CHAPTER 33

THE INFLUENCE OF INDUSTRIAL CONTAMINANTS ON THE RESPIRATORY SYSTEM

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INTRODUCTION

Those persons responsible for evaluation and control of the industrial environment for the purpose of preventing respiratory injury should have (1) knowledge of the anatomy of the respiratory system, (2) an understanding of the factors governing entry, deposition, removal and retention of gases and particles presented to the system and (3) some knowledge of the way in which tissues of the respiratory system react to gases and particles.

The type and severity of tissue response is related to the dose and the nature of the specific agent present. Air, which looks dirty or has an offensive odor may, in fact, pose no threat whatsoever to the tissues of the respiratory system. In contrast, some gases essentially odorless or at least not offensive, and some particles even when present in numbers too small to make the air appear dirty, can cause severe and serious tissue injury. Information about these matters provides an essential motivation to the industrial hygienist and his co-workers and gives them a more balanced approach to their activities. Lack of such knowledge converts a responsibility which should be most interesting and rewarding into a series of rather dull activities.

The progenitors of man evolved in an environment which probably contained a higher concentration of particles and noxious gases than exists now. One could anticipate therefore, that man might retain some ability to overcome such hazards or his genetic precursors would not have survived during that distant period. The fact is, man does possess anatomical and physiological mechanisms which protect the tissues from injury by many airborne agents. The multiple branchings and tortuous course of the narrow passageways through which air is conducted on its way to the deeper portions of the lungs favor the deposition of particles upon the more resilient surface of the proximal conducting tubes, rather than the fragile, more distal gas exchanging surface. The entire surface of the air-containing parts of the lung is covered by a thin layer of fluid which, not only serves as a protective layer, but also as a carrier or vehicle upon which particles are transported from the lung to the pharynx via the mucociliary escalator. This mechanism, plus that of the phagocytic system, is extraordinarily efficient in removing particles or storing them within cells, the macrophages, which are capable of tolerating many kinds of particles without injury. The surface cells of the lung replicate at a high rate and when they are injured, they are rapidly replaced by normal cells. Recovery from tissue injury via these regenerative forces is often surprisingly complete. These various mechanisms of upper airway deposition, surface protection, particle transport and cell regeneration make it possible for man to tolerate surprisingly high concentrations of airborne particles and noxious gases. Nevertheless, the system can be overwhelmed with subsequent persistent injury depending upon the concentration and the kind of gases and airborne particles to which it is exposed. This chapter is aimed at setting forth the principles governing the reactions of the respiratory system to the environment. It is not a compendium of occupational respiratory diseases, nor is it in any sense a textbook of pulmonary anatomy and physiology.

PERTINENT FEATURES OF THE ANATOMY AND PHYSIOLOGY OF THE RESPIRATORY SYSTEM

The human lung is much like a fish's gill, developed in the course of our evolution in a position inside rather than outside the body. It is a gas-exchanging mechanism comprised of a large membrane, on one side of which blood flows and on the other side of which there is a gas phase. A high gradient for oxygen and CO_2 exchange is maintained by the flow of venous blood over one side, and by the pumping of air into and out of the lungs, thus maintaining an optimum concentration of oxygen and CO_2 in the gas phase overlying the other side of the membrane. The gas-exchanging surface is comprised of blood capillaries overlaid by a very thin single cell layer having an effective surface of approximately seventy square meters. Blood is brought to this membrane via pulmonary arteries and conducted away from it via the pulmonary veins. A second system of tubes, the bronchial system, conducts air to and from the gas phase contained in the alveoli, the thin walls of which contain the capillaries. The heart pumps blood through the system, and the muscles of respiration move the chest bellows, and thus pump the respired air to and from the gas phase in the alveoli. One of the marvels of animal construction is that this highly complex and effective system is housed in a relatively small space and is protected from mechanical injury by being contained within the chest cavity. In this discussion we will be concerned chiefly with the air-conducting system and the terminal air spaces or alveoli, the walls of which constitute the membrane separating the gas from the blood phase.

The air-conducting system begins with the

nose, mouth and pharynx. The mouth and oral-pharynx are a globular, open chamber. The nasal and naso-pharyngeal chambers, in contrast, contain ridges or projections, the turbinates. The passages through the nose are semi-separate, narrow and tortuous and this causes the airstreams traversing this system to be turbulent and to change direction frequently, and to be so narrow that the center of the moving airstream is close to the wall of the passages. This arrangement favors deposition of the particles and makes for a more effective gas absorbing surface in this region than exists in the mouth and oral pharynx.

A single tube or airway, the trachea, emerges from the pharynx. This tube divides into the right and left bronchus, each of which further divides into branches entering each lobe of the two lungs. The bronchial system undergoes twenty-three branchings, each of slightly smaller diameter than its parent. The walls of the bronchial tubes become progressively thinner and at the seventeenth branching, small out pouchings or chambers — the alveoli — begin to appear. Subsequent branchings have walls composed essentially of alveoli. Progressing from the trachea toward the ultimate end structures, all divisions devoid of alveoli are called bronchi or bronchioles. When a few alveoli appear in the wall of the conducting system, the tube is designated as a respiratory bronchiole and, when many are present, the tube is an alveolar duct. The ultimate structure at the very end is a wider chamber, the atrium, and from this room only alveoli project.

Air conduction or mass movement of air traverses all bronchi and bronchioles, but at the alveolar duct, or some more distal point, mass movement of air ceases. Further movement of gas molecules into the alveoli, or from the center of the alveoli to the surface of the alveolar membrane, is by diffusion. The anatomical point at which the transition from mass movement of air to pure diffusion occurs is uncertain and probably varies with depth of breath. The location of this interface where mass movement of air ceases and diffusion becomes the only mechanism for more distal movement of particles is of some importance. Particles larger than 0.5 microns do not diffuse, but move through the airways by being entrained in mass movement of air. Hence, particles larger than 0.5 microns penetrate the lung only where mass movement of air occurs and the majority that are deposited fall on the walls of the conducting tubes. Based upon position, some undoubtedly fall by gravity effect into the alveolar openings and thus onto the alveolar surface. A relatively small proportion of the total particles larger than 0.5 microns in diameter entering the lung actually reach the alveolar surface.

The surface of the nasal passage is approximately 160 cm^2 and in most places the air flows through channels approximately one millimeter in diameter. These dimensions, plus the fact that the air stream changes its direction several times and is turbulent at various points during its passage through the nasal structures, makes the nasal passageway effective as a filter for airborne particles and also as a gas absorber, particularly for those gases such as sulphur dioxide which have a rather high solubility in the fluids covering the inner surface of the nose. In adults, the trachea is approximately twenty millimeters, the third or fourth branching of the bronchi five millimeters, and the sixteenth branching 0.5 millimeters in diameter. Further branchings arrive at a tube approximately 0.4 millimeters in diameter. The conducting tubes become slightly wider during each inspiration and narrower during expiration. The frequent change of direction of the branching air tubes and their small diameter greatly favors the deposition of particles from the air passing through them. Thus, those airways proximal to any point in the conducting system act as a filter protecting those passages located more distal to that point.

The nasal passages and the air conducting tubes are lined by a mucous membrane having most important characteristics. The surface of the membrane is covered by mucous, a liquid which arises in part from cells making up the surface of the membrane, and in part from secreting glandular structures located beneath the surface of the membrane, but connected to that surface by a tubular structure. The mucous forms a sheet overlying the tissue surface and would rather rapidly fill the lumen of the conducting system if it were not for the fact that a mechanism exists for propelling the mucous from the deeper parts of the lung towards the pharynx, where it either can be swallowed or expectorated. The majority of the cells making up the surface of the mucous membrane lining the nasal passages and conducting tubes bear a multitude of cilia on their luminal surface, located just underneath the mucous blanket. The cilia beat rhythmically in a fashion which propels the overlying mucous sheet in the direction of the mouth and thus constantly removes the secretions. The mucous blanket serves two obvious purposes. First of all, it acts as a protective layer on top of the delicate cells which line the respiratory conducting system. Equally important, the blanket provides a vehicle for removal of particles which are continuously deposited upon it from the overlying air mass. Thus the mucociliary escalator system becomes a very potent mechanism whereby the lung undergoes continuous self-cleansing. The mucociliary apparatus extends from the pharynx down through the fifteenth or sixteenth generation of branching. The surface of subsequent branchings, including that of the alveoli, is lined by a thin liquid film, which according to recent studies, is constantly being replaced but at a far slower rate than that of mucous secretion. This thin lining probably is removed by a push from the film-forming cells combined with a pull by its attachments to the mucociliary sheet. In essence, there is a continuous cleansing phenomenon provided by removal of a film of varying thickness and composition, extending all the way from the alveolar surfaces up to the pharynx.

A second cleansing mechanism is provided by phagocytic cells, the macrophages, which are found primarily in the alveolated parts of the lung. The origin of the macrophages is not certain, but the

evidence suggests there are always some present, and these can be enormously and rapidly augmented by local cell division and, via the blood stream, by cells of a similar nature formed in other parts of the body. Macrophages are large enough to engulf particles measuring as much as fifteen microns in their largest aspect. These cells also form clusters around even larger particles and produce giant multinucleated cells. The macrophage individually or in clusters, may live for a long period of time with their engulfed particles, provided the nature of the particle is not such as to cause the death of the macrophage. Some macrophages, since they are mobile, find their way out onto the mucociliary escalator and are excreted together with their engulfed particles by that cleansing mechanism.

A third mechanism of lung cleansing is provided by the lymphatic system. There is a liquid filled space between the capillary blood vessels and the surface of the alveoli, into which particles can penetrate or perhaps be carried by phagocytic cells. This liquid filled space is in direct continuity with the lymphatic tubular system which provides for the flow of a liquid, the lymph, in a direction paralleling the bronchi and directed towards progressively larger tubes. Ultimately the lymph is discharged into the venous system, but enroute it passes through aggregates of lymphoid tissue cells, including the large aggregates or lymph nodes at the lung root. Some of the particles that penetrate into this tissue space just below the alveolar surface ultimately appear in and are held by these collections of lymphoid cells. Other particles appear to traverse the lymph nodes and ultimately are discharged into the venous system. The exact mechanism of this transport of particles and their storage is unknown. A substantial proportion of the particles suspended in the inhaled air remain airborne and leave the lung during exhalation. Those particles which are deposited on the surface of the conducting and more distant portions of the airway are removed by the mucociliary escalator, engulfed by macrophages, pass into the lymphatics, become retained in the lymph nodes or enter the blood stream and some portion of the total remain free in the tissues of the lung.

It is worth noting that particles deposited on the distal portions of the mucociliary escalator can traverse the distance from the fourteenth or fifteenth generation of bronchi up to the pharynx within as little as thirty minutes. Cleansing of this portion of the air-conducting tubes therefore is quite rapid. Those particles deposited in a more distal area move much more slowly; it may take days or weeks in order to be cleared or sequestered. Some particles, either naked or engulfed by macrophages, simply remain indefinitely on the surface or in the interstitial tissues between the alveoli.

Bands of smooth muscle encircle the conducting system throughout its entire length. The utility of this muscle tissue is unclear but because of its presence, the lumen of various portions of the conducting system can be markedly narrowed when this muscle contracts. The mucous producing cells can respond quite rapidly to stimuli of various kinds with an augmentation of flow of mucous. Under the influence of some kinds of stimulation, the mucous membrane becomes engorged with blood retained in the capillaries and by an excess production of interstitial tissue fluid. These various mechanisms lead to some degree of narrowing of the airway and consequent elevation of resistance to airflow through the conducting system. These phenomena likewise can be reversed quite rapidly, either by removing the stimulus or by applying appropriate drugs. The muscular system is under the control of nerve impulses and the same appears to be true, to some degree at least, of the mucous secreting glands and possibly even of the ciliary action.

The surface cells, blood vessels, lymphatics and conducting tubes, especially those that are thin walled, are supported by an interlacing network comprised of strands of collagen, reticulin and elastic tissue, termed the connective tissue. This tissue also has its substrate of cellular components, chiefly fibroblasts. The replication rate of this tissue is slower than that of the surface cells or blood vessels, but can proceed in an orderly fashion. If injured, however, the replacing tissue may lose its properly organized structure, and instead form masses of fibrosis or scar tissue. The precise mechanism whereby this occurs is uncertain. As will be discussed later, some kinds of particles evoke a rather marked fibrosis and persistent cellular reaction, while other particles are quite inert and produce little or no such reaction. This brief account of the anatomy and physiology of the conducting system and alveolar structures should be of help in understanding the manner in which the respiratory system reacts to inhaled gases and particles.

Behavior of Gases Which Enter the Respiratory System

Gases are made up of particles of molecular size which move both by mass transfer, as in the flow of gas along a tube, and also by diffusion under the influence of the gravitational forces between molecules. If one breathes back and forth into a bag containing a foreign gas of low solubility, the mass movement of air and diffusion forces will lead to an even distribution of gases through the lung-bag system within three or four minutes of quiet breathing, and within a matter of a few seconds if one takes rapid deep breaths. If the foreign gas has a high index of solubility in the fluids lining the conducting system and the gas is of relatively low concentration, the major portion of the inhaled gas may be absorbed in the upper airways, especially in the nose, and the concentration of the gas reaching the depth of the lung will be lower than at the point of entry. This is particularly true during breathing through the nose. For this reason, gases such as SO_2 will predominantly affect the nose and upper airways, whereas gases of low solubility, such as nitrogen dioxide, will affect the airways rather evenly throughout their entire length. Some gases, as for example nitrogen and carbon monoxide,

appear to be totally inert insofar as their influence on the cellular structure of the respiratory system is concerned. Other gases such as phosgene, nitrogen dioxide, sulphur dioxide, and ozone may have a profound effect on the tissues dependent upon the concentration presented to the cells making up the tissues at the point of contact.

Behavior of Particles Which Enter the Respiratory System

If, by suitably gentle technique, one digests the lungs of fifty- to sixty-year-old individuals, including those who may have worked in the dusty trades, one will obtain a residue which can be assumed to have come from exogenous sources via the airborne route over the years. These tiny particles have a most interesting size range. Many will be found to be so small as to be visible only by electron microscope magnification, while others, generally those larger than 0.5 microns in diameter, can be visualized by appropriate illumination and 450x magnification. Of this entire population of particles retained over a period of many years, approximately half will be smaller than 0.5 microns in diameter. Of those that are larger, almost all will be between 0.5 and 5.0 microns in diameter. Fewer than 0.2 of a percent of the total will be larger than 5 microns in diameter, and less than 0.002 percent will be larger than 10 microns in diameter. If one defines a fiber as a particle having an aspect ratio such that the length is three or more times its diameter, one will observe fibers for the most part to be less than fifty microns in length, although some may be as much as two hundred microns long. Even so, the diameters of these fibers will be distributed as indicated above. If, in contrast, one samples the ambient air to which the general public or those who work in the dusty trades are exposed, one finds particles of these dimensions, but in addition, many of much larger diameter and length. It is incumbent upon us, therefore, to reach an understanding of why it is that the long term retention of particles is limited to the sizes just described, in spite of the fact that millions of particles of greater diameter become airborne and, therefore, have the potential for entry into the respiratory system. The explanation for this arises from our knowledge of the behavior of particles suspended in air (aerosols) and the anatomical and physiologic peculiarities of the lung as described in the preceding paragraphs.

For the immediately ensuing paragraphs we will consider particles to be of a non-fiber character. Particles can vary markedly as to shape and, dependent on composition, as to density; both of these factors play a role in the behavior of particles in air suspension. For our purposes we will consider all particles as being spheres of unit density with the understanding that there could be some variation between particles as to speed of settling, depending on their shape and density. For this part of the discussion we will also think of particles as being far larger than those of molecular size. In this respect the major point would be that those particles larger than 0.5 microns will exhibit essentially zero diffusion activity, and even those down to 0.1 will have minimal such reaction. Those of electron microscope size down to .01 microns and lower will respond to molecular bombardment, and thus exhibit a considerable diffusion activity.

Several physical forces are conducive to the removal of particles from an airborne suspension and their deposition upon surfaces of the respiratory system. Particles suspended in a moving air stream possess inertial forces tending to maintain the direction of motion of the particle. When the air column changes its direction, as at a branching point of the conducting system, or in the tortuous passages of the nose, the entrained particle will tend to continue in its previous direction and be precipitated upon the surface. This effect is directly proportional to the size of the particle, the speed of the air stream, and thus of the particle, and inversely proportional to the radius of the tube. Gravitational forces also remove particles from the air stream and precipitate them on the surface of the respiratory system.

The terminal settling velocity of a particle is directly related to its density, the gravitational constant and to the square of the particle diameter. It is inversely related to air viscosity. Since the gravitational constant and air viscosity are the same at all times, the terminal velocity is in fact predominantly related to particle density and diameter. The degree to which deposition on the basis of gravity will occur is thus related to these two factors, plus the distance through which the particle must fall and the time permitted for the event to occur.

Particle deposition by diffusion is limited essentially to those particles having a diameter smaller than 0.5 microns and, in fact, smaller than 0.1 micron. The smaller the particle the more rapidly diffusion movement can occur. The electron microscope size particles are relatively uninfluenced by any deposition force other than that of diffusion, and the fact that such large numbers of electron microscope sized particles are found in the lung residue indicates that diffusion can play a major role in the deposition of this size particle. Electrostatic and thermal forces have been thought possibly to play a role in deposition of particles in the lungs, but this is still uncertain.

On the basis of known behavior of particles in air suspension and the anatomical arrangement of the conducting tubes, it was predicted that particles larger than ten microns in diameter would be removed completely in the passage of the air stream through the nose and upper airways and that particles between five and ten microns in diameter would be deposited primarily in the upper airways on the mucociliary escalator. Only those particles in the range of one to two microns would be likely to penetrate into the deeper portions of the lung where some deposition in the alveoli might occur by gravity. Particle deposition would, on the basis of these calculations, be least for those particles having a diameter of 0.5 microns. Deposition of particles smaller than this might be increased by diffusion, particularly in

the most distal portions of the air system.

Numerous actual experimental determinations have confirmed this general distribution of location of deposition. For nasal breathing it has been shown that particles larger than ten microns in diameter are almost completely removed and few, if any, reach the conducting tubes of the lungs per se. Some smaller particles also are deposited in the nose, but the majority of these pass through and then are deposited, dependent primarily upon their diameters, along the upper or lower airways. It can be seen from these studies that particles greater than three microns in diameter will have very little opportunity to penetrate deeply and be deposited in the most distal portions of the conducting tubes, where the cleansing action and mucociliary apparatus would be less effective. Since almost 100% of the particles larger than three microns in diameter would fall on the mucociliary escalator and be removed, there is a reasonable explanation for the fact that so few particles of larger size are found in the lung residue after a lifetime of exposure to aerosols of ambient air which undoubtedly contained particles of larger size.

A fiber, defined as a particle the length of which is three or more times its diameter, represents a special case in terms of deposition. As is true of other particles, the settling velocity of a fiber is dependent primarily upon its diameter. One can think of a fiber as being a string of non-fibrous particles insofar as the settling velocity is concerned. In a moving air stream, fibers tend rather strongly to align their length parallel to the direction of air flow. Those fibers that are straight and rigid will therefore present an end-on aspect essentially that of their diameter. Fibers that are curved, curled, or bent in a U shape will have an end-on aspect equal to the width of the curl or curvature. Insofar as interception is concerned there thus will be a much greater chance for deposition of the non-straight fibers, a factor of considerable importance in the narrow airways and in the boundaries of air flow close to the surface. It has been demonstrated that curly fibers penetrate to the deeper portions of the lung much less readily than do straight fibers of equivalent diameter. Length becomes important also to the degree that the fibers are distributed in a random way in the moving air stream. Thus a fiber one hundred microns long oriented at right angles to the direction of flow will have a much greater change of impacting on the surface than will those oriented parallel to the direction of flow. While one does observe an occasional fiber two hundred microns long in the lung dust residues, by far the majority are shorter than fifty microns in length.

Factors Governing the Reaction of the Lung to Gases and Particles

The recognition of whether or not a lung reaction in response to a stimulus has occurred is to a high degree dependent upon the tools and criteria used for such recognition. This is a matter of great importance and often ignored when determining the significance of a specific reaction with respect to whether or not the cellular changes have led to impairment in terms of function, life expectancy, and employability. Cell death and replacement by replication characterizes the organism from conception to death. Physical factors and external agents, such as bacteria and viruses, constantly influence the orderly progression of cell death and replacement during the state that we call good health. From time to time these external agents may exert an influence of sufficient magnitude to interfere with function or life expectancy, and these episodes are thought of as representing disease.

When one examines the body of a healthy person, utilizing the light microscope one can always find some areas of inflammation and scarring and mild disorder of cell replication which is termed metaplasia. As one examines tissues with the electron microscope, one can recognize alterations of cell structure under circumstances which the light microscope will not recognize. During life one is usually limited to the use of less refined tools in order to recognize the presence of abnormalities, and in general our concept of disease is based upon these tools. Such tools are coarse to the degree that the quantitative aspects of abnormality must reach a certain extent before they will disclose the presence of injury. There is thus a quantitative aspect as well as a qualitative aspect in our concept of disease. There is a further factor involved in deciding whether or not an injury is meaningful and thus deserves the appellation of disease. This has to do with whether or not the impairment is of sufficient magnitude to interfere with life and normal pursuits which make up one's life style. For example, some scars representing the end stage of injury are found in the lungs of every adult. Nevertheless, when these scars are minor in extent they do not in any way interfere with function or shorten the life expectancy of the person. In the light of these statements, it is imperative that one realize there is no sharp line of demarcation between being healthy or ill, normal or diseased, injured or uninjured. We can speak in rather broad terms of the way in which gases and particles may or may not injure the lung, but one must bear in mind that the quantitative aspects are probably more important under most circumstances than are the qualitative ones.

The above comments are germane to a balanced understanding of the factors that govern the import of tissue reaction to external agents. Three characteristics determine whether or not tissue injury will occur and be of an extent great enough to impair function, or shorten life. These factors are (1) the nature of the agent, (2) the quantity or dose of the agent brought to bear in action upon the tissues, and (3) the reactivity of the tissues, oftentimes referred to as the host-factor.

Particles and gases vary as to their inherent physical and chemical nature, and this influences whether or not injury occurs. There are some gases as for example nitrogen, and particles such as carbon and most silicates, which under almost all circumstances are essentially inert in terms of evoking tissue reaction. Such particles, when retained in the lung, are engulfed in macrophages

and ultimately come to reside in the tissue, or in lymph nodes where the reaction is either nonexistent, or at most a mild foreign body inflammatory process. Under unusual circumstances of exceedingly high concentration, as for example nitrogen under several atmospheres of pressure, or carbon particles in extra-ordinarily excessive amounts, a cell reaction of greater significance may occur. In contrast, there are some gases such as phosgene and particles such as free crystalline silica, which, because of their inherent quality, are biologically quite active and when present in high enough concentrations can evoke a biological reaction of important magnitude. Bacteria are a special case because these particles, when deposited in the lung, may either be destroyed by macrophages or may grow in large enough numbers to produce disease. In ordinary life pursuits most particles and many gases are inert or relatively inert in the concentrations commonly met with.

On the basis of much evidence, it is generally held that there is a dose or quantity of potentially biologically active particles that will be tolerated without overt evidence of tissue reaction. In terms of an important reaction, this is certainly the case. In terms of recognition of a cellular reaction such as macrophage accumulation in the lung, or subtle changes recognized only by electron microscope or biochemical disturbance of cell structure or function, there is some question as to whether or not this is true. It must also be recognized that the cell reaction to the agent may be an appropriate one and considered a normal reaction rather than an abnormal one. For example, premature death of a cell and its replacement by a normal cell can be thought of as a normal body mechanism for tolerating exogenous agents. In the same sense, the phagocytic action of macrophages with storage of inert particles therein is a normal body function and can scarcely be considered an injury. For our purposes, all injury of a meaningful sort is dose related. This appears to be the case, at least in the minds of most students of the problem, even with respect to carcinogenesis.

The host factor plays an important role, but unless the dose can be accurately measured it is very difficult to quantitate the host reactivity. There are striking examples of true allergic hypersensitivity causing a person to react violently to doses of allergen readily tolerated by the nonallergic. There is also a considerable variation from individual to individual in terms of their immune responses and cellular responses, which is not on an allergic basis. This ordinarily is referred to as hyperreactivity and it accounts for the fact that more serious tissue injury may develop in one individual than in another even though the dose administered to both individuals is the same. This is an important phenomenon because it requires us either to set safe levels for specific agents in terms of the effect on those who are most reactive, or it requires us to find some means of excluding from contact with such agents, those people who are hyper-reactors.

Taking into consideration these three major factors it is no wonder that there is considerable personal variation in terms of whether or not disease occurs in response to deposition of particles or exposure to gases, and that there should be some confusion in the minds of the uninformed with respect to the fact that some gases and particles can exist in high concentrations without ensuing disease.

VARIOUS WAYS IN WHICH THE RESPIRATORY SYSTEM CAN REACT TO AIRBORNE PARTICLES AND NOXIOUS GASES

All parts of the respiratory system can be injured with consequent impairment of function as a result of the inhalation of certain kinds of gases and particles. Among the manifestations of such injury or stimulation are (1) changes of resistance to airflow through the conducting tubes, (2) hypersecretion of mucous, (3) paralysis of the mucociliary escalator, (4) mobilization of macrophages in the tissues and air spaces of the lung, (5) cell injury with consequent acute inflammatory processes or pulmonary edema, (6) chronic inflammation of a granulomatous nature, (7) the development of pulmonary fibrosis or scar tissue, and (8) cell transformation or carcinogenesis. As indicated at the outset of this chapter, it would not be appropriate to discuss all these in detail, but some comments with respect to each of these will be useful.

Changes of Resistance to Airflow

An increase of resistance to airflow, either of an acute and reversible nature or of a chronic and persistent nature, may develop as a result of inhalation of certain noxious gases and particles. It has been shown that deposition of finely divided particles or the inhalation of certain gases such as SO_2 or hydrochloric acid mist will appreciably increase the resistance to airflow and that this is readily reversible following removal of the stimulus or by the use of appropriate drugs.

The site of the stimulation is both in the nose and along the course of the tracheo-bronchial tree. It is presumably caused by contraction of the circular smooth muscle plus some engorgement of the mucosa with consequent anatomical narrowing of the lumen of the conducting system. It is probable that all kinds of finely divided particles may do this to some degree. The dose required for this reaction is usually quite large except in those persons truly allergic. If a specific allergen is deposited in the nose or upper airways, the sensitized person will respond with rapid and oftentimes very severe bronchial narrowing. In this instance the dose may be extremely small. It is also of interest that in this circumstance the particle size can be quite large. Most pollens are greater than ten microns in diameter. These are readily deposited in high concentration in the nose and upper airways where they trigger the acute response. The ability to cleanse these areas by the mucociliary escalator removes the pollens and terminates the episode. Perhaps the most exquisite example of this in an industrial setting is the severe asthmatic response of those who have been sensitized to toluene-2, 4-diisocyanate (TDI).

Hypersecretion of Mucous

Many gases and most particles are irritating to the mucosa of the nose and conducting system of the lungs. When the dose is sufficiently large and the stimulus strong enough, there is an outpouring of mucous from the appropriate cells, leading to cough and an increase of sputum. Acute short term exposures produce a reaction that is fully reversible and in all probability this should be considered a normal phenomenon and not a disease manifestation.

There is some evidence, especially among heavy cigarette smokers, that a persistent stimulation by irritant gases and particles will produce persistent hyper-secretion and enlargement of the mucous secreting glandular system. This is to some degree reversible on removal of the stimulus, but in some individuals there appears to be a persistent hypertrophy and hypersecretion even after the stimulus is removed. The excess secretion leads to chronic productive cough and this condidition is termed chronic bronchitis. The accumulation of secretions in the lumen of the air tubes and the thickening of the mucosa consequent to hypertrophy of the glandular system causes a reduction in the lumen of the air tubes and therefore an increase of resistance to airflow. Such individuals not only have chronic cough and excess sputum production, but also evidences of chronic obstructive airway impairment. There is controversy as to whether this occurs as a result of industrial exposure to gases and particles, but it is generally agreed that industrial environments characterized by high levels of irritant gas or particles aggravate chronic bronchitis.

Paralysis of Mucociliary Escalator

There is evidence in experimental animals that gases such as SO_2 and NO_2 paralyze, at least temporarily, the cilia and thus interfere with the effective removal of mucous secretions. There is some evidence that in response to certain doses there may be a stimulation of the cilia. Recovery from this kind of paralysis appears to be rapid and there is no evidence to indicate that persistent or permanent paralysis of cilia occurs under ordinary life circumstances. The combination of daily excess mucous production and impairment of ciliary action, however, leads to an excessive accumulation of mucous in the conducting tubes. This in turn leads to an increase in resistance to airflow and to inadequate cleansing of the lung with the result that colonization of bacteria can occur with greater ease. As a result, acute bronchitis or pneumonia may ensue. Prolongation of the "residence time" of some biological agents also may occur and be an important influence in causing tissue injury.

Mobilization of Macrophages

Though essentially all of the particles larger than ten microns, and a large proportion of those two to five microns, lodge on the mucociliary escalator and thus are removed, a substantial proportion of those under five microns, and particularly those that are under two microns in diameter, will penetrate far enough out into the lung to be deposited beyond the mucociliary escalator and in the alveolous bearing portion of the lung. Under normal circumstances there are relatively few macrophages in this portion of the lung at any one time. These are present in part for the purpose of sequestering, removing or digesting foreign material taken into the lungs from the general environment. When greater numbers than usual of particles are deposited, there is an augmentation of the macrophage population and in some circumstances the numbers can become very large. This macrophage response is a normal function and cannot, in itself, be considered to constitute a disease.

Macrophages engulf the particles either as single cells or functioning as clusters of cells and retain the particles for the lifetime of the macrophage. The exact life of the macrophage is unknown, but it is measured in weeks and probably in months. Presumably when the macrophage dies and the particles are released, they are rephagocytized by another macrophage.

When inert particles are injected intratracheally into the lungs, there is an initial massive outpouring of macrophages in the regions where the particles are distributed. Over an ensuing period of weeks and months the number of macrophages becomes less and the number of free particles in the lung tissue becomes smaller. One can at this later time observe numerous macrophages filled with particles lying on the surface of the alveoli or in the interstitial tissues and large numbers of particles may be seen in the regional lymph nodes. Fibers shorter than ten to fifteen microns also are phagocytized. Segments of longer fibers may be incorporated in one or more macrophages or entirely surrounded by a cluster of macrophages. At any one time, particles, including fibers, may be seen entirely outside of macrophages even years after they have been introduced into the lung. It is not known whether they have never been phagocytized or are at that moment between periods of residence within a macrophage.

Macrophage reaction is clearly a very important one for removal and sequestration of particles. It is tempting to speculate that the macrophage surrounds the particles and either coats the particle or, by surrounding it with its own protein, breaks the direct contact between the surface of the particle and other cells in the tissues and therefore renders the particle innocuous. There are some circumstances, as for example, free crystalline silica, where those particles small enough to be phagocytized by the macrophage actually kill the macrophage within a matter of a few days. The released particles are rephagocytized and again kill the macrophage. The importance of this phenomena will be discussed under the paragraph on pulmonary fibrosis.

Cell Injury with Acute Inflammation or Pulmonary Edema

Acute cell injury is limited essentially to reaction to noxious gases rather than to particles. Exception to this would be a consideration of bacteria as particles. Gases such as phosgene and nitrogen dioxide and to a lesser degree sulphur dioxide or sulphurous acid mist will, dependent

upon the concentration, produce anything from a mild irritation manifested by hypersecretion of mucous to a severe reaction characterized by death of the cells lining the airways and most distal portions of the lung. In the latter circumstances the lining cells of the conducting tubes are destroyed with the exception of the most basal layer of cells. From this basal layer there is the potential for a reconstitution of the normal cell system lining the conducting tubes. In the alveolar bearing areas, cell injury may lead to destruction of the alveolar surface cells and also of the capillary cell wall with a resultant pouring out of blood plasma or whole blood leading to hemorrhagic pulmonary edema. Depending upon the severity of the reaction, there can be a very rapid outpouring of liquid with death virtually due to drowning in the accumulation of fluid in the deep portions of the lung. With lower concentrations of these gases, the death of the cells making up the alveolar wall is slower and there is a delayed pulmonary edema occurring four to six hours after the exposure. This can be just as fatal as the more acute and sudden reaction. When there is a still lower intensity of exposure, the walls of the alveoli may maintain their physical integrity and gradually be reconstituted in a normal fashion. It is of interest that when particles are inhaled, their distribution within the lung is localized or patchy in nature. The same is true for the inhalation of gases, if the period of inhalation is rather brief, as for example, only a few minutes rather than hours. For this reason not all parts of the lung are involved equally in the severe reaction, and a patchy distribution of pulmonary edema is the rule. If the individual survives the acute reaction, the subsequent course is one of recovery with little or no residual injury. This kind of chemical pneumonia in its earliest stage is a hemorrhagic edema, but in the later stage there is an outpouring of leukocytes and sometimes actual bacterial infection supervenes followed by lobar or bronchial pneumonia. In some unusual circumstances, as for example, exposure to the salts of beryllium, there may be a more gradual or sub-acute development of the chemical pneumonia. Experiments have shown an astonishing ability of animals to recover from this kind of acute cell injury with reconstitution of lung tissue that has in all facets the appearance of normal lung tissue.

Chronic Inflammation of a Granulomatous Nature

This is sometimes termed "chronic interstitial lung disease" and it occurs in individuals exposed to some salts of beryllium, farmers exposed to moldy hay and in certain other occupations such as the handling of bagasse and removal of bark from trees. The exact nature of this disease from an etiologic point of view is uncertain, but it would appear to be predominantly a hypersensitivity reaction with the development of a chronic inflammatory disease in the distal parts of the lung. The nature of the injury is such as to lead to more or less persistent changes which can fluctuate in severity and be reversed to some degree by steroids. Occasionally the injury is such as to lead to the development of pulmonary fibrosis. In some circumstances the exciting agent is thought to be a thermomycete. In all of these cases there appears to be a rather high degree of individual susceptibility. Depending on the extent of the disease, clinical manifestations can either be absent or severe.

Pulmonary Fibrosis

A classical example of pulmonary fibrosis secondary to the inhalation of particles is the reaction to the inhalation of substantial amounts of free crystalline silica. The hypothesis for pathogenesis of this disease, silicosis, having the strongest scientific support is as follows. The particles of free silica, when deposited beyond the mucociliary escalator and picked up by the macrophages, appear to kill the macrophage and in the process release a material capable of stimulating the connective tissue of the lung to produce fibrous scars. This clearly is a dose-related disease.

There are two kinds of scar production, probably based on two separate mechanisms. The particles of silica appear to be collected in focal areas in the lungs inside the macrophages and, at the death of the macrophage, they release the fibrogenic agent which leads to the development of a nodular kind of dense connective tissue characterized by a proliferation of fibrous tissue elements and the laying down of a central area of collagen. These focal points of fibrosis, scattered through the lung, characterize what is termed simple discreet nodular silicosis. In many individuals this is the only reaction that occurs.

In some such individuals, however, a second reaction characterized by the development of a massive irregular scar sometimes reaching five or more cm. in diameter develops. The nodular character is lost and the predominant feature is the large mass of scar tissue. Around the periphery the reaction is more cellular in nature. In contrast to the simple discreet nodular reaction, which appears to be self-limited after removal from exposure to dust, the massive scars tend to continue to enlarge and hence the term "progressive" massive fibrosis. It is postulated that the discreet nodular lesion is the reaction to a fibrogenic material released locally and hence its discreet focal character. In contrast the progressive massive fibrotic lesion is thought to be caused by coalescence of the simple nodular lesions plus the laying down of large amounts of gamma globulin. In other words, the progressive massive fibrosis is in part an immunological reaction and hence its progressive nature. There is not full agreement with this hypothesis. It is of considerable interest that the coal miner, whose nodular lesion is very different from that of the silicotic nodule in that far less scar tissue develops in the "coal worker nodule," nevertheless may go on to develop the large scars of progressive massive fibrosis. The same can apparently occur following unusually heavy exposure to iron oxide or to pure carbon black. It would appear that progressive massive fibrosis is an immunological reaction and thus is a manifestation of hyper-reactivity or an unusual host factor.

In contrast to the nodular lesion produced by the focal collection of free crystalline silica and the reaction to silica in the lung, the reaction to asbestos fiber is of a quite different nature. In this case, the very short fibers, less than five microns long, are phagocytized by the macrophage and appear to reside in the macrophage without harming it. Longer fibers which cannot be totally enclosed within the macrophage and remain naked in the lung tissue or on the surface of the alveolus lead to a cellular reaction which is of a granulomatous nature. If the reaction becomes mature enough, actual fibrous tissue is laid down in a non-nodular manner creating a pattern distinctly different from that of silicosis. Progressive massive fibrosis does not appear to develop as a result of exposure to asbestos, but the question of whether or not the granulomatous and fibrotic reaction to the asbestos fiber is progressive even after removal from exposure is unsettled. The reaction to asbestos fiber is not as focal and is much more generalized than is the reaction to free crystalline silica. In all of these examples where extensive scar tissue forms, lung substance is lost and a restrictive type of pulmonary function impairment occurs. Because of the focal nature, with normal intervening lung tissue, the silicotic reaction is accompanied by less impairment of blood gas exchange than occurs in the more generalized kind of tissue reaction characterizing the response to inhalation of asbestos fibers.

Carcinogenesis

There are numerous cell types in the lungs, most of which undergo division or replication within the lung in order to replace the senile and dying cells or, under intermittent stress, to augment certain cell types such as the macrophage. The epithelial or lining cells of the airways and alveoli are estimated to replace themselves completely every few weeks. It is probable that this rate of replication is accelerated under the stimulus of surface cell injury or irritation. Normally, cell division proceeds in an orderly fashion with the continuous development of identical, normally formed and constituted cells. In response to the influence of irritation and other factors, the cells may gradually change their character and organization and undergo metaplasia. If the alteration is of a particular kind, the cells lose their customary organization and orderly replication and undergo malignant transformation. The frequency with which this happens is unknown, but in some individuals the cancerous cells survive, become established and propagate to produce clinical malignant tumors. It is known that some kinds of inhaled particles foster the development of metaplasia and cancer. For example, the frequency of lung cancer is excessively high in workers exposed to particles of chromium, nickel, asbestos, uranium and other agents. Cigarette smoke, a complex of irritating gases, including nitrogen dioxide, when combined with small particles and hydrocarbons, has carcinogenic action. While single agents have been shown to be carcinogenic in experimental animals, a much higher yield of tumors is obtained if agents are combined. For example, if the surface cells of the bronchi are caused to replicate in an accelerated manner by SO_2 or trauma, benzo (a) pyrene becomes a potent carcinogen, even though it is a weak one when used alone. It would appear that cells are more vulnerable to malignant transformation when they are replicating at a high rate. The concept of co-carcinogen action and multi-factorial influences in carcinogenesis seems to be well established. That there is a host factor as well is very likely.

RESIDENCE TIME AND COMBINED EXPOSURE

Two other concepts with respect to the action of particles deposited on the surface of the lung need to be pointed out in order to establish a better understanding of the possible biological effects of dust and gases. While the deposition of particles on the surface of the proximal conducting airways protects the more distal air tubes and favors particle removal by the mucociliary apparatus, there is an appreciable "residence time" of such particles. During that period of minutes to hours, the biological effects leading to chronic bronchitis, metaplasia and lung cancer could be initiated. If co-existing gases paralyze the cilia and reduce their effectiveness, the residence time would be prolonged.

A second potentiating effect might occur by reason of the fact that particles, which might ordinarily be inert, can become carriers by having biologically active agents adsorbed upon their surface. This might concentrate the active agent and prolong the effect when the coated particle is deposited in the lung.

These two factors might play a role not only in carcinogenesis but also in the other biological effects discussed in this chapter. The importance of taking into account multiple co-existing exposures is becoming more and more apparent and reveals a heretofore inadequately appreciated responsibility of the industrial hygienist.

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CHAPTER 34

OCCUPATIONAL DERMATOSES: THEIR RECOGNITION, CONTROL AND PREVENTION

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INTRODUCTION

Occupational diseases of the skin comprise a broad assortment of skin changes caused by an infinite number of substances or conditions encountered in the work environment. Because of the varied clinical displays, they are appropriately termed "occupational dermatoses," but other titles as industrial dermatitis, occupational eczema, occupational contact dermatitis, and professional eczema are also used. More specifically related to cause are such descriptive titles as cement eczema, chrome dermatitis, chrome holes, fiberglas dermatitis, oil acne, rubber itch, and tar cancer, among others.

These disorders have plagued mankind since antiquity, but little was written about them until 1700 when Ramazzini published his classical text entitled, "De Morbis Artificum," (A Treatise of the Diseases of Tradesmen). Contained within this book are descriptions of occupational skin diseases which remain remarkably accurate today. In 1755, Percival Pott described cancer of the scrotum among chimney sweeps. This was probably the first report dealing with an occupationally induced cancer. Throughout the 18th and 19th century, interest in occupational diseases of the skin was prominent in England, France, Italy and Germany. As industrialization spread to other countries, more people were employed and occupational diseases, including those affecting the skin, occurred in greater numbers. With the advent of World War I, industry expanded enormously in the United States and with it there developed a strong interest in diseases of occupation which has led to better understanding of the work hazards, the diseases they produce, and what could be done to control them.

That occupational skin disease is an important sector in dermatology is without question. However, these disorders are equally important in the field of occupational medicine, industrial hygiene, occupational health nursing and to the insurance companies. This is readily understood because dermatoses are by far the most common of the occupational diseases, numbering no less than one-half to three-fourths of all industrial illnesses reported. They are no less important to the working population, who number between 73 and 80 million. About one-third of this number, 23 million, work in big industry. The remaining 40 to 50 million work in small plants (500 employees or less). Anyone who works is a potential candidate for an occupational skin disease. If he works in a large industrial plant, the chance of developing a skin ailment is less because the medical, nursing and hygienic services are keyed to prevent occupational diseases. Conversely, work in a small plant often is attended by greater risk to health because protective measures generally are poor in quality, if present at all. In any event, it has been estimated that 1% of the working population suffers from occupational skin disease during the course of the year. Thus, with our present employment level, we can expect the occurrence of between 730 and 800 thousand cases. The resulting economic impact of this disease frequency is unknown, but it is estimated that the amount of money required to compensate for lost time and medical care associated with occupational dermatoses each year is in excess of 150 million dollars. Further, there is no way of calculating the dollars lost because of occupational diseases which result in job changes, loss of efficiency and production, or the rehiring and retraining of help.

DEFENSE MECHANISMS OF THE SKIN

The skin is the largest organ of the body. It has approximately 20 square feet of surface area for potential contact with foreign substances in nature and in the industrial environment. It is a multi-functioning organ whose anatomical and physiologic properties subserve protection by regulating body heat, receiving sensations, secreting sweat, manufacturing pigment, and replenishing its own cellular elements. Each of these functions is important in the maintenance of a healthy skin and any deviation from normal can alter the health of the skin and sometimes that of the entire body.

The structure or anatomical design of the skin is protective because of its thickness, resiliency, and the capacity of certain of its layers to inhibit the entrance of water and water-soluble chemicals. Its thickness and elasticity protect the underlying muscles, nerves and blood vessels. Additionally, the thickness and color of the skin afford protection against the effects of sunlight and other sources of physical energy.

Structurally, skin is composed of two layers — the epidermis and the dermis. Epidermis has two essential levels — an outermost stratified layer of horn cells called the "stratum corneum" and the inner living cells from which the horn cells arise. Stratum corneum cells are shed, yet replenished continually because the inner living epidermal layer keeps reproducing cells which eventually become stratum corneum cells. In short, the epidermis has its own self-support system. The stratum corneum layer is essential for protection,

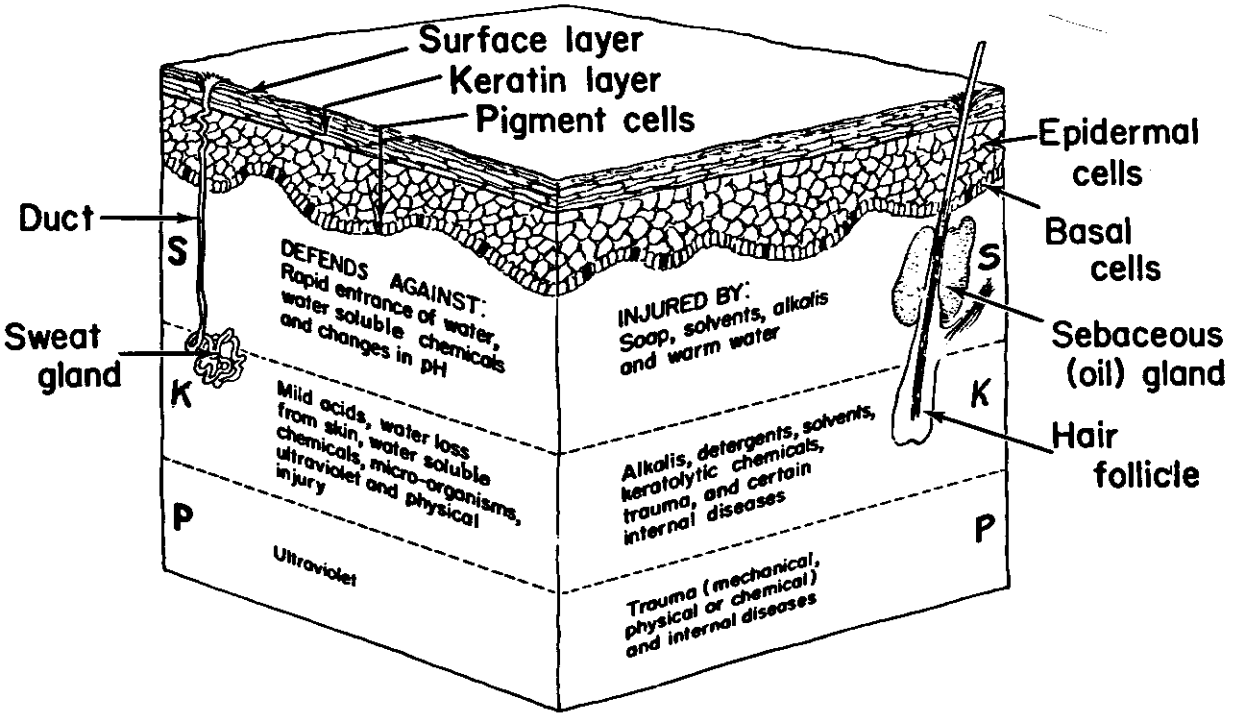

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Figure 34-1. Diagram of the Skin's Protective Layers.

being thickest on the palms and soles. Chemically, it is a complex protein structure which is relatively resistant to mild acids, to water and water-soluble chemicals; but vulnerable to alkaline agents, strong detergents, desiccant chemicals, and solvents (Figure 34-1).

In the lowermost region of the epidermal layer are the basal cells from which all of the epidermal cells arise. Nestled within the basal cell layer are melanocytes or pigment-producing cells which furnish protection against ultraviolet radiation. This comes about through a complex enzyme reaction leading to the production of pigment or melanin granules which are engulfed by the epithelial cells which, in turn, migrate to the upper level of the skin and eventually are shed. Melanin serves as a protective screen against sunlight because the granules absorb photons of light. This mechanism occurs naturally throughout the lifetime of an individual. Sunlight and certain chemicals stimulate pigment formation and, at times, its activity can be inhibited.

Dermis is thicker than epidermis and is composed of elastic and collagen tissue which provide the skin with its resiliency. Invested also in the dermis are sweat glands and ducts which deliver sweat to the surface of the skin; hair follicles in which hairs are encased; sebaceous or oil glands which excrete their products through the hair follicle openings on the skin; blood vessels; and nerves.

Body temperature is regulated by the excretion of sweat, circulation of the blood, and the central nervous system. Blood is maintained at a relatively constant temperature even though the body can be exposed to wide ranges of temperature variations. Sweat facilitates greatly the cooling of the overheated skin surface by evaporation. At the same time, dilation of the blood vessels within the skin also permits heat loss. Conversely, when the body is exposed to severe cold, blood vessels will contract to conserve heat. Nerve endings and fibers present in the skin participate in the receptor and conduction system which allows the individual to differentiate between heat, cold, pain and sense perception. This latter quality allows one to discriminate between dryness or wetness, thickness or thinness, roughness or smoothness, hardness or softness.

Secretory elements within the skin are the sweat glands and the sebaceous glands. Perspiration or sweat contains products from the body's metabolic function, but 99% of sweat is water. Excessive or inadequate sweating can be harmful not only to the skin, but to the general health. Sebaceous or oil glands are situated in the dermis and connect to hair follicles which exit on the surface of the skin. They manufacture an oily substance called "sebum," whose precise physiologic function is not well-understood. Present in normal amounts, it appears to offer some surface protection to the skin. Over-function of these glands is associated with acne.

Coating the outer surface of the keratin layer is a waxy type of mixture composed of sebum, breakdown products of keratin, and sweat. It is

believed that the emulsion-like mixture impedes somewhat the entrance of water and water-soluble chemicals, but its actual protective quality is minimal. It does assist in maintaining the surface pH of the skin, which is normally in the range of 4.5 to 6. Its protective capability is minimized because it is easily removed by soaps, solvents and alkalis. None the less, it is continually replenished under normal conditions and does constitute an extra layer of protection which must be removed before keratin cells can be attacked.

Absorption of materials through the skin occurs when the continuity of the skin is disrupted by an abrasion or a laceration or a puncture. Absorption of fat-soluble chemicals, fats and oils can occur via the hair follicle which contains the hair bulb and a portion of the hair shaft. Some substances as organophosphates are absorbed directly through the intact skin; further, skin permits the ready exchange of gases, except for carbon monoxide. Sweat ducts offer little, if any, avenue for penetration. From the above it is evident that the skin has its own built-in defense mechanism. However, many direct and indirect causes of occupational skin disease can alter this normal defense pattern.

CAUSES OF OCCUPATIONAL DERMATITIS

Indirect or predisposing factors which lead to the development of an occupational dermatosis are generally associated with race, age, sex, texture of the skin, perspiration, season of the year, lack of cleanliness and allergy.

An outstanding example of how racial characteristics predispose to the development of an occupational dermatosis is seen in the marked reaction of the red-head or blond, blue-eyed, light complexioned individual to sunlight. The converse is seen in the resistance to sunlight or ultraviolet displayed by dark or melanotic skin. This racial difference is true in the case of sunlight and in the handling of certain chemicals as tar and pitch which react with sunlight, but dark skin is not universally resistant to the industrial environment.

It has been noted that young workers develop occupational dermatoses more readily that the older workers. This is not a predisposition associated with any peculiar structure of their skin, rather it is the direct result of their frequent disregard for exercising caution in handling injurious materials at work.

Women are just as prone as men to develop occupational skin diseases. In manufacturing plants they work at many of the same jobs and come into contact with chemicals — organic and inorganic, solvents, machine oils, plastics, etc. Women have a natural tendency to be more fastidious in their cleansing habits at work; but, as a group, they also experience additional exposure to cleansers, detergents, waxes and other agents in the household.

Workers with naturally dry skin are less able to tolerate the action of solvents and detergents. Those with oily skins can resist solvents more readily, however, they also are predisposed to developing acne-like lesions induced by cutting oils. Insoluble oils collect within the hair follicle openings and irritate that area sufficiently to cause an inflammation of the hair follicle. Permitted to continue, oil acne will result.

Although sweating is a normal physiologic action, in excess it is often detrimental. Increased perspiration in the armpits and in the groins may cause a breakdown of the skin surface which allows chemicals and bacteria to be more active at those sites. Excessive perspiration also can cause prickly heat, particularly among workmen exposed to high degrees of temperature.

Occupational dermatitis is generally more common during warm weather. When the work area is hot, workmen become lax in the use of protective clothing and, thereby, overexpose themselves to hazardous substances. Warm weather also means greater exposure to sunlight, poisonous plants and insects, the effects of which may or may not be related to the job.

Keeping the skin free of harmful agents encountered at work is readily accomplished by frequent washing and the proper use of protective devices. However, workmen with poor cleansing habits are prone to develop an occupational skin disease. These same individuals tend to wear soiled work clothing for prolonged periods of time. This practice enhances the amount of contact between the skin and chemical contaminants in the soiled clothing.

It is a natural tendency on the part of many people to believe that all dermatitis is based on allergy. Among the working population, allergy accounts for about 20% or less of all the occupational dermatoses seen. Certain individuals known as "atopics" are born with a predisposition for the development of allergic diseases such as hay fever, asthma, hives and eczema. However, these people are no more disposed to allergic contact dermatitis in industry than are nonallergic workmen. Certain industrial or environmental substances are well-known allergens, but these materials can cause a contact allergy in anyone. When one develops an occupational contact allergy it does not mean that he has to quit work. By using good protective measures, he can generally continue at his job. However, there are certain people who develop such a high level of allergic reaction that they must seek other types of work.

Direct Causes

In order of their importance and frequency, the direct causes of occupational dermatitis can be classified as chemical, mechanical, physical and biological.

Chemical

Organic and inorganic chemicals are the major dermatoses hazards present in the work environment. They constitute a never-ending list because each year the chemical spectrum gains additional agents capable of injuring the skin. Chemicals act as primary irritants or allergic sensitizers or photosensitizers. A primary irritant is a substance which, if permitted to contact the skin in sufficient concentration for a sufficient length of time, will produce a demonstrable effect upon the skin at the site of contact. In short, a primary irritant

will affect the skin of anyone. Some irritants are strong or absolute in their action; for example, chromic acid, nitric acid, sodium hydroxide or chloride of lime can produce their effect within moments after contact, or at least within a few hours following initiation of contact. Other substances act as relative or marginal irritants and require several contacts before any of their effects are seen; for example, prolonged exposure to soap and water or to soluble cutting fluids or mild solvents as acetone.

About 80% of all occupational dermatoses are caused by primary irritants. Most inorganic and organic acids act as primary irritants. Certain inorganic alkalis as ammonium hydroxide, calcium chloride, sodium carbonate, sodium hydroxide are skin irritants. Organic alkalis, particularly the amines, also are active irritants. Metallic salts, notably the arsenicals, chromates, mercurials, nickel sulphate and zinc chloride, produce severe irritant effects on skin. Organic solvents represent a large number of substances, such as the chlorinated hydrocarbons, petroleum base compounds, the ketones, the alcohols, terpenes, among others, which irritate skin because of their solvent qualities.

Primary irritants damage skin because they have an innate chemical capacity to do so. Many primary irritants are water-soluble and, thereby, actively able to react with certain tissue within the skin. Even the water-insoluble compounds which comprise many of the solvents react with the lipid elements within skin. We do not know the precise mechanism of primary irritation on the skin, but some useful generalizations serve as indices to explain the activity of groups of materials in the irritant category.

Keratin Solvents

All of the alkalis, organic and inorganic, injure the keratin layer when concentration and exposure time is adequate. These agents soften the keratin cells and succeed in removing many of them. At the same time, they bring about considerable water loss from this layer resulting in dry, cracked skin which prepares the way for secondary infection and also, at times, for the introduction of allergic sensitization.

Fat and Oil Solvents

Just as organic solvents dissolve oily and greasy industrial soils, they remove the surface lipids and disturb the keratin layer of cells so that they can no longer maintain their water-holding capacity. Workmen exposed each day to the action of the organic solvents develop exceedingly dry and cracked skin.

Protein Precipitants

Several of the heavy metal salts precipitate protein and denature it. Best known for this action are the salts of arsenic, chromium, mercury and zinc.

Reducers

Salicylic acid, oxalic acid, urea, as well as other substances, in sufficient concentrations, can actually reduce the keratin layer so that the latter is no longer protective and an occupational dermatosis results.

Keratin Stimulants

Several chemicals stimulate the skin so that it undertakes peculiar growth patterns which may lead to tumor or perhaps cancer formation. Certain petroleum products, a number of the coal tar based materials, arsenic, and some of the chlorinated hydrocarbons can stimulate the epidermal cells to produce these effects.

Primary irritant chemicals are commonly encountered in industry; they account for about 80% of all of the occupational dermatoses seen; they attack the skin in various ways; strong irritants can injure the skin in a matter of moments or hours; weak or marginal irritants may require several days. Because a workman must come into contact with a primary irritant does not mean that he will necessarily develop an occupational dermatitis. Exposure to irritant materials can be controlled when proper precautions are taken.

Sensitizers

Chemicals which cause allergic contact dermatitis are far fewer in number than are the primary irritant substances. Best known among the allergenic agents are such plant toxins as poison ivy, poison oak, and poison sumac. Other well-known cutaneous sensitizers are the alkaline dichromates, epoxy resin systems, hexamethylene tetramine, phenolformaldehyde resins, among others. A sensitizer does not cause visible change on the skin following first contact; but after several contacts, which may require days or sometimes months, it causes specific changes in the skin so that further contact on the same or other parts of the body will induce a dermatitis. Allergic contact dermatitis is rarely seen amongst workers before the fifth or seventh day after exposure is initiated, whereas primary irritant dermatitis can occur within a few hours or a few days. Only in a few instances, as for example, when exposed to poison ivy or epoxy resin systems or phenol-formaldehyde plastics, do large numbers of workers become allergically sensitive.

Photosensitivity

There are two forms of photosensitivity dermatitis — phototoxic and photoallergic. Excess exposure to sunlight or artificial ultraviolet can injure the skin through a phototoxic effect. Many workmen are exposed to various forms of natural and artificial light, for example, farmers, policemen, road builders, telephone and electric linemen and sailors. Additional to exposures from natural and artificial light sources are numerous chemicals, plants and drugs which react with selected wavelengths of natural and artificial light to cause phototoxic or photoallergic dermatitis. The best-known industrial chemicals with this capacity are derivatives of coal tar, as anthracene, phenanthrene, and creosote; and certain dyes — acridine, eosin and rose bengal. Further, a host of topically applied and ingested drugs can interact with specific wavelengths of light to produce these effects. Examples of these are certain chlorinated compounds (present in soaps for antibacterial purposes), tranquilizers of the phenothiazine type and drugs related to sulfonamid and some antibiotics.

Mechanical Causes

Anyone who works experiences some type of a mechanical trauma involving friction or pressure. Friction may result in an abrasion or, more commonly, a callus, produced by repetitive types of hand motions or through using a certain type of tool. Those who work with pneumatic tools may experience untoward effects of the hands and forearms, depending on the type of tool being used. High frequency tools can produce what is called "painful white fingers," a disorder accompanied by spasmodic pain in the fingers of the hand operating the tool. Heavier pneumatic instruments, like hammers, riveters and chisels, can cause painful tendinous or muscle or bone injury to the hands and forearms.

Physical Causes

Heat, cold, sunlight, artificial ultraviolet and ionizing radiation are capable of injuring the skin. Jobs involving exposure to high temperature induce excessive sweating and prickly heat. High levels of heat may also cause systemic symptoms and signs as heat cramps, heat exhaustion and even heat stroke. Burns of the skin can result from electric shock, sources of ionizing radiation, molten metals and glass, and solvents or detergents being used at elevated temperatures.

Low temperatures may induce frostbite and permanent damage to blood vessels. The ears, nose, fingers and feet are common sites for this type of cold injury. Electric and telephone linemen, highway maintenance workers, farmers, fishermen, policemen, postmen, among other outdoor employees, may experience this type of skin injury.

Many people who work outdoors are exposed to sunlight and increasing numbers come into casual or prolonged contact with artificial ultraviolet sources as molten metals and glass, welding operations and the plasma torch. A newer light source is found in operations using the laser apparatus. Since these monochromatic beams can injure skin and other biologic tissue, appropriate protective devices should attend their use.

Numerous ionizing radiation sources are being used in industry. Alpha radiation, though not injurious to skin, is dangerous if inhaled or ingested. Beta radiation can injure skin and the body in general if inhaled or ingested. Gamma radiation and x-rays are well-known skin and systemic hazards when sufficient exposure occurs. X-ray diffraction instruments pose a potential source of skin injury to those employed in the operation of these devices.

Biologic Causes

Bacteria, viruses, fungi and parasites attack the skin and sometimes produce systemic disease of occupational origin. Animal breeders, agricultural workers, bakers, culinary employees, florists, horticulturists, laboratory technicians and tannery workers are among the ones who may develop an occupational dermatosis or a systemic disease caused by one of the biologic agents. Where these substances are known to be connected with the work, all necessary precautions for preventing disease must be exercised. A common type of skin infection seen among workmen is caused by staphylococci invading the site of a previous wound.

Clinical Types of Occupational Dermatoses

Several clinical variations of occupational skin disease are known to occur; however, the lesions produced on the skin rarely are characteristic of a specific chemical. Nevertheless, certain types of skin changes do suggest contact with certain classes of agents, for example: (a) Acute contact dermatitis is generally caused by a primary irritant or a sensitizing chemical, a poisonous plant or a photosensitizing agent. (b) Acne-like skin diseases usually mean contact with petroleum oils and greases, tar or pitch or certain chlorinated hydrocarbons which induce chloracne, for example, chlorinated diphenyls and triphenyls, chlorinated diphenyl oxide, among others. (c) Pigment changes in which there is a loss or gain in pigmentation. Several complex phenolic compounds present in germicidal agents have caused loss of skin pigment. Some examples are tertiary butyl phenol, tertiary amyl phenol, tertiary butyl catechol. Conversely, petroleum oils, asphalt, pitch, photoreactive chemicals and sunlight produce gains in pigment formation. (d) New growths. Sunlight, x-ray, tar, arsenic trioxide, impure paraffins and certain shale oil fractions are known to cause skin tumors, which may become cancerous. (e) Ulcers. Arsenic trioxide, chromic acid, sodium chromate, potassium dichromate, lime, thermal burns and forceful injury can cause ulceration of the skin.

These five examples of occupational skin changes (dermatoses) are presented in a decreasing order of frequency. To recognize them and understand their causation requires a familiarity with diseases of the skin and the environmental factors which influence their development. It involves understanding the nature of the lesion, the site of the eruption, the course of the disease and the correct interpretation of any clinical tests found necessary to aid in the diagnosis. Occupational skin problems are best managed by physicians familiar with them.

PREVENTION OF OCCUPATIONAL SKIN DISEASE

Dermatoses caused by substances or conditions present in the work environment are largely preventable, but only through the conjoint effort of management, supervision and the workman. That such can be accomplished is best demonstrated in large industrial plants, while the converse is demonstrated in hundreds of small work establishments where little, if any, interest is shown in preventive measures. Two major approaches to the control of occupational diseases, in general, or dermatoses, in particular, are: (a) environmental control measures and (b) personal hygiene methods.

Engineering Controls

The best time to introduce engineering controls is when a plant is being designed. At that

time, control measures can be integrated more readily into the operations than after the plant has been built. Ideally, operations would be conducted in entirely closed systems, but not all industrial processes lend themselves to this approach. When closed systems are used, raw materials can be brought to the manufacturing site in sealed cars or containers and their contents emptied into storage tanks or bins, and later cycled through retorts or other reaction apparatus, meanwhile preventing contact with the material being processed. If this type of control system is unattainable, it is generally possible to install local systems which collect the irritant dusts, vapors, fumes and mists. In any event, it is recognized that elaborate ventilation systems are costly and most plants cannot afford this method of control. Smaller plants may have many devices intended for control purposes, but experience shows generally that small plants depend more upon personal hygiene practices than on environmental control measures.

Personal Hygiene

If a workman is to minimize contact with harmful agents, he must have access to facilities for washing his hands and be furnished other means of keeping clean at work. It is up to the plant to provide adequate washing facilities and good cleansing materials. Washbasins must be well designed, conveniently located and kept clean, otherwise they will be used infrequently, if at all. The farther a workman must walk to cleanse his skin, the less likelihood there is of his doing so. Inconveniently located washbasins invite such undesirable practices as washing with solvents, mineral oils or industrial detergents, none of which were intended for skin cleansing. For workmen to keep their skin reasonably free of injurious agents, they must use washing facilities at least three times a day — during work, before lunch, after lunch and before leaving the plant. Working with toxic chemicals and radioactive substances requires the daily use of showers.

Many industrial hand cleansers are available as plain soap powders, abrasive soap powders, abrasive soap cakes, plain soap cakes, liquids, cream soaps and waterless hand cleaners. Workmen generally like powdered soaps because they gain a sense of having removed soils because of the frictional element. Most powdered cleansers with abrasives will remove tenacious soils, but waterless cleaners have become very popular because they remove greases, grimes, tars, paint and some plastics with relative ease. However, care must be exercised in selecting waterless cleaners because many of them contain excess alkalis and solvents, which cause excessive drying of the skin and sometimes contact dermatitis.

Management should have more than a passing interest in providing good washing facilities and good cleansing products. All too frequently, the cleansing agents are purchased by people having no familiarity with their quality. The practice usually results in procuring industrial hand cleansers which are cheapest in price.

Disposable hand towels are desirable because they can be discarded after use. They are an excellent replacement for the old fashioned machinist waste.

Protective Clothing

It is not necessary that all workmen wear protective clothing, but for those jobs in which its use is required, good quality clothing should be obtained. Manufacturers now provide a large selection of protective garments of rubber, plastic films, leather, cotton or synthetic fibers designed for specific purposes. For example, we now have access to different clothings which protect against acids, alkalis, extreme exposures of heat, cold, moisture, oils and the like. When such garments must be worn, management should purchase and control the use of the protective gear. They should see to it that the clothing is serviced and laundered often enough to keep it protective. When workmen are required to purchase their own protective clothing, they generally buy the cheaper materials with little thought being given to the purpose for which it is intended. Further, if work clothes are laundered at home, it can cause contamination of family wearing apparel with chemicals, fiberglas or other dusts.

Protective clothings include hair covers as caps and nets, coveralls, smocks, aprons, sleeves, gloves and shoes. Protective sleeves and gloves are helpful devices, but care must be exercised in their use. Unless they are made of tear-away fabric or film, a sleeve or glove may cause serious injury to an arm or a hand. Cotton or leather gloves are useful for protecting the hands against friction and dusts. Synthetic rubber gloves are used for protection against acids and alkalis. Neoprene dipped cotton gloves will protect against most liquid irritants. Some workmen do not like to wear rubber gloves because the rubber causes the hands to perspire excessively. Gloves with built-in liners are probably less efficient and comfortable than plain plastic or rubber gloves which can be worn over replaceable cotton liners. Each workman requiring this type of protective gear can have three or four pairs of washable cotton liners which can be changed when his hands become saturated with perspiration. Major manufacturers of protective clothings have descriptive catalogs which provide useful information in selecting the best protective apparel for certain exposures.

Barrier Creams

Generally speaking, a barrier of protective cream is the least effective way of protecting skin. Nevertheless, there are instances when a protective cream may be the best method available for preventing contact with harmful agents, for example, if the face cannot be covered by a shield or gloves cannot be worn. There is no all-purpose protective cream. Several manufacturers compound a variety of products, each designed for a certain type of protective purpose. Thus, there are barrier creams for protecting against dry substances and those which protect against wet materials. Using a barrier cream to protect against a solvent is not as effective as using an impervi-

ous glove; however, there are compounds which offer some protection against solvents, providing the creams are used with sufficient frequency. To use a protective cream correctly, it must be applied on clean skin at the beginning of the work shift, removed and reapplied at the break, removed at lunch, reapplied after lunch, again in the afternoon and, of course, removed at the close of the work shift. When barrier creams are used, they should be selected because of a particular need by workmen who cannot wear other types of equipment. They should not become the substitute for protective clothing.

In summary, management is responsible for furnishing the facilities and products required to keep the work-place safe. Similarly, the workman has certain responsibilities in a prevention program. He must wear protective clothing if it is required; he must wash with frequency if he is working with irritant or toxic chemicals. If he develops an occupational dermatitis in spite of his attempts to prevent its occurrence, he should report immediately to the plant dispensary or to his physician for prompt diagnosis and medical treatment.

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CHAPTER 35

PRINCIPLES FOR CONTROLLING THE OCCUPATIONAL ENVIRONMENT

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INTRODUCTION

Hazards and potential hazards in the occupational environment can be purely mechanical in nature, or they can take the form of materials which are capable of causing fire or explosion, or of producing injury by inhalation, skin or eye contact, or by ingestion. Physical forms of energy such as noise, non-ionizing and ionizing radiation, and heat are also potential hazards. Most basic to the control of any hazard is the concept that it can be controlled. Once the hazard is defined properly and the need for and the degree of necessary control is determined, then the only requirements are imagination, trained personnel and money to put the control methods to work.

The basic principles for controlling the occupational environment consist of substitution, isolation and ventilation. Not all basic control principles are applicable to every form of hazard, but all occupational hazards can be controlled by the use of at least one of these principles. Ingenuity, experience and a complete understanding of the circumstances surrounding the control problem are required in choosing methods which will not only provide adequate control, but which will consider installation, operating and maintenance costs and personal factors such as employee acceptance, comfort and convenience. Furthermore, hazards, costs and benefits can change with time so that hazard control systems need continuous review and updating. The aim, then, must be not only to devise efficient hazard control methods, but to evaluate the effectiveness of those methods at regular intervals.

SUBSTITUTION

Usually, when one thinks of controlling a hazard he thinks automatically of adding something to do the controlling. For example, an engineer is more likely to think of controlling a vapor hazard by ventilation than by substituting a less hazardous material for the one which is causing the problem. Yet, substitution of less hazardous materials or process equipment, or even of a less hazardous process, may be the least expensive as well as the most positive method of controlling an occupational hazard.

Unfortunately, substitution is not a technique easily taught. No one can sit down with a slide rule, pencil and paper and decide how to best use substitution to eliminate an occupational hazard. Instead, the principle of substitution is demonstrated best with examples so that by analogy the student may apply what he has learned to his particular problem.

Process

One of the main hazards to our atmospheric environment results from the use of gasoline-powered internal combustion engines in nearly all of our automobiles.[1] Control of this source of air pollution is being attempted in many ways, from the passage of laws to the modification of gasoline to the substitution of a less hazardous process. Substitute processes range from diesel engines to electric motors, and even include the greatly increased use of mass transit systems. That there is no agreement on the best "less hazardous process" (or in fact, that process substitution is necessary) indicates that more study is needed and problem solutions may be political as well as scientific.

Choosing a substitute process is not always difficult. For instance, dipping an object into a container of paint almost always creates much less of an inhalation problem than does the process of spraying that object. Cutting is usually less noisy than breaking or snapping; mechanical stirring causes less material to become airborne than does sparging; generating electric power from nuclear energy causes less air pollution than does the use of fossil fuel, but hydroelectric power is less polluting than either; and distillation usually causes fewer problems than does crystallization.

After considering many examples of process substitution, one principle appears to stand out: the more closely a process approaches being continuous (as opposed to intermittent), the less hazardous that process is likely to be. This principle is a fairly general one and applies to energy hazards such as noise, as well as to the more familiar material hazards. This principle is not always useful, but its application should be considered whenever hazard control by process substitution is attempted.

Equipment

Where the process itself does not need to be changed to reduce hazards, the needed control often can be achieved by substituting either equipment or materials handled, or both. Substituting equipment is nearly always less expensive than substituting processes and often can be done "on the job." On the other hand, finding a substitute material may be easy or may require extensive research and/or process changes. For these reasons, equipment is substituted more often than either processes or materials.

Equipment substitution is often the "obvious" solution to an apparent hazard. An example might be the substitution of safety cans for bottles to store or contain flammable solvents, or the substitution of safety glass for regular window glass in the sash of a "fume" hood. Examples such as these can be multiplied indefinitely because they are obvious on inspection.

One of the main requirements for efficient equipment substitution is the awareness of alternates. Persons concerned with hazard reduction must familiarize themselves with all kinds of "safety" equipment as well as with the processes and process equipment in their jurisdiction. For example, sideshield safety glasses are unlikely to be substituted for regular spectacles unless someone knows the need for, as well as the existence of, the side-shield glasses. Unless someone knows that neoprene gloves are being ruined by contact with chlorinated hydrocarbons, and also knows that polyvinyl alcohol gloves are available and impervious to this kind of attack, a substitution is unlikely.

Realistic suggestions for process equipment substitution are often based on a background in both engineering and industrial hygiene, but even without an extensive background, a fresh look at an old process or problem can pay large dividends. The man who gets out and around within a plant, a company, a city or a nation is likely to observe new solutions to problems and thus is likely to be able to apply them elsewhere. Good equipment substitution is based on common sense, ingenuity, keeping up with the state of the art, and the experience of working with people, processes, and the equipment used by both.

Material

After equipment substitution, material substitution is the technique most often used to reduce or to eliminate hazards in the occupational environment. Examples abound. The substitution (forced by a tax law in 1912) of red for white phosphorus in matches drastically reduced both an industrial and a "general" hazard. Substitution of perchloroethylene for petroleum naphtha in the dry cleaning industry essentially eliminated a serious fire hazard. Using tritium-activated phosphors instead of radium-based paint for watch and instrument dials has reduced the hazards associated with the manufacture of the dials, and in addition has reduced by a small amount the background radiation experienced by the general public. Removing beryllium phosphors from fluorescent lamps not only eliminated a hazard to the general public, but also eliminated a more serious hazard to the men manufacturing such lamps.

Many years ago the principal cold cleaning solvent was petroleum naphtha. Because of its fire hazard, a substitute material was sought. Carbon tetrachloride appeared to be ideal because of its low flammability, good solvent power, and low price. Experience and a great deal of research, however, showed that a serious fire hazard had been traded for a perhaps even more serious vapor inhalation hazard. Today, carbon tetrachloride is being supplanted by several other chlorinated hydrocarbons, notably 1, 1, 1-trichloroethane, trichloroethylene, perchloroethylene and methylene chloride. Each of these substitutes is far less toxic and far less hazardous to handle than is carbon tetrachloride, although each has its own hazards. In addition, the fluorinated hydrocarbons are being used more and more despite their expense, mainly because their inhalation and fire hazards are so low.

The principle of material substitution carries with it the same type of reward and the same potential hazards as other kinds of substitution. Substitution of a different material can reduce or eliminate hazard, but one hazard can be substituted for another inadvertently. A careful watch must be kept for unforeseen hazards that may crop up when any kind of substitution is used. An excellent source of information about the toxic properties and hazards of materials and their substitutes is the Hygienic Guide series published by the American Industrial Hygiene Association.

ISOLATION

Isolation is the term applied when a barrier is interposed between a hazard and those who might be affected by that hazard. The barrier may be physical, or distance or time may provide the isolation considered necessary.

Stored Material

Stored material rarely poses an overt hazard, and therefore, whether it is raw material or finished product, those concerned are likely to take it for granted and to assume that it poses no threat. This assumption can be dangerous.

When flammable liquids are stored in large tanks above ground, common practice is to group the tanks on a "tank farm" but to isolate each tank from the others by means of a dike made of earth or concrete. If a major spill does occur, the (possibly flaming) liquid is restrained by the dike from coming close enough to other storage tanks to affect them. For more positive protection, tanks are buried to interpose an even more formidable barrier between their contents and the general environment. A further example is to restrict the volume of material stored in a single container. This exemplifies the use of isolation to reduce a hazard by imposing many small barriers rather than one large one between the contents and the environment.

Where the principal hazard of a liquid arises from inhalation rather than from fire, the imposition of a physical barrier becomes much more difficult than simply building a dike. When the quantities are relatively small (up to a few tens of gallons, perhaps) the best storage technique uses both isolation and ventilation. An example of this practice is the more and more common use of ventilated storage cabinets in laboratories.[2] Such cabinets are usually made of fire resistant material and air is drawn through them constantly by means of a fan which discharges out-of-doors. This type of arrangement interposes both a physical and a ventilation barrier between the contents of storage vessels and the laboratory environment and in ad-

dition, may free much valuable hood space for other than storage use.

Solids usually are stored either in original containers (bags, cans, or drums), bins, or simply in piles which may even be out-of-doors. Except in unusual cases, solids rarely pose problems in storage which compare in magnitude with those of liquids and gases. Outside storage piles can be unsightly and can be the source of air pollution problems; in such cases a physical barrier is the usual answer. The barrier may be as simple as a tarpaulin or as complex as a storage building with several kinds of materials handling equipment.

Equipment

Most equipment used in processing operations is designed to be safe if it is used properly. On the other hand, there are times and cases where this is far from true. Equipment that is operated under very high pressure, for instance, may well pose a severe hazard even when operated correctly. In such cases, the proper action to take is to isolate the equipment from the occupational environment. Usually physical barriers are used and the barriers may be very formidable ones, indeed. Extensive use may be made of armor plate as well as reinforced concrete, mild steel, and even wood. Viewing the work area may be done by remote controlled television cameras, simple mirrors or periscopes.

Equipment isolation may be the easiest method of preventing hazardous physical contact, for instance with hot surfaces. Insulating a hot water line may not be economical from a strictly monetary standpoint, but may be necessary simply because that line is not sufficiently isolated from people by distance.

Inhalation hazards can often be reduced markedly by equipment isolation. One example is that of isolating pumps. Nearly all pumps used in industry can leak and will do so, at least occasionally. Proper planning should take this fact into consideration, perhaps by arranging vessels and piping so that pumps handling hazardous materials can all be located in one area. That area, then, can be isolated physically from the remainder of the process equipment. If, then, the pump room (and/or each pump) is ventilated properly, minor leaks will be of no consequence, and major ones will be repairable without a serious inhalation hazard to the mechanic.

Process

Process isolation is usually thought to be the most expensive of the isolation methods of hazard control, and thus is probably the least used. Nevertheless, with today's space-shot-perfected techniques, some extremely complex processes and equipment have been shown susceptible to remote control, and in principle there is probably no process which cannot be operated remotely if the expense of remote operation is justified.

Process isolation techniques were given great impetus when men sought ways in which to handle radioisotopes safely. They found that the hazard from external radiation sources could be attenuated with shielding and distance, but both of these techniques required the development of very sophisticated methods of remote operation. Master-slave manipulators were designed to allow direct "handling" of equipment from very remote locations and this, in turn, accelerated the development of different viewing methods, complex electronic systems, and the theory and philosophy of remote operation.

The modern petroleum processing plant is an example of the use of remote processing. Many of the newer plants are based almost completely on centralized control with automatic sampling and analysis, remote readout of various sensors, on-line computer processing of the data, and perhaps actual computer control of process equipment. These techniques were not developed with hazard control uppermost in mind; instead, economy of operation was the spur, but safety was a by-product.

Computer-controlled processing also appears to be gaining acceptance in the chemical industry. For the most part, this change has been in response to economic pressures because, despite their high initial costs, computer-controlled continuous processing plants can be operated with much less expense than that associated with manual operation, and at the same time produce a superior product. Such plants enjoy the advantages of remote operation and also those of continuous processing with attendant relatively low volumes of materials actually being handled. This combination can result in a very low hazard potential.

Process isolation, however, by its very nature can pose some rather extreme hazards. That is, when human intervention is required, the potential hazard may rise abruptly from near zero to near certainty. In such cases, full use must be made of techniques of isolating the man from his environment.

Workmen

Isolating workmen from their occupational environment has been used since antiquity, and will continue to be necessary in the foreseeable future. The first blacksmith to don an apron of hide was using this principle just as certainly as is the present day radioisotope handler with his plastic airsupplied sealed suit and its connecting "tunnel."[3] Pliny, the Elder, wrote about the use of pig's bladders by miners to reduce the amount of dust inhaled[4] and today advertising men extol the virtues of masks made of polyurethane foam to accomplish the same thing.

Using personal protective equipment of any sort exemplifies the principle of isolating man from his occupational environment. Protective equipment for workers should usually be designed for emergency or temporary use, but this does not always hold true. Experts in the safety field stress the continual use of some sort of eye protection if only because loss of vision is such an extreme penalty to pay for a moment's inattention. Hard hats and safety shoes with steel toecaps are other examples of protective equipment designed to be cheap insurance against severe loss. Some kinds of personal protective equipment are so ubiquitous as to be almost a badge of the trade. The butcher's

apron, the chef's tall hat, the welder's helmet, the first baseman's glove, the logger's boots and the fullback's shoulder pads are all devices designed to help isolate man from his occupational environment.

Today it is possible to isolate anyone from practically any environment for nearly any length of time. We can send men through the vacuum of space to the moon, for instance, or send them to the depths of the sea, completely protected from rather extreme environments. Nevertheless, even though essentially complete protection is possible, it is rarely used.

Completely isolating a man from his occupational environment is difficult and expensive; therefore, when worker isolation is necessary, it is usually partial rather than complete. Even partial isolation can result in discomfort (consider wearing a gas mask all day, for instance), and in such cases other techniques of controlling the environment should be considered seriously. Face shields, ear plugs, rubber gloves and the like should always be available if their use is warranted, but the aim of the engineers and planners should be to make their continual use unnecessary. Furthermore, all emergency protective equipment should be inspected periodically and tested if necessary to assure that it will perform its intended function in use.

Testing of protective equipment and planning for its proper use (see Chapter 36) are both very complex fields. By its nature, most equipment of this type is designed for use at times when all of the hazards are not delineated readily — where, in fact, the real hazards may never be known. For instance, canister-type gas masks have been regarded as suitable for respiratory protection in emergencies provided that the air still contains enough oxygen to sustain life. Chemical reactors, tanks, sewers and buildings on fire don't always provide enough oxygen to sustain life, and therefore, injuries do occur from asphyxiation. Furthermore, the canister on the mask may not be designed to protect against the air contaminant(s) actually present and again people are injured despite their gas masks. While the traditional gas mask still has uses, in many cases it should be replaced by one of the supplied-air type which can be worn in an oxygen-deficient atmosphere which contains unknown concentrations of unknown gases, vapors and particulates. This type of mask will do a good job in such atmospheres provided that it fits,[5] that the reservoir contains sufficient air for the necessary time, and that the regulator is functioning properly.

Gas masks are not the only pieces of protective equipment that actually may not protect in the emergency where they are used, but they exemplify the idea that obtaining equipment for protection is no guarantee that the equipment will be effective. Judicious testing of equipment designed to isolate man from his occupational environment is a necessity.

VENTILATION

Ventilation (see Chapters 39 and 42) can be used to insure thermal comfort as well as to keep dangerous vapors from the breathing zone of a worker. It can be misused in an attempt to blow away radiant heat or used properly to control the dust hazard from a grinder. Ventilation equipment is found everywhere, much of it designed, engineered, and used improperly, even though a similar expenditure of time, effort and money could well have resulted in adequate or better-than-adequate control of the occupational environment.[6]

From the point of view of the engineer, ventilation systems can be either local or general in nature, and they can attempt control mainly by exhausting or supplying air properly. These designations cannot, of course, be absolute because, for instance, local supply for one area is general supply for any other part of that room or building. Nevertheless, the intention of the planner will control this discussion.

Local Exhaust and Supply

Localized ventilation systems nearly always attempt to control a hazard by directing air movement. The velocity of the moving air may also be a consideration, but except in high velocity-low volume systems, it is used only to assure that the direction of movement is the correct one.

There are two main principles governing the correct use of local exhaust ventilation to control airborne hazards. The first is to enclose the process or equipment physically as much as possible. The second is to withdraw air from the physical enclosure (hood) at a rate sufficient to assure that the direction of air movement at all openings is always **into** the enclosure. All other considerations are secondary. If these principles are followed, no airborne material will escape from the enclosure so long as the enclosure is intact and the ventilation system is operating properly.

There are times where no enclosure is possible and where control of airborne hazards must be accomplished simply by the direction and velocity of air movement. These cases are not exceptions to the basic principle because, at the point where control must be assured, if the direction of air movement is always into the hood there will be control of materials suspended in that air. Similarly, if an air-tight enclosure were to be used, then no air need be moved to assure control of a vapor or an aerosol, but the principles have not been violated.

Three of the problems associated with local exhaust systems stand out. First, and most obvious, is that of poor design. All too many ventilation systems appear to have been laid out by someone who has no knowledge of how to handle air properly. These systems abound in abrupt expansions and contractions, in right-angle entries, in the overuse of blast gates to attenuate problems, and so on. Since the advent of the ACGIH Ventilation Manual,[7] poor exhaust or supply system design has had no excuse because good technique is so easily available.

The second problem is that of inadequate exhaust. It is exemplified by the exhaust system which has been added to from time to time, until nothing associated with the system works at all

well. The solution is simply to make sure that all systems, old as well as new, are well engineered.

The third problem of local exhaust systems is that of inadequate supply. People who are willing to install extra hoods at the drop of a hat (probably adding them to an already overloaded exhaust system) almost uniformly seem to feel that adequate supply air is a luxury or frill which they can do without. This tendency is accentuated by the widespread knowledge of a "rule of the thumb" which states that so long as the number of air changes per hour in the building is less "X" there is no need for a separate supply system. (The value of "X" varies from thumb to thumb, but is likely to be from 2 to 4.) This rule assumes that the building isn't "tight" and that infiltration of air will equal or exceed that exhausted.

Almost all buildings "leak" a little, and some leak a lot of air. Nevertheless, another principle of controlling the occupational environment by local exhaust is "always supply at least as much air as will be exhausted." A mechanical air supply system can and will do many things that infiltration cannot. A mechanical system can supply air that is filtered (and thus clean), tempered (warmed or cooled as necessary) and in the proper location to eliminate drafts and to avoid excessive disturbance of air at the faces of local exhaust hoods. None of these benefits can be gained by counting on infiltration for supply.

Local supply in itself is used occasionally to effect control or to assist in control of local exhaust. A combination of supply and exhaust, for instance, is sometimes used as a "push-pull" system to control vapors from large open tanks,[8] the supply air being used to "push" vapors into the exhaust system. If properly engineered, such systems can work well and can effect control by the movement of much less air than would be necessary if only exhaust were used.

The main use of local supply systems is not, however, to control hazardous vapors but, instead, to reduce heat stress problems. For this application, air is usually supplied on an individual basis and each man is allowed to control the direction and/or the velocity of air impinging on his work station. The air used is not cooled, but is supplied at high velocities (up to 500 fpm); it cools by sweat evaporation and by convection, if its temperature is below the man's skin temperature (as is usually the case).

General Exhaust and Supply

General exhaust and supply systems attempt to control the occupational environment by dilution. This principle can be used for many types of problems, ranging from hazardous vapors to locker room odors to problems of dust, humidity and temperature. A principle of general ventilation is that it be used to control problems that inherently are widespread. That is, it makes sense to use general exhaust and supply ventilation to control the temperature and humidity of all the air in an office building, but it does not make sense to try to control the fume generated by one welder with an exhaust fan located in the opposite wall. General ventilation is almost always unsuccessful when used to control "point" sources of airborne contaminants, and in addition, is very wasteful of air when used for such purposes.

Even local systems must have air to exhaust, and usually that air is supplied by a general system — one that is not associated with any particular hood or exhaust port. Some dilution of air contaminants will take place because of the general supply system, but its main purpose is simply to provide air to be thrown away by the exhaust system.

Air moving equipment can be expensive, and air filtering and tempering equipment can be even more so. Therefore, some engineers attempt to save money by recirculating some exhaust air back into the supply system. While this practice is standard in office buildings, it is rarely applicable in factories and shops because the air handled by the exhaust system cannot usually be cleaned adequately. Once-through systems, therefore, are standard except where the contaminant in the exhausted air is an easily handled particulate with a low inhalation toxicity. Sawdust, for example, is usually low in toxicity (although some woods are sensitizers), and the particles may be large enough to be removed easily from an air stream. In such a case, recirculation of some part of the exhaust air could be considered.

Inadvertent recirculation of exhausted air is a growing occupational health problem. When exhaust stacks and supply inlets are not separated adequately, part of the exhaust air will be captured by the inlet and recirculated to the building. This problem is prevalent in buildings designed by architects who are more concerned with the appearance of a roofline than they are with the health of those who will work in the building.[9] The problem also occurs between buildings, especially when roof elevation differences are not great, and elsewhere when little or no attention has been paid to the possibility of recirculation.

Recent work has shown that the best way to prevent recirculation is to discharge exhaust air in such a manner that all of it will escape from the "cavity" which forms as a result of wind moving over and around buildings.[10, 11] The intake can then be located at any convenient place, usually close to the roof, with assurance that recirculation will be negligible. Unfortunately, the prediction of cavity height above a roof is not yet an exact science, but enough is known so that intelligent decisions can be made. The recirculation problem must be considered whenever highly toxic, highly hazardous, or highly odorous materials are discharged by an exhaust system, whether or not a mechanical supply system is present.

EDUCATION

The first and most basic principle of almost any discipline is that knowledge is needed in order to apply that discipline to practical problems. Some knowledge comes with experience, but experience can be a poor teacher. More or less formal education can supplement experience and can direct it into the most productive channels. Nearly all people with line responsibility in indus-

try, and many with staff responsibility, can become involved with controlling the occupational environment. All of these people can profit from education in this area.

Management

Few managers become involved directly in the practical aspects of hazard control, yet very little hazard control is done without management backing. Managers exist mainly to motivate people (or to allow people to motivate themselves), but even expert motivators cannot channel activity into areas of which they are ignorant. Education of management should deal much more with the "why" of hazard control than with the how, when, where or whom.

There has been very little effort to formalize the education of managers in most industries; usually they are taught about hazards in meetings, conferences and personal chats by men who work for them. Informal education is better than no education at all, but the present best hope is the recent proliferation of short courses prepared and presented for representatives of high echelon management. A short course is the easy way to obtain quite a lot of valuable information with a small expenditure of time. This approach has been used successfully in the field of hazard control and much more use of it should be made in the future.

Short courses for managers should identify hazards in broad areas; details should be reserved for examples. The courses should concentrate particularly on the costs and benefits of controlling the environment, but should not completely neglect humanitarian aspects. Legal requirements which must be met should also be a part of the course content, but where a "carrot" exists, its use will almost always produce better results than will a club. Particularly for managers, the carrots (rewards) should be searched out, found and emphasized.

Engineers

At least a portion of the work of every industrial hygienist can be traced to equipment and/or process design failure. In many "failure" cases the person who designed the equipment or process simply was not aware of the potential consequences of the failure, or that such a failure was possible. Examples range from the purchase of equipment noisy enough to be hazardous, to the use of carbon tetrachloride or benzene as solvents, to the specification of gasoline-powered lift trucks for an enclosed warehouse, to the omission of a necessary fire door. In general, these failures arise from ignorance rather than from malice or from a "devil-may-care" attitude. Furthermore, the decision which resulted in a failure probably was made by someone quite far removed from the consequences of the decision — a planner, perhaps, or an engineering designer.

Educating engineers in regard to environmental hazards has, in the past, taken place mainly on the job by association with more experienced people. In recent years a few short courses have been given to supplement on-the-job training, but all too often any remedy applied is both too little and too late.

The logical place for engineers to be exposed to the knowledge that the environment abounds with hazards is when they are students at the undergraduate level. What is necessary then is not a program designed to turn these people into industrial hygienists or safety engineers, but instead, a course or courses which tend to open their eyes to the consequences of decisions they may make in their professional capacity. Undergraduate engineers (and most graduate engineers, for that matter) simply are not aware that it is perfectly possible to write noise specifications for much equipment; that carbon tetrachloride and benzene have excellent, much less hazardous, substitutes; that LPG fueled lift trucks generate much less carbon monoxide than do gasoline-powered lift trucks, that electric lift trucks are available and entirely suitable for most lift truck tasks; or when and where to install fire doors. The hazard gamut is so large that the typical short course can only scratch the surface, and a semester-long exposure stands a much better chance of getting the idea across.

Several colleges and universities already offer one or more courses surveying the fields of industrial hygiene for undergraduates especially in engineering curricula. With such courses as the foundation, short courses later in professional life should be able to keep engineers reasonably well up to date on environmental hazard control provided, of course, that they regularly read the literature related to the field.

Supervisors

In most circumstances, the further a supervisor is from actual control of a process, the more he deals with men and the less he deals with things. Supervisors usually work only through other people and consequently, they become aware of most environmental hazards from other people, or through their actions. In the case of an obvious hazard within his jurisdiction, a supervisor either can deal with the hazard with his own resources or he can solicit aid from others. Generally, which action to take is rather obvious, but some of the hazards posed by the occupational environment are subtle rather than obvious, and most supervisors are not equipped to deal with the subtle variety at all.

Education of supervisors usually should be process and process equipment oriented. The aim of the education should be to teach them about the subtle hazards that may be found in the environment of their employees and when and under what circumstances to request aid in solving the problems those hazards pose. Supervisors who are knowledgeable and well informed about hazardous processes, operations and materials are often able to control hazards early enough so that outside aid is not necessary except for periodic checks or reviews.

Workmen

Traditionally, little effort has been made to teach workmen about either the equipment or the materials that they handle. In the past few decades, safety engineers have shown over and over again that there are direct benefits to be gained

from teaching workmen about the physical hazards in their environment and how to avoid those hazards. More recently, industrial hygiene engineers have begun, usually in periodic safety meetings, to teach workmen about the hazards of materials and energies and, perhaps not surprisingly, have found similar benefits.

Hazards associated with the occupational environment impinge first on the men who work directly with materials, process equipment and processes. As these men are the first affected, they may well be the first to recognize adverse effects, and if so, if they are knowledgeable about the effects of the materials and energies they work with, they may be able to pinpoint problems before those problems become severe.

The main arguments against educating workers about the real and potential hazards of the materials and energies to which they are exposed have been that such knowledge would create apprehension, cause malingering, and give the unions another club to hold over the head of management. Where worker education has been used, however, groundless fears have evaporated, attendance has improved, and unions have been more cooperative, especially in matters concerning the health and safety of workmen.

An aware workman can often anticipate and circumvent hazards before they become serious to him, his fellow workers, or to the physical facilities. Furthermore, once the source of a hazard has been found, workmen, rather than supervisors or engineers, quite often have the best ideas of how to eliminate the problem with the least effort and expense. And finally, aware workmen often can be used to assist in industrial hygiene surveys,[12] thereby freeing the industrial hygiene engineer for perhaps more productive tasks.

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