

- occur after long-term administration of salbutamol controlled release (Volmax). *Eur Respir J* 1988;1(2):202.
19. Tsanakas JN, Baxter PS. A comparison of salbutamol controlled release and slow-release theophylline in exercise-induced asthma. *Eur Respir J* 1988;1(2):356.
 20. Larsson S, Svedmyr N, Thiringer G. Lack of bronchial beta-adrenoceptor resistance in asthmatics during long-term treatment with terbutaline. *J ALLERGY CLIN IMMUNOL* 1977;59:93-100.
 21. Hendeles L, Weinberger M. Theophylline, a "state-of-the-art" review. *Pharmacotherapy* 1983;3:2-44.
 22. Powell ML, Weisberger M, Dowdy Y, Gural R, Symchowicz S, Patrick JE. Comparative steady-state bioavailability of conventional and controlled-release formulations of albuterol. *Bio-pharm Drug Dispos* 1987;8:461-8.

Occupational asthma caused by latex in a surgical glove manufacturing plant

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*A 33-year-old latex glove inspector was diagnosed as having occupational asthma on the basis of peak flow and methacholine-responsiveness changes related to workplace exposure. She had latex sensitivity by skin prick testing. This finding led to a survey of her workplace. Of the 81 workers, 84% completed a questionnaire to assess work-related respiratory symptoms, 79% underwent skin prick testing to latex and six other workplace chemicals, and 62% performed workshift spirometry. Seven workers had spirometric changes consistent with asthma, of whom five had $\geq 15\%$ fall in FEV₁ during the workshift. Six of these workers underwent methacholine-challenge testing, and three were found to have a significant improvement in responsiveness to methacholine away from work, suggestive of occupational asthma. Two of these workers had positive skin test responses to latex. Including the index case, 6% of workers who had pulmonary function testing had findings of latex-related occupational asthma. Skin test responses to latex occurred in 11% of workers tested. Our findings suggest that latex should be considered among the causes of occupational asthma. (*J ALLERGY CLIN IMMUNOL* 1990;85: 626-31.)*

Case reports of immediate hypersensitivity responses to natural latex have been published mainly since 1979,¹ most case reports of contact urticaria¹⁻⁸ with a few case reports of allergic rhinitis or anaphylaxis.⁹⁻¹³ Only recently have there been case reports of asthma.^{14, 15} The diagnosis in our center of a worker with occupational asthma from latex sensitivity has prompted a survey of the patient's workplace, a surgical glove manufacturing plant, to identify other workers with IgE-mediated responses to latex and occupational asthma.

Abbreviations used

FEF₅₀: Forced expiratory flow at 50% vital capacity
FEF₇₅: Forced expiratory flow at 25% vital capacity
PC₂₀: Provocation concentration inducing a 20% fall in FEV₁
MOL: Ministry of Labor, Ontario, Canada

CASE REPORT

A 33-year-old woman was first observed for assessment of occupational asthma. She had been working for 7 years as an inspector at a medical supply company. Her job involved inflating natural latex gloves that were coated with cornstarch.

For the 6 months before she was observed, there was episodic cough, shortness of breath, chest tightness, and occasional wheezing. Symptoms were of variable severity. The symptoms began within 10 minutes of the onset of work and became worse later (90 minutes after leaving work). Symptoms cleared completely while she was away on a 12-

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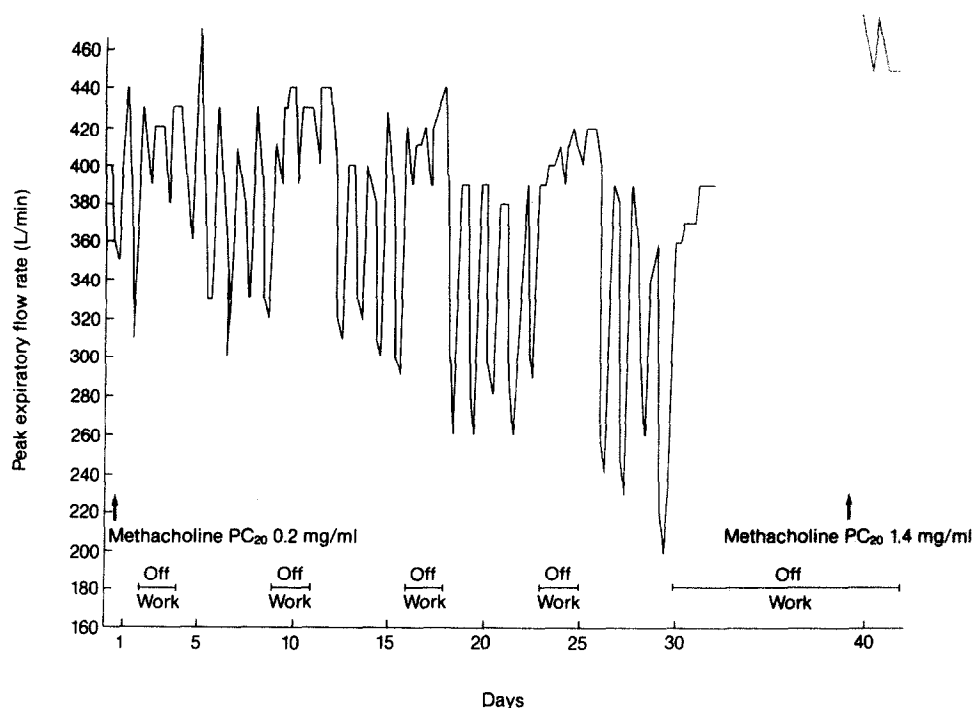


FIG. 1. Peak flow readings and methacholine responsiveness in the index case at work and away from work.

day holiday and recurred the first day back at work. There were no cutaneous or nasal symptoms. Past medical history was positive for migraine headaches.

On physical examination, she was obese. Her blood pressure was 100/60 and her pulse was 68/min and regular. Physical examination was completely normal. In particular, there were no abnormal findings on examination of the lungs.

Skin tests by the prick method to 16 common food and inhalant allergens, as well as corn and cornstarch,¹⁶ were negative. The chest radiographs were normal. FEV₁ was 3.1 L (129% of predicted), and FEV₁/FVC was 74%. FEF₅₀ was 94% predicted. FEF₇₅ was 44% of predicted. There was no significant change after inhaled bronchodilator. These results demonstrate mild airflow limitation at low lung volumes. In a methacholine-inhalation test the next day, there was a 24% drop in the FEV₁ at 0.25 mg/ml (PC₂₀, 0.2 mg/ml), consistent with severe airway hyperresponsiveness.

The peak flow was reduced at work compared to values at home (Fig. 1). There was a concomitant increase in medication requirements at work. During the course of a month of follow-up, the asthma significantly worsened, and there was a major reduction in FEV₁ to 1.9 L (79% of predicted); FEV₁/FVC ratio was 65%. After avoidance of work for 2 weeks, baseline FEV₁ was 3 L, and methacholine, 1.4 mg/ml, was required to elicit a 20% drop in FEV₁ (PC₂₀ 1.4 mg/ml), demonstrating a reduction in the airway hyperresponsiveness.

Inhalation-challenge tests were performed on separate days with the cornstarch glove powder and the coagulant.

On the first challenge day, the patient was exposed to the coagulase liquid undiluted from the workplace, aerosolized with a spray bottle for 30 minutes. Spirometry was performed before exposure, immediately after exposure, and at 30-minute intervals for 5 hours. On the second day, the exposure was repeated with powdered cornstarch, microtalc, and the dried coagulase from the workplace, airborne by shaking the powders from one beaker to another, with each powder used for 10 minutes. Spirometry was performed before the exposure, after each exposure, and at 30-minute intervals for 5 hours. These challenges were negative. On each day the FEV₁ fall was <8% from baseline before the exposure of the day.

A prick skin test with the industrial latex solution undiluted was positive with a 5 mm wheal and flare. Three similarly tested control subjects were negative.

MATERIAL AND METHODS

Workplace inspection

After diagnosis of our initial patient with latex-induced occupational asthma, a workplace inspection was conducted by MOL to assess the nature and use of workplace chemicals. The company employed 87 workers. The plant produced about 53,000 pairs of gloves per day.

The glove molds were first dipped into a tank of coagulant (lactic acid, ethyl and methyl alcohol, calcium nitrate, and magnesium silicate) and then into a tank of latex compound mixture (water, low ammonia latex, and accelerators, including dipentamethylenethiuram hexasulfide, zinc 2-mercaptobenzothiazole, zinc diethyldithiocarbamate, and

alkylated diphenylamines). The latex compound solidified around the mold forming the glove. The coagulant was then leached out of the gloves in a water bath at 165° F. The gloves were dried in an oven at 195° F and were then cured in an oven at 240° F. Gloves were sprayed with a corn starch, alcohol, and water solution and were then air blown off the molds and were tumbled at room temperature to polish them.

The gloves were then inspected by inflating them like a balloon with compressed air. A small cloud of dust potentially containing traces of the glove chemicals, as well as glove powder, was released from the glove as it was inflated in front of the worker. Local ventilation was provided but was impaired if screens became caked with dust or if cross-drafts were present from a fan in the aisle. At the time of the visit, inspectors did not wear the respirators that were available.

The inspection area and the packaging area were adjacent to but separated by a partition from the manufacturing area. At the time of the visit, management and the worker representative reported that eight of 28 inspection workers had reported respiratory problems. Further investigation was initiated by the MOL Medical Service Chest Clinic because of the number of workers with significant respiratory complaint.

Study protocol

Consent was obtained from representatives of the workplace management and workers for a survey of the plant to investigate the presence of IgE-mediated latex-allergic responses and occupational asthma. The study protocol was approved by the ethics committee of the University of Toronto. All the plant workers were invited to participate in the study.

A questionnaire modified from the American Thoracic Society questionnaire was used to determine symptoms of rhinitis, asthma, and urticaria, and any temporal relationship of these symptoms to the workplace. Other questions included medication requirements, smoking history, and whether the worker had been diagnosed previously as having asthma.

Skin test materials were obtained as used in the workplace. A low ammonia latex solution was used (Dynatex GTX, supplied by Guthrie Latex, Inc., Woodbridge, N.J.) as obtained in the plant before addition of other chemicals. This contained 0.55% ammonia (percent weight per weight on water) and a combination of zinc oxide and tetramethylthiuram disulfide (0.1% wt/wt on latex). It was used in "industrial strength" without further dilution for skin testing. Chemical solutions added to the latex in the plant were also used for skin testing: agerite stalite (alkylated diphenylamines), zetax (zinc 2-mercaptobenzothiazole and zinc diethyldithiocarbamate), sulfad (dipentamethylenethiuram hexasulfide), triethanolamine, and ethyl zimate. These were diluted from the "industrial strength" to 1:10 vol with 50% glycerin. A solution of glove powder starch was also used, supplied by the plant and then similarly diluted, as well as a diluent control (Bencard Allergy Service, Mississauga, Ontario, Canada).

Skin tests were performed by the prick test method, and the mean wheal diameter was recorded. A wheal ≥ 2 mm larger than the control response with surrounding flare was considered a positive response. Antihistamine use was recorded at the time of skin testing.

Spirometry was performed in the workplace with a calibrated portable wedge spirometer. Forced expiratory maneuvers were repeated until at least three acceptable curves were obtained, including two tracings that corresponded within 5% both for FVC and FEV₁. The largest FVC and FEV₁ from any acceptable curve were recorded. Maximal expiratory flows between 25% and 75% of the FVC were taken from the acceptable curve with the largest sum of FVC and FEV₁. These measurements were corrected to body temperature, atmospheric pressure of 760 mm Hg, and water pressure of 47 mm Hg.

Spirometry was performed on all consenting workers immediately before a Monday workshift, 1 hour into the shift, and at the end of the 8-hour shift. At the end of the shift, spirometry was also repeated 10 minutes after administration of inhaled salbutamol, 200 μ g.

Consent for methacholine challenge testing was requested from all workers with positive skin tests to a workplace chemical, workers with a fall in FEV₁ of at least 15% during the work shift, and workers who had at least a 15% increase in FEV₁ after a bronchodilator (salbutamol) at the end of the work shift. The methacholine challenges were performed initially 2 weeks before the Christmas holidays during a working day in the workplace.

Methacholine challenge testing was performed according to the method of Juniper et al.¹⁷ The methacholine PC₂₀ was obtained from the dose-response curve. A methacholine PC₂₀ of 8 mg/ml or less is considered consistent with a diagnosis of asthma.

Workers in whom the methacholine PC₂₀ was 8 mg/ml or less were requested to keep records of peak flow readings for 2 weeks at work and then for 10 to 14 days away from work during the Christmas plant closure. Workers were instructed in the correct use of a Spira peak flow meter (Trudell Co., London, Ontario). Workers were asked to record the highest of three readings at least four times a day before the use of any medication, as well as at the time of any nocturnal respiratory symptoms. These workers were also requested to return for repeat methacholine-challenge testing at the end of their holiday period before returning to the workplace.

Air sampling for total dust was performed on 2 of the testing days at work to assess worker exposure. Personal and area samples for total dust were collected by drawing air through preweighed polyvinyl chloride filter cassettes (based on National Institute for Occupational Safety and Health method 0500¹⁸).

Our criteria for occupational asthma were arbitrarily set as (1) supportive evidence of asthma with at least 15% variability in FEV₁, either during a workshift or in response to inhaled bronchodilator, as well as (2) supportive evidence of occupational change with twofold or greater improvement in methacholine responsiveness after removal from the workplace.

RESULTS

Air sampling

Air sampling by the MOL for total dust to assess worker exposure to the dusting powder was done on the 2 days that clinical testing on the workers was being done. The airborne levels of cornstarch/dust were well within the allowable exposure level of 10 mg/m^3 .¹⁹ The dust levels in the production areas were consistently low on both days of testing. However, there was a wide variation in dust exposure among the inspectors, ranging from 0.4 mg/m^3 to 5.5 mg/m^3 . This variation was probably due to individual work habits.

Survey of workers

The total number of workers in the plant during three shifts was 81, excluding all six office workers who did not wish to participate in the study. Of the workers, 30 were glove inspectors, 29 worked in the production area, nine worked in packaging, four worked in quality control, eight worked in maintenance, and one worked in compounding.

Sixty-eight workers (84%) completed the questionnaire, 22 men, mean age, 27 years, and 46 women, mean age, 35 years. Most workers stated that they had upper and lower respiratory symptoms, worse at work (Table I), whereas fewer workers (10%) stated that they had hives while they were at work.

Skin test responses to the latex solution were observed in seven of the 64 workers tested (11%). Four of these responses were 2 mm in mean wheal diameter, two were 3 mm, and one response was 5 mm in mean wheal diameter. Six of these seven workers worked with the finished glove, whereas only one worked in production. All but one worker had upper and lower respiratory symptoms related to work. Two additional production workers had skin test response to agerite stalite (3 mm and 2 mm wheal diameters), and one of these workers also responded to zetax (2 mm wheal diameter). One additional inspector responded to sulfad (2 mm wheal diameter). No other skin test responses were present.

Workplace spirometry demonstrated a 15% or greater fall in FEV_1 during a workshift in five of 50 workers (10%) (Table I). Four of these five workers had upper and lower respiratory symptoms, worse at work, and the fifth worker had a cough at work. Two of these workers had skin test responses, one to latex and one worker to zetax and agerite (2 mm and 3 mm wheal diameter). A bronchodilator response was assessed in four of the five workers at the end of the workshift and demonstrated $\geq 15\%$ increase in FEV_1 in two of these workers, both skin test negative. Among the 50 workers undergoing spirometry, an ad-

TABLE I. Surgical glove workers

	No.	Percentage
Questionnaires completed	68	
Respiratory symptoms at work	52	76.5
Upper	52	76.5
Lower	41	60.3
Upper and lower	39	57.3
Urticaria at work	7	10.3
Urticaria, upper and lower respiratory symptoms at work	6	8.8
Skin tests performed	64	
Positive skin tests to latex	7	11.0
Positive skin tests and upper and lower respiratory symptoms at work	6	9.0
Pulmonary function tests during shift	50	
FEV_1 shift changes	5	10
FEV_1 shift change, upper and lower respiratory symptoms at work	4	8.0
FEV_1 shift changes, and upper and lower respiratory symptoms and positive skin tests	2	4.0

ditional two subjects (4%) had no significant workshift changes in FEV_1 but did have a bronchodilator response at the end of the shift.

A history of diagnosed asthma from questionnaire was present in eight subjects, none of whom responded to latex on skin testing but one of whom had the positive skin test to zetax and agerite and demonstrated a workshift fall in FEV_1 . Two other workers demonstrated a bronchodilator response at the end of the shift and two refused spirometry.

Methacholine-challenge testing was performed on a workday in 11 subjects (Table II), five of the seven latex skin test responders, two of the three other skin test responders (including the subject skin test positive to zetax and agerite), and four of the additional five workers, with spirometric findings of asthma, that is, workshift or bronchodilator FEV_1 changes. Five of those workers challenged had a methacholine PC_{20} in the asthmatic range, $\leq 8 \text{ mg/ml}$. Three of these workers were skin test negative, and two workers were positive to latex (5 mm and 3 mm wheal diameters). Repeat methacholine challenge after 10 days away from work demonstrated a greater than twofold increase in PC_{20} in three of these five workers, including the two workers skin test positive to latex, one of

TABLE II. Results of methacholine/peak flow studies in 11 surgical glove workers

Subject	Methacholine PC ₂₀		Peak flow change	Shift FEV ₁ change	Bronchodilator	Skin test positive
	Work	Home	Work/Home			
1	1.9	6.0	Negative	ND	ND	Latex
2	2.25	5.6	? (12%)	ND	ND	Latex
3	0.05	0.5	Negative	Positive	Positive	—
4	0.8	0.7	Negative	Positive	Positive	—
5	12.0	8.0	Negative	Negative	Negative	Agerite
6	4.2	ND	ND	Negative	Positive	—
7	17.0	ND	ND	Negative	Negative	Latex
8	20.0	ND	ND	Positive	? (13%)	Zetax, agerite
9	>32.0	ND	ND	Positive	Negative	Latex
10	>32.0	ND	ND	Negative	Negative	Latex
11	>32.0	ND	ND	? (11%)	Negative	—

ND, Not done.

whom also had improvement in peak flows away from work, not observed in the other workers.

A diagnosis of latex occupational asthma was made in three workers, including the index case (6% of workers having pulmonary function tests), on the basis of the skin test response to latex in the presence of work-related respiratory symptoms, spirometry changes, and methacholine changes related to workplace exposure. All were female workers, aged 33 and 34 years, and none of the workers were current smokers. Two of these workers were glove inspectors, and one worker was a quality control worker. A fourth worker had findings suggestive of occupational asthma but no identified workplace allergen. She was a production worker, aged 30 years, with a 25-pack year smoking history. An additional five workers had small skin test responses to latex, and four of these workers had upper and lower respiratory workplace symptoms. Three of these four workers had no objective evidence of asthma. The remaining worker of these four symptomatic workers declined pulmonary function testing.

DISCUSSION

Natural latex, a polymer of isoprene, as used in the production of surgical gloves, is obtained from the sap of the rubber tree, *Hevea brasiliensis*. Despite its widespread use in industry, occupational asthma from latex has only recently been reported in one case.¹⁴ Our study of three cases in the same factory indicates that latex should be included among the increasing number of agents that can cause occupational asthma.

The latex solution used for skin testing in this study did contain small amounts of additives: ammonia, zinc oxide, and tetramethylthiuram disulfide. Of these ad-

ditives, the thiuram could be antigenic, but to our knowledge in contrast to latex itself, thiurams have only been reported to cause delayed dermal responses and have not been reported to cause immediate hypersensitivity responses or asthma. In addition, skin tests with a thiuram, dipentamethylenethiuram hexasulfide, also used in the plant, were negative in all workers who were skin test positive to latex solution. Therefore, the responsible antigen in these workers is likely to be in the latex solution itself. This may be an isoprene-derived contaminant, as suggested in one previous study of rubber glove asthma,¹⁴ or may be natural latex proteins, as suggested by findings of Carrillo and Wrangsjö et al,⁹ and by our RAST findings in a previously reported patient.¹¹ This finding is further supported by recent immunoblot studies demonstrating that at least four soluble polypeptides from natural latex can bind specific human IgE.¹⁵

The route of sensitization to latex in these workers is unknown. Seven of the eight workers, including the index case with skin test responses to latex and all three workers with latex-related asthma, were working with the finished gloves as inspectors, packagers, or in quality control. This suggests that the exposure occurs from the cured latex. None of the subjects had known skin disease, unlike previous subjects with anaphylactic reactions to latex.^{10, 11} In addition, the respiratory manifestations would suggest that exposure to the latex antigen was by inhalation, possibly in association with the glove powder.

The prevalence of occupational asthma to latex in the plant surveyed can only be estimated, since the participation by the workers in this study was incomplete. Nevertheless, it occurred in at least 3.7% of

workers (three of 81) and possibly in 6% or more, depending on how representative was the sample of 50 workers who underwent spirometry. This finding is similar to the estimated 5% prevalence of occupational asthma related to isocyanates²⁰ and 4% prevalence related to red cedar.²¹

Our findings suggest that, as with other allergens, skin sensitivity to the latex solution was more commonly found than occupational asthma (11% of workers tested), including marginal 2 mm wheal responses. Six of the eight workers who had skin test responses to latex solution also had symptoms compatible with occupational rhinitis. However, these symptoms could not be clearly related to latex because they were present in most workers and therefore more likely reflected an irritant workplace factor.

Skin test responses to other chemicals used in making latex gloves were found in three workers, suggesting that alkylated diphenylamines, zinc diethyldithiocarbamate, or zinc 2-mercaptobenzothiazole and dipentamethylenethiuram hexasulfide might uncommonly cause immediate skin sensitization.

Immediate hypersensitivity causing occupational asthma has been reported with azodicarbonamide²² used in rubber and plastics industries. However, the many other chemicals used in rubber production have not, to our knowledge, been reported to cause asthma.

The questionnaire used by the authors in this survey yielded a high percentage of workers whose symptoms would be consistent with occupational upper or lower respiratory symptoms. This may, in part, have reflected worker bias in participation, since the purpose of the study was known to the workers. It may have resulted from a nonspecific irritant effect of workplace chemicals, although the scope of this study did not permit extensive investigation of this hypothesis. One skin test negative worker who demonstrated significant improvement in bronchial reactivity away from work had findings consistent with an occupational exacerbation of underlying asthma. This finding would be consistent with our recent findings of work-related changes in bronchial reactivity in some patients with irritant-induced occupational asthma.²³

Our findings indicate that latex should be included as a potential cause of occupational asthma. With recent increases in production and use of latex gloves and other rubber products, clinical sensitivity may be found more commonly than in previous years.

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REFERENCES

1. Nutter AF. Contact urticaria to rubber. *Br J Dermatol* 1979;101:597-8.
2. Forstrom L. Contact urticaria from latex surgical gloves. *Contact Dermatitis* 1980;6:33-4.
3. Kopman A, Hunnaksela M. Contact urticaria to rubber. *Duodecim* 1983;99:221-4.
4. Meding B, Fregert S. Contact urticaria from natural latex gloves. *Contact Dermatitis* 1984;10:52-3.
5. Kleinhans D. Contact urticaria to rubber gloves. *Contact Dermatitis* 1984;10:124-5.
6. Frosch PJ, Wahl R, Bahmer FA, et al. Contact urticaria to rubber gloves is IgE mediated. *Contact Dermatitis* 1986;14:241-5.
7. Carrillo T, Cuevas M, Muñoz T, et al. Contact urticaria and rhinitis from latex surgical gloves. *Contact Dermatitis* 1986;15:69-72.
8. Turjanmaa K. Incidence of immediate allergy to latex gloves in hospital personnel. *Contact Dermatitis* 1987;17:270-5.
9. Wrangsjö K, Mellström G, Axelsson G. Discomfort from rubber gloves indicating contact urticaria. *Contact Dermatitis* 1986;15:79-84.
10. Axelsson JG, Johansson SG, Wrangsjö K. IgE-mediated anaphylactoid reactions to rubber. *Allergy* 1987;42:46-50.
11. Spaner D, Dolovich J, Tarlo S, Sussman G, Butto K. Hypersensitivity to natural latex. *J ALLERGY CLIN IMMUNOL* 1989;83:1135-7.
12. Jancelowicz Z, Sussman G, Tarlo S, Dolovich J. Clinical presentation of five patients allergic to latex [Abstract]. *J ALLERGY CLIN IMMUNOL* 1989;83:267.
13. Slater JE. Rubber anaphylaxis. *N Engl J Med* 1989;320:1126-30.
14. Seaton A, Cherrie B, Turnbull J. Rubber glove asthma. *Br Med J* 1988;296:531-2.
15. Morales C, Basomba A, Carreira J, Sastre A. Anaphylaxis produced by rubber glove contact: case reports and identification of the antigens involved. *Clin Exp Allergy* 1989;19:425-30.
16. Fisher AA. Contact urticaria due to cornstarch surgical glove powder. *Cutis* 1986;38:307-8.
17. Juniper EF, Frith PA, Hargreave FE. Reproducibility and comparison of responses to inhaled histamine and methacholine. *Thorax* 1978;33:705-10.
18. NIOSH method 0500. NIOSH manual of analytical methods, vol 2. 3rd. ed. Washington, D.C.: U.S. Department of Health, Education, and Welfare. 1984 (NIOSH publication No. 84-100).
19. Regulation respecting control of exposure to biological or chemical agents made under the Occupational Health and Safety Act. Ontario regulation 654/86.
20. NIOSH criteria for a recommended standard. Occupational exposure to diisocyanate. Washington, D.C.: U.S. Department of Health, Education and Welfare, September 1978 (NIOSH publication No. 78-215).
21. Chan-Yeung M, Vedal S, Kus J, MacLean L, Enarson D, Tse K. Symptoms and pulmonary function in western red cedar workers related to duration of employment and dust exposure [Abstract]. *Am Rev Respir Dis* 1984;129(suppl A152):909.
22. Slovak AJM. Occupational asthma caused by plastics blowing agent azodicarbonamide. *Thorax* 1981;36:906-9.
23. Tarlo SM, Broder I. Irritant-induced occupational asthma. *Chest* 1989;96:297-300.