

# Clinical Surveillance and Management of Occupational Asthma\*

## Tertiary Prevention by the Primary Practitioner

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FEV<sub>1</sub>=forced expiratory volume in 1 s; FVC=forced vital capacity; NIOSH=National Institute for Occupational Safety and Health; RAST=radioallergosorbent test; TDI=toluene diisocyanate

Asthma and other idiosyncratic respiratory diseases have become an increasingly important component of the occupational lung disease burden in the United States and other developed countries as more cases are recognized and the pneumoconioses come under control (see Malo, "Compensation for Occupational Asthma in Quebec," this issue). Unfortunately, control of occupational asthma cannot be achieved by the same strategy that has begun to prove effective in reducing risk for the dust diseases. This report reviews the theoretical and practical reasons why alternative approaches must be sought and delineates one modality that may well be the cornerstone in the foreseeable future: early recognition and management by primary care practitioners.

Since occupational asthma has been the subject of numerous and excellent recent papers,<sup>1,2</sup> I will not review current data on its prevalence and causes; for convenience, a list of known causes is summarized in Table 1. For present purposes I will define occupational asthma broadly, as the development or recrudescence of documentable, reversible drops in air flow (>10% FEV<sub>1</sub>) during or after a work shift in a previously unaffected worker or one who has been free of bronchospasm for a prolonged time (eg, since childhood). As such, I am adopting the current (albeit untested) criteria developed by the National Institute for Occupational Safety and Health (NIOSH) Surveillance Subcommittee of the Board of Scientific Counselors for use in the SENSOR reporting system (see article by Matte et al, this issue) rather than the perhaps more restrictive criteria used by various immunologists or inhalational challenge-based investigators. Present information would suggest that occupational asthma, defined according to NIOSH criteria, is common and widespread throughout the work force.<sup>3</sup>

### THE BASIS FOR A TERTIARY PREVENTION STRATEGY

#### Limits of Primary Prevention

Based on the experience with disease prevention for the pneumoconioses, it is obvious that a strategy to eliminate the causes of occupational asthma at the source by reducing exposures below "no effect levels" would be ideal if it were feasible. Unfortunately, there are ample reasons to suspect

that such a strategy may not be feasible. The range of agents capable of causing asthma in the workplace is large and rapidly expanding, with many new ones added every year. Furthermore, unlike diseases caused by dust exposures, the agents are extremely widely distributed, with hardly a sector of the work force, standard industrial code, or even individual workplace free of potential culprits. This was well illustrated by the compilation of the eight-year experience of our own clinics in Connecticut, where some 150 cases were caused by over 30 different substances in over 60 different settings, far different from the situation for other occupational diseases, which were heavily concentrated in a few industries or sites.<sup>4</sup> If this were not problematic enough, many of the potential causes such as animal and vegetable materials and pharmaceuticals are unimaginably difficult to

Table 1—Materials Causally Linked to Asthma in the Workplace

Vegetable material	Animal material
Grain dust	Danders
Flour	Insects
Fig plants	Silkworm larva
Wood dust	Shellfish
Seaweed	Excreta (pigs, chickens)
Green coffee beans	Fish feed
Fungal spores	Animal enzymes
Gum tragacanth	
Castor bean	<i>Metals</i>
Tea	Stainless steel
Tobacco	Galvanized steel
Flax	Aluminum fluoride
Hemp	Vanadium
Cotton	Cobalt
Hops	Tungsten carbide (cobalt)
Bacterial enzymes	Platinum salts
Colophony	Nickel
	Chromium
<i>Plastics/chemicals</i>	<i>Pharmaceuticals</i>
Acid anhydrides	Penicillins
Epoxy resins	Cephalosporins
Diisocyanates (TDI, MDI, HDU)	Piperazine
Persulfate salts	Psyllium
Paraphenylene diamine	Methyldopa
Phthalic anhydride	Spiramycin
Dimethyl ethanolanolamine	Tetracycline
Azobisformamide	Amprolium
Azodicarbonamide	Cimetidine
Formaldehyde	Isoniazid
Ethylenediamine	Phenylglycine
Acrylates	
Henna	

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eliminate.

Even if one could identify a finite set of agents on which to focus attention, the pathogenesis of asthma appears to be quite different from that of other forms of toxicity for which dose-response relations have been defined. Whereas cumulative or average dose would appear to be the best determinant of risk for most environmental diseases, best evidence suggests that transient bursts of exposure are more important in causing asthma than total or typical dose; such bursts are extremely difficult to control in most work settings, let alone to regulate short of continuous monitoring with alarms, for example. Further, once a pathogenic burst has occurred, reexposure at doses far below that which may incite sensitization appears to be capable of perpetuating the cycle leading to clinical asthma. It is hard to imagine that the broad range of agents could soon be controlled at such levels in the vast majority of settings in which they appear, and harder still to believe that such an approach would be economically feasible, given the relatively small numbers in most settings who would actually benefit from such rigid environmental control.

An alternative primary preventive approach that has received much attention is to protect the at-risk population from exposure by preemployment testing. Even without regard for the ethical problems that arise from such a posture, the strategy at present lacks any scientific foundation. Careful review of the experience with major small-molecule causes of asthma such as toluene diisocyanate (TDI) and western red cedar dust has suggested that the likeliest at-risk group, atopic subjects, are not significantly more susceptible than their nonatopic counterparts.<sup>5,6</sup> Studies in settings in which atopics do harbor the major risk, such as animal handling, have proved that exclusion of atopic subjects does not appreciably reduce incidence over time; a strong consensus has emerged repudiating the idea, however cost-effective it may have appeared on the surface.<sup>7</sup>

Another approach has been preemployment determination of airway function and reactivity. The theory behind this strategy is that individuals with reactive airways or preexisting obstructive defects would be those most likely to develop asthma and therefore should be excluded from opportunities for exposure to asthmogenic substances. Here, too, the data from work to date have not been promising. There are no data that demonstrate that asthma develops more frequently with controlled exposures to any primary causal agent in individuals who have obstructive lesions to begin with. Furthermore, the concept that overt asthma occurs principally in individuals with preexisting bronchial reactivity has yielded to an understanding that the reactivity itself is generally acquired as part of the pathogenic process, rather than being an inherent, preexisting host factor.<sup>8</sup> For this reason, sophisticated preemployment physiologic testing seems unlikely to be an effective means to control disease either.

None of these objections should be taken to suggest that asthmogenic substances should not be rigorously controlled at the source, or that efforts to reduce exposure in those at highest risk are not without some benefit. On the whole, however, it is clear that with present knowledge, technology, and economic feasibility, in the short run primary prevention is unlikely to succeed in limiting the occurrence of this

serious occupational disease.

#### *Current Limitations to Secondary Control Strategies*

Considerable research has been invested in the study of serologic markers of sensitization to some of the more prominent causes of occupational asthma, including small molecules such as TDI and larger proteins such as animal danders or grains. If such markers could be identified, made available to most exposed workers, and correlated with a strong predilection to develop asthma, such tests might serve as a basis for the secondary prevention of asthma in the workplace. Specifically, workers could be routinely screened for the development of such markers—presumably before the onset of overt symptoms—and removed, if positive, after exposure but before disease develops. Control by this means would be ethically easier to accept than broad preemployment selection, and more efficient.

Unfortunately, none of the 3 conditions enumerated above yet exists for workers exposed to the majority of the agents recognized to cause asthma. Antibodies typically of the IgE class and measurable by RAST have been well characterized for some of the large protein antigens such as grain flours and laboratory animal products but generally appear to have little relationship to asthma, correlating far better with upper respiratory symptoms.<sup>9</sup> For the ubiquitous small molecules, the search for the appropriate serologic test has been frustrated by lack of knowledge of the physiologic moiety that is apparently sensitizing, the agents themselves lacking obvious antigenic properties, owing to their small size.<sup>10</sup> The possibility that classic IgE or IgG mechanisms are not involved has been repeatedly raised, some investigators proposing an alternative immunologic mechanism, others suggesting nonimmune possibilities<sup>11</sup> (see article by Patterson et al, this issue).

Furthermore, even where a test of sufficient reproducibility and predictive value may exist, it is not obvious that laboratories are available or that workers are under sufficient surveillance to render this approach feasible from a public health perspective. Although further research and changes in occupational health services could render these approaches viable in the future, alternatives are clearly necessary at this time.

#### *The Possibility of Tertiary Prevention*

Rarely in occupational health practice are tertiary preventive strategies deemed important as more than backup for the more highly regarded primary and secondary approaches. Examples of this limited role are current Occupational Safety and Health Administration/Mining Safety and Health Administration (OSHA/MSHA) rules for the prevention of pneumoconioses and lead poisoning. For tertiary prevention to be effective, it is necessary that the disease be readily detectable at a stage when intervention has a high likelihood of reversing or substantially modifying the disease course. Further, there must be appropriate surveillance so that most affected workers will be evaluated appropriately during this window of opportunity.

Although the data are far from complete on the subject, most observations to date do suggest that occupational asthma tends to have a favorable prognosis when exposure to the sensitizing agent is halted within the first several

months of symptoms. The most complete observations are the longitudinal studies of Chan-Yeung of populations exposed to western red cedar wood (see article by Chan-Yeung et al, this issue). Among those removed from exposure within six months of first symptoms, most have experienced reversal of symptoms, with many demonstrating loss of even bronchial reactivity after a while. Conversely, among those not initially recognized or those who continue to work despite symptoms in a one-industry region, the prognosis was far worse, with the majority going on to full-blown asthma precipitated by multiple agents and stimuli. Similar observations have now been made in workers exposed to other small molecules, such as TDI,<sup>12</sup> as well as large proteins, such as snow crab processors.<sup>13</sup>

Assuming that these findings can be reasonably generalized, it must be considered whether sufficient opportunity exists for most workers to have the diagnosis made early and the necessary job intervention undertaken. Most workers exposed to even known asthmogenic substances are unlikely to be under direct surveillance at the workplace. Such services, directly linked to employment, would be the best guarantee that examinations would be undertaken at adequate intervals and that subtle early symptoms or signs would not be overlooked or misconstrued. More important, such surveillance would almost certainly guarantee that appropriate action would follow evidence of early disease, with removal of affected workers from exposure in time to maximize good outcome. Realistically, however, this is unlikely to be available except in limited settings such as large corporations or those under specific forms of regulatory scrutiny, *eg*, laboratories using animals, which require regular certification or licensure.

Fortunately, there is an alternative that may be feasible short of massive regulatory overhaul or sweeping changes in the way health services are delivered to workers. For although most workers at risk are not under regular surveillance for occupational asthma, the majority do have access to general health care and most probably have personal or family physicians whom they consult when they have a problem. It is the central premise of this article that persistent asthmatic symptoms, whether overt wheezing and shortness of breath, or more subtle coughing or chest discomfort, are typically sufficient to bring workers to medical attention and probably soon enough after onset that effective intervention could be undertaken, with good results. If this is true, the challenge for tertiary prevention becomes one of educating primary physicians to recognize early manifestations of the disease, to appreciate the significance in terms of long-term health, and to make the major intervention of removing a patient from noxious exposure, or promptly refer that patient to an appropriate specialist who can. The second section of this article presents practical strategies for the primary physician to accomplish these crucial tasks. My contention is that such effort can realistically be expected from well-trained primary care givers.

#### APPROACH TO RECOGNITION AND TREATMENT OF OCCUPATIONAL ASTHMA

##### *Developing a High Index of Suspicion*

Primary physicians, especially those who see large numbers of patients with urgent needs, must develop and

maintain a high index of suspicion about occupational asthma. In practical terms, this means awareness that the first physician-patient interview regarding a respiratory symptom or complaint may also be the last chance for a correct and complete diagnosis to be made during the relatively short window of opportunity described above. Although occupational asthma rarely presents as a life-or-death medical emergency, the brief window of opportunity dictates an alertness not different from that for other, more serious conditions: there may not be a second chance.

Since awareness among primary physicians about occupational diseases in general is presently quite low,<sup>14</sup> perhaps the best approach is to change physician thinking about asthma generally. Although it is unlikely that new asthma cases in adults are occupational in more than 5% to 20% of cases, it is probably true that many cases of new-onset asthma can be traced to environmental causes which, if eliminated, could dramatically alter prognosis. For this reason, it would be reasonable that traditional teaching about asthma, which centers on treatment rather than prevention, be modified to stress the urgency of early etiologic investigations and intervention for care rather than the choice of pharmacologic agent merely to control symptoms. Physicians must appreciate that new-onset asthma requires more than strictly medical control, which remains the present emphasis, new cases being handled little differently from flares in known asthmatics. New bronchospasm should receive the same urgent attention as, say, a new finding of blood in stool and should receive the same timely workup as is typical for that ominous sign.

A more straightforward approach is available for the many workers who receive routine preventive care from their doctors. For these workers it would be highly feasible to incorporate current data on occupational asthma into a planned care approach. Specifically, if the physician becomes aware that a particular patient is exposed to a substance that may cause asthma, it would be reasonable to see the patient every 6 months or so for evaluation for early signs of asthma. Increasingly, this could happen because primary physicians are taking more careful occupational histories from their new patients and because workers are being told on the job that they face this risk and they may convey the information to their doctors. In this situation, the patient could be more or less under surveillance by his personal physician, perhaps a reasonable substitute for more formal surveillance at work.

##### *Recognition*

When a working adult presents with respiratory problems, there are 2 components to physician recognition of the possibility of occupational asthma. The physician must first consider that the symptoms could represent asthma, then consider that the asthma may be due to a chemical in the workplace. For many reasons, the first component of physician recognition of asthma may be as formidable a challenge as the second component. In fact, premature attention to the role of the workplace may obscure recognition of the true nature of the problem and lead to long delays before the correct diagnosis is considered and appropriate management is instituted.

Published case series of occupational asthma, as well as anecdotal reports from experienced clinicians, suggest that

occupational asthma frequently manifests with atypical symptoms—cough, chest discomfort, or easy fatigability—rather than the more classic symptoms of wheeze and breathlessness. Whether this is more true of occupational asthma than of asthma of other causes is uncertain. However, it is clear that a large proportion of occupational asthma cases may challenge the clinician's diagnostic skill at the time of presentation. Not only will symptoms be atypical, but very often, especially if the patient has taken a day off from work, the typical signs may be absent and spirometry unremarkable. Because of the high prevalence of cough and chest discomfort in the adult population, many atypical presentations may be written off as upper respiratory, or nonspecific in origin and attributed to infection, common allergy, or smoking without further evaluation until symptoms persist and simple remedies fail. Patient accounts of the presence of irritating chemicals in the workplace may contribute to this reasoning and forestall recognition of the more serious lower respiratory locus of involvement.

Deciphering the occupational relationship also presents a significant challenge in many cases unless the physician is already aware that exposure to known asthmagenic substances is occurring. Unlike recognition of most other occupational diseases, where the main clue comes from awareness that exposure to a particular harmful agent has occurred, the key to occupational asthma is symptom periodicity, with recurrence of symptoms in a fixed temporal pattern in relationship to work and relief of symptoms, at least during early stages of the disease, in a similarly fixed pattern in relationship to periods away from work, such as weekends or holidays. Unfortunately, the pattern may not be trivial, such as symptoms all day on workdays with relief on weekends. The relevant exposure may not occur every day or at the same time every day, depending on the nature of the work and the offending agents. In addition, many agents appear to cause bronchospasm delayed by as much as 6 to 24 hours after exposure, leading to symptoms that paradoxically only occur out of the workplace. Furthermore, shift work and overtime may totally obscure a pattern if, for example, the patient never takes a day off or works variable shifts, a notorious problem for such susceptibles as scientists, health care workers, artists, and so on. Finally, it must always be borne in mind that after some months of occupational asthma, bronchospastic responses may be elicited by a wide range of precipitants, including cold, exercise, infection, and irritants, which may totally obscure the initial pattern, if one were ever obvious.

Although these pitfalls may suggest that recognition of occupational asthma is unlikely to be possible for most primary clinicians who specialize neither in occupational nor in pulmonary medicine, there are certain aspects of occupational asthma that vastly simplify life for practitioners. With respect to physiology—*ie*, recognizing atypical symptoms to asthma—very simple tests exist, accessible to almost every physician in developed countries, to rule it in or out. Thus, the only real impediment to recognition of asthma *per se* is failure to consider it as a possibility. With respect to occupation, the large and growing list of causal agents, combined with the possibility that very small exposures may be sufficient to cause asthma in a sensitized individual, means that primary clinicians need not worry too much

about the complexities of the workplace or trying to ascertain every exposure to which the patient may have contact. It is generally sufficient, for recognition purposes, to decipher the pattern linking work or some task at work to the symptoms under diagnostic consideration. Occupational asthma is one of the very few occupational diseases that can be diagnosed easily without a thorough review of work exposures, and the clinician is advised to concentrate more on the patient than on the imponderables of the workplace.

#### *Documenting Occupational Asthma*

If local expertise in occupational medicine is available, patients with suspected occupational asthma are probably best referred. Nonetheless, it is important for the primary physician to understand what will go on so that he or she may advise and co-manage the case. As with the recognition phase, documenting occupational asthma also consists of two distinct components. First, it is necessary to prove that asthma—*ie*, reversible bronchospasm—is occurring; second, the relationship to workplace exposure must be demonstrated. Since most of the steps in the latter phase are time-consuming and resource intensive, it is sensible to confirm the asthma before getting too deeply into proving a workplace cause.

Clinically, asthma is readily demonstrable by auscultation of audible wheezes. In the absence of available spirometry, the finding of wheezes in association with typical symptoms that remit with treatment or when the patient spontaneously improves is generally sufficient. Since wheeze can represent an upper respiratory source in an occasional patient, spirometric confirmation of at least 15% improvement in FEV<sub>1</sub> on valid tracings is valuable support and worth the small expense. Often spirometry may be normal when the patient is initially seen, and sometimes repeatedly thereafter. In this case, and only in this case, nonspecific challenge with methacholine may substitute, since airway responses to low or very low doses correlate well (although not perfectly) with the presence of recent bronchospasm. Equivocal or intermediate dose responses to methacholine should prompt more intense efforts to reevaluate the patient during symptomatic periods, if possible.

Once asthma has been documented, attention may turn to the issue of work-relatedness. The most desirable approach is to measure pre- and post-shift flow rates and vital capacity in a pattern consistent with the history. Ideally, this should be done on 2 separate days of the week (1 early in the week, 1 late) for 2 or 3 consecutive weeks. A consistent pattern of a greater than 10% drop in FEV<sub>1</sub> or FVC between the pre- and post-shift trials is diagnostic; smaller cross-shift drops associated with a marked decline in baselines between the early and later part of the work week are highly suggestive.

An alternative that avoids testing at hours inconvenient to the practitioner is to instruct the patient in the use of a peak flow meter in conjunction with an activity and symptoms diary. Although this test is effort-dependent and data are recorded by the patient, introducing the potential for unreliable results, the approach offers more complete data points (*eg*, every 4 hours while awake every day, including weekends) and may unearth a pattern that would elude the traditional strategy. In either event, the patient should be

instructed not to use bronchodilating medication on days on which data are to be recorded, and preferably not at all during the diagnostic period.

Occasionally, neither of these alternatives is practical, perhaps because the patient is too sick to work at all, cannot manage peak flow meters, or the like. In such situations one may have to rely on clinical judgment, which, in turn hinges heavily on the patient history. In these cases, diagnostic trials of removal from work or a move to an exposure-free job for 1 to 2 weeks, followed, if necessary, by return to work under close observation, are often very revealing. Specific knowledge of the actual exposure history may also be influential. In particular, a clinical diagnosis is far more likely to be correct when it has been demonstrated that the patient is exposed to a well-known and potent asthmogen, like grain dust, animal danders, or TDI, than if no such well-characterized agent has been identified.

#### *Identifying the Causal Agent*

I have said little about specific exposures. There are 2 very important reasons for relegating this aspect to a relatively minor role in the scheme of things. First, it is often extremely challenging, especially when a patient works in a milieu in which many chemicals are used or when work materials such as machining fluids or resins contain large numbers of additives, any of which could be responsible. Since even small exposures could cause bronchospasm once sensitization has occurred, one cannot assume, as in the search for other occupational toxicities, that only major exposures are of consequence. More important, and perhaps more surprising, identification of the specific agent is usually not very important for management. In my experience, far too much time is often wasted in the search, long after the diagnosis of occupational asthma is obvious and often to the detriment of the patient by forestalling urgent, outcome-influencing intervention.

Nonetheless, there are reasons to identify causal agents, and for certain patients this search may play a vital role in management. The most important general reason for a search is public rather than individual health. In particular, an employer may be using a potent asthmogen without knowing it or without having a satisfactory reason not to substitute a safer alternative. In a similar vein, identification of the agent responsible for a case of occupational asthma may prompt surveillance steps by government, employer, union, or physicians of individual workers if the information is well publicized.

From the patient's perspective, the main reason for taking steps to identify the causal agent relate to future work possibilities. This is especially crucial for workers in regions where one industry dominates, for example, lumbering of western red cedar in British Columbia; sensitization to the wood would place a substantial limitation on future safe work alternatives. Other subsets of workers who may need to know are artists and professionals. In these cases, specific knowledge of a sensitivity may allow continuation of career by job modification or substitution rather than a blanket restriction on activities.

Once the decision has been made to pursue this issue, the approach is relatively straightforward. The first and most difficult step is identification of all materials in the work

environment to which the patient may be regularly exposed. Included are not only materials handled by the patient, but also those which may be used by others in neighboring areas or departments. If such materials are likely to be composed of mixtures of chemicals (oils or paints, for example), material safety data sheets or other information will usually be necessary to delineate the actual constituents.

Next, this list must be compared against a list of established causes of asthma such as that in Table 1. The result of this comparison will dictate the next steps. In the easiest case, a single agent will appear which will at once be among the chemicals most commonly used by the patient and a potent asthmogen. In this case, it is overwhelmingly most likely that this is the cause and it would be appropriate to presume as much without further testing unless there are compelling social or medical reasons for confirmation. At the other end of the spectrum are cases in which there are no known asthmogens to which the patient has been exposed. After ensuring that the exposure list is, in fact, complete, it is worth reviewing the evidence for the diagnosis of occupational asthma. If convincing, the search for a cause becomes tantamount to a search for a new cause of asthma, an occasion which yields success about a dozen times per year in the published literature. In general, attention is best focused on chemicals that are large proteins or are highly reactive and bear some relationship to other substances known to cause asthma. Final proof, if it is clinically or scientifically important, will typically demand some confirmation with direct inhalational challenge with the single agent or some immunologic test (*eg*, RAST) demonstrating specific biologic sensitization to the substance in the patient. If multiple potential agents are present, the possibilities may be honed down by careful review of work activities with the patient, which may reveal that some are inconsistent with the history of symptoms in relationship to work. Failing that, it may be necessary to resort to some biologic discrimination, either single agent challenge or a search for specific antibodies.

Unfortunately, when it would be desirable to have available safe, sensitive, and specific tests to identify causal agents, the options are poor. For some substances, notably the large antigenic proteins like animal or vegetable products, reasonably reliable and sensitive IgE titers may be obtained by commercial RAST testing. The major limitation is the lack of specificity of these tests for occupational asthma *per se*; positive results are seen in the presence of sensitization and allergic syndromes of all kinds. However, once the diagnosis of occupational asthma is secure, a positive RAST result in high titer probably has good predictive value for the agent.

The theoretical reference standard is specific inhalational challenge. While it is fair to say that much of what we now know about occupational asthma was generated in part by use of this technique, the practical value is limited at present, for several reasons. Competent and qualified laboratories are scarce and are rarely available on a commercial basis. Probably not unrelated is the extraordinarily cumbersome nature of the testing, requiring sequential doses (starting at a very low dose), with each challenge observed in the hospital for up to 24 hours (for late reactions); tests may take many days. Despite these precautions, the risk of

overdose with unpredicted clinical bronchospasm requiring major therapy makes the procedure potentially harmful. In addition, false negative results have been common in the study of at least some important agents, such as TDI.

The use of laboratory testing in the research for a cause for occupational asthma must be viewed in the context of what information can be expected. The question must always be asked: to what extent will the result change management? In particular, to what extent will a positive test help or a negative one dissuade from a particular course of action? Given the risks, limitations, and pitfalls of the available choices, in all but a few circumstances it is best not to depend too heavily on these tests to decide on proper management.

#### MANAGEMENT OF SUSPECTED OCCUPATIONAL ASTHMA

The most important issue in the management of any case that may turn out to be occupational asthma is urgency. Although it is impossible to reliably predict outcome in any given case based on the prognostic data enumerated earlier—patients with early asthma may do poorly and those with advanced cases may do well—it is imprudent to knowingly prolong the period of symptomatic exposure before removing the variable that correlates best with outcome. On the other hand, it may be worth a few weeks of examination while exposure continues to simplify and secure the right diagnosis, given the limited possibilities for doing this while the patient is out of work.

To accomplish both of these objectives simultaneously requires a clear plan developed at the first suspicion of occupational asthma. The first step is proof that bronchospasm is occurring, which may require additional visits, rapid review of prior medical evaluations, or a nonspecific inhalation challenge. Even as this phase is proceeding, steps should be taken to assess work-relatedness: exposure records should be sought from the employer or other potential sources (with appropriate patient consent), pre- and post-shift spirometry may be arranged or a peak flow meter and diary provided. In any event, it is essential at the outset to consider which patterns of results would prompt immediate removal and which would prompt watching while exposure continues. In general, medication is avoided during this period, except as a diagnostic challenge, but in practice it is often hard to resist the use of at least short-acting inhaled bronchodilators on nontest days.

However the plan works, soon the point comes when a decision has to be made despite the fact that all available information is not in or the results are equivocal. One can rarely accomplish cessation of an occupational exposure without simultaneously setting in motion huge changes in the patient's life; this decision cannot be taken casually, and referral to an experienced consultant is advised if one is available. In the best circumstance, removal may entail as little as a small modification in daily routine or a simple job change for an employer with available alternative work. This is ideal as long as it can be readily predicted that likely causal agents (which may yet not be obvious when decisions have to be made) will not also be present at the new position. Much more frequently, removal from exposures means removal from the workplace altogether, a step that cannot be taken responsibly by the patient unless there is confidence that he or she will get every support in obtaining

compensation of other benefits, which will be crucial during a job transition.

In the latter case, the clinician's management responsibilities clearly extend into the social sphere. Social workers or others capable of assisting with the inevitable financial and emotional disruptions associated with job loss or change may be very useful in helping the patient accept seemingly severe treatment for a not-so-severe disease. Similarly, the treating physician's careful explanation of the situation to the patient and, when necessary, the employer and insurance carriers is important in guaranteeing compliance and the most favorable outcome possible.

Rarely will the patient not accept a transfer and choose to continue to work with symptoms. Unfortunately, there is no evidence that medication of any kind, including agents used prophylactically, such as sodium cromolyn, in any way mitigate the likelihood of occupational asthma progressing to generalized asthma and ultimately perhaps fixed obstructive diseases. On the other hand, even early removal is no guarantee of therapeutic success, and patients, employers, and others in the picture must always be advised cautiously, lest expectations for recovery exceed reality.

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