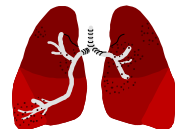




Occupational Airways



A quarterly newsletter of the Occupational Health Surveillance Program, Division of Environmental Epidemiology and Occupational Health (EEOH), Connecticut Department of Public Health, 410 Capitol Avenue, MS# 11OSP, P.O. Box 340308, Hartford, CT 06134-0308 (860) 509-7744

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OCCUPATIONAL ASTHMA: SOME CLINICAL OBSERVATIONS

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There is still vigorous debate among experts with respect to how broad a definition of occupational asthma should be employed, and whether occupational exposures that represent only irritants to already reactive airways should represent examples of occupational asthma. A more serious clinical confusion exists among health providers with respect to how occupational asthma "ought to behave" as a clinical entity. Should occupational asthma behave at all like the routine extrinsic asthma seen in adults? Or like the atopic asthma common to the life-long symptomatic allergic patients? And if not, why not? I would propose, from my experience, that asthma due to occupational exposures or that of nonoccupational etiology is almost identical in clinical patterns and response to treatment.¹

The following discussion represents an attempt to de-mystify the behavior of occupational asthma that is perceived to be inconsistent with the diagnosis, and more typical of allergic or post viral extrinsic asthma in adults. These behaviors frequently lead to questioning of the diagnosis and to the erroneous conclusion that routine management procedures are not applicable to occupational asthma.

The mechanism for asthma is the presence of hyperresponsive airways associated with inflammation, increased bronchomotor tone, edema, and increased mucous gland volume and productivity with a more mucoid than usual secretion. The search for significant differences in the morphology or pathophysiology between occupational and nonoccupational asthma has yielded few real differences. Differences in the cellularity and the character of the basement membrane have been identified, but are not consistent through all studies. The involvement of the standard immune system in the process varies widely between patients having both forms of asthma. Once the hyperreactivity of the airways is established, the patients often display remarkably similar symptom patterns, intolerances, and various identified causes for exacerbations. Therefore, once the link to occupational exposure is adequately established or ruled out, the separation between occupational and nonoccupational asthma becomes somewhat artificial and less than useful with respect to medical management.²

The principles in diagnosis and treatment of both forms of asthma are virtually identical and include an accurate clinical diagnosis of asthma (versus chronic bronchitis or other disorders), identification of the causative agent where possible, environmental controls, education of the patient to the mechanisms of asthma to permit better insight and environmental controls, and

REMINDER

Report all known or suspected cases of occupational disease!

For more information on reporting requirements or to obtain a copy of the *Physician Report of Occupational Disease* form, call the Occupational Health & Special Projects Program at DPH at 860/509-7744.

appropriate pharmacologic therapy once the other aspects have been addressed. While removing the offending agent from the patient's environment is critical in occupational asthma, the management of occupational asthma is otherwise remarkably like the management of atopic or extrinsic asthma in adults. They usually share the pattern of disease and susceptibility to the common causes of asthma exacerbations such as exercise, exposure to cold air and chemical irritants like cigarette smoke and diesel exhaust, viral infections, strong perfumes, aerosols like hair spray, and the ever present stress. Stress is a factor in both forms of disease. It is especially treacherous as a mechanism because most patients do not wish to acknowledge that stress is a factor. To them it implies significant psychological disease rather than viewing stress as an integral part of daily life, both at work and at home.

The failure to recognize how important common irritants are in exacerbating occupational asthma may lead to doubting the diagnosis when it is, in fact, accurate. Furthermore, asthma is a dynamic disorder. Remissions and exacerbations need not be due to removal or re-exposure to a causative agent alone. These may in fact be related to other environmental exposures. Such events as intercurrent viral infections with bronchial epithelial injury or the addition of a nonspecific "irritant" to the home such as an insensitive smoker increases airway reactivity. Many asthma exacerbations occur with a physical move such as from one home or workplace to another, especially with major geographic changes. Such transitions may either greatly improve or greatly exacerbate asthma. They are testimony not only to the reactivity to environmental agents such as mold, mildew and specific allergens, but also to the stresses associated with these social and geographic changes.

Once occupational asthma is established, the disorder confuses patients and healthcare givers alike by appearing to exacerbate with numerous environmental factors, as in the case with patients who have typical allergic asthma. A list of common irritants that exacerbate asthma are in Table 1. All asthmatics are capable of reacting to these fairly nonspecific irritants that offend the hyper-responsive airways while being only nuisances to

the nonasthmatic. Further, asthma is exacerbated with exercise.

Table 1

Common Irritants That Exacerbate Asthma	
Cigarette smoke	Hairspray
Diesel exhaust	Perfumes
Wood stove or fireplace	Detergents
Dust	Glass cleaners
Mold, mildew	Ammonia
Cold air	Paints
Solvents	Oven cleaners

I have also found in some thirty years of evaluating patients with occupational asthma that removal from the offending exposure may result in only a modest improvement in symptoms. This is most striking in isocyanate-induced asthma. It is a common pattern that leads to the erroneous conclusion that the asthma may not be occupational after all. Patients with isocyanate-induced asthma are frequently disappointed with the results of withdrawal from the workplace because their expectations of rapid and complete clearing of symptoms with cessation of exposure are unmet.

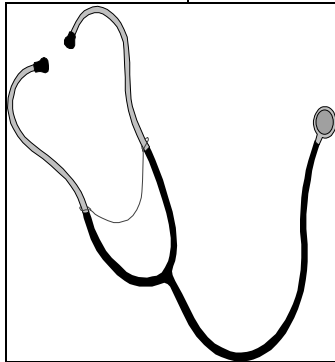
Since both immunological and non-immunological mechanisms are postulated for asthma, that is, for example, isocyanate-induced, patients may be confused by a sudden sensitivity to other agents such as freshly cut grass, ragweed and dust. These agents may become causes for exacerbations only after increased airway reactivity has been established by the occupational exposure. The treating physician or other health care provider should not be "put off" by this apparent increased sensitivity to environmental elements. Once airway reactivity is established, the asthmatic patient's list of offending agents to be avoided is remarkably uniform. Of course, there will be variations from patient to patient, as is typical of human disease.

The tendency for night time cough, wheezing and chest tightness is as common in patients who have a day job and have occupational asthma as in those with nonoccupational forms. The reduction in catecholamines and steroid levels occur in both forms in the evening hours. In addition, patients ingest most of their food from dinner to bedtime, producing a continued cholinergic stimulation.

Many handle the mucoid secretions characteristic of asthma by following their treating physician's advice and increasing their hydration in the afternoon or evening. Unfortunately, this is coincident with the increased airway reactivity of the evening hours. This often results in an increase in evening chest tightness and cough induced by the ingestion of iced drinks during that period. Some claim they are "allergic" to milk since they appear to have the same reaction to milk and ice cream, both of which are invariably refrigerated. I have found in practice that pointing this out to patients has resulted in their suddenly discovering why some of their evenings have been more symptomatic, especially with regard to the unexplained increase in paroxysmal cough. It is uncommon that their physicians have ever advised them on the effects of ingesting cold food or fluids or the therapeutic value of warm drinks. Some patients have discovered that hot fluids are therapeutic but ascribe the benefits to the pharmacological effects of tea or other beverages.

It is also necessary to point out to asthmatics that they are sensitive to cooling of their skin as well as to the cooling of their upper airways by ingesting cold drinks and being in a cold environment. Patients with any form of asthma should be educated to use a dust mask, or other suitable shields that cover the mouth and nose in cold weather or in wind, to maintain adequate temperature and humidity in the breathing zone. Although all asthmatics seem partially vulnerable to this problem, it varies from patient to patient and in a single patient may vary widely from time to time, based on their current level of airway reactivity. The etiology of their asthma is unimportant with respect to this characteristic.

Since the airway hyperreactivity and symptoms may occur through an immediate reaction or a delayed reaction in both occupational and nonoccupational asthma, the evaluator must be alert to symptoms exacerbating hours after the worker has left the workplace. There is a tendency for the evening exacerbation, which is common to all asthmatics, to be viewed as evidence that the cause is not occupational. There are two factors to be considered pertaining to evening symptoms:



1) the nocturnal increase of symptoms which occurs in all asthma; and 2) the possibility of a delayed reaction from late day shift exposures. This pattern is a major reason for workers ruling out work exposure as a likely cause for their symptoms, thus greatly delaying the evaluation and diagnosis of work-related asthma. Another confusing aspect of occupational asthma is its tendency initially to clear overnight and on weekends, but then to become more persistent in a pattern no longer having the distinct relationship to specific exposures or periods of exposures. This is confusing to both the worker and the treating health care provider. The change in circadian rhythm and exposure patterns for evening and night shift workers is another element altering the pattern of symptoms and misleading the treating physician into not considering work exposures as the underlying cause.

Common problems encountered in the diagnosis and treatment of asthma in adults are an inadequate history, ignoring of paroxysmal cough, an inadequate review of work and home exposures to known sensitizing agents or irritants, and a failure to identify a key viral infection that may have caused the sudden onset of airway responsiveness. When these have been overcome, two major management problems remain: (1) denial, and (2) a refusal to use adequate environmental controls and medication to suppress the disorder. The diagnosis of asthma is rarely accepted with equanimity by adults. They usually refuse to believe the diagnosis for periods varying from days to literally months and years. It is therefore important to make a firm diagnosis with clear descriptions of why the diagnosis is likely correct. The uncertainty on the part of the health care provider permits patient denial to continue and renders suboptimal care.

The patients must also be made to realize the importance of environmental controls once the common offending agents have been properly identified. They must be warned not to experiment with re-exposures in an uncontrolled manner if they are to avoid raising the level of their disease or prolonging their symptomatic period. It is important that patients realize that no one is capable of predicting how long their symptoms will

continue, the extent to which they will respond to medication, or if they will have a prolonged period of relief versus continued need for medication and environmental controls.

It is my habit to remind patients that up to 15% of children have asthma at some time, and between 8-10% of adults have asthma that usually begins with a single event, a viral infection, or recurrent exposures with or without an allergic background. This is helpful in allowing them to feel less different or paranoid about their new diagnosis, thus reducing the ever present risk of persistent denial. In some patients an element of denial will remain despite our best efforts since the diagnosis of asthma causes some adults to feel imperfect or "damaged goods."^{3,4}



Patients should be told that the same medication will be effective in both occupational asthma and other forms of the disease, but the specific first goal of management is identification and removal of all known or suspected occupational exposures. Patients must not be allowed to run the risk of even an occasional accidental exposure to an established causative agent, especially to such agents as isocyanates, epoxy resins, epichlorohydrin, latex or common degreasing agents. Continued exposure to these agents may produce a sudden exacerbation to a higher level of disease, which then may or may not remit. Patients should be discouraged from increasing their medication on their own while attempting to continue working with the exposure on a trial basis. The patient may try this approach in the hope of protecting their job and not being required to make an occupational change.

In the author's opinion, the use of safety devices to avoid exposures to causative agents which remain in the work place is asking a worker to accept an undue risk of permanent injury. In such a setting, the worker is often part of the problem, being unwilling to leave a job that is very remunerative. While understandable, this philosophy is one common barrier to proper management of demonstrated occupational asthma. If the disease is more than intermittent and mild, I most often urge retraining and transferring to another area where there will be no

risk for re-exposure when the causative agent is identified with reasonable certainty.

As with any form of bronchial asthma, the best policy is to first utilize as much environmental control, then as much medication as is needed to fully suppress the disease. "Yo-Yo" therapy, that is, inconsistent and less than adequate medication, only perpetuates the asthmatic response and permits it to smolder, reducing the likelihood of full control or a prolonged subsidence of the disorder. An outline of a proper approach to the diagnosis of occupational asthma may be found in the March 1996 issue of *Occupational Airways*.

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ADDITIONAL READING

Bardana Jr. EJ (1995): Occupational asthma and related respiratory disorders. *Disease-A-Month* 41:143-199.

Summary of Number of Reported Cases of Selected Respiratory Diseases				
CT DPH Occupational Disease Surveillance Data				
	1994	1995	1996*	ODSS Total**
Asthma	13	34	15	102
RADS***	1	1	4	12
Silicosis	4	1	0	7
Asbestosis	3	5	3	38
Asbestos-related pleural diseases	17	8	5	120
Total	38	49	27	279

* As of September 30, 1996. Data subject to change.

** Occupational Disease Surveillance System (ODSS) total as of 11/91

*** Reactive Airways Dysfunction Syndrome

CASE REVIEW

Asthma in a Body Shop Worker Due to Isocyanate Paints



A 35 year old male was seen with a one year history of asthma that appeared to exacerbate with exposure to auto body painting operations where he was employed as a shop foreman. He had worked in body shops doing initial metal repairs and using "Bondo" as well as employing spray paints for touch-ups in repair operations since age 18. He was a nonsmoker. He had worked in six different shops over the preceding 17 years, several with a spray booth properly vented in what he regarded as "clean shops" and where no spray painting occurred outside the booth. In his current place of employment the shop had become very busy and spray painting was occurring outside the booth on a regular basis.

The patient's symptoms were cough, shortness of breath, wheeze and chest tightness that had been present intermittently for some three months, but gradually became more constant. His symptoms at first cleared overnight and seemed related to specific spray paint exposure incidents, fully clearing on weekends and vacations. The relationship of his symptoms to isocyanate paints had been further established by demonstrating reductions in peak flows after exposures compared to his pre-exposure status. In the last three months, his symptoms had become daily in spite of a late diagnosis and treatment by the treating physician. The patient was now steroid dependent, on full treatment, out of the workplace, and still symptomatic in spite of full medication and no known exposure for three months. Interestingly, he had a history of chronic nasal congestion, initially intermittent, and present for three months before the onset of cough and wheeze. The patient now fears that he is no longer rational because not only has he failed to get better with withdrawal from the workplace, but he has begun to have exacerbations of asthma when exposed to a cat, freshly cut grass and dust, reactions he had never before experienced.

This case illustrates the progressive nature of isocyanate sensitization and the need for early diagnosis and removal from further exposure if permanent injury is to be avoided. It also

illustrates the common reactivity to other environmental allergens or irritants once the disease is established. It is noteworthy that the patient had initially intermittent and then chronic nasal congestion for months prior to the onset of the asthma, which could have served as a warning of a progressive sensitization process, all too obvious in retrospect, but its significance not appreciated at the time.

For more information, Dr. Godar can be reached at St. Francis Hospital, Hartford at (860) 714-4055.

TURNING DIAGNOSIS INTO PREVENTION

Conference Review and Evaluation

A day-long occupational medicine conference was held on Wednesday, June 26, 1996 at the Connecticut Hospital Association in Wallingford, CT. It was sponsored by the CT Department of Public Health, CT Department of Labor, CT Workers' Compensation Commission, UCONN Health Center, Division of Occupational & Environmental Medicine, Yale Occupational & Environmental Medicine Program, the Occupational Medicine Auxiliary Clinics Network, and the Occupational and Environmental Medicine Association of CT. The conference was attended by a total of 90 participants, including physicians, nurses, public health professionals and industrial hygienists.

The conference was organized to educate health care providers about the many different agencies involved in occupational health in CT, to discuss prevention strategies for workers after diagnosis of an index case, and to discuss emerging issues in occupational health. Case studies of prevention efforts were presented for silicosis, mercury toxicity, chemical hepatitis, latex exposure and back injuries. Workshops included:

- Reducing latex exposure & patient identification
- Managing a workplace surveillance program
- Occupational asthma
- Industrial hygiene in a clinic setting
- Evaluating intervention outcomes
- Intervention for repetitive strain injuries

Of the 90 participants, 48 (53.5%) completed a conference evaluation form. Of the 48, 22 participants were nurses, 10 were physicians, and 12 represented public health professionals, industrial hygienists and other occupations. Fifty-four percent of those responding rated the conference as "Excellent" and 44% rated it "Above Average". Overall, 85% of those responding found the conference to be educational, and 95% found the material presented to be useful.

DPH plans to organize similar occupational health conferences, workshops or seminars in the future. Future topics requested by attendees were: regulations/guidelines, ergonomic/repetitive motion injuries, occupational diseases, environmental factors/indoor air quality,

psychosocial issues of the workplace, medical issues, and environmental epidemiology.

The Occupational Health & Special Projects Program invites your comments and suggestions for future events. If you have comments or suggestions or if you are interested in providing guest commentary on a topic of occupational respiratory disease for the newsletter, please call Marian Heyman or Juanita Estrada at (860) 509-7744.

Occupational Airways is produced by the Occupational Health Surveillance Program, Division of Environmental Epidemiology & Occupational Health, Connecticut Department of Public Health.

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