Occupational and Environmental Asthma*

Legal and Ethical Aspects of Patient Management

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CBC = complete blood cell (count); FEV_1 = forced expiratory volume in 1 s; FEV = forced expiratory volume; MDI = metered dose inhaler; RV = residual volume; TCE = 1,1,1-trichloroethane; TLC = total lung capacity

In the coming decade, it is likely that occupational asthma will replace pneumoconiosis as the most common occupational lung disease.[1,2] This supplantation will occur as a result of increasing rates of introduction of new substances into the workplace and, by extension, into the environment at the same time that occupational and environmental factors become increasingly recognized as causes of asthma. In order to adequately manage the patient with asthma, the practicing physician must be aware of these associations. He or she should have knowledge of the legal remedies potentially available to the patient; and physicians should have some understanding of the ethical issues likely to be encountered. The present article focuses on legal and ethical aspects of management of the patient with occupational or environmental asthma. Although these issues will be discussed in the context of occupational asthma, the same principles can be applied to the patient with environmental asthma, with the exception of certain legal remedies which will be discussed separately. Two case studies will be used to illustrate.

Case Studies

Case 1. A 29-year-old baker referred by a local physician presented with the chief complaint of wheeze and shortness of breath, and a history of asthma since age 5. Allergy testing in the past had revealed allergy to grass. No testing for flour or grain dust allergy was done. There was a history of stage I sarcoidosis, diagnosed on the basis of chest radiograph and mediastinoscopy. Transbronchial lung biopsy revealed no endobronchial lesions and no interstitial fibrosis. The patient never smoked cigarettes. There was no family history of allergies or asthma. The patient was currently taking Theo-Dur and albuterol by metered-dose inhaler (MDI).

Physical examination was normal, with the exception of congestion of the mucous membranes of the nose, deviation of the nasal septum, and wheeze at the end of forced expiration on chest auscultation. A chest radiograph revealed hilar lymphadenopathy and hyperaeration. Pulmonary function tests revealed moderately severe obstructive airways disease, with a significant positive response to an inhaled bronchodilator: FEV_1, 2.41 L (55% P[= predicted]); FEV_1/FVC (%), 56; PEFR, 4.94 L/s (52% P); TLC, 8.4 L (122% P); RV, 4.08 L (232% P). The single-breath diffusing capacity for carbon monoxide (Dco) was normal. CBC was normal. The total eosinophil count was elevated at 960/mm^3 (normal, less than 440/cu mm); the quantitative IgE was elevated at 167 IU/ml (normal, less than 103 IU/ml).

In January 1981 the patient began work on a full-time basis in a bakery, with heavy exposure to dust from bleached and unbleached flour. He worked as a packer, alternating with machine operator. Flour was ubiquitous in the plant, with heaviest dust levels occurring in the mixing area. The ventilation was reported to be fair. Dust masks had been available for respiratory protection for 5 years. Approximately 1 year after he began to work in the bakery, the patient noted an increase in wheeze and shortness of breath, with gradual progression since. The symptoms were reported to improve slightly on his 2 days off work and during vacations. Symptoms increased during the first day back at work. Cromolyn by MDI failed to relieve the symptoms. From 1978 to 1979 the patient attended a junior college, studying computer science. From 1979 to 1981 he worked in the furniture department of Bargain Centers, without known exposure to potentially toxic gases, dust, or fumes. Despite his difficulty breathing at work, the patient was reluctant to leave his job because he could see no way to support himself while retraining for another job. He had no knowledge of the legal remedies potentially available to him.

Case 2. A 55-year-old man referred by his attorney presented with a history of dyspnea on exertion after climbing less than 1 flight of stairs, constant wheeze, and daily cough and sputum production for 2 years. The symptoms remained unchanged over that period of time. A diagnosis of asthma was made by his own physician, and the patient was taking Theo-Dur and albuterol by MDI. He had no personal or family history of asthma or other lung diseases, allergies, or hay fever. The patient had smoked cigarettes, approximately ½ pack per day of filtered cigarettes, until the onset of respiratory symptoms. At that time he discontinued cigarette smoking altogether.

Physical examination revealed mild kyphoscoliosis, with a respiratory rate of 16/min. Expiratory wheezes were present at rest. The I:E ratio was slightly increased at 1:2.5. Pulmonary function tests revealed a severe obstructive defect: FEV_1, 1.3 L (32% P); FEV_1/FVC (%), 47; PEFR, 2.23 L/s (24% P). The single-breath Dco was mildly reduced (21.6 ml/min/mm Hg, 71% P) and the KCO was normal (4.91 ml/min/mm Hg/L, 113% P). There was a significant positive response to inhaled bronchodilator. A chest radiograph was normal. The total eosinophil count was normal at 71/cu mm. Quantitative immunoglobulin counts, including IgE, were normal.

The patient had worked as an electrician in a large New England shipyard from 1969 until his retirement in August 1987. The onset of his respiratory symptoms was associated with the inhalation of a "strong blue smoke" at work. The patient reported that he had worked in proximity to a pipefitter who was cutting threads on a pipe, using an aerosolized lubricant. Heat from the cutter burned the lubricant. A Material Safety Data sheet which was subsequently obtained revealed the lubricant to contain 29% volatile 1,1,1-trichloroethane (TCE). In association with this exposure, the patient had experienced a "funny taste" in his mouth and profound fatigue. On the following day the exposure recurred. At that time, he developed difficulty breathing, with cough, wheeze, shortness of breath, and tightness in his chest. A co-worker experienced similar distress, and both men were evaluated in the First Aid department of the plant. The patient reported he had been given oxygen and

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sent home. Treatment on the following day by his own physician included amoxicillin and triamcinolone. Pulmonary function tests at that time revealed severe airways obstruction, not significantly different from that observed 2 years later. In order to return to his previous job, the patient was required to wear a respirator. Because of wheeze and shortness of breath, he was unable to comply. At the time of his office visit, he had been out of work for 2 years and had tried unsuccessfully to obtain workers' compensation.

Analysis

Both patients reported symptoms of wheeze and shortness of breath that were temporally associated with work. Patient 1 had a prior history of wheeze; patient 2 did not. Patient 1 was exposed to flour used in baking, a well-recognized cause of occupational asthma.[3,4] The patient had a nonspecific increase in IgE and an increase in total eosinophil count, providing evidence of the predicted immunologic effect of his exposure to a high molecular weight substance.[4] He was actively employed at the time of his diagnosis of occupational asthma. Patient 2 was exposed to an unknown gas. In all likelihood the gas was phosgene, a known potent respiratory tract irritant. Although aliphatic hydrocarbons are reported to release phosgene as a byproduct in the presence of ultraviolet radiation or heat, the available medical literature does not include TCE.[5] Its structural similarity to these chemicals provides the principal basis for extrapolation to the current situation.

Both patients had partially reversible airways obstruction. Patient 1 was still working. Patient 2 was unable to work at his usual or any job because of wheeze and breathlessness. Patient 1 was 29 years old and single. Patient 2 was 59 years old and married with 2 children.

In each case the examining physician must make decisions regarding diagnosis, medical treatment, and short- and long-term management. A detailed discussion of diagnosis and treatment is beyond the scope of this paper. The legal and ethical aspects of management will be discussed as they relate to the physician responsible for patient management.

Legal Remedies

Two kinds of legal remedies are available to individuals with work-related injury, illness, or disease: workers' compensation and toxic tort remedies (Table 1). The 2 differ in basis for recovery, method of adjudication, and potential monetary award. Physician familiarity with each is important to the successful management of the patient with an adverse health effect from an occupational or environmental exposure.

Workers' Compensation

Workers' compensation is a no-fault system that virtually eliminates the worker's right to sue the employer. Thus, participation in the system protects the employer from liability under the tort system. Bewteen 1910 and 1920, state governments in the United States began to enact worker compensation statutes. The impetus for such legislation is somewhat controversial. On the one hand, altruism is set forth as the motivating factor, the intent being to free the worker of the need for burdensome litigation in order to recover for work-related injury. On the other hand, between 1875 and 1905, workers were increasingly successful in recovering damages in negligence suits against their employer.[6] The workers' compensation system was established as a no-fault system in an attempt to eliminate the need for litigation. Employees lost the right to bring negligence suits against their employer. Employers lost their use of the following common-law defenses: employee negligence; the fellow traveler rule, *ie*, another employee, a co-worker, is at fault; and assumption of the risk—"the employee knew what he was getting into." Worker compensation law was originally intended to apply to traumatic injury, as opposed to occupational disease. In 1914 Massachusetts was the first state to interpret the law in such a way as to extend coverage to work-related disease.[6]

Table 1—*Legal Remedies*

Worker's Compensation
No-fault system
Eliminates worker's right to sue employer
Eliminates employer's use of common law defenses
Sine qua non: disability arising "out of and in the course of employment"
Recovery based on wage
Adjudication before administrative law judge
Toxic Torts
Liability based on negligence
Defective product design
Failure to warn
Negligent use or disposal of toxic chemicals
Interference with use or enjoyment of property
Sine qua non: exposure-induced injury causally related to negligence
Recovery determined by jury
Adjudication in a court of law

The sine qua non for recovery is disability that arises "out of and in the course of employment." Although no proof of employer negligence is required, the patient must show proof of causal association with work. Herein lies the rub for occupational diseases. Latencies may be long, as long as 20 to 30 years, and other causes of the same disease common. In order to prove his case, the patient with work-related disease must too often become a plaintiff in the legal system. Issues such as amount and schedule of recovery, method of adjudication, and statute of limitations may vary from state to state. In all states, the amount of money recovered is based upon and limited by wage. In Massachusetts, recovery is ⅔ of the patient's weekly wage at the time of the accident (or exposure).[7] If 5 years or more elapses before a claim is filed, recovery is based on the benefit schedule in effect at the time of eligibility. Adjudication is before an Industrial Accident Board, a 3-member panel made up of administrative law judges. The statute of limitations in Massachusetts is 4 years.

Toxic Torts

An alternative avenue of recovery exists for the injured

worker in toxic torts. For an individual suffering from an environmental exposure, this system may provide the exclusive legal remedy. Theories of liability derive from the circumstances of the exposure and are basically 2.[8] If the injury occurs as the result of exposure to a product containing a toxic substance, products liability law applies. If the injury is the result of exposure to a toxin in the environment, such as gas released from a paper mill or dust from a grain mill, liability derives from "more traditional concepts of negligence and the duties of property owners."[8]

Under product liability law, the principal causes of action are defective design and failure to warn. In the workplace each can result in toxic exposures and/or accidental injuries. Although the individual is barred under workers' compensation from suing his or her employer (the second party), he or she is not barred from suing the manufacturer of the toxic substance (the third party), whether or not the exposure occurred as a result of defective design or as a result of failure to warn. Toxic environmental exposures are actionable as tort cases when the toxic substance is improperly handled, processed, disposed of, or dumped. In addition to personal injury, individuals may recover under toxic torts for damage to personal or real property as a result of "exposure" to hazardous materials.

In contrast to the workers' compensation system, the tort system does not require "disability" for monetary recovery. The sine qua non is injury that is causally related to negligent activity, including a defectively designed product. Adjudication is in a court of law. Recovery is determined by jury. The statute of limitations is 3 years.

Role of the Physician

In the management of the patient with occupational or environmental asthma, the role of the physician extends beyond that required in other types of asthma (Table 2). Effective management may involve not only appropriate diagnosis and treatment in the short term, but also medical advocacy for the patient within the legal and regulatory systems in the long term. It may be the case that the worker must change jobs or leave the work environment altogether in order to avoid the exposure that is causing disease. Such a step should be viewed as a necessary part of treatment and one in which the physician participates actively when other measures such as pharmacologic therapy, respiratory protection, medical removal protection, or workplace cleanup are ineffective or unattainable. The patient may need assurance of income in order to be able to take this necessary and health-preserving step, both in the event of total and permanent disability from work and in the event of temporary or partial disability. The described legal remedies can provide this income, as well as additional income to pay related medical expenses.

Table 2—*The Physician's Role*

To determine diagnosis and nature and extent of impairment
To determine causality "with a reasonable degree of medical certainty"
To provide written opinion
To provide examining physician or expert witness testimony
To facilitate workplace evaluation and toxic exposure control, where indicated

The role of the physician in this aspect of patient management is critical. Determination of the nature and extent of impairment requires that the physician diagnose asthma and determine the extent of respiratory impairment in the context of the special conditions that exist for asthma because of its quixotic nature.[9] Theoretically, determination of disability (as opposed to impairment) is not within the purview of the physician, as it involves such nonmedical issues as age, education, and socioeconomic status.[10] But in fact, physicians are called on to make decisions about disability in cases of occupational disease, identifying whether disability is present or absent, total or partial, permanent or temporary. Eligibility for vocational rehabilitation must also be evaluated.

Determination of Causality

Determination of causality is perhaps the most difficult of the physician's responsibilities. But this determination is essential to effective treatment. If the diagnosis of occupational asthma is incorrect (the patient is diagnosed as having a nonoccupational cause of asthma, or vice versa), 1 of 2 outcomes is likely. The causal exposure will continue and the patient's asthma will persist or progress, perhaps to irreversible disease.[11] Or, alternatively, the patient will change jobs or leave the workplace inappropriately, and not only will his asthma persist, but the patient will have taken a major and perhaps irrevocable life step unnecessarily. The physician's job is made somewhat easier by certain realities of the legal system. Specifically, "causal" association means that the likelihood of a true association (between exposure and disease) is at least 50%.[6] Is it "more likely than not" that the described exposure was causally related to the observed disease? Further, the physician is directed by the system to "take the patient as you find him/her." In other words, the exposure in question has only to contribute to the patient's impairment. It does not have to be the sole cause. Interpretation of the extent to which exposure must contribute to the disease process will vary on a state-by-state basis. For example, patient 1 brought to the workplace a history of asthma since childhood. Exposure to flour dust was neither the precipitating nor the sole cause of his asthma. The exposure did, however, exacerbate his underlying disease and contribute to the observed impairment.

Having determined the diagnosis, the extent of impairment, and the causal relationship to employment, the physician must then step more visibly into the legal arena by (1) providing a written opinion, and (2) providing examining or expert witness testimony, should the situation require it. Because statutes of limitation, both under workers' compensation and toxic torts, often begin to run at the time the individual is informed of his or her diagnosis, it is extremely important that the physician be clear, both orally and in writing, what the diagnosis is and what the physician's opinion is regarding causality. Related legal testimony may take 1 of 3 forms: testimony at a hearing before an Industrial Accident Board in the case of workers' compensation, testimony in a court of law in the case of toxic tort action,

or testimony at a deposition in both cases.

Finally, it is incumbent upon the physician to remain involved with the patient whom he or she has determined to be disabled from work. Responsibilities include periodic reevaluation and written opinion regarding the level of the patient's impairment and the extent of disability.

Ethical Issues

Ethical issues faced by the physician managing the patient with occupational asthma revolve around determination of causality and willingness to advocate for the perceived truth, both in the legal arena and in the regulatory arena or the workplace. Pressure may be brought to bear on the physician by the patient, the employer, the product manufacturer, and lawyers for both sides. Legal testimony is rigorous and often stressful. It requires diligent preparation. The physician who is uncomfortable with his or her decision regarding causality or impairment will have a difficult time and will, in all likelihood, be a poor manager of the patient.

It is not the diagnosis of asthma that presents the difficulty in most cases. Rather, it is the determination of causality—the strength of the association between asthma and occupational environmental exposures. In some cases, the association is strong and the level of certainty on the part of the physician is high. For example, in case 1, the reported exposure was to flour during the course of work in a bakery. Pulmonary function tests revealed reversible airways obstruction. The patient's eosinophil count and IgE level were elevated. The wheeze was worse at work. Baker's asthma is well-described in the scientific literature.[3,4] The physician's role with regard to determination of causality is relatively easy, as is his task of providing examining physician or expert witness testimony.

In case 2, on the other hand, the exposure is less certain. Phosgene, the alleged exposure, is a highly toxic gas produced when certain aliphatic hydrocarbons are heated to 400°C. The substances most commonly reported to release phosgene as a byproduct under these circumstances are trichloroethylene, tetrachloroethylene, and chloroform.[5] The aerosolized lubricant used in this case contained TCE. The examining physician must thus extrapolate from what has been reported for structurally similar substances to the substance in question, and rely on a single published report of the production of phosgene by photo-oxidation of TCE under experimental conditions.[12] To further complicate matters, the patient was a cigarette smoker until the time of the accident. Premorbid pulmonary function is unknown. The physician knows only that the patient was free of respiratory symptoms or known respiratory disease until the time of the accident. Inhalation challenge with phosgene will not add useful information in this case and is potentially dangerous.

To answer the question of causality more definitively is often difficult but may be aided by recreation of the workplace situation and exposure in the laboratory under carefully controlled conditions, an expensive and time-consuming proposition not available to most practicing physicians. The level of certainty on the part of the physician is lower in case 2 than in case 1. As the level of certainty decreases and approaches the decisive 50%, the physician's task becomes increasingly difficult. This aphorism applies whether the point of absolute certainty is yes, a causal relationship exists, or no, there is no causal association. Physicians as a group are intolerant of diagnostic uncertainty.[13,14] Anderson and associates advise that there is "an irreducible necessary fallibility emanating from the uncertainties inherent in medical predictions based on human observations and the laws of natural science."[15] Physicians managing patients with occupational asthma must come to accept their own fallibility and be willing to assign causality (or lack thereof) when certainty is less than absolute. Because of the nature of the disease and the likelihood that its occurrence will increase in association with the introduction of new substances into the workplace, the diagnosis of occupational asthma will only be made by physicians willing to listen to their patients, to accept some degree of uncertainty, to extrapolate from existing bodies of knowledge to current situations, and to create new bodies of knowledge based on clinical experience. These physicians will be well-served by remembering Osler's dictum that "medicine is a science of uncertainty and an art of probability."[14,16]

In most cases, physician action in the face of uncertainty results in an outcome that is apparent in the short term. The patient improves or gets worse. Although there is a risk of anger on the part of the patient and of eventual malpractice suit if the physician is wrong, there is most often no immediate personal discomfort or stress for the physician. In the case of occupational asthma and other occupational diseases, the physician encounters a number of decision points, each of which leads to more or less involvement with medical-legal and regulatory systems and to more or less stress for the physician. These decision points occur at (*a*) determination of causality, (*b*) determination of impairment/disability, (*c*) provision of written opinion, (*d*) agreement to offer legal testimony in support of the written opinion, (*e*) intervention on behalf of the patient in the workplace, directly or indirectly through government agencies, and (*f*) agreement to provide ongoing documentation of the patient's disability for employers or insurers. At any of these points the physician can take the easy way out. He or she can opt for absolute certainty at point (*a*), so that points (*c*) and (*d*) become less difficult. Or he or she can proceed with less than absolute certainty at point (*a*) but refuse to provide either (*c*) or (*d*), so that the physician's course becomes easier but the decision at point (*a*) becomes meaningless in terms of benefit to the patient. To take action at point (*e*) is not necessarily uncomfortable or stressful for the physician, but it is time-consuming. It offers the opportunity, however, not only to increase the level of certainty at point (*a*), but also to prevent similar disease among co-workers of the patient and to reduce exposures for the patient, thus obviating the need for the patient to seek job transfer or other employment. Similarly, action at point (*f*) does not create a stressful situation for the physician, but it is also time-consuming and annoying to deal with the chronic influx of disability forms.

These decision points bring into focus the ethical issues facing the physician manager of the occupational asthma patient. Moral values influence whether the physician will behave in such a way as to be most consistent with the truth as he or she sees it and to provide the greatest benefit to the patient and to others at risk from the same or similar exposures, or in such a way as to provide the easiest way out

of a potentially difficult and stressful situation.

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