are not the focus of this book.

Occupational lung diseases usually develop when the lungs are con-

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Solvents used by industry in cleaning and in thinning paints, greases and the like, are generally volatile—giving off vapors even without heating. Breathing the vapors can cause various lung diseases as well as diseases elsewhere in the body, such as liver and blood disorders; skin contact is also dangerous.

stantly or repeatedly exposed to hazards like mineral or agricultural dusts, gases, chemical vapors, fumes, and sprays. Some conditions, such as chronic bronchitis and emphysema, seem to stem mainly from the lungs' continued reaction to irritation; these conditions can lead to secondary diseases of the heart. Other diseases, such as fibrosis and cancer, are associated with individual hazards, like silica and coal tar distillates, that seem to provoke these conditions; irritation may also play a role. And occupational lung diseases can also result from the lungs' overreacting to certain hazards to which they are overly sensitive; these are called hypersensitivity diseases.

Many adults suffer from a group of chronic lung disorders that have no single cause but that result in persistent air-flow obstruction. The term chronic obstructive pulmonary disease (COPD) is usually applied to chronic bronchitis, emphysema, and asthma, and combinations of these diseases, which are discussed individually below.

Chronic Bronchitis

Chronic bronchitis is characterized by persistent oversecretion of mucus. The mucus-producing cells eventually enlarge, the excess mucus tends to clog the airways, and the mucociliary escalator becomes overloaded and functions less efficiently. The mucus forms an ideal medium for micro-

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organisms to grow on, and the lungs begin to suffer from repeated infections. These infections, together with the continued irritation by the hazard, produce inflammatory changes in the airway walls. The small airways eventually become clogged, the amount of air flowing into and out of the gas-exchange areas is reduced, and breathing becomes an increasing effort.

The victim becomes short of breath on exertion, has a persistent and productive cough, and frequently has to resort to spitting out the mucus in his throat. (The mucus is then referred to as sputum.) Episodes of bronchitis and occasionally pneumonia punctuate the progressively worsening disease.

Cigarette smoking alone can produce chronic bronchitis. When cigarette smoke is combined with occupational hazards like dusts and fumes, the disease develops and progresses more quickly than in nonsmokers.

Emphysema

Emphysema can follow chronic bronchitis or any other lung condition in which the airways become seriously clogged or obstructed. (A few people have an inherited enzyme deficiency that predisposes them to emphysema, regardless of other lung diseases or exposure to hazards.)

The development of emphysema isn't completely understood. Air seems to become trapped inside the alveoli and comes under increasing pressure as the chest cavity contracts. This pressure stretches the alveolar walls, which lose their elasticity; some alveolar walls rupture, destroying both the pulmonary membrane and the capillary bed. Other alveoli then become overdistended, losing their elasticity or rupturing.

The emphysema victim becomes increasingly breathless, often developing a barrel-chested appearance because the lungs are overdistended. The disease is progressive and often leads to heart disease.

Heart Disease

Cor pulmonale is the medical term for heart disease that is caused by lung disease. When lung disease causes blood to be low in oxygen, the heart tries to compensate by pumping harder at higher pressure. This sustained work by the heart frequently causes it to become enlarged and eventually diseased. Many lung disease victims finally die from heart failure, although the underlying cause of death was the lung disease.

Pulmonary Edema

In edema, plasma, the liquid part of the blood, leaks into the airways and the alveoli. It is a serious complication of heart or lung disease,

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rather than a disease entity in itself. Edema occurs when the pressure of blood in the capillary bed rises high enough to force plasma across the pulmonary membrane or when the capillary walls are damaged or destroyed and plasma leaks into the alveoli.

Plasma becomes frothy when mixed with air, and the froth then hinders the passage of air into and out of the alveoli. Even if the cause of the edema is corrected, it takes a long time for the fluid to be cleared out of the lungs. While there is plasma in the lungs, the patient experiences severe breathing problems, especially if large areas are involved.

Pulmonary Fibrosis

Fibrosis is a disease process that seems to be associated with certain occupational hazards, notably silica and asbestos. It is also seen occasionally in people with hypersensitivity diseases caused by agricultural dusts. No one knows yet how and why fibrosis begins.

In fibrosis a thickened, nonelastic tissue appears in the lungs. Rather than obstructing the airways, as chronic bronchitis and emphysema do, fibrosis increases the stiffness of the lungs, restricting the ability of the small airways and the alveoli to respond to pressure changes. It can also thicken the pulmonary membrane, making it harder for gases to pass through the membrane.

Fibrosis may be discrete—that is, confined to certain lung areas—or diffuse—that is, widespread through the lungs. Some hazards, notably silica, can produce a fibrosis that spreads rapidly and inexorably in the lungs; this disease is called progressive massive fibrosis (PMF). Although PMF is usually fatal, it occasionally stops progressing; the reason is unknown but is the subject of much research.

Cancer

Cancer, an uncontrolled growth of abnormal cells, may develop as a reaction by the nose, throat, and lungs to inhaled hazards. The cause of cancer still eludes researchers, but present evidence indicates that several factors are involved, rather than a single agent.

One factor may be persistent irritation of the respiratory tract. The irritation may produce inflammatory changes within the cells and make them more susceptible to cancer-provoking agents, called carcinogens. Another theory is that dusts retained in the lungs absorb carcinogens—from tobacco smoke, for instance—and hold them in contact with lung tissues for relatively long periods.

Known carcinogens—arsenic, asbestos, some chromate compounds, and many others—are found in some workplaces and in tobacco smoke. Pol-

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lution of the general environment also seems to increase the incidence of lung cancer. When two or more carcinogens assault the same set of lungs, the threat of cancer increases dramatically.

Lung cancer remains one of the least curable cancers. Occupational lung cancers often develop in lungs that are already diseased, further reducing the chances of successful treatment.

Hypersensitivity Diseases

Hypersensitivity diseases develop because the lungs overreact to specific hazards. The best known example of hypersensitivity disease is hay fever, an overreaction of the nose and sinus linings to inhaled pollens. Not everyone is hypersensitive to pollen. And the same goes for occupationally encountered hazards. Only a small percentage of the people exposed to a hazard develop a hypersensitivity to that hazard. Allergy is the commonest type of hypersensitivity reaction, but there are others. They produce similar symptoms but occur through different physiological mechanisms, a difference that may be important in treatment.

OCCUPATIONAL ASTHMA. Occupational asthma is a hypersensitivity reaction that occurs mainly in the small bronchi and bronchioles. In response to some specific hazard in the workplace, the muscles in the walls of these airways contract in a spasm that narrows the airways and leaves the victim wheezing as he or she struggles for breath. A thick, sticky mucus is secreted, and this mucus is difficult to clear from the airways.

ALVEOLITIS. Alveolitis is a hypersensitivity disease that occurs in the alveoli and surrounding tissues. The disease is usually caused by fine organic dusts that are inhaled into these areas.

In contrast to asthma episodes, alveolitis episodes usually begin several hours after exposure to the causative hazard. The alveolar walls become swollen, and the distribution of white cells in the walls is disturbed. The symptoms of coughing, fever, and difficulty in breathing may last for hours or several days. The symptoms are sometimes serious enough to warrant emergency treatment.

Repeated episodes, especially if they are mild enough to go unnoticed and untreated, sometimes lead to a diffuse fibrosis of the alveolar walls. This fibrosis restricts the functioning of the lungs and frequently leads to cor pulmonale.

FACTORS INFLUENCING THE DISEASE PROCESS

What are the chances of any one person getting a work-related lung disease? Which disease will it be? And how serious will it become? Will

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it be a barely noticeable cough signaling that mild chronic bronchitis may develop long after the victim retires? Or will it be a progressive fibrosis that leads to cancer and death at the age of 55?

There are no simple answers to those questions. Occupational diseases depend on many factors. Hazards vary in their ability to cause disease. They have different chemical and physical properties, and they interact in different ways with body systems and tissues. Lungs vary, too, in their ability to handle hazards. Their ability to resist occupational diseases is also affected by other hazards they are exposed to at the same time.

Even hazards that are similar in physical form can vary considerably in their disease-causing powers. Some mineral dusts are a nuisance, rather than a danger to health; other mineral dusts irritate lung tissue and lead to chronic obstructive pulmonary disease; still others induce fibrosis or cancer. Some gases suffocate; others poison body systems; still others produce chemical burns in the lungs and other tissues. When a group of hazards are closely related both chemically and physically, like the vapors from a series of similar chemical compounds, they usually have similar effects on health—but this is by no means an iron-clad rule. Sometimes a substance causes health effects vastly different from those caused by related substances.

The particle size of dusts, fibers, and sprays affects the type and the severity of the occupational lung disease because the size of a particle determines how far it penetrates into the lungs. Inhaled hazards pass through the airways rather like driftwood floating down a stream. The large and heavy pieces tend to catch on the sides or sink to the bottom. The smaller and lighter pieces continue in the mainstream of the current. Fine dusts, therefore, tend to reach the smaller airways and alveoli, where the clearing mechanisms are slower and the delicate tissues more susceptible to damage. Coarser particles are usually deposited in the upper airways, from which they are more readily cleared. The most dangerous particles, in fact, are those that are so small that they cannot be seen with the naked eye.

The period of exposure to a hazard and the concentration of the hazard also affect the severity of disease caused within the lungs. Someone who has worked around fumes or dusts for many years is more likely to have lung disease than is someone who started work in the same place only three months ago. Someone who is exposed to a concentrated acid vapor is more likely to incur disease than is someone exposed for the same period of time to a weak solution of the same acid.

Another variable is the difference in lung health from one person to another. Some people are able to withstand considerable hazard exposures with little effect; others succumb to disease relatively rapidly. Cigarette

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In dozens of occupations, irritating gases rise from the host of materials the workers stir, heat, pour or mix. Many cause chronic lung disease after short- or long-term exposure; some are damaging even in small amounts.

smoke and, to a smaller extent, pollution of the general atmosphere also adversely affect the ability of the lungs to withstand occupational lung hazards.

Within a group of people who live and work under similar conditions and are exposed to the same hazards, an occupational health specialist can expect to find the same type of lung disease but to different degrees. In coal miners, for example, one may find some workers with light deposits of coal in their lungs, some with heavier deposits, and a few with very heavy deposits. Among the miners who also smoke cigarettes heavily, one finds some workers with chronic bronchitis and others who also have emphysema. If the group of miners is large enough some will have progressive massive fibrosis.

These guidelines on the factors influencing the development of occupational lung diseases are generalizations. It can be difficult, occasionally impossible, to correlate hazard exposures with the prevalence and severity of disease. There are always exceptions to the overall pattern, which may be blurred to begin with. Some people remain obstinately healthy, despite long service in a hazardous occupation; others are seriously disabled by a job that is normally associated with only mild effects on the health.

Often, the most definitive statement that can be made about a job-disease relationship runs something like this: "People who work with this substance under these conditions tend to develop disease X, which usually progresses fairly slowly but may be complicated by disease Y; a few instances of disease Z have also been reported."

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Chapter 3

TYPES OF LUNG HAZARDS

The miner breathes in coal dust. The silo filler breathes in nitrogen oxide. The painter breathes in a chemical spray. The auto mechanic breathes in sulfuric acid fumes. And all may eventually develop lung disease.

Dusts, gases, sprays, vapors, fumes, radiation—lung-damaging agents come in many forms. Some of these hazards can be seen; others smell terrible; still others irritate the nose and throat. But many can be detected only by special equipment. Where lung hazards are concerned, you may get more than you see—or smell or feel.

This chapter describes the physical forms in which lung hazards occur, some of the workplaces where they may be encountered, and their harmful effects on the lungs. The objective here is to point out the widespread and diverse nature of occupational lung hazards and the enormous amount of unnecessary disease they cause—not to provide a comprehensive list of hazardous occupations or dangerous substances. The fact that a particular substance is not mentioned here does not mean that it is nontoxic.

For the sake of simplicity, the harm that each type of hazard can do to the lungs is considered separately. This approach tends to minimize the total effect of the workplace on lung health, for few hazards occur in isolation. One process or one work area may generate several hazards. One person may work in several hazardous areas, each with a different hazard, during a normal day's work. Or the worker's job—and the hazards he or she encounters—may change with the seasons, when absenteeism occurs, or as production priorities vary. Hazards encountered in the workplace may also interact with cigarette smoke and with general air pollutants.

At best, the damaging effect of one hazard is added to or superimposed on the havoc wrought by another hazard. At worst, the effect of two

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hazards is synergistic—the total effect is much greater than the effect of one hazard added to the effect of a second; one plus one may equal five. The phenomenon of synergism has been best documented in cancer, but it is also found in other diseases.

The range of hazards and hazardous occupations covered in this chapter emphasizes the need for caution in the workplace. Even the many apparently harmless pollutants that have been in use for decades or longer should be regarded a little warily; there is always the remote possibility of long-delayed or very rare effects, as happened with vinyl chloride gas and liver cancer.

Improved hazard control should be a constant objective. In some instances, available information is not being used to maximum effect. In other instances, economic feasibility and market factors are forcing a decision on the balance between risks, benefits, and hazard-control costs. And in many cases, much more research is needed.

One guideline that is unlikely to be disproved is this: The fewer hazards people are exposed to, including smoking and general air pollution, and the lower the levels of exposure, the lower the incidence and severity of any resulting disease.

The hazards discussed in this chapter are dusts, fumes and smoke, gases and vapors, mists (sprays), and radiation. In many respects, the insults afforded to the lungs by dusts, fumes, and smoke are similar; the same is true of gases and vapors. The hazards are discussed individually, however, because they are produced in different ways and are, therefore, associated with different workplaces.

DUSTS

At birth, the human lungs are pale pink. By middle age, the lungs are dirty gray, even in someone who lives and works in a relatively unpolluted area. The reason? Dusts.

Dusts are found everywhere. They are formed whenever solid matter is broken down by natural or mechanical forces. The resulting collection of small, dry particles remains suspended in the air for some time once stirred up. The larger, heavier particles tend to settle first; smaller, lighter particles hover in the area for a longer time and drift with air currents.

Dusts abound in the general atmosphere and in many workplaces, both industrial and agricultural. As discussed in Chapter 2, whether a dust causes disease depends on many factors, including the properties of a particular dust, the amount inhaled, the efficiency of individual lung-clearing mechanisms, and the size of the dust particles—with the very fine, often invisible, respirable particles being most likely to induce disease.

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Types of Dusts

Dusts fall into three basic groups: mineral, organic, and chemical. Each type tends to cause a different pattern of disease. (Disease processes are discussed in detail in Chapter 2.)

MINERAL DUSTS. Mineral dusts are formed from the rocks, stones, and ores found in the earth's crust. These dusts are often called inorganic dusts because they are formed from nonliving materials.

Coal is an unusual case. In occupational disease studies it is usually grouped with the mineral dusts, because it behaves as inorganic dusts do. Strictly speaking, however, coal is derived from formerly living materials and might well be called an organic dust.

Mineral dusts are generated in the mining or quarrying process, during blasting and tunneling, and during the transportation of the raw materials. Subsequent processing is also dusty—milling, crushing, and grinding the crude ore into smaller pieces; mixing and loading the ore for foundry operations; preparing clays for pottery, tiles, and brick; carving monuments; and countless other processes. Smelting and other techniques are used to obtain pure metals or compounds from the crude ores. Dusts are generated during these operations and, later, in metal-working and numerous other manufacturing industries.

ORGANIC DUSTS. Organic dusts are derived from living materials—plants, animals, microorganisms—and from such natural products as wood and leather. Dusts are generated during the harvesting, storage, transportation, and processing (milling, chopping, spinning, sawing) of plant products. These products include cereal grains, coffee beans, paprika, cotton, flax, and timber. Animal and poultry husbandry produces dusts consisting of animal hair and dander, bird feathers, and droppings. Woodworking and leatherworking entail considerable exposure to organic dusts. And all these dusts are recognized causes of lung disease.

Another troublesome group of organic dusts consists of the minute seeds or spores produced by fungi. If hay, straw, or other crops are stored when damp, they provide excellent conditions for fungal growth. Later, when these crops are handled, billions of spores that can cause disease are released. In some industries, such as cheese processing and mushroom farming, fungi are essential; nevertheless, their spores are a danger to workers' lungs.

CHEMICAL DUSTS. In addition to the naturally occurring mineral and organic dusts provided by nature, humans have invented many of their own. An increasing number of synthetic chemicals are used in industry, business, agriculture, and the home. Many of these synthetic chemicals are in powder form—bleaching powders, catalysts, pesticides, dyes—and their dusts may cause disease if inhaled.

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Effects of Dusts

Susceptibility to dust-induced diseases varies widely from one person to another. Although two people work under the same conditions for many years, one may have only early symptoms of disease, while the other has severe respiratory disability. The efficiency of the lung-clearing mechanisms varies from person to person, and any other hazards that the lungs are also dealing with—notably smoking and general air pollution—also have significant effects on the development of disease. Without exception, occupational dusts induce disease more rapidly and more frequently in smokers than in nonsmokers, and cigarette smoking hastens the progression of lung diseases. General air pollution may also bolster the effects of occupational dusts but to a smaller degree.

As a general rule, mineral dusts can produce pneumoconioses (from the Greek *pneumon,* lung and *konis,* dust); obstructive lung conditions, such as chronic bronchitis and emphysema; and sometimes fibrosis and cancer. Hypersensitivity reactions are uncommon with mineral dusts but not unknown; beryllium, cobalt, and some platinum salts are suspected of provoking such responses.

Most mineral dusts are relatively inert; they are not dissolved or broken down by lung tissue, and they begin to accumulate once they overload the worker's lung-clearing mechanisms. The damage a dust does to the lungs depends on the characteristics of that particular dust.

All mineral dusts seem to irritate the lungs to some degree, but some, such as tin dust, have such mild effects that they are generally considered benign or nuisance dusts. The irritation from others can lead to chronic bronchitis, emphysema, and cardiac complications.

Many mineral dusts also initiate other disease processes. Silica, for example, tends to induce fibrosis; aluminum also induces fibrosis but less frequently and less severely than silica does. Certain chromate compounds appear to initiate cancer within the lungs; an even more powerful carcinogen is asbestos, which also causes fibrosis.

Unlike mineral dusts, organic dusts are usually either dissolved in the lung fluids or broken down by physiological mechanisms. They do not usually accumulate in the lungs; instead, they may evoke hypersensitivity reactions. Coarse dusts tend to act on the bronchi (the major subdivisions of the windpipe), provoking occupational asthma or, in the case of cotton, byssinosis. Fine dusts tend to cause reactions within the small airways and alveoli (the tiny air sacs in the lungs), provoking allergic alveolitis. Repeated mild or untreated attacks of asthma may eventually lead to permanent obstructive disease; repeated mild alveolitis attacks can lead to diffuse lung fibrosis. Certain wood dusts also seem to be associated with nasal cancer.

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Chemical dusts produce various kinds of lung damage, depending on the properties of the chemical involved. Some chemical dusts are irritants, such as fluoride dusts and the dusts of some selenium and vanadium compounds. Other chemical dusts—such as the dusts of chloride of lime (bleaching powder), sodium hydroxide, potassium hydroxide, and phthalic anhydride—have a caustic effect; they cause chemical burns when inhaled in quantity, and prolonged exposure to low levels of those dusts is thought to contribute to the development of laryngitis and bronchitis.

A number of chemical dusts provoke allergic reactions within the lungs. Others are absorbed through the lungs and cause cancer elsewhere in the body; then, when the cancer spreads, secondary tumors can appear within the lungs. It is possible that some of the many chemical powders introduced in the past 15 years can induce lung cancer, but these powders have not been in use long enough for this latent effect to be recognized.

FUMES AND SMOKE

These two lung hazards are similar in some respects, and laymen often use the terms interchangeably. There are, however, technical and medical differences.

Smoke is formed from burning organic material—cigarettes, fireplaces, and incinerators being the most familiar examples. To the lungs, smoke is a mixture of solid particles—carbon dust—and gases like carbon monoxide, carbon dioxide, and possibly others, depending on what material is burning.

Fumes are formed when very hot vapors—usually metallic vapors—cool rapidly and condense into very fine solid particles. To the lungs, a fume is a very fine mineral dust with a high potential for causing disease that is often mixed with hazardous gases, like ozone and nitrogen oxides, which are formed at the same time.

Where Fumes Are Found

Fumes are produced mainly in industrial high-heat operations, like welding, smelting, and furnace work. In welding, several metals and coatings—from the surfaces being joined or repaired and from the welding rod or electrode—are heated to high temperatures. The metals often vaporize, react with oxygen in the air, and form a fume consisting of fine particles of metal oxides. The fume has a blue-gray appearance and often hangs in the air and drifts into patterns, just as cigarette smoke does.

Smelting involves heating ores, usually to separate and refine the metals present. A variety of fumes and gases may be produced.

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In welding and soldering, protecting the eyes from glare does not save the lungs from the many gases that may be created by heat working on the soldering material, the metal being worked or the coatings and paints involved.

Effects of Fumes

It is difficult to determine the degree of hazard posed by the individual constituents of fumes, since several hazards are almost invariably formed simultaneously. Welding and smelting fumes usually contain the oxides of several different metallic elements; at the same time, a mixture of gases—such as carbon dioxide, ozone, and nitrogen oxides—is frequently formed. In copper smelting, for example, metal fumes, sulfur dioxide, and arsenic trioxide may all assail the lungs. None is beneficial.

Oxides of iron, tin, zinc, copper, nickel, and some rarer metals used in welding cause fume fever when inhaled in sufficient quantities. Fume fever, a flu-like illness of several days' duration, is well known to welders and metalworkers.

Cadmium fumes not only cause a severe case of fume fever but are also thought to have a direct effect on the pulmonary membranes. In addition, some authorities think that prolonged low-level exposure to cadmium fumes causes emphysema; others hold that repeated, infrequent acute exposures are responsible for the emphysema.

Coal carbonization, as in coke ovens, produces some gaseous hydrocarbons that condense into particulates on cooling and are associated with an increased incidence of lung cancer—particularly in smokers. There may also be more chronic respiratory disease in coke oven workers.

Chromium fumes, formed in smelting and furnace operations, can

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produce both asthma and lung cancer. Whether the chromium itself or some contaminant is responsible has not yet been determined.

Beryllium is recognized as a potent cause of both acute and chronic lung disease when its dust or fumes are inhaled. Safer substitutes have replaced beryllium in many instances, but beryllium is still valued in other processes.

Arsenic occurs as a contaminant in many commercially used ores and is released during smelting. It is also used in the chemical industry. Arsenic can cause lung cancer when its dust or fumes are inhaled; if handled, arsenic can also produce skin cancer.

GASES AND VAPORS

Scientists define a gas as a fluid that expands to fill the space that contains it. A gas can travel quickly and sometimes undetectably far from its point of origin. High flammability is characteristic of many gases; some are explosive when mixed with air in certain proportions; and many gases are very reactive, both chemically and physiologically. Gases are among the most valuable and the most dangerous agents found in the workplace.

Poor ventilation, confined working areas, and heat frequently increase gas hazards, but danger can be present even without those factors. The very nature of gases makes them formidable hazards.

"Vapor" is the technical name for the gaseous form of a liquid that always exists above that liquid—just as water vapor always exists over water. The closer a liquid is to its boiling point, the more it vaporizes. Liquids with boiling points just above room temperature vaporize readily and are called "volatile." Liquids heated in the workplace vaporize increasingly as their temperatures rise. A few solid substances tend to vaporize at room temperature, but this phenomenon is unusual.

To the lungs, vapors and gases are essentially the same. The practical difference is that vapors are encountered wherever their parent liquids are found; gases aren't necessarily associated with their liquid forms.

Where Gases Are Found

The main sources of gases are chemical reactions and high-heat operations. Gases that are both colorless and odorless are particularly dangerous unless special detection equipment is used; early recognition of the initial effects of such gases may be lifesaving, as with carbon monoxide and drowsiness.

CHEMICAL REACTIONS AND GASES. Gas-producing chemical reactions occur in nature and as a result of technology.

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Natural fermentation processes produce gases. The methane that occurs in coal fields and oil wells is one example; the explosive hazard to miners and drilling personnel effectively outweighs its health hazard. The fermentation of fresh silage results in highly toxic nitrogen oxides, and newly filled silos are a serious threat to farm workers and their families; even entering an empty but badly ventilated silo has sometimes proved fatal.

Sewage treatment and the decay of sanitary landfills and other organic matter may produce both methane and hydrogen sulfide gases. Hydrogen sulfide smells like rotten eggs—an odor so foul that it is easily detected and avoided except when the odor is drowned by other smells, as it may be in sewage processing, septic-tank cleaning, slaughterhouse work, and fish-product industries.

Ozone, a toxic and irritating gas, is produced in the upper atmosphere by the interaction of solar ultraviolet radiation and oxygen; it has proved troublesome to the occupants of high-flying aircraft. It is also produced by the action of the sun on photochemical smog from automobile emissions and during certain arc-welding operations.

Man-made chemical reactions frequently produce gases—not always within the confines of a factory. House cleaning people have been exposed to dangerous levels of chlorine after mixing household cleaning agents, especially those with bleaching properties. Cleaning or repairing operations inside industrial vessels have resulted in accidental gas exposures when cleaning agents or welding techniques have interacted with traces of chemicals previously stored in the vessels. And the astronauts on one of the Apollo space missions were exposed to toxic nitrogen oxides when they omitted a safety check during re-entry.

Gases are often produced by industrial chemical reactions and other technological processes. Often, they are collected as they bubble off from a process and used elsewhere, either as fuel sources or as ingredients in other chemical reactions. Sometimes they are merely disposed of in some way. Wherever gases are produced, handled, or transported, there are potential dangers to both life and health. Small leaks may subject nearby workers to low doses of hazardous gases. Equipment breakdown or malfunction may result in sudden, massive exposures that are classed as industrial accidents.

The manufacture of gases for industrial or domestic consumption is a sizeable industry in itself. Some gases—acetylene, propane, butane—are used as fuel sources. Other gases—oxygen, hydrogen, ammonia, chlorine, fluorine, bromine, acrylonitrile, phosgene, and countless others—are used as ingredients in chemical reactions. Gases are also manufactured for other specialized purposes, such as life-support systems and anesthetics in medicine. Exposure to those gases can occur at any stage during the

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manufacture, storage, transportation, and use of the products.

HIGH-HEAT OPERATIONS AND GASES. High-heat operations produce gases, usually because of chemical reactions that don't occur at lower temperatures. Welding and brazing can, in badly ventilated areas, allow a build-up of dangerous gases like ozone and nitrogen oxides. If these operations are conducted anywhere near chlorinated hydrocarbons, such as degreasing agents, phosgene—a highly dangerous gas—may be produced. Smelting, oven-drying, and furnace work are other potential sources of hazardous gases.

The deliberate or accidental burning of some synthetic materials can produce phosgene, nitrogen oxides, and other dangerous gases. Those gases are occupational hazards for fire fighters, who may also encounter such gases as hydrogen cyanide, carbon monoxide, and carbon dioxide.

The internal combustion engine is essentially a high-heat burning operation, producing both carbon monoxide and carbon dioxide. Whenever coal or oil is burned, sulfur dioxide is also produced; this gas is a noted lung hazard at both low and high concentrations.

Effects of Gases

Gases endanger life in general and lung health in particular in many ways. Gases that are physiologically fairly inert—such as nitrogen and methane—can suffocate a person simply by displacing oxygen from the air. Others are poisonous to various body systems: Hydrogen cyanide interferes with the use of oxygen; hydrogen sulphide is toxic to the central nervous system; carbon monoxide prevents the bloodstream from picking up oxygen. The principal threat of these gases is to life itself, rather than to lung tissues. Oxygen itself can be toxic at elevated pressures, as in diving.

Many other gases can damage or cause disease within the lungs themselves. These gases fall into three categories: those that are immediately irritating to the respiratory tract, those that have no immediate irritating effect, and a small group of gases that induce lung cancer.

The gases that have an irritating effect on the lungs and upper respiratory tract include ammonia, chlorine, bromine, and sulfur dioxide. Because they dissolve readily in the watery mucous lining of the nose and throat, they rapidly penetrate to the underlying tissues and irritate them. A sudden, intense exposure, which sometimes occurs in an industrial accident, poses a life-threatening situation. Then these gases either penetrate and burn the lungs or produce such a marked reaction that the trachea closes completely, cutting off the supply of air to the lungs.

Whether there is any danger to health from prolonged exposure to low levels of these gases—levels that produce little or no irritation—is a

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question that has not been answered. Only one gas—sulfur dioxide—has been studied in detail, largely because of concern over its presence in the general atmosphere. Low levels have been experimentally shown to constrict the airways; there is evidence that such levels can also aggravate established obstructive lung disease but not necessarily cause it. The sulfur dioxide levels permitted in occupational settings would cause alarm in the general environment.

The gases that do not have an immediate irritating effect on the upper respiratory tract include phosgene and the nitrogen oxides. Because they are less soluble in water, they don't rapidly reach the tissues beneath the watery lining of the respiratory tract. Hence, there is no immediate irritation to provide an early warning of danger. Instead, these gases penetrate deeply into the lungs, slowly attack the tissues, and may do extensive damage before the first symptoms occur—often many hours after exposure. Edema (excess fluid in the lungs) is one of the main medical problems after inhalation of these gases. Nitrogen dioxide has a heavy, red-brown appearance, and phosgene has a faint but characteristic smell of new-mown hay, which may give some warning of their presence but cannot be relied upon.

A few gases cause lung cancer, as do the radioactive gases. (Radioactive gases are discussed later in this chapter, under Radiation.) The first carcinogenic (cancer-producing) gas to be recognized was mustard gas, which was used in chemical warfare during World War I; many workers who manufactured the gas later died of lung cancer. Nickel carbonyl, which is produced in certain nickel furnace operations, has a similar effect. Vinyl chloride gas, which was recently discovered to produce a rare liver cancer after many years of exposure, is now suspected of causing both brain and lung tumors as well. Other gases may have carcinogenic properties that have not yet been recognized because the gases have not been in use long enough for such latent effects to appear. Frequently, cancer is first detected 20 or more years after exposure to the carcinogenic agent.

Where Vapors Are Found

Vapors are found in most industries. Although commonly pictured as clouds rising from large vats or tanks of liquids, vapors are also found wherever the parent substance is used—over parts that have been dipped into cleaning or curing solutions, around air hoses used to dry off excess fluid, near ovens that have been opened after heat processing. Exposure to vapors is by no means confined to people who handle liquids in bulk.

Vapors can be divided into two chemical groups: those from inorganic substances, which have mineral or metallic origins, and those from organic substances, which are derived from carbon.

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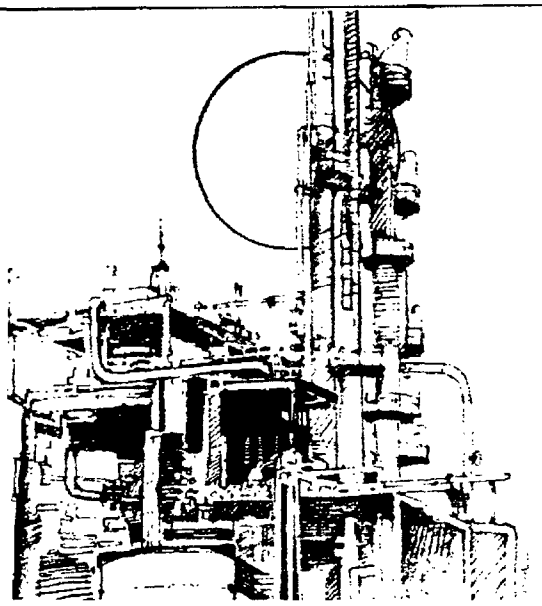

Workers in the petrochemical industry may breathe in a variety of vapors and gases—hydrocarbons such as benzene, sulfur dioxide, ammonia and more. Each can do damage; a worker breathing several or all of them faces an even greater danger of damage.

Most inorganic substances in industrial use have relatively high boiling points and don't vaporize much at normal working temperatures. Few are recognized lung hazards.

Organic vapors are more widespread than inorganic vapors, partly because many organic liquids are used in industry and partly because many of these liquids have relatively low boiling points and vaporize readily at normal working temperatures. Many organic liquids are valued for their solvent properties and are used to dissolve chemicals, pigments, grease, and other industrial residues. Chemically, the liquids are classified into numerous groups, including acetates, alcohols, ketones, and aromatic hydrocarbons. They have many different and highly individual effects on the body.

Effects of Vapors

In general, vapors capable of seriously damaging the lungs at high concentrations cause nose and throat irritation at lower levels—a warning sign that should never be ignored.

Mercury and mercury compounds vaporize readily and are notorious for their effects on the brain and the nervous system; mercury can also cause tracheobronchitis (inflammation of the windpipe and the bronchi) and lung edema. Osmium, a rare metal with specialized uses, slowly oxidizes in the air and forms oxides that are intensely irritating to the eyes and

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lungs, causing bronchitis and tracheitis; vapors from some other osmium compounds may cause asthma.

Hypersensitivity reactions to vapors are common; for example, the heating of polyvinyl chloride produces a vapor associated with meat wrapper's asthma.

Like some gases, some vapors are carcinogenic. The vapors of benzidene and related compounds can cause cancer in several body organs, and secondary tumors may develop in the lungs. All aromatic compounds (derived from the basic benzene ring structure) should be treated with great caution. Many have been in use for too short a time to allow carcinogenic effects to be detected.

Many organic vapors do not directly harm the lungs, but they do enter the system through the lungs and cause disease elsewhere in the body. Carbon tetrachloride is highly dangerous to the liver, especially if alcohol is also imbibed. Many other vapors affect the functioning of the liver, kidneys, brain, and nervous system.

MISTS (SPRAYS)

If a painter uses a spray gun to paint something, he is probably making his job considerably easier. With the spray gun, he can coat large surfaces and complex shapes quickly and efficiently, and he can penetrate easily into areas inaccessible to a paint brush. But at the same time, he may also be exposing his lungs to a dangerous mist—a fine suspension of liquid droplets in air or some other propellant gas.

Where Mists Are Found

Sprays are widely used in industry and are especially useful for applying substances that would damage the skin if applied by hand. Acid sprays are used to remove rust from metals; organic solvents are sprayed in many cleaning operations. Mists may also be nuisance by-products of other operations, like the cutting oil mists in machine tool workshops.

Small businesses also value sprays. Beauticians and exterminators use a variety throughout their working days.

In the home, many people use aerosol spray cans to dispense cleaning agents, cosmetic products, paints, varnishes, lacquers, insecticides, and many other products. These aerosol cans have been the subject of considerable consumer concern, but the total domestic exposure remains well below the exposure in workplaces where sprays are regularly used.

Effects of Mists

Mists are like dusts in one important way. The finer the spray or dust, the farther the droplets penetrate into the lungs. The amount and the

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nature of the damage a mist causes depends on the properties of the liquid being sprayed and to some extent on the temperature and the concentration of the spray. Many sprays, like paints and varnishes, are a mixture of several ingredients—solvent, pigment, stabilizing agents. The lungs are, therefore, exposed to all these substances—and to the propellant gas. Any propellant other than compressed air may irritate the lungs. The dangers posed by the innumerable combinations of ingredients in sprays are still unknown.

Both acid and alkaline mists can produce chemical burns within the lungs; severe exposure can result in fatal edema. The effects of chronic, low-level exposure are not yet proved, but some evidence indicates that such sprays contribute to the development of obstructive lung disease. Many chromium compounds, which are widely used in a variety of industries, are irritant to both the nose and the lungs; some also cause lung cancer. Some of the organic compounds used in sprays—especially toluene diisocyanate (TDI) and methylene bisphenyl isocyanate (MDI), which are used in polyurethane paints—provoke hypersensitivity reactions and can lead to asthma.

Some recently introduced organic compounds, particularly the aromatic ones, probably have latent carcinogenic properties that are still undetected; such cancer-causing properties may be enhanced by other substances present in the sprays. The cutting oil mists formed in machine tool shops are currently under investigation for both malignant and nonmalignant effects on the lungs.

RADIATION

Radiation is forever associated with the atomic bomb and the cities of Hiroshima and Nagasaki. But some people are exposed to radiation in the workplace.

Where Radioactive Materials Are Found

If mine ventilation is inadequate, miners extracting radioactive ores can be heavily exposed to radioactive gases. Uranium is associated with a group of such gases known as radon daughters. There have been instances of increased rates of lung cancer in a few other mines producing non-radioactive materials; in each case, radioactive contamination of either mine air or mine water was discovered.

Radioactive materials are increasingly used in weapons, power plants, industry, and medicine. With the aid of special devices, these materials can be detected through layers of metal or skin. Therefore, radioactive substances are valuable aids in checking industrial processes or physio-

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logical mechanisms that cannot be seen directly. For example, they are used to measure the corrosion inside pipes and to measure the amount of material taken up by an endocrine gland.

Exposure to radioactivity can occur in the extraction, processing, shipping, and end-uses of radioactive materials.

Effects of Radiation

There are different types of radiation: radiation from electromagnetic waves—including ultraviolet, infrared, radar, microwave, laser, and radio frequency waves—and ionizing radiation, which includes radiation from X-rays, gamma rays, and alpha, beta, and neutron particles.

Electromagnetic waves are relatively low-energy forms of radiation, and they have not been considered a significant health hazard, except to unprotected eyes. However, questions have recently been raised about the possible adverse effects of laser, microwave, and radio frequency waves. The full picture has yet to unfold.

Ionizing radiation has more inherent energy than do electromagnetic waves and can damage human tissue. The physiological effects of ionizing radiation are varied. The most important effect is the mutation of normal cells, which allows cancers to develop and may produce genetic defects in the affected person's children.

The lungs are affected principally by radioactive gases, which cause lung cancer. Among those exposed to such gases, smokers have a higher incidence of tumors than do nonsmokers, probably because cigarette smoke acts as a tumor-promoter and enables cancers to develop faster.

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Chapter 4

MAJOR OCCUPATIONAL LUNG DISEASES

In the past decade we have heard a great deal about black lung—the vividly descriptive popular name for a disease that afflicts coal miners. But black lung is only one of a large group of diseases known as pneumoconioses, diseases caused by dust in the lungs, just as coal dust is only one of the many disease-causing dusts that people may inhale on the job. Taken as a whole, the pneumoconioses make up one of the two major groups of occupational lung diseases.

The other major group of occupational lung diseases consists of the hypersensitivity diseases. These diseases—occupational asthma and allergic alveolitis—are caused by the lungs' overreaction to airborne pollutants that workers breathe in while at work.

Falling outside these two categories are three other common occupational lung diseases: byssinosis, a disease that afflicts cotton workers and that resembles the pneumoconioses in some ways and the hypersensitivity diseases in other ways; industrial bronchitis, a disease entity that has aroused a great deal of controversy; and occupational lung cancers, which are showing up in many industries.

PNEUMOCONIOSES

The pneumoconioses were among the first diseases to be recognized as job-related. They were discussed by Hippocrates more than 2,000 years ago, but they became of great importance at the start of the industrial revolution, 200 years ago. It was then that the workers suffering from job-related lung diseases began giving such descriptive names as grinder's

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rot, miners' phthisis, and miners' asthma to the pneumoconioses.

Translated from the original Greek, the literal meaning of "pneumoconiosis" is "dust in the lungs." Not all dusts cause disease, however, and the present-day tendency is to restrict the term to those dust accumulations that provoke a lung-tissue reaction and ultimately either interfere with lung function or shorten the life span. A truly international problem, pneumoconioses beset the employees of mineral industries throughout the world.

Silicosis

More than a million people in the United States are believed to run some risk of developing silicosis, a pneumoconiosis caused by inhaled silica dust. This figure reflects the widespread use of silica, which is by far the commonest material in the earth's crust. Silica occurs in many different forms, such as sand, quartz, tridymite, cristobalite, and kieselguhr.

Silica is used in foundry work, pottery making, and the manufacture of glass, tiles, and bricks. Finely ground silica is used in abrasive soaps, polishes, and filters, and it is especially dangerous when inhaled. Workers in a number of other occupations, such as stonemasonry and sandblasting, also run varying risks of silicosis.

The mining and quarrying of siliceous ores is clearly hazardous, but the risks of developing silicosis extend far beyond the production and use of silica itself. The ores of most other minerals, from coal to gold, are found either mixed with silica or embedded in silica-containing rocks. Sooner or later, silica dust gets into the air of most mines and, therefore, into the lungs of miners. The risks don't stop underground. Anyone working where ores, sands, clays, or soils are transported, milled, ground, mixed, or bagged is likely to inhale silica dust.

MEDICAL ASPECTS OF SILICOSIS. Silicosis occurs in various forms. Which form a worker develops depends on the extent of his exposure to silica.

The commonest form, chronic or classical silicosis, is seen in miners, foundry workers, and other people who have inhaled relatively low concentrations of dust for 10 to 20 years. In the earliest and mildest form of chronic silicosis, the accumulated dust and the tissue reaction result in small, whorl-shaped nodules scattered throughout the lungs. At this stage, these nodules have little effect. The only symptom may be some breathlessness during exercise.

However, chronic silicosis may become more serious—usually when silica exposure continues but sometimes even if the victim changes jobs and is no longer exposed to the dust. Progression of the disease may be

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so slow that the victim enjoys a normal life span with little or no respiratory difficulty. But in other instances, progression is more rapid. The nodules in the lungs enlarge, become more numerous, and coalesce; breathlessness becomes more of a problem and may be accompanied by a cough and sputum production. The more silica there is in a person's lungs and the longer he lives with it, the greater the chances that complicated silicosis, a much more lethal form of the disease, will develop. Statistically, some 20 to 30 per cent of all chronic silicosis victims progress to complicated silicosis.

In complicated silicosis, a fibrosis—progressive massive fibrosis (PMF)—spreads inexorably through the lungs, restricting their function and frequently leading to cor pulmonale (heart disease caused by lung disease). Why fibrosis starts in some lungs and not in others that are equally laden with silica remains a mystery. The victim of complicated silicosis complains of breathlessness, weakness, chest pain, a cough, and sputum—becoming a respiratory cripple and likely to die of heart failure.

Another type of silicosis, acute silicosis, is seen only in people whose occupations entail an intense exposure to silica dust over a relatively short time. For example, acute silicosis is seen in sandblasters, who use a high-speed jet of sand to clean and polish bricks and the insides of corroded tanks; in tunnelers and rock drillers, especially if they drill through sandstone; and in workers who produce finely ground silica. Most people who develop acute silicosis have been exposed to high concentrations of silica dust for several months or longer, but the disease has been reported in people who worked only a few weeks in such conditions.

Acute silicosis is a rapidly progressive disease, currently as intractable as lung cancer and usually leading to severe disability and death within five years of diagnosis. The early symptoms are difficulty in breathing, loss of weight, fever, and coughing. There is an inflammatory reaction within the alveoli and a diffuse fibrosis. X-rays do not resemble those obtained in chronic silicosis, and the whorled nodules characteristic of the chronic disease are usually absent. Unfortunately, many victims of acute silicosis are young, otherwise healthy people.

Tuberculosis was formerly a very common complication of silicosis. It is much less common today. Tuberculosis as a complication of silicosis has declined as tuberculosis has declined in the community at large.

There is extensive research into the association between silica and fibrosis. After silica particles have been ingested by macrophages (as explained in Chapter 2), the macrophages die, and something liberated from the macrophage as it breaks down appears to be able to induce fibrosis. Since the macrophages are also responsible for protecting the lungs from tuberculosis, the silica-macrophage interaction may also ac-

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count for the increased susceptibility of silicotics to tuberculosis.

PREVENTION OF SILICOSIS. Dust control is essential for the elimination of silicosis. The wetting-down of mines and improved ventilation have done much to reduce dust levels. But in other situations much more is needed. For example, sandblasters working in enclosed vessels can be protected only if they wear special suits and breathing apparatuses; some authorities think that such protection is still inadequate.

Coal Workers' Pneumoconiosis (CWP)

Coal miners who have spent much of their working lives underground may be afflicted with coal workers' pneumoconiosis. This disease is popularly called black lung, a nontechnical name that accurately describes what the surgeon finds in the operating room and what the medical examiner finds during an autopsy. The coal miner's lungs are often as black as coal—and for good reason; they are filled with coal dust.

CWP is a potential danger for 150,000 coal miners in the United States today, and the energy crisis and the resulting demand for more coal from America's vast reserves could increase the number at risk. The efficacy of ventilation and other dust-control measures will determine the incidence of CWP in the future.

Many studies were designed to determine what percentage of miners develop CWP and how severe the condition becomes. The results have shown that the prevalence of CWP and its severity vary from one mine to another, even in regions producing the same type of coal. The amount, size, and nature of the dust in the air vary according to the type of coal mined, the machinery and techniques used, the efficiency of ventilation and other dust-control systems, and the type of rock surrounding the coal seam. The large number of variable factors probably accounts for the wide range of estimates on the prevalence of CWP; the simple form of the disease presently affects an estimated 10 to 30 per cent of all coal miners. In general, CWP seems to be more prevalent in miners of anthracite or hard coal than in miners of bituminous or soft coal.

Coal is not the only mineral found in coal miners' lungs. Silica, kaolin, mica, beryllium, copper, cobalt, and others have been reported. Whether any of these materials contributes to the development or the progression of CWP isn't known. Silicosis is occasionally seen in coal miners, usually in those mining anthracite coal—a coal that occurs in comparatively irregular seams, with the result that miners burrow more frequently into the silica-containing rocks surrounding the seam.

Before discussing the medical aspects of CWP, we should clarify the term "black lung." "Black lung" is a legal term, one applied to coal

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Coal dust breathed in by the men and women who work in coal mines accumulates in their lung tissues. For some of them the result is coal workers' pneumoconiosis.

miners who have a respiratory condition that is legally compensable. The term has no medical significance.

MEDICAL ASPECTS OF COAL WORKERS' PNEUMOCONIOSIS. Simple CWP is the earliest and commonest consequence of the continued inhalation of coal dust. The dust accumulates in the lungs and can be seen on X-rays; the heaviest deposits are around the respiratory bronchioles. Over the years, as more coal dust piles up, some of the respiratory bronchioles become dilated, a condition called focal emphysema.

Unless other lung conditions are also present, simple CWP causes virtually no symptoms and no respiratory difficulty. Different groups of miners have shown some slight changes in various respiratory mechanics, but no consistent pattern has emerged. Simple CWP alone doesn't shorten the life span, and comprehensive studies have shown that coal dust inhalation alone carries no increased risk of lung cancer.

In about 3 per cent of all simple CWP victims, a more serious type of disease develops, one known as progressive massive fibrosis (PMF) or complicated CWP. In this disease, fibrosis begins to develop in some of the dust accumulations. Each fibrotic area spreads and then merges

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with other fibrotic areas; eventually, most of the lungs are filled with stiffened, useless tissue. This PMF considerably shortens the life of its victim, who may die from respiratory failure, cor pulmonale, or severe infection.

Complicated CWP most often afflicts miners with heavy deposits of coal dust in their lungs, but from time to time it occurs in miners with less coal dust in their lungs. How and why progressive massive fibrosis gets started is a medical mystery and a research challenge. It may appear suddenly, years after a miner has left the mines; it does not necessarily strike those who have the heaviest deposits of coal in their lungs; and, perhaps most surprising of all, it sometimes stops suddenly for no discernible reason.

For a long time, fibrosis in coal miners was attributed primarily to silica in the lungs. Nowadays, silica is not considered the principal agent but is thought to play some role in the fibrotic process—a process that still defies explanation, despite extensive international research.

Smoking has an adverse effect on the overall health of a miner's lungs, although it apparently has little effect on the development of CWP itself. Cigarettes cause chronic bronchitis and emphysema in miners, just as they do in nonminers. Sophisticated tests have shown that nonsmoking miners have a slight degree of lung obstruction and only minimal disturbance in the way their lungs fill with air; both of these changes are five to six times more marked in miners who smoke.

PREVENTION OF CWP. Dust control is of paramount importance in coal mining. Reduced levels of coal dust can lower the percentage of miners afflicted with simple CWP (now estimated at 10 to 30 per cent), reduce the number of miners with heavy accumulations of coal dust in their lungs, and thereby probably reduce the number of miners likely to develop complicated CWP.

Asbestos-Related Lung Diseases

Asbestos is a versatile product, unsurpassed as a durable fireproofing and insulating agent. It has been mixed into paints and cements, wrapped around water pipes, compressed into floor tiles, used to line furnaces and air-conditioning ducts, and woven into theater curtains, hot pads, and ironing board covers. It has even been used to filter soft drinks and medicines.

But asbestos, that durable and valuable material, is one of the most dangerous occupational hazards. It can cause both fibrosis and cancer in those who work with it. Moreover, the dangers of asbestos are not confined to the workplace. It pervades the general environment because it was extensively used before its health hazards were realized.

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Asbestos dust, like the dusts of many other mined materials, clearly jeopardizes those who mine the ores and process the crude material into pure form. But, although asbestos deposits do exist in the United States, very little of the world's supply is mined here. Instead, Americans are endangered mainly by the processing and end-uses of asbestos.

Asbestos is spun, woven into fabrics, compressed into blocks and tiles, and purchased by a variety of industries for inclusion in countless products. An unknown number of workers are exposed to asbestos in manufacturing industries. It is used in insulating materials, paints, cements, clutch and brake linings, and innumerable other products.

Processed asbestos is used in many trades and occupations. Heating and insulation workers, electricians, and clothing ironers are frequently exposed to asbestos. The exposure of construction workers, shipbuilders, decorators, and painters is more sporadic but likely to be intense. Fortunately, the once-common practice of applying asbestos paints and insulating material by spraying, which exposed both workers and passersby to high concentrations of the material, has been largely but not completely discontinued.

The durability for which asbestos is prized makes it a hazard for many people who do not work directly with the material. The asbestos that was incorporated into walls, floors, siding, and insulation decades ago is still there today; no one knows exactly where it is or what form of asbestos was used. Demolition workers, other crafts workers, and do-it-yourself home owners intent on destroying or renovating old structures are quite likely to inhale asbestos. Public buildings—schools in Wyoming and New Jersey, university buildings in Connecticut and California, for example—have been temporarily closed because asbestos was found to be flaking from the walls or ceilings.

For a long time after asbestos exposures were controlled in mining and the principal asbestos-processing industries, asbestos usage in such products as textured paint and spackling compounds was largely uncontrolled. Federal agencies and many state governments are now moving to place much stricter controls on all uses of asbestos.

MEDICAL ASPECTS OF ASBESTOS-RELATED DISEASES. Asbestos-related diseases have been the subject of a good many epidemiological and animal studies. These studies have shown that asbestos poses different dangers to different groups of people. Which danger a particular person faces depends on the quantity of asbestos inhaled and the nature of the asbestos fibers found in the ore handled. Asbestos occurs in the earth's crust in several different forms or ores. They have in common a densely interlocking, fibrous structure, which makes them virtually indestructible. Commercially, the most important ores are chrysotile, croci-

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dolite, and amosite. Medically, the most hazardous of these asbestos ores are crocidolite and amosite.

Asbestosis, the fibrosis caused by asbestos, develops after exposure to all types of asbestos fibers, although chrysotile may be somewhat less fibrogenic (fibrosis-causing) than are the other types of asbestos. The inhaled fibers accumulate around the lungs' terminal bronchioles. The body, in an effort to isolate the fibers, surrounds them with iron-rich tissue, producing an asbestos body with a characteristic appearance. As more fibers are inhaled, more asbestos bodies appear in the lungs. Eventually, usually after 10 to 20 years of exposure to asbestos, fibrosis begins in the lungs; if exposure is heavy, fibrosis may appear in only four or five years. The victim's symptoms, which begin some time after the onset of fibrosis, include a cough, sputum, weight loss, and increasing breathlessness. The disease usually kills its victim within 15 years of the onset of symptoms.

Today, almost every asbestos miner with more than 20 years of exposure to the material has asbestosis to some degree. Present evidence indicates that the disease is related primarily to the total amount of dust inhaled and that it develops soonest in workers who are heavily exposed to asbestos.

Lung cancer is associated with all types of asbestos. Between 20 and 25 per cent of deaths among workers heavily exposed to asbestos are due to lung cancer. The disease is related to the degree of asbestosis present in the lungs and also to cigarette smoking, which greatly enhances the carcinogenic (cancer-causing) properties of asbestos. The asbestos worker who smokes is estimated to be 90 times as likely to get lung cancer as a smoker who has never worked with asbestos.

Mesothelioma, a cancer of the pleura (chest lining), was almost unknown before asbestos appeared on the scene. Now it is estimated that this disease, which is inoperable and always fatal, will attack 7 to 10 percent of asbestos workers.

Most cases of mesothelioma occur in people exposed to crocidolite fibers of a certain size, but a few cases occur in handlers of amosite fibers. Crocidolite fibers are fine and straight; the fibers found in other ores are fatter or coiled in shape. The needlelike shape of crocidolite fibers seems to enable them to pass right through the soft lung tissues to the rib cage, where they remain and initiate cancer.

One of the nastier features of this disease is that it can occur after very little exposure to crocidolite. Mesothelioma has been reported in the nonworking wives of asbestos workers and in people living near plants but not working in them. Cigarette smoking doesn't seem to be a contributing factor. *All that's needed are a few fine, straight crocidolite fibers.*

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Auto repair workers in poorly ventilated garages are subjected to carbon monoxide, other car fumes, and solvent vapors; those who work on brake linings encounter asbestos.

There is a strong case for leaving crocidolite undisturbed and underground.

Asbestos workers also have a higher-than-average rate of other cancers, notably of the esophagus, stomach, and intestines. The swallowing of asbestos-contaminated mucus that has been cleared from the lungs is probably responsible for these cancers.

PREVENTION OF ASBESTOS-RELATED LUNG DISEASES. In spite of the present recognition of the hazards associated with asbestos and in spite of strict dust-control measures, the number of asbestos-related diseases has been increasing. This rise probably reflects the increasing use of asbestos, better recognition and reporting of these diseases, and the difficulty of controlling asbestos exposure in people who use the substance only occasionally or unknowingly. Much tighter controls on all uses of asbestos are clearly essential. Also essential is a determined policy of using substitute materials wherever possible. Some countries have already taken such steps, which are strongly advocated in the United States. The potential harm caused by asbestos that is already in the environment can be minimized only by a massive effort to educate the general public to its dangers.

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Other Pneumoconioses

Many other dusts accumulate in workers' lungs. On the basis of present evidence, a number of these dusts are currently regarded as benign—that is, their accumulation does not seem to produce lung disease. Barium, pure iron, tin, and titanium dusts fall into this category. But a number of other dusts do have recognized adverse effects on lung health, especially aluminum, beryllium, carbon black, fiberglass, fuller's earth, kaolin, mica, talc, and tungsten carbide.

Aluminum, a widely used metal, seems to be dangerous when its finely powdered pure metal is inhaled, notably in the manufacture of fireworks and explosives, and when fumes of aluminum oxide are produced in the making of alumina abrasives, especially corundum. The dust accumulates in the lungs, and fibrosis follows in a number of cases. In most other circumstances, aluminum is harmless to the lungs; only inhaled particles of a certain size seem to induce disease.

Beryllium, a light but strong metal, is used in metallurgy, certain machine tools, the making of ceramics, and the nuclear power industry. Before its toxicity was recognized, it was widely used in fluorescent light tubes. The greatest risk of exposure today is probably in the plants that extract pure beryllium from the crude ore.

Beryllium affects most body systems. In the lungs, its main effects are a severe chemical pneumonia after an acute exposure and a chronic form of disease, berylliosis, after recovery from an acute exposure or after repeated exposures to low levels of beryllium dust. Chronic berylliosis consists of difficulty in breathing, diffuse fibrosis over a period of 15 to 20 years, and, finally, cor pulmonale. The disease seems to be one of hypersensitivity, for the level of exposure does not necessarily correlate with development of the disease. Beryllium exposure is also associated with increased rates of lung, liver, and gall-bladder cancers.

Carbon black, used in the rubber industry and printing inks, is produced by burning certain organic materials. Graphite, the form of carbon used in lead pencils, is a naturally occurring ore that is also used in steel production, as a lubricant, and in electrical generators. Prolonged inhalation of either dust produces a pneumoconiosis that is identical to coal workers' pneumoconiosis in both its simple and its complicated forms.

Fiberglass is known to be a skin irritant, but it is also under investigation as a possible lung hazard. Although animal tests have shown that fiberglass can cause cancer when implanted into the pleura (chest lining), extensive work has failed to produce any evidence that working with fiberglass carries an increased risk of either lung cancer or pleural cancer in humans.

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Fuller's earth, an absorbent clay, is used in foundry molds and oil refining. Its dust may be inhaled during the mining and transportation of the crude ore when the dried clay is ground to a powder. The pneumoconiosis produced by fuller's earth is fairly benign, but some cases of progressive massive fibrosis have been reported.

Kaolin, a silicate obtained by quarrying, is ground to a fine powder and used in ceramics, papermaking, paints, and the pharmaceutical industry. Pneumoconiosis is rarely seen, but a high percentage of victims do develop progressive massive fibrosis.

Mica, another silicate, is mined, ground, and used in the electrical and furnace industries and in the manufacture of paint and paper. Mica seems to produce a degree of pneumoconiosis in people who handle it, and a few cases of fibrosis have been recorded.

Talc is inhaled by those who mine and mill the crude ore, usually soapstone, and by workers in the cosmetic, paint, pottery, asphalt, and rubber industries. Pure talc produces a characteristic pneumoconiosis. Less fibrosis is seen after talc inhalation than after silica inhalation, but an evaluation of the fibrosis risk is difficult because most commercially used talc contains traces of both asbestos and silica, each of which can induce fibrosis. There is a high incidence of lung cancer among people exposed to talc, but whether this high rate is caused by talc or by asbestos contamination, as well as increased by smoking, is not known.

Tungsten carbide manufacture involves the fusing of tungsten and carbon, with cobalt used as a binding agent. Workers processing the alloy suffer from a hard-metal disease that seems to have two distinct components: a pneumoconiosis with some risk of fibrosis and an obstructive disease that seems to be some type of allergy, manifested by wheezing, coughing, and breathlessness. Experimental work indicates that both these diseases may be caused by cobalt, rather than by tungsten or carbon.

Mixed-Dust Pneumoconioses

Many workers are exposed to a mixture of dusts. Foundry, steel, and iron workers encounter dusts from a variety of ores and other ingredients, and they may also inhale fumes. Miners are often exposed to mixed dusts in compound ores and as they dig through one mineral bed to reach another. And many workers who are exposed to one dust on a certain job may change jobs and begin to inhale a different dust.

A pneumoconiosis caused by a mixture of dusts can be very difficult for a physician to diagnose. Individual dusts, when inhaled alone, usually deposit in patterns that can be recognized on chest X-ray films. But when mixtures are inhaled, the patterns are different. Knowledge of the patient's working history, important in diagnosing all occupational lung diseases,

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plays an even greater role in the diagnosis of mixed-dust pneumoconioses.

Whether the effects of the mixed dusts in the lungs are additive (one plus one equals two) or potentiating (one plus one equals five) is largely unknown. Not enough people have been studied, and the variable factors are numerous. However, as a general rule, the amount of fibrosis present in a mixed-dust pneumonoconiosis depends on how much silica has been inhaled.

HYPERSENSITIVITY DISEASES

Many people suffer from asthma and allergies, but these hypersensitivity diseases fall into the occupational category only when the antigen (the agent causing the overreaction) is found primarily in the workplace, rather than in the home or the general environment. Pollen allergy, for example, is not an occupational disease in an office worker, but it is an occupational disease in a grain farmer; and asthma provoked by dog hair is an occupational disease in a kennel worker but not in a miner or a dentist.

Lung hypersensitivity reactions can occur in the bronchi (the large air passages), in the bronchioles (the smallest airways), or in the alveoli (the terminal respiratory sacs). In general, coarse dusts tend to evoke bronchial reactions, and fine dusts provoke small-airway and alveolar reactions.

Occupational Asthma

Occupational asthma, an overreaction of the bronchi and bronchioles to some airborne pollutant, can happen to anyone, but it is seen more often in certain people and certain industries than in others.

About 10 per cent of the population have a genetic tendency toward allergies; hay fever and asthma tend to run in their families. Such people are called atopic, and they are more likely to develop occupational asthma than are nonatopic people.

Lung hazards also vary in their ability to provoke hypersensitivity reactions. Some hazards are called highly antigenic because they have a marked tendency to produce allergic reactions.

As a result, atopic persons working with highly antigenic substances are the ones most likely to develop occupational asthma, often in less than five years. But nonatopic persons can also become sensitized to occupational lung hazards, especially if the hazards are highly antigenic.

Substances noted for their antigenic properties include detergent enzymes, platinum salts, cereals and grains, certain wood dusts (western red cedar and species of pine, birch, and mahogany), isocyanate chemicals

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used in polyurethane paints and other products, several agents in the printing industry, and some pesticides. It is probably advisable for people with allergic tendencies to avoid jobs that involve the handling of known antigens.

MEDICAL ASPECTS OF OCCUPATIONAL ASTHMA. The main symptom of occupational asthma is wheezing. The patient struggles for breath and often coughs up a sticky mucus as the attack ends. His symptoms, which are caused by the constriction of his air tubes, begin only in a certain work area or when he uses a particular agent. Once away from the provocative antigen, he has no respiratory problems—at least not in the early stages of the disease. Repeated attacks, especially if the cause is unrecognized and the asthma is untreated, may lead to permanent obstructive lung disease and eventual disablement. Once an asthmatic response becomes well-established, similar symptoms may be provoked by other factors, such as house dust and cigarette smoke, and by fatigue, breathing cold air, and coughing.

PREVENTION AND TREATMENT OF OCCUPATIONAL ASTHMA. The simplest and most effective treatment of occupational asthma is complete avoidance of the responsible antigen. Attacks caused by inadvertent exposure can be treated by bronchodilator drugs, antihistamines, and steroids, but the patient should not rely on these drugs for continuous control. Desensitizing treatments have not been notably successful. Nothing short of total elimination of the hazard from the air will prevent asthma attacks in those who are already sensitized to a particular hazard, for hypersensivity reactions can be provoked by minute traces of the antigen.

Allergic Alveolitis

Even the farm, supposedly one of the healthiest places to work, is not immune to occupational lung diseases. Farmer's lung is the best-known example of allergic alveolitis, a hypersensitivity disease caused by fine organic dust that is inhaled deep into the lung's smallest airways. Farmer's lung is caused by moldy hay. Other dusts known to cause allergic alveolitis include those from moldy sugar cane and barley, maple bark, cork, animal hair, bird feathers and droppings, mushroom compost, coffee beans, and paprika. Often, the disease is named for its cause—mushroom-worker's lung, paprika-splitter's lung. But in many cases, fungus spores growing in the apparent antigen are thought to be the real cause of the disease.

In recent years, the isocyanate chemicals, which are known to induce asthma, have been recognized as additional causes of alveolitis. It is likely that other chemicals will be associated with this disease in the future.

MEDICAL ASPECTS OF ALLERGIC ALVEOLITIS. The symptoms of allergic alveolitis begin some hours after exposure to the offending dust.

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The patient feels tired and short of breath, and has a dry cough, fever, and chills. Inside his lungs, the alveoli are inflamed, inundated by white blood cells, and sometimes filled with fluid. His symptoms may last from one to 10 days. Sometimes the symptoms are serious enough to warrant emergency treatment and hospitalization.

If exposure to the causative dust is infrequent or the dust concentration is low, the symptoms may be so mild that the worker does not seek treatment or realize that he has a problem. When that happens, a chronic form of allergic alveolitis smolders unnoticed for some time. The alveoli and surrounding tissues become mildly inflamed, and eventually fibrosis may set in. By the time the victim has serious symptoms, the fibrosis may be so well-established that the disease cannot be arrested.

TREATMENT OF ALLERGIC ALVEOLITIS. Acute attacks of allergic alveolitis can be treated with steroids and other drugs. However, recovery may take as much as six weeks, and the patient may suffer from residual lung damage even after his recovery. The real cure, of course, is permanent separation of the patient and the antigen. This separation does not necessarily mean leaving agricultural work. Properly dried and stored farm products—hay, straw, sugar cane—do not cause allergic alveolitis, presumably because fungi grow only in moist conditions.

BYSSINOSIS

Byssinosis is an occupational disease which occurs particularly in the primary textile industry. It is best known in cotton workers, but it also afflicts those in flax and hemp industries.

The early symptoms of byssinosis occur only on Mondays—or the first day back at work after a vacation or long weekend—and disappear on subsequent workdays. Hence, the layman's term "Monday fever." Medically, this term is misleading for two reasons: First, it implies a rise in temperature, which is not a feature of byssinosis; second, it gets confused with mill fever, a true fever seen occasionally in certain textile workers and probably caused by a microorganism that thrives on damp fibers. Another popular term for byssinosis is "brown lung."

In the cotton industry, the most commercially important natural textile industry in the United States, the cause of byssinosis is found in the bales of raw cotton. These bales contain mostly cotton fibers, but they also contain some fragments of the cotton plant's parts, including the bract, which surrounds the cotton boll. And something in the other plant matter, rather than in the pure cotton, is the main cause of byssinosis.

The disease is most prevalent among workers involved in the early stages of processing, where the bales of raw material are opened, combed,

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and carded. Workers involved in the later stages, in the spinning and weaving rooms, are less often afflicted unless the ventilation system allows fine dust from the combing and carding rooms to permeate into these areas. The people who run the greatest risk of developing byssinosis are those in the opening rooms of cotton mills and those who clean the machinery in the combing and carding rooms. Their work stirs up and dislodges the raw cotton dust, exposing them to heavy concentrations of the disease-causing material.

Medical Aspects of Byssinosis

The onset of byssinosis is characterized by a feeling of tightness in the chest on returning to work after a weekend. The tightness may last only one day, and the victim may feel fine for the rest of the week. This Monday symptom may develop after working only a few months in the industry or after many years.

There is a strong relationship between the amount of dust breathed in and the symptoms.

The problem is that Monday's symptoms eventually may persist throughout Monday and Tuesday. Gradually, the victim's troubles extended throughout the working week. Later, he can get no relief, even when away from work. A worker who leaves the textile industry before chronic bronchitis and emphysema set in may regain a fairly healthy set of lungs. But if he does develop chronic bronchitis and emphysema, his breathing difficulties become continuous, his chest hurts, and he has a persistent productive cough. Then he leaves the textile industry as a respiratory cripple, never to regain lung health.

Byssinosis is a curious disease from a medical point of view. It has been classed as both a pneumoconiosis and a type of asthma. But nowadays it is recognized as a disease entity in its own right. As in the pneumoconioses, the textile dusts do accumulate in the lungs, but the disease pattern in byssinosis differs considerably from that normally seen in the pneumoconioses.

And, although the symptoms of byssinosis do resemble those of asthma in some respects, byssinosis also differs significantly from the classic hypersensitivity diseases. As in asthma, the sensation of chest tightness characteristic of byssinosis results from the constriction of the bronchi and bronchioles in response to something in crude cotton dust. However, the symptoms in asthma and allergy do not disappear while exposure to the antigen continues, but the symptoms of byssinosis do disappear—at least, in the early stages.

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Some people never develop Monday fever; others get it soon after starting work; still others get it only after many years on the job. Many cotton workers suffer only mild symptoms for many years and don't get any worse; in others, the disease worsens within a few years.

Prevention of Byssinosis

Dust control measures can significantly reduce the incidence of byssinosis. It may become possible to detect people who are likely to become sensitized to cotton dust by pretesting techniques; such people could then be kept out of the high-risk areas. Handpicking of cotton, rather than machine-picking, results in fewer pieces of bract in the bales and less byssinosis in mill workers, but this is not a practicable approach for obvious economic reasons. Pretreating bales of cotton by washing with steam or other agents may inactivate the causative agent in some way and help to reduce the incidence of byssinosis.

INDUSTRIAL BRONCHITIS

Industrial bronchitis is a controversial topic. Physicians disagree about whether it exists at all and about whether it can truly be designated an occupational disease.

Bronchitis is an accepted and well-defined disease entity. The symptoms are a persistent cough and the production of sputum, which is caused by the oversecretion of mucus within the lungs. Bronchitis is a generalized response of the lungs to any form of continued irritation—cigarette smoking, general air pollution, dusts, irritant gases, vapors. Since it has so many causes, bronchitis and chronic bronchitis are usually termed nonspecific responses of the lungs to irritation.

A number of authorities think that many cases of bronchitis are caused mainly by workplace pollutants that irritate the lungs; therefore, they hold that such cases are really occupational and should be called industrial bronchitis. Other authorities think that so many hazards contribute to the disease that bronchitis can seldom, if ever, be proved to be purely or even mainly occupational in origin. Moreover, the effects of cigarette smoking overwhelm the contributions of other factors to the disease.

Two observations are indisputable: The prevalence of bronchitis is much higher in industrialized areas than in rural areas, and occupational lung hazards do contribute to the development of bronchitis. Even if cigarette smoking and its effects could magically be banished, together with all air pollution outside the workplace, it is possible that people working in polluted areas would still develop bronchitis.

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OCCUPATIONAL LUNG CANCER

Cigarette smoking is acknowledged to be the single most important cause of lung cancer, but a considerable amount of evidence now indicts workplace air pollutants as significant causes and contributing causes of cancer. One estimate is that between 70 and 90 per cent of all cancers are caused or aided by environmental factors, including life-style, dietary factors, environmental and occupational exposures. Although the contribution made by each of these factors is hard to determine, Nicholas Ashford, in a Ford Foundation report on occupational disease and injury (*Crisis in the Workplace*, M.I.T. Press, 1976), reported a general consensus among cancer researchers and environmentalists that occupational factors probably contribute to about half of these cases.

Many cancers, including lung cancer, occur more frequently in industrialized and urban areas than in rural areas. And studies of workers in specific occupations have shown that lung cancer rates are above average in occupations that involve the handling of arsenic, bis-chloromethyl ether, coal tar and pitch volatiles, petroleum, mustard gas, coal carbonization products, chromates, asbestos, X-rays, radium, uranium, nickel, and isopropyl oil. Vinyl chloride gas and mineral-cutting oils are currently under investigation as possible causes of lung cancer. Chemists, painters, and printers also seem to have an increased risk of lung cancer, but the specific carcinogens have not yet been identified.

Role of Cigarette Smoke

The role of cigarette smoke in causing lung cancer cannot be overstated. Alone and unaided cigarette smoke is a potent cause of lung cancer and cancers of other sites. When the effect of cigarette smoke is combined with the effects of occupational carcinogens, the incidence of lung cancer rises dramatically. In several of the jobs, trades, and professions found to be associated with a high risk of lung cancer, there is considerable debate among experts as to whether the occupational carcinogen can, in fact, be proved to cause lung cancer in the absence of cigarette smoke as a co-carcinogen.

Medical Aspects of Occupational Lung Cancer

There are several different types of lung cancer, each an abnormal mass of cells with an uncontrolled growth pattern. Lung cancer is one of the more intractable cancers—in part because its symptoms seldom appear until the disease is well-advanced. Even periodic X-rays may not detect certain types of lung cancer until the disease is inoperable.

More than half of all occupational lung cancers are small-cell carcinomas, a type that has the poorest response to any form of treatment.

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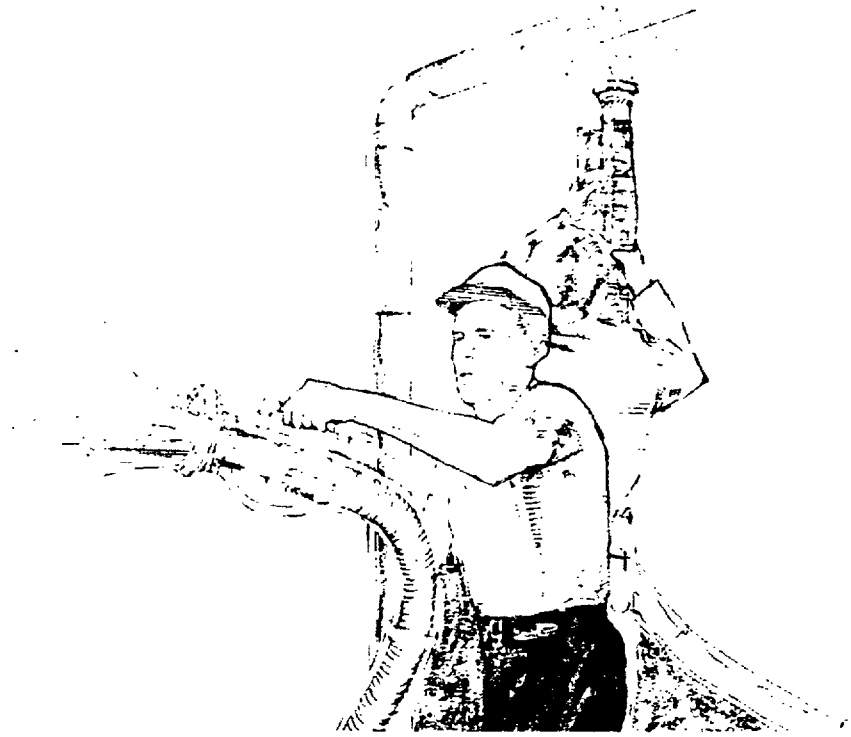

Asbestos is most dangerous to workers who mine it and handle it in the making of asbestos products. Using the products has its dangers wherever dust is created, as in spraying asbestos insulation or blowing away brake-lining dust in a machine shop.

Many cases also occur in lungs already damaged or diseased by occupational hazards—further reducing the likelihood of cure.

Prevention of Occupational Lung Cancer

The most important point about lung cancer is that it is largely preventable but very seldom curable.

Cigarettes are the single most important factor in lung cancer. People can greatly reduce their chances of contracting this disease by avoiding or giving up smoking; the lung cancer rate for heavy smokers is higher than the rates for light smokers and ex-smokers, and the rate for light smokers is much higher than that of nonsmokers.

Occupational factors can undoubtedly contribute to the development of lung cancer, and carcinogenic substances must be strictly controlled if they cannot be replaced by safer materials. Continual medical surveillance is essential, both to verify that existing standards are effective in preventing cancer and to detect previously unrecognized carcinogens.

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Chapter 5

CONTROLLING HAZARDS

When lung hazards and their resulting diseases are identified, what then? Few people can afford to quit their jobs, even if hazardous. And few industries can afford to close down hazardous processes. Does that mean that everyone simply has to accept lung diseases?

Not at all. The answer lies in controlling lung hazards in the workplace.

Hazard control can be approached in many ways. For the purpose of this handbook, the approaches have been simplified into three categories: on-site hazard controls, statutory controls, and hazard monitoring.

On-site hazard controls are the practical technological and operational measures designed to reduce the amount of hazard that actually gets into people's lungs.

Statutory controls are the legislative and regulatory actions designed to ensure that on-site control measures are put into effect and that hazards are reduced to acceptable levels.

Monitoring is the surveillance of hazards and the effectiveness of hazard controls. It includes checking the actual levels in the workplace and the physiological effects of continued exposure. The information gleaned by monitoring is essential to the evaluation of both on-site and statutory controls.

ON-SITE HAZARD-CONTROL MEASURES

Physically keeping hazards out of workplace air is not necessarily complicated or expensive—although it may sometimes be both. Hazard-control systems can have secondary benefits that help offset the costs involved—pleasanter working conditions may mean less absenteeism, formerly wasted

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materials may be recovered and economically recycled.

How a hazard is controlled depends on many factors: the nature of the hazard (gases, for example, cannot be contained as easily as dusts); how the hazard is produced (dusts arising from ores or powders being poured into hoppers cannot be handled in the same way as dusts flying horizontally off a grinding wheel at high speed); and whether people have to work close to the hazard source (vapors from a solvent tank constantly used for hand-dipping operations cannot be controlled in the same way as vapors from a tank used only for solvent storage).

Hazard-control systems are as varied as the hazards produced by a modern industrialized society. They may be applications of modern technology, like vast ventilating systems incorporating air-cleaning or scrubbing devices. Or they can be time-honored practices, like storing only thoroughly dried hay or sugar cane, weather permitting. Or they can be a mixture of long-used measures and modern technology, which are combined in modern continuous-mining machines. These machines churn out many tons of coal a minute and generate vastly more dust than did old pick-and-shovel methods, but they simultaneously spray down the dust with a fine water spray, a method of dust control known for centuries.

There are three basic approaches to on-site hazard control:

Operational procedures—changes in machinery, ingredients, or work practices that eliminate, reduce, or better contain a hazard or separate people from the hazard.

Ventilation systems, which remove hazards after they are generated, thereby reducing exposure and preventing hazard build-up.

Personal protective equipment, which protects an individual worker from a hazardous environment that cannot be otherwise controlled.

Sometimes one of these approaches alone controls a hazard. Sometimes a combination of methods is more effective, more practical, or more economical.

Operational Procedures

A variety of on-site changes can improve hazard control. The possibilities are probably best indicated by a few examples.

Sometimes a hazard can be reduced or eliminated completely by changing a process. The dust generated during the handling and transporting of a dry ore can be minimized if the ore is handled, instead, as a wet slurry. Loose powders can be handled in solution or as compressed pellets or briquettes. Using electrostatic techniques for spray coatings greatly reduces the amount of spray escaping into the surrounding air.

Another way to avoid or reduce a hazard is to substitute safer materials. Steel shot or steel grit may be used instead of silica sand in abrasive

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blasting. Sandstone grinding wheels can be replaced by wheels of boron nitride or other synthetics. Toluene can often be used instead of the more toxic and highly volatile benzene.

Changing a process or an ingredient to deal with one hazard is not necessarily a simple decision. Joining metals by riveting instead of welding eliminates lung-damaging fumes and gases but introduces a noise hazard that must be dealt with. And substituting a safer ingredient in a process may not eliminate all risk.

Equipment changes may be possible. For example, forklift trucks powered by electricity or liquified petroleum gas generate less carbon monoxide and other exhausts than do gasoline-powered trucks and are, therefore, preferable in confined or badly ventilated areas.

In many instances, improving the way hazards are contained can markedly affect hazard levels. The manual batch-filling of vats and hoppers can be replaced by continuous or automatic filling, in which the vessels remain closed, preventing the escape of dusts or vapors into the general atmosphere. Tanks of solvents or other hazardous liquids can be closed or covered with a layer of foam or inert oil. Premeasured, bagged ingredients can be used instead of bulk powders. Asbestos fibers can be transported inside disposable plastic bags, dampened inside the bags, and subsequently handled as a paste, thus reducing the number of fibers in the air.

Physically separating people and hazards is sometimes a practical approach. Highway toll collectors and overhead-crane operators in foundries are enclosed in protective booths. Sometimes it is easier to enclose a process. Small-scale abrasive and polishing operations, for example, can be done inside cabinets, the operators remaining outside and working through gloves that project into the cabinet.

Hazardous processes can often be isolated from other work areas, so that no one is needlessly exposed to a hazard. Plating and dipping tanks, for example, can be located in a separate, well-ventilated area away from other work areas. Two or three cans of a degreasing agent or other solvent stored alongside various workbenches can be relocated in a central, well-ventilated cabinet, where exposure to the vapor is periodic, rather than constant.

Administrative decisions can sometimes do much to reduce the effect of a hazard. For example, scheduling underground blasting or foundry shake-outs for the end of the day or the last shift can allow the dust produced to settle or be removed before most people return to the area. Nonessential workers should leave an area that is about to become hazardous, such as a tank where a welding repair is about to be made.

Reducing the amount of hard manual work that must be performed

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around a hazard can also benefit the lungs. In an eight-hour shift a sedentary worker inhales about five cubic meters of air, much of it filtered through the nose. Someone who is laboring hard for the same time period inhales about 20 cubic meters of air—much of it through the mouth—thereby exposing his or her lungs to more pollution.

As this discussion shows, there are many ways of reducing hazards by changing the way in which things are done. Ingenuity and a modest investment can frequently bring major health benefits. More than one occupational health specialist has commented that the people who are actually exposed to a hazard can often make the most practical suggestions on how to control that hazard efficiently.

Ventilation

There are two basic approaches to ventilation: general ventilation (overall ventilation of the workplace) and local exhaust ventilation (on-the-spot extraction of polluted air from a hazardous process or machine). Each approach has limitations, and frequently the two methods of ventilation are used together to ensure a healthy workplace.

GENERAL VENTILATION. Some general ventilation is needed in every building, from homes to factories, to ensure the replacement of stale air. For some businesses, city and state codes specify how often the air in a room must be changed. The specifications usually depend on what the room is used for, ranging from once every one to three minutes for boiler rooms to once every five to 10 minutes for warehouses.

Besides replacing stale air, general ventilation can also remove health hazards. General ventilation can be relied on only when hazards of very low toxicity are produced at a more or less constant rate. Whenever low levels of a hazard are toxic or a build-up of hazard levels would endanger life or health, additional means of hazard control, such as local exhaust ventilation, are absolutely essential.

Whether it serves a single room or many miles of underground tunnels, a good general ventilation system doesn't just happen; it needs careful, knowledgeable planning. The essential elements include a planned source of clean, fresh air; a fan system capable of changing the air as often as necessary; a correlation between work operations and air currents that ensures that air flows first through clean areas of the plant and last through areas where hazard generation is heaviest (and always to the worker first and over his work area later); and an exhaust outlet located so that the discharged air does not re-enter the system. The discharged air may have to be passed through an air-cleaning system to prevent occupational hazards from entering the general environment.

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Because heating, cooling, and conditioning air to provide comfortable working conditions is expensive, some air is often intentionally recirculated. Industrial hygienists discourage recirculation unless the air is passed through a high-efficiency cleaner and air quality is automatically monitored. Without such precautions, hazard levels can rise undetected.

The only way of knowing that a general ventilation system is handling hazards effectively is to constantly check—or monitor—the quality of the air, as outlined later in this chapter.

There are many reasons for inefficient ventilation. A system may have been badly designed from the beginning. Common faults are: no planned source of clean air, air intakes close to exhaust outlets, workplace air currents that take hazards into workers' faces or into cleaner work areas, fans too small to move enough air. Or an initially well-designed and adequate system may have been overloaded as business expanded or operations changed.

The commonest cause of poor ventilation is neglect of the system, which is usually accorded a low priority on maintenance schedules. Grills become clogged, ducts fall apart, fans stop working or run slowly. Lung hazards build up, and the lungs suffer.

LOCAL EXHAUST VENTILATION. When a hazardous substance is produced in fits and spurts, rather than at a steady rate, or is produced in large amounts, general ventilation alone is not enough. It must be supplemented by local exhaust ventilation to remove the hazard as close as possible to the point at which it is generated, so that the hazard cannot escape into the general air in the workplace.

The variety of exhaust systems on the market parallels the variety of hazards produced in today's industrial society. The design and placement of the system must be tailored to both the properties of the hazard and the way it is generated. And, of course, the hazard handled by the machinery should not itself corrode or otherwise damage the system controlling it.

Most local exhaust systems have three basic elements: a hood to catch the hazard, a duct to carry it away, and a fan to draw air over the hazard and carry it into the duct. A properly planned local exhaust system draws clean air—that is, air not already polluted by some other process—first into the workers' breathing zone, then over the hazard-generating area, and finally into the exhaust duct. Regular inspection and maintenance of local exhaust systems is essential if they are to operate effectively.

Last but by no means least important is the fact that excellently designed and well-maintained ventilation systems—both general and local—are absolutely useless unless they are turned on.

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Personal Protective Equipment

A respirator is a portable device worn by an individual worker for protection against hazardous air. A respirator should be used only when no other means of protection can be reasonably devised or as a temporary expedient during repairs or while other hazard-control systems are installed. Respirators should be viewed as a last resort, rather than a quick and relatively inexpensive fix for hazardous conditions.

In the situations in which respirators must be used, one of the main problems is their unpopularity. Most people find them bothersome and uncomfortable, especially if they must be worn for more than short periods of time. Unfortunately, it is all too common to see a respirator hanging decoratively around the neck of the person it is supposed to be protecting. A better realization among wearers about the health risks involved and more care in the design and selection of respirators should improve their acceptability.

Anyone who is to use a respirator should take the time and trouble to understand how it works and what it is designed to do—information that management is obliged to provide. The respirator may be the only thing between the wearer and illness or even death. Programs to educate the worker about the why and the how of respirators should be of concern to both management and labor unions.

SELECTION OF RESPIRATORS. Respirators must be selected according to the type of hazard to which the wearer will be exposed. The federal government's Occupational Safety and Health Administration requires that respirators approved by the Bureau of Mines or the National Institute for Occupational Safety and Health be used whenever they are available.

A large variety of respirators are on the market. Notwithstanding the great variety available, respirators fall into two basic groups: air-filtering devices, which simply clean the air as it passes from the workplace into the lungs, and supplied-air systems, which insulate the wearer from the surrounding air and supply him with a separate, clean source of air or oxygen.

Air-filtering devices. These simple respirators are useful only when the hazards are in small doses and pose no immediate threat to life or long-term danger to health and when the air is otherwise fit to breathe—that is, when plenty of oxygen is available.

In theory, the air-filtering respirator consists of a mask that fits snugly over the nose and the mouth and that allows air to enter only through a filter. In practice, however, faces vary so greatly that unfiltered air often leaks in around the edge of the mask; sideburns and beards pose more

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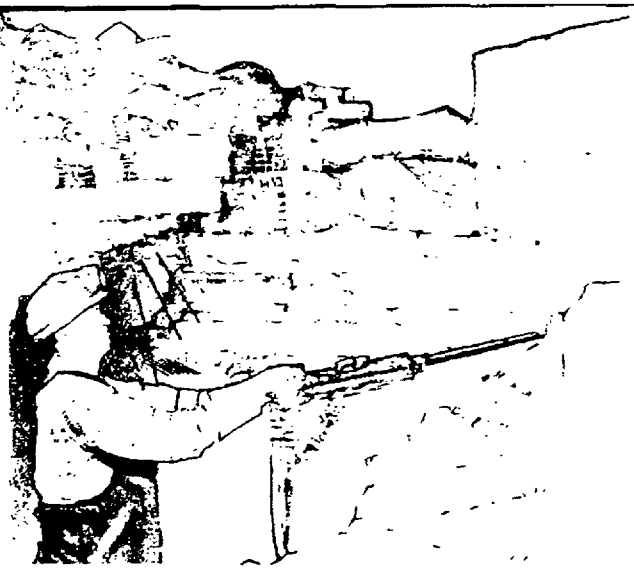

Silica is the earth's most common material, often encountered in mining and stoneworking activities, such as quarrying. Silicosis from breathing in silica affects workers in many industries.

problems. As a result, air-filtering devices cannot be used around a dangerous hazard. An air filter can be used, for example, if traces of chlorine gas might be encountered in otherwise normal air but not if the air is deficient in oxygen or if the chlorine levels are high.

Commercially available filters can handle only one specific hazard or a group of hazards with similar properties, such as respirable dusts or solvent vapors from a particular chemical family. Filters should never be used for hazards other than the ones they are claimed to handle. A filter designed for silica dust probably allows welding gases to pass through readily; one intended for ammonia gas may be useless against hydrogen sulphide. An ineffective filter is worse than no respirator at all if the wearer thinks that it is working.

Air-filtering devices have a limited capacity. They can be clogged by dust, making breathing difficult, or the canisters used to absorb gases, vapors, and mists can become saturated and useless. Most filters are designed to provide some sort of signal when they are approaching capacity. The worker should be sure he understands the signal before he starts wearing the device.

Supplied-air systems. These respirators fall into two groups: demand-type respirators, which supply air as the wearer needs it, and positive-pressure respirators, which continuously pump air into the mask, maintaining a higher pressure inside than outside.

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Like filtering devices, demand-type respirators are limited by the fact that their masks often fit poorly and allow air to leak in around the edges. They should not be used when the workplace air is dangerous to life or health. But they are valuable whenever conditions are such that filtering devices rapidly become overloaded and when the workplace is low in oxygen. A demand-type respirator suffices in a tank full of coarse, flying silica dust and fresh air or in one containing 90 per cent carbon dioxide and 10 per cent oxygen. Neither a few particles of coarse silica nor a little carbon dioxide poses a threat to life or health if the mask leaks. But if the tank contains any phosgene or hydrogen cyanide, which are immediately toxic, a demand-type respirator is dangerous.

Positive-pressure respirators are the only ones that can safely be used in air that is immediately hazardous to health. Because these respirators maintain a higher pressure inside the mask than outside, air leaks out, rather than in, if the mask doesn't fit tightly, thus keeping dangerous air away from the lungs.

With either type of supplied-air system, the fact that the air supply may fail must be considered. The wearer may need a second, emergency source of oxygen to enable him to escape from the hazardous area.

Expert advice is essential when choosing respirators. The total circumstances prevailing in the workplace have to be considered. Take the case of an abrasive blaster. If he is using sand to clean a small area of an outside wall, a simple air-filtering device and a mask that includes goggles for eye protection will probably suffice. If, however, he is cleaning the inside of a tank that formerly held a chemical and the residual fumes from that chemical could both burn his lungs and irritate his skin, a positive-pressure supplied-air respirator is essential, and he also needs protective suiting. If he is using steel shot, instead of sand, in the same chemical tank, the positive-pressure air supply should be into a helmet, not a hood.

CARE OF RESPIRATORS. All respirators must be carefully cleaned after each use and meticulously checked and maintained to remain in good working order. Many companies have a central respirator-servicing unit under expert control. Where such a service isn't available, respirators are all too often carelessly stored and badly maintained.

STATUTORY CONTROLS

Historically, legislative controls over workplace conditions proved essential because, without legislation, practical measures to control both health and safety hazards simply weren't routinely used.

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It would be impossible in this handbook to describe in detail all the laws affecting workplace conditions and the ways in which they have been interpreted by various courts when challenged. This handbook simply outlines the main pieces of legislation, describes the basic mechanisms by which statutory controls operate, and shows how these controls tend to work in practice.

The three most important and far-reaching federal acts in this field are the Federal Mine Safety and Health Act of 1977 (which amended the Coal Mine Health and Safety Act of 1969), the Occupational Safety and Health Act of 1970, and the Toxic Substances Control Act of 1976. Before these laws were passed, federal legislation had been piecemeal, affecting small groups of workers in specific industries. The Occupational Safety and Health Act applies essentially to all workplaces—farm, factory, and office—left uncovered by previous legislation.

The Occupational Safety and Health Act also allows state controls to be substituted for the federal controls, provided the state controls promise protection at least equivalent to that afforded under the federal law. It offers financial aid to states wishing to develop or administer their own occupational safety and health programs. For a variety of political and financial reasons, the status of individual state plans has fluctuated; up-to-date information should be sought from the appropriate state agency.

Statutory Control Mechanisms

How can a health hazard—a gas, dust, or fume—be controlled by legal means? First, hazards have to be detected and then evaluated—how much is dangerous, and what are the effects? Second, a standard has to be established—a permitted level of exposure that will not affect either life or health, even if a person is exposed to it throughout his entire working life. And third, some mechanism must be established to enforce compliance with the official standard—some means of checking that hazard levels are really kept at or below permitted levels.

DETECTING AND EVALUATING HAZARDS. Federally, the responsibility for hazard identification and evaluation falls on the National Institute for Occupational Safety and Health (NIOSH), which is part of the Center for Disease Control in the Department of Health, Education, and Welfare. NIOSH is the official source of information on health hazards. The agency is authorized to detect them by industry-wide studies and other methods; to evaluate them by toxicological, epidemiological, and other methods; and to publish an annual register of all known toxic substances and their effects. NIOSH provides the data and recommendations on which hazard standards are based.

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SETTING STANDARDS. Standards for health hazards in underground mines (methane gas and respirable dusts) were embodied in the Coal Mine Health and Safety Act of 1969, amended in 1977 by the Federal Mine Safety and Health Act. For other workplaces, standards are set by the Occupational Safety and Health Administration (OSHA), which is part of the Department of Labor. OSHA must publish proposed standards in the Federal Register, with provisions for public hearings before the standards become law. Chemicals which are hazardous to health are also regulated by the Environmental Protection Agency under the Toxic Substances Control Act of 1976.

OSHA uses data provided by NIOSH when formulating standards. NIOSH is also responsible for monitoring the effectiveness of standards established for underground mines and recommending changes if necessary.

Standards for lung hazards are usually in the form of threshold limit values, called TLV's for short. The TLV is the maximum concentration of a hazard that a person can be continually exposed to, without impairment of health, throughout every day of his or her working life. TLV's are expressed in whatever form is readily measured—as the number of parts of hazard per million parts of air (ozone, 0.1 ppm; ammonia, 50 ppm); as the number of milligrams of hazard per cubic meter of air (cotton dust, 0.5 mg/m^3; nickel carbonyl, 0.007 mg/m^3); as millions of particles per cubic foot of air (Portland cement, 50 mppcf; natural graphite, 15 mppcf).

For hazards that pose no immediate or serious threat if permitted levels are temporarily exceeded, the TLV represents an average exposure level, meaning that levels above the TLV are permitted if they are counterbalanced by an equivalent period of exposure to levels below the TLV.

Hazards that have acute toxic effects are controlled by more rigid standards, including a ceiling concentration that hazard levels may not exceed, even if the time-averaged exposure is within limits. Cadmium fume standards are an example; the permissible average exposure over an eight-hour shift is $0.1 mg/m^3$, but the acceptable ceiling concentration is 3 mg/m^3.

ENFORCING STANDARDS. Two government agencies are primarily responsible for enforcing occupational health standards and other statutory controls: the Mine Safety and Health Administration (MSHA) in underground mines and the Occupational Safety and Health Administration (OSHA) in most other workplaces.

Both administrations have extensive powers to detect noncompliance with standards and to make sure that corrective steps are taken. Both

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can inspect workplaces to check conditions. MSHA is required to inspect all underground mines at least four times every year.

How Statutory Controls Work

How do the complex written statutes, government agencies, and enforcement mechanisms mesh together to ensure that workplace health hazards are controlled? What, briefly, are the rights and responsibilities of the people involved—the employers, the employees, and the government agents?

ROLE OF THE EMPLOYER. The employer has the basic responsibility for workplace conditions under federal law. He or she must know the health and safety hazards in his or her business and must take steps to make sure that employees are aware of and protected from these hazards. The employer must provide instruction in hazard-control techniques if necessary, provide any essential protective clothing or equipment, allow government officials to inspect the workplace at an employee's request either voluntarily or in response to a warrant, keep comprehensive records of hazard levels and the exposures of individual workers, and conduct medical examinations or biological tests if needed to monitor the effects of hazards. All these records must be made available to both employees and government agents if they so request. The employer must also report all illnesses, injuries, and deaths occurring as a result of occupational factors.

Besides complying with official standards for specific hazards, the employer also has a general duty under the Occupational Safety and Health Act to see that workplaces are free from recognized hazards that are not subject to formal standards. This provision is an attempt to ensure that common sense prevails in the absence of specific standards.

ROLE OF THE EMPLOYEE. The employee has many rights under federal statutes—and some obligations, too. Most important, he or she has the right to know if workplace conditions are potentially hazardous and to be protected from any hazards that may be encountered. Under the common law of arbitration, workers have the right to refuse exceptionally hazardous work, a right that has been interpreted differently by different courts.

An employee who feels that the place of work may be hazardous has the right to complain to the appropriate enforcement agency (MSHA or OSHA) and request an inspection. The law forbids discrimination against anyone making such a complaint and endeavors to ensure anonymity for complainants if they request it.

When an inspection is conducted, employees have the right to talk privately with the inspector and to accompany the inspector on the tour

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—a right that may be delegated to an authorized employee representative.

All employees have the right to be informed of standard violations discovered by inspectors. If a complaint is found to be unjustified, the complainant must be given the reasons for this finding in writing. An employee may appeal if the inspector decides to take no action.

Federal statutes also require that employees comply with all hazard-control measures designed for their protection and with efforts to monitor hazard levels and health effects. This provision means that the employee must wear a respirator where one is required, even if it's uncomfortable.

Employees may also ask NIOSH to undertake a hazard evaluation if something in the workplace seems to be affecting health but is not subject to standards.

ROLE OF THE GOVERNMENT AGENTS. Government agents must see that both employers and employees are meeting their respective responsibilities—although, in practice, virtually all enforcement measures are directed at employers.

Both OSHA and MSHA may inspect workplaces at random or in response to specific complaints. If an employer refuses consent to an inspection, the OSHA inspector must obtain a warrant. Inspectors can check hazard levels, inspect hazard-control systems, look at the records that employers are obliged to keep, and discuss conditions and practices with any employee.

If standards are being violated or statutes contravened, inspectors can act according to the degree of danger involved. Both MSHA and OSHA inspectors can issue warning notices and citations for less dangerous violations, giving employers a period of grace in which to take corrective measures and letting employees know their findings. Notices about violations must be posted in the area where the violation is occurring. When an employer delays getting into compliance with standards, penalties become progressively more severe.

If dangerous conditions are found, MSHA inspectors have broader powers than do OSHA inspectors. If an MSHA inspector discovers conditions that, in his or her opinion, are imminently dangerous, the inspector can stop the work at once and order everyone out of the mine. The inspector may also stop the work if a mine operator has delayed unreasonably in correcting a less hazardous violation. OSHA inspectors cannot stop the work, even in dangerous conditions, without obtaining a court order.

Statutory controls over workplace conditions are relatively new. Changing technology and materials create new and different hazards; research constantly provides better information on hazard effects and on the interaction between occupational and environmental hazards. The result is a

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continually changing milieu for statutory controls to contend with.

The present legislation and enforcement procedures are often criticized. Most of the affected parties feel that the procedures are inadequate or unfair in different respects. Nevertheless, existing statutory controls, although far from perfect, are a good tool with which to attack the problem of occupational disease.

MONITORING

Monitoring—the process of checking on hazard levels and their physiological effects—is an essential part of the overall concept of hazard control. No one can be sure that engineering, administrative, and legislative efforts are truly protecting the health of everyone in the workplace unless he or she knows which hazards are still in the air, at what levels, and whether such exposure is currently affecting health. Only with this knowledge can the effectiveness of existing control mechanisms be evaluated. Only this information can indicate whether more needs to be done.

The earliest and most primitive occupational health monitor was probably the first canary carried underground by miners. Since the canary was usually more sensitive to carbon monoxide than were the miners, it would topple off its perch in low levels of this gas, giving the miners a chance to escape before they, too, were affected.

Monitoring today is a vastly more sophisticated science. There are air-quality monitors: machines, samplers, rapid-reading tubes, and other devices designed to provide continuous or periodic readings of how much dust, gas, vapor, or radiation is in the air. Medical and biological monitoring techniques are also used. They can measure how much of a hazard is accumulating in the body and how and to what degree it is affecting individual body functions and overall health.

When information from both approaches to monitoring can be correlated, a picture of the relationship between hazard exposures and physiological effects begins to emerge. Monitoring activities—samplings, readings, record-making, and medical checks—are sometimes viewed with disdain or impatience by both employees and employers. It must be remembered that the information gleaned, intelligently used, may prove a vital element in achieving the ultimate goal of a workplace that is both profitable and healthy.

Air-Quality Monitoring

A measure of hazard levels in the air is useful in many ways. It can show whether hazard-control systems are working properly, indicate the main sources of a hazard, and be used to document compliance with

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hazard-control laws. Comprehensive records can give occupational health professionals a good picture of a person's working history—which hazard or hazards the person has been exposed to, at what levels, and for how long. Records can also help determine disease patterns, assess required compensation, and indicate if legislative controls should be imposed, tightened, or loosened.

Monitoring methods range from very simple to highly complex. People may be employed to walk around looking for visible hazards escaping from leaky valves or to check if other people are wearing respirators in designated areas. Highly complicated monitoring machines, which are becoming increasingly specialized, may provide continuous readings and printouts. Other monitoring machines are linked to automatic alarm systems that are activated if conditions become dangerous. The canary in a cage has been replaced, in some places, by a continuous monitor that not only sounds an alarm but also automatically shuts down the machine or process that is generating the hazardous matter. Some chemical carcinogens are also monitored in this way.

Two important points about air-quality monitoring should be noted. First, whatever method is used, several readings are necessary to provide a meaningful measure of average hazard levels. A single, isolated reading seldom has any significance, because levels of most hazards vary throughout the working day or week, depending on what operations are being conducted. Second, monitors and samplers should be located so that they check hazards in the breathing zone—at face level.

There are two basic ways to approach the problem of air-quality monitoring: area monitoring and personal monitoring. Each is useful in different ways.

Area monitors remain stationary in a work area and are used to provide information on the distribution of a hazard in both time and space. Techniques can be adapted to provide a variety of information. For example, one monitor taking repeated readings shows how hazard levels vary in that area throughout the day. Several monitors taking repeated readings simultaneously but in different places show how a hazard is distributed through the different areas at different times. What an area monitor may not show, however, is the amount of hazard encountered by a person who moves around from one area to another, perhaps spending 10 minutes here and two hours there. For this information, personal monitors are used.

A personal monitor is worn by an individual worker. The air-intake portion of the device is usually fastened to the lapel, close to the breathing zone. The monitor is operated for some time, often four to eight hours, to provide details on how much hazard that person inhales during a

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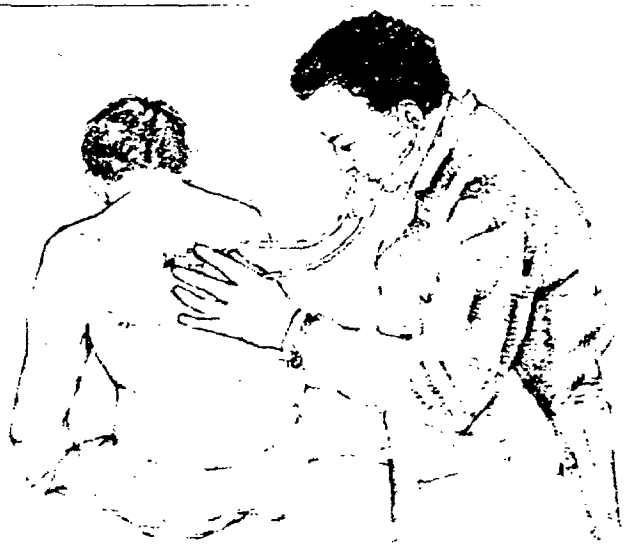

Medical monitoring of workers in hazardous occupations may find occupational lung disease early. Prompt action sometimes can be taken to halt or minimize the disease. Specialized tests may be necessary such as analysis of blood and sputum samples, lung function tests, and bronchoscopy.

shift. Personal monitors are also used when area monitors are impractical.

Most air-quality monitors operate by drawing a measured amount of air through the device and collecting the hazard therein in some manner. Respirable dusts are collected by filters, organic vapors are absorbed by activated charcoal, gases are absorbed by liquids, and so on. The total amount of hazard collected is then measured by some appropriate technique, and the amount per cubic meter or other unit of air is calculated.

Some air-quality monitors use other techniques. Some gases and vapors can be checked with rapid-reading tubes, which change color according to the concentration of hazard present. Lapel badges, known as passive monitoring devices, are available to show accumulated exposure to radiation and to some vapors, including mercury; these badges progressively change color or appearance as hazard exposure continues.

As this brief outline indicates, the monitoring of air quality can be a complex and highly specialized business affected by what the hazard is, how it is generated, the air temperature and currents, other airborne hazards in the same area, and many other factors. Techniques and machines should be selected only with expert advice. As a general rule, the more dangerous a hazard is—whether the danger is immediate, like carbon monoxide, or delayed but medically serious, like carcinogens—the more frequently it should be monitored.

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Medical Monitoring

Medical monitoring, sometimes called biological monitoring, is the science of investigating hazard effects in people—taking samples of body fluids, such as blood and urine, to determine hazard levels; checking the functioning or organs like the liver, kidneys, and lungs to see if they are being adversely affected; looking for other signs or symptoms of occupational disease.

Evaluating the effects of a hazard on the body may seem in some respects to be a retrospective exercise, rather than a preventive one. But medical monitoring is valuable in many ways. It may be the only real way of measuring hazard exposure. For example, air-quality monitoring is a highly unsatisfactory way of measuring pesticide exposures, but the level of exposure can reliably be judged by monitoring an enzyme (cholinesterase) in the bloodstream. Lead provides another example; it is accumulated in the body, usually by the inhalation of dusts or fumes, and may be stored for long periods. The only way to be sure of the amount of lead in a lead worker's body is to regularly monitor urine or blood levels.

Medical monitoring can also be used to check the effectiveness of hazard-control mechanisms. If a supposedly controlled hazard accumulates in the bodies of people exposed to it, several things may be happening. Engineering controls may not be working effectively; there may be some additional unsuspected and uncontrolled source of exposure; the permitted exposure levels may not be low enough to protect health. To identify the problem, an occupational health professional needs information from air-quality monitoring and medical findings. Such professionals frequently find themselves playing detective, confronted by contradictory evidence and hunting for additional clues to solve a medicotechnological mystery.

Early recognition of the signs of occupationally induced disease can sometimes be followed by actions to halt or minimize the progression of the disease. If detected at a very early stage by medical monitoring, asbestosis may sometimes be prevented from becoming severe by immediately removing the victim from all asbestos exposure. If people who are developing a hypersensitivity to cotton dust or isocyanates are identified before symptoms become serious, permanent damage to the lungs may be avoided by a change of job. This situation can become controversial if the transferred employee suffers a loss in seniority, prestige, or pay.

Medical monitoring is also valuable when the toxicity of a substance is unknown or when more information is needed. Many enlightened industries, in an ongoing search for information about toxic effects, monitor

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their employees carefully and regularly not only during their working lives but also after they retire. The monitoring of asbestos workers throughout many years of dust-control measures has now produced indications that existing standards for asbestos exposure may not be sufficient to prevent asbestosis.

A number of specialized tests are used to detect early signs of disturbances in lung function and to accurately diagnose specific occupational lung diseases. Most tests or diagnostic procedures provide only a small amount of information about the underlying state of the lungs. The results of several tests usually have to be put together before a disease process can be identified with reasonable certainty.

Many lay people have the idea that a chest X-ray shows if anything is wrong with the lungs, but this is far from true. An X-ray can show patterns of dust deposition and some reactions to this dust, like established fibrosis. It can also show areas of lung congestion, collapsed lung lobes, and most cancers. It can't show the early stages of most diseases, for which lung-function tests are usually needed.

Lung-function tests are designed to measure the performance of the lungs. By means of a breathing tube connected to a special apparatus, lung-function testing measures how elastic the lungs are, how quickly air can flow in and out, and, if tests are repeated while the patient is exercising, how well the lungs can respond to an increasing need for oxygen. These tests often show early signs of lung disease and can indicate whether the problem is a restrictive one, like fibrosis (which interferes with the elasticity of the lungs), or an obstructive one, like chronic bronchitis or asthma (which interfere principally with the flow of air).

Sometimes other techniques are needed to provide additional information. Blood and sputum samples may be analyzed; bronchoscopy may be used to examine the large airways; skin and lung reactions to suspected antigens may be needed to identify hypersensitivity diseases.

Comprehensive medical monitoring, including lung-function tests, is essential if occupational lung diseases are to be detected in the early stages, when measures can be taken to stop their progression or, at least, slow down their progress. By the time many of these diseases show up on X-rays, it is often too late to do anything except let the disease run its crippling course.

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Chapter 6

PROBLEMS IN OCCUPATIONAL HEALTH

No one who seeks to improve the occupational environment can move far toward that goal without encountering resistance of one kind or another. The national occupational health problem of the late 1970's is serious, large, and complex. It has social, economic, technical, logistical, and political dimensions. If occupational disease is to be effectively combatted, some understanding of the underlying problems is essential.

The problems that are touched on briefly in this chapter include:

History, which sets the stage for present-day problems.

Differing perspectives on occupational health, for few people view the problem objectively.

Attitudes and traditions toward work and workplace conditions, which not only militate against improved working conditions but also have proved to be strong enough to affect occupational health legislation.

Technical problems of linking jobs and diseases, which are often thrown into the public limelight because of the economic and social consequences of hazard control.

Information problems, the difficulties of identifying hazards and obtaining and understanding information relevant to hazard toxicity and control.

Economic factors, which affect the willingness of industries to control hazards and the effectiveness of agencies charged with administering legislative controls.

HISTORY

Today's national occupational health problem, with its large but ill-defined dimensions, has been a long time in the making. The roots are far back in the development of this country and its economic system, when

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the fact that air pollutants can be hazardous to health was unknown to most people.

For a long time the symbols of prosperity were soot and fly ash all over a town. Industry brought jobs, and jobs brought money—money for food, housing, clothing. Business owners ran their operations as they chose, maximizing profits by minimizing costs, using whatever materials and processes produced the best financial returns.

Yet, as industrialization proceeded and technology diversified, occupational disease was following suit—affecting more people and appearing in new forms. New materials, processes, and stresses assaulted the human frame, producing effects previously unknown. The common and easily attributable diseases, like silicosis and coal workers' pneumoconiosis, eventually produced demands for compensation and, later, for preventive measures. Those diseases helped to change the social climate, moving it away from the view that occupational disease is part of the privilege of having a job and toward the idea that work shouldn't damage health.

As knowledge of the relationship between work and health improved, more occupational hazards began to be recognized. With help from the environmental and labor movements, occupational disease was belatedly brought to national attention in the 1960's.

But even as occupational health surfaced as an important issue, scientific advances were greatly enhancing the concept of toxicity. Some chemicals were found to cause chromosome damage that shows up in animal and human offspring; other chemicals, such as kepone, cause temporary or permanent sterility. Anesthetic gases appear to cause an increased rate of miscarriages and other problems in operating room personnel. Studies of the origins of cancer revealed to a skeptical world that some carcinogens take 20 to 40 years to exert their effects and that a few carcinogens can, after only brief exposure, leave sufficient imprint on a tissue to result in cancer many years later. These were new concepts in toxicology. Many effects were also impossible to prove in the general population and were detectable in the laboratory only by sophisticated and often expensive testing.

Although in all probability few of the substances used in American workplaces have bizarre toxicological properties, recognition of them and of other agents with more straightforward toxicological effects poses enormous technical and logistical difficulties.

Workplace conditions, too, are intricately interwoven with the economic viability of individual businesses and the nation. Economic, social, and political repercussions predictably follow any proposed change in the status quo.

Some long-established traditions toward work and the workplace are

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Firefighters encounter a host of irritating gases resulting from the combustion of all kinds of materials. Deadly phosgene, for example, results when something containing chlorine, such as degreasers and dry cleaning materials, are in a burning building.

also prevalent and powerful. They have markedly affected the legislation that was passed to improve workplace conditions.

DIFFERING PERSPECTIVES ON OCCUPATIONAL HEALTH

Whether campaigning for improved working conditions at the state or the national level or in an individual industry, granary, or workshop, we must remember the other pressures affecting the people approached, for no one can view the occupational health problem in a vacuum.

The worker on the shop floor, the farm hand, or the beautician using cosmetic sprays may or may not be concerned about health hazards. He or she is subject to prevailing traditions and attitudes toward work and the workplace. These people are also concerned with keeping their jobs. They may not wish to be classed as troublemakers. They may have no desire to antagonize their employers or supervisors, and they almost surely do not want their employers to go out of business, for a job change would probably mean the loss of seniority and a reduction in salary.

A supervisor or line manager may be better informed about job-related health risks. But his or her prime concern is to run the operation efficiently and to meet quotas and deadlines. The improvement of the working conditions may be at the back of this person's mind, but this goal is often overlooked in the everyday pressures of getting the job done—pressures that are also felt by the people under his or her supervision.

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Higher management and business owners are likely to know even more about health risks and their own legal responsibilities. But they also have to retain their profitability to keep their stockholders happy and to keep ahead of the competition and stay in business.

Then there are the legislators who are trying to balance different demands that are often perceived as conflicting. Voters want unemployment reduced; workers want better, healthier working conditions; owners and corporations want to retain their competitive business positions; environmentalists are concerned with the pollutants an industry discharges to the world outside the workplace.

And the industrial physicians, hygienists, and other occupational health professionals wrestle with the problems of identifying, quantifying and controlling hazards, try to strike a balance between scientifically acceptable standards of proof of harmful effects and the point at which it seems imperative to take preventive action based on the evidence already in hand; and try to convince business and government agencies to accept their recommendations.

In short, everyone involved in and affected by the occupational health issue tends to see it in a different perspective. What is needed is a broader commitment to the idea that work should not adversely affect anyone's health or life span unnecessarily. (Some jobs, such as fire-fighting, will always have some element of danger.)

Adherence to this principle entails some rearrangement of the economic order. But this order is not static. Changes occur because of material shortages, energy availability, demand fluctuations, and other factors. There is no reason why changes designed to improve the health of workers should be regarded as merely optional.

ATTITUDES AND TRADITIONS

One of the most persistent and negative attitudes pervading the occupational health area, from the shop floor to the legislative floor, is the notion that work is necessarily dirty and unpleasant—that workplaces have to be dusty, smelly, fume-filled, or foggy. This attitude leads to a lot of apathy among those who work in such conditions. They feel that putting up with the working conditions is partly what they are paid for.

This fatalistic attitude was well summarized in the findings of a 1974 Department of Health, Education, and Welfare survey on the acceptability and effectiveness of respirators worn by abrasive blasters, people who use jets of high-speed sand or other abrasive material to clean industrial tanks, masonry, and other surfaces:

"The average firm [company] safety man, where one exists . . . seems unaware of the problems of respirable dust and noise. The average blaster

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seems unconcerned by equipment deficiencies. His trade has always been dusty and noisy."

The general acceptance of the idea that workplaces have to be unpleasant has even pervaded legislative efforts to improve both the general environment and the occupational environment. As a 1976 congressional report on chemical dangers in the workplace pointed out, there are marked discrepancies in the protection afforded different populations under existing legislation.

Consumers of manufactured goods—such as aerosols, pesticides, and cleaning agents—are often warned of possible hazardous effects by mandatory warnings on labels, but few such warnings are given to those who manufacture the products, despite the fact that their exposure is likely to be to higher levels and for longer periods of time. And both the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA) set air-pollution standards in their respective jurisdictions, but OSHA allows much higher levels of pollutants within the workplace than EPA allows outside.

These dual standards for air pollution are sometimes justified on the grounds that only fit and healthy people are exposed to workplace conditions but that children, the elderly, and those in poor health are exposed to the general environment. Another rationale used is that exposures in most workplaces are for only eight hours a day but exposure to the general environment is continuous. These arguments can be challenged on several grounds. Many workers aren't healthy. Their lungs are already diseased from prior occupational exposures and smoking. And many workers frequently work overtime and live close to the industries they're employed by; thus, they inhale high levels of pollutants at work and additional but lower levels in their home environments.

The notion that the workplace has to be dirty and unpleasant may have been true early in the process of industrialization, when technology was in its infancy, but it certainly isn't true today. In most instances we now have the technology to reduce and eliminate the pollution we create, but we lack the determination, motivation, and economic incentive to use this technology effectively. Only education can counteract this acceptance of workplace pollution and the apathy and legislative quirks that it spawns.

A number of other traditions also tend to militate against a healthier working environment. One is the fact that, from the very beginning of legislative attempts at better conditions, health and safety considerations have always been lumped together, and safety or accident prevention has always overshadowed health protection. Accidents and their prevention have always been more dramatic, more evocative, more newsworthy. A person who loses an arm in an unguarded machine arouses more sym-

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pathy and indignation than does a person with occupational lung disease who is slowly coughing and wheezing his way to an early death.

One of the main findings of the congressional investigation into chemical dangers in the workplace was that, from its inception in 1971 until 1976, the Occupational Safety and Health Administration had concentrated on safety issues and had paid little attention to health. Only 20 per cent of OSHA's compliance officers in 1976 were trained to detect health hazards during their safety-oriented inspections.

The same bias can be detected in engineers, buyers, planners, and plant managers; whenever they turn their attention to workplace conditions, they tend to concentrate on safety issues and to ignore or neglect health considerations.

Again, only increasing recognition of the full extent of the occupational health problem will produce the pressures to rectify this situation and elevate health maintenance to the same prominence enjoyed by accident prevention.

Another tradition that hinders attempts to control occupational health hazards is the innocent-until-proved-guilty status historically enjoyed by substances used in the workplace. Until the passage of the Toxic Substances Control Act in 1976, the onus of proof was always on those who believed it necessary to curtail use of a substance. They had to prove that it was harmful; the producer or user was under no legal obligation to demonstrate the substance's safety.

While this situation prevailed, industrial practices varied widely. At one end were enlightened and responsible industries that routinely tested any substances they contemplated using and that conducted their own epidemiological studies and monitored their employees for signs of adverse health effects. At the other end of the spectrum were the industries, often small ones, that through ignorance or disregard used whatever materials brought them the most profit. And in between these extremes were most businesses, which showed varying degrees of concern for health protection.

This innocent-until-proved-guilty status meant that the National Institute for Occupational Safety and Health (NIOSH), at its formation in 1971, faced a formidable array of about 12,000 substances that were already in use and that required toxicological investigation. The logistic and economic difficulties of this task forced NIOSH to rank research priorities according to the apparent risk factor posed by each hazard. Priorities are determined by a complex formula that weighs the suspected toxicity of a substance, how many people are exposed to it, whether it is being increasingly or decreasingly used, and other factors.

The end result is that NIOSH's efforts are largely devoted to investigating carcinogenic properties and to answering straightforward toxicity

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questions. There is little research into more complex toxicological effects, such as chromosomal damage, or into the additive or potentiating effects that may follow simultaneous or subsequent exposure to more than one hazard, despite the fact that in real life—out there on the factory floor, atop the furnace, at the workbench, or in the auto-body repair shop—people are seldom exposed to a single hazard in isolation.

The Toxic Substances Control Act of 1976, although born of concern about the effects of synthetic chemicals on the general environment, also extends to the working environment. It attempts to make sure that all substances not listed as already in use by November 1977 undergo at least some toxicological testing before entering general use. If a chemical substance of mixture presents an unreasonable risk to health or the environment, EPA may prohibit or limit its manufacture, processing, distribution, use, and disposal. This provision should at least keep NIOSH's work load from expanding indefinitely. But it still leaves many substances of unknown toxicity around in America's workplaces.

TECHNICAL PROBLEMS

Establishing a link between someone's job and his or her state of health can be a difficult scientific exercise. Investigative methods have their limitations, often leaving gaps in the scientific picture and differences of opinion about the interpretation of the available information. This situation is far from unusual in scientific circles, but the political repercussions attendant on the control of occupational health hazards tend to bring scientific arguments into the public spotlight. Lay people are then confronted by disputes between experts about what has and what hasn't been proved. Some appreciation of the limitations of investigative techniques can help one understand such arguments.

Linking jobs and diseases is a twofold task. The scientists must first recognize that a disease risk exists in a working population and then identify the causative hazard so that preventive measures can be devised. The first task is mainly the province of epidemiologists, who compare the prevalence and distribution of disease within different population groups; the second task is largely the province of toxicologists, who investigate the harmful effects of chemicals on biological tissues.

The epidemiologist who studies occupational lung disease seeks to compare the prevalence rates of a particular disease in two populations that are alike in all respects except for the exposure of one group to the workplace conditions under investigation. Normally, this means matching the populations in terms of age, sex, and length of employment. If it weren't for the occupational exposure, the two groups would presumably demon-

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Cotton dust is dangerous mostly because of the plant remnants that come with the cotton from the field. Resulting byssinosis afflicts flax and hemp workers, too, especially those who clean the raw product, but also those who card and spin it.

strate a similar prevalence of disease.

But in the case of occupational lung diseases, both smoking and general air pollution are associated with an increased prevalence of chronic lung disease. So it is difficult to establish to what extent these diseases are caused by occupational factors and to what extent they're caused by smoking and general air pollution. To eliminate the effect of smoking and air pollution on the prevalence of chronic lung diseases, epidemiologists must also match their populations in terms of smoking habits and exposure to air pollution outside the workplace.

In practice, it can be almost impossible to match two populations in which someone cannot find some possibly relevant differences other than the hazard under investigation. People are complex. They have home environments, hobbies, part-time jobs, inherited traits, and other non-occupational aspects to their lives that may affect their health.

Although epidemiological studies often provide important clues, toxicological studies are usually the ones that pinpoint and quantify specific health hazards in the workplace. Such toxicological studies aren't easy. Workplaces are diverse—hot or cold, wet or dry, noisy, subject to vibration, perhaps dusty in one corner and fume-filled in another. Trying to prove that one factor is a definite health hazard can be a challenging exercise.

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Toxicologists test suspected health hazards, usually on animals and sometimes on specially cultured tissues, to see if harm develops. Their results are often disputed on the grounds that what happens in animals may not necessarily happen in humans. When a toxic effect is manifested in several species, however, the odds are that it is occurring through some biochemical mechanism or cellular process that also occurs in humans.

Both epidemiological studies and toxicological studies are limited by the inability to experiment directly on humans. People can't be ordered into controlled groups in which their total environment can be monitored. Nor can they be subjected to experimental doses of suspected health hazards. Hence the highly politicized disputes about what has and hasn't been proved by scientists.

Leaving aside the merits of epidemiological and toxicological techniques, one should look at the important overall picture of a suspected health hazard. What percentage of people who are exposed appear to have incurred occupational disease? Is the severity of the disease correlated to the intensity of the hazard exposure? Can the disease be found in other populations exposed to the same hazard? Do animal tests seem to confirm that the hazard is a cause of disease?

Positive answers to those questions can present a convincing argument in favor of a link between a job and a disease, even if there are highly technical questions about the significance or the validity of a single piece of evidence. At some point, it becomes reasonable to take preventive measures based on available knowledge, without waiting for absolute proof, which may, for practical reasons, be unattainable.

INFORMATION PROBLEMS

To understand and combat any known health hazard, we need three pieces of information: exactly what the hazard is, what sort of danger it poses, and how it can be controlled, eliminated, or countered.

Identifying a hazard may be very difficult. From polishing powders and grease solvents to fumigants and cutting oils, many substances used in America's workplaces are known to their purchasers and users and sometimes to the manufacturers only by a trade name or a code number. No other identification of the substance appears on the container, except perhaps a mandatory warning about flammability, corrosive properties, or lead content. Whether this practice really protects trade secrets in this age of sophisticated analytical techniques is debatable, but it does effectively prevent lay users from knowing what hazards they are exposed to.

NIOSH conducted a national occupational hazards survey of 5,200 workplaces in an attempt to determine the extent of the trade-name prob-

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lem. In 70 per cent of the instances in which they found a person exposed to a potential hazard, the hazard was known only by a trade name or code number. In 90 per cent of those cases, NIOSH could not determine the contents of the trade-name substances either from the container or from information available at the plant. In short, neither employers nor employees knew what substances people were being exposed to, much less their toxic effects.

Seeking more detailed information, NIOSH asked the manufacturers of the mystery trade-name substances for a list of ingredients. The investigators learned that 45 per cent of the products in question contained substances supposedly regulated by OSHA because of their recognized toxic potential. About 30 per cent of the products contained another trade-name product purchased from a different manufacturer, with the complete contents of the final product known to no one. And, most disturbing of all, 427 different trade-name products encountered in the survey contained one of the 15 carcinogens (cancer-producing agents) that are supposed to be strictly regulated by OSHA. Neither the buyers nor the handlers of the products knew about the carcinogens.

The congressional inquiry used the word "unconscionable" to describe the practice of including toxic and carcinogenic substances in trade-name products that have neither a list of ingredients nor appropriate warnings on the container. The committee members also expressed concern about the secret formulations included in other secret formulations; they also noted that, when warnings do appear on containers, they are almost invariably concerned with immediate dangers, such as swallowing poisons or splashing corrosive material into the eyes. Information on the chronic effects of low-level exposure is seldom given; often, it simply isn't known.

There is clearly an urgent need either for a central clearinghouse to supply information on the contents and toxicity of all products used in the workplace or for legislation mandating that this information be included on container labels. Since formulations change frequently, the latter course seems more practicable and less expensive. Including long-term toxicity information on labels would highlight the dearth of information in this field and would produce a demand for more and better testing.

To be of maximum benefit, toxicity information must be supplied in language that the public can understand. For example, "progressive dyspnea and pneumonitis" should be replaced by "difficulty in breathing that gets worse and inflammation of parts of the lungs." Unfortunately, toxicology reference texts and the annual NIOSH registry of toxic substances and their effects are not readily understood by people without scientific training.

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There is also a problem with the transfer of the information that is available. Once researchers identify a hazard and determine its effects, the information needs to percolate through all the industries and situations in which the hazard is encountered. Sometimes word spreads through the industry that is the main user of the hazard concerned, but small companies and those that use the same or similar materials in a different context may not receive or act on the information. There is now no formal network through which such information is transmitted.

Occupational health specialists have also noted that information passes slowly from one country to another. Policies adopted by one country that has extensively studied a hazard go unnoticed in other countries. Certain amines used in dye manufacturing, for example, continued to be used in the United States long after several other industrialized nations prohibited their use.

ECONOMIC FACTORS

As mentioned earlier, workplace conditions are intricately interwoven with the national economic system. Efficiency is king; the goal is maximum productivity at minimum cost. Investors and, therefore, owners, directors, and managers look for concrete, visible, and fast returns from capital investments in plant and equipment—reduced operating costs, improved productivity, a product with a sales-plus that gives it a competitive advantage in the marketplace.

The cost of hazard-control equipment or of modifying processes to reduce hazard emission doesn't fit neatly into the recognized system of values. The immediate capital outlay is deducted from the bottom line, with no compensating, visible benefit in the immediate future. The long-range benefit of lower health-care costs accrues to someone other than the company that installed the hazard control. Although better working conditions mean fewer working days lost, lower compensation payments, and improved employee morale and productivity in the long run, the business and financial worlds tend to operate in a much shorter time frame.

This conventional economic perspective gives rise to the now-familiar arguments about the effects of pollution-control equipment, both inside and outside the workplace, on the profitability of business operations. In extreme cases the costs of cleaning up the environment inside the plant and of cleaning up the emissions into the general environment have been claimed to impose an intolerable burden on a business's finances, resulting in the highly political jobs-versus-pollution-control arguments.

The passage of the Occupational Safety and Health Act in 1970 clarified some of those arguments. The law requires that recognized hazards be

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reduced to acceptable levels, and many concerns that refused to find the money for control equipment, monitoring devices, and services before the law was enacted managed to put in the necessary investment once the bill was passed—without going out of business. In other instances, compromises have been made, with companies working toward the desired goal in stages.

But the effectiveness of OSHA is affected by economic factors in another manner. Hazards cannot be controlled under the law until standards are set, standards cannot be set without the background research and information on which to base the standards, and compliance with existing standards cannot be ensured without effective inspection and enforcement mechanisms. The effectiveness of OSHA and NIOSH, which have prime nationwide responsibility for the protection of occupational health, is directly affected by the resources allocated to them. Only increased awareness of the occupational health problem and increasing demands for improvements will direct more of our national resources into health protection on the job.

Economic factors also contribute to the medical profession's lack of leadership in occupational health. Occupational medicine does not bring funds into a medical school in the same way that departments like medicine and surgery do. Occupational medicine residents sometimes have to pay for their first two years of training; in other specialties, residents are paid while training. And the qualified industrial physician usually does not enjoy either a salary or a status equivalent to that of other specialists. Too often, he or she is perceived as a company person, rather than as a practitioner with integrity. If the poor image of industrial medicine in the medical profession is to be improved, financial and prestige adjustments must be made. These adjustments should follow from a greater awareness of the occupational health problem by the community in general.

As a conclusion to this brief discussion of some of the many problems besetting those who try to improve the working environment, a remark by Willard Wirtz, a former United States Secretary of Labor, provides food for thought:

"There is nothing more critically imperative today than this society's assertion of the absolute priority of the individual over institutional interests and of human over economic values."

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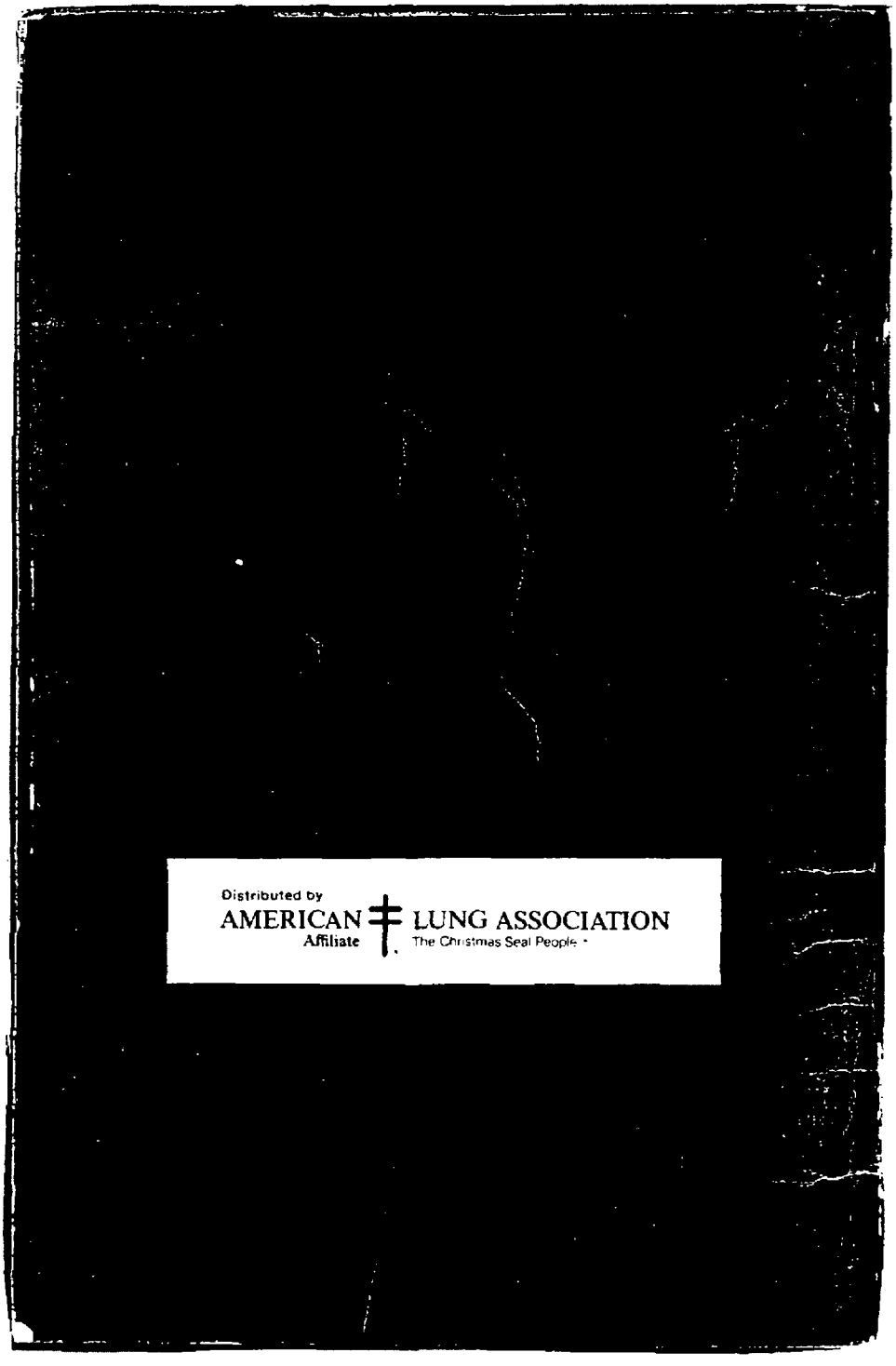
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