41 | Environmental Inhalation

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Definition

Environmental inhalation deals with abnormalities, both pulmonary and systemic, that result from inhalational exposures to dusts, gases, chemicals, allergens, and infectious agents in the home, at work, and in the general environment.

Technique

As in most of medicine, the history, accompanied by a high index of suspicion, is the key to implicating environmental exposure in the pathophysiologic process. Because a detailed history of environmental exposure can take hours, clinicians must exercise judgment regarding detail. They must, however, at least ask the patient to list the jobs he or she has held and whether the job setting involves exposure to dusts, fumes, gases, and the like.

The clinician who cannot explain the cause of a patient's problem or who obtains a history of environmental exposure or who serendipitously finds an unexpected abnormality in a lab test such as a screening chest x-ray must then pursue a more detailed history. This involves asking the patient to iterate every job ever held (full- and part-time) along with the description of tasks, products manufactured, and substances used in production. In identifying a potentially pathogenic substance, the physician should be extraordinarily explicit. Accepting a history of "coal dust exposure" from a miner, for instance, is inadequate because anthracite coal mining is associated with a fibrogenic pneumoconiosis, whereas soft coal mining is associated with only a simple coal worker's pneumoconiosis. Accepting a history of exposure to "chemicals" from a worker in the chemical industry is also insufficient; many specific chemical substances (e.g., toluene diisocyanate, which causes asthma in susceptible workers), are well-defined pathogens.

Because workers are not necessarily legally entitled to know the generic names of workplace substances, and because these substances are often referred to with slang terms or coded numbers, the clinician must be creative in defining the generic ingredient of trade-name products. The appendix at the end of this chapter lists reference sources that are useful in pursuing an inhalational exposure.

Once the history and nature of an inhalational exposure are established, the clinician must delineate the intensity and duration of exposure. Since an individual's estimate of degree and length of time of exposure to a toxic substance is notoriously inaccurate, the physician must insist on specific information, which may only be supplied by a patient's company and/or union. The use of protective measures to prevent inhalational exposure is important, as is a general estimate of ventilation and exhaust status of a plant where a patient works. Materials used by other workers in a plant or work site may also be important.

The timing of symptoms in relation to work may provide a clue to an occupational inhalational exposure. A symptom such as wheezing that is relieved on weekends and during vacations may point toward an occupational etiology. For instance, patients with byssinosis classically have wheezing that resumes on Monday morning and diminishes as the week goes on. Symptoms accompanying a change in job or occupation should make the clinician think of a pathogenic respirable agent in the patient's new workplace. Whether fellow workers are afflicted with symptoms similar to the patient's is also of interest.

The home environment can be an important source of inhalational exposure. Presence of pets, use of potentially pulmonary toxic substances (e.g., ammonia and solvents used in cleaning), use of a potential pathogen (e.g., formaldehyde in the pursuit of a hobby like photography), and presence of air conditioning units are all of obvious interest. More subtle are exposures to dusts from spouse's work clothes, exposures that have been documented to be of significance for substances such as asbestos and beryllium. The neighborhood in which a patient lives may be important. For instance, in one study, people living near a plant that produced castor oil had a higher incidence of asthma than expected.

Pulmonary abnormalities that modify the significance of exposure to a pathogenic substance are also important. For example, a history of lung disease in nonsmoking relatives may indicate an alpha-1 antitrypsin deficiency and the presence of emphysema. A history of hay fever, asthma, or eczema in the family may indicate an increased susceptibility to bronchospasm and even the presence of asthma. A history of chronic cough and sputum production in a smoker will suggest bronchitis. All these diseases may cause abnormalities in the deposition and clearance of inhaled substances.

Smoking, both active and passive, affects many inhalational exposures, possibly working in combination with other inhaled substances to produce disease. The synergistic effects of asbestos exposure and smoking to produce mesothelioma is an example.

Socioeconomic factors such as extreme crowding substantially increase the risk of communicable diseases such as tuberculosis and should be considered in a good inhalational history.

Table 41.1 summarizes the requisites for a thorough inhalational history.

Basic Science

The variety and range of pulmonary and systemic abnormalities that environmental exposure can cause are vast. Knowing the pathogenic mechanism of an environmental inhalation can assist the clinician in keeping a high index

Table 41.1
Requisites of a Thorough Inhalational History

Work environment
Timing of respiratory symptoms in relation to work
Listing of all jobs (full- and part-time)
Description of all activities at work
Identification of materials worked with and exposed to
Estimation of duration and degree of exposure to possible pathogens
Estimation of ventilation and exhaust status of plant
Use and condition of protective equipment at plant (e.g., respirators or masks)
Characterization of respiratory symptoms of fellow workers
Home environment
Questions regarding:
Presence of pets
Agents used in cleaning, restoring, or repair
Exposure to dusts from spouse's work clothes
Hobbies and materials used
Materials used in construction of the dwelling
Important individual factors
Smoking
History of lung disease in relatives
History of atopy in relatives
Crowded living conditions

of suspicion for possible environmental exposure in illnesses whose etiologies are obscure.

The lungs can react to a pathogen in a variety of ways: bronchospasm, proliferation of reticulin and/or collagen fibers, edema, inflammatory reaction, and neoplastic formation. Asthma, defined as a reversible airway obstruction, is a common manifestation of an inhalation exposure and may occur soon after an exposure (i.e., within 30 minutes) or be delayed for 8 or 9 hours. The basis for the bronchospasm can be immunologic, pharmacologic, or simply irritative. Pneumoconioses can be fibrogenic or nonfibrogenic. People with nonfibrogenic pneumoconioses present with an abnormal chest x-ray due to proliferation of reticulin but have no respiratory symptoms referable to inhalational exposure, no abnormalities on pulmonary function testing, and no increase in morbidity and mortality compared to control populations. An example of this benign pneumoconiosis is stannosis, a consequence of working with tin. In contrast, workers with fibrogenic pneumoconioses, for example, asbestos workers with asbestosis, have a protracted course characterized by increasing dyspnea and possibly cough.

Allergic alveolitis is characterized by cough, fever, and dyspnea 4 to 6 hours after exposure to an antigen. Examples include "farmer's lung" and office workers' exposure to thermophilic actinomyces from the air conditioning system.

Granulomatous lung disease may be a manifestation of pathogens as different as beryllium and tuberculosis. Frank pulmonary edema and/or chemical pneumonitis are often the consequences of exposure to irritant gases such as ammonia and nitrous oxide. Lung cancer is a long-term consequence of exposure to such agents as asbestos, chromates, and bischloromethyl ether.

Systemic complications of environmental inhalation are common. A high concentration of carbon monoxide in the environment, for instance, can cause exacerbation of preexisting coronary artery disease and/or chronic obstructive pulmonary disease. Exposure to mercury vapor can cause renal and neural toxicity, and solvents such as carbon tetrachloride can affect practically any organ system of the body. Examples of neoplastic pathology that are consequences of environmental inhalation include bladder cancer in alanine workers and liver cancer in polyvinyl chloride workers.

Clinical Significance

As the number of new substances used in industry proliferates, the challenge to identify pathogenic substances increases. Many new materials are being substituted for known pathogenic substances, but the possibility that these new substances may be pathogenic requires vigilance. Continued epidemiological monitoring is crucial to identify possible new pathogens, but the individual clinician should also serve an important role in this surveillance. For instance, experts estimate that 10 to 15% of all people with asthma have bronchospasm on the basis of occupational exposure, and some data indicate that continued exposure may lead to chronic, irreversible airway obstruction. The challenge to the individual clinician to identify such an asthmatic is obvious.

Even if the clinician is unable to help an individual who has incurred an irreversible process such as mesothelioma, he or she should realize that identifying such a pathological process may be the impetus for an investigation and subsequent elimination of the hazard for other workers. It is conceivable that disease from environmental inhalation is grossly underestimated.

Appendix: Inhalational Exposure Resources

Books

Gardner W, Cooke I, Cooke RW. Chemical synonyms and trade names, 8th ed. New York: International Publications, 1978.

Gleason MN, Gosselin RE, Hodge HE, Smith RP. The clinical toxicology of commercial products, 4th ed. Baltimore: Williams and Wilkins, 1976.

Hawley GG. Condensed chemical dictionary, 10th ed. Princeton: Van Nostrand Reinhold, 1981.

Haynes W. Chemical trade names and commercial synonyms: a dictionary of American usage, 2d ed. Princeton: Von Nostrand, 1955.

NIOSH Registration of Toxic Effect of Chemical Substances, 1979.

Zimmerman, Latine. Handbook of material trade names. Dover, NH: Industrial Research Service, 1953–1968.

Agencies

Environmental Protection Agency, National Institute for Occupational Safety and Health, and Occupational Safety and Health Administration. These are government agencies with regional offices throughout the country.

Poison Control Center. There is a center in every major U.S. city.

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Dosman JA, Cockcroft DW, Hoeppner VH. Airways obstruction in occupational pulmonary disease. Med Clin North Am 1981;65:691–706.

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