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**C H E S T**

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# Machine Operator's Lung\*

## A Hypersensitivity Pneumonitis Disorder Associated With Exposure to Metalworking Fluid Aerosols

David I. Bernstein, MD; Zana L. Lummus, PhD; Greg Santilli, MD;  
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Six auto parts manufacturing workers were referred for evaluation of a 6-week history of work-related dyspnea, cough, and fatigue. Two workers also reported fever and weight loss. All six worked in a machining area where a waterbased metalworking fluid was used and recirculated under high pressure, thereby creating an aerosol. Chest radiographs revealed pulmonary interstitial infiltrates in four workers. Lung function tests showed that four workers had decreased diffusing capacity. After removal from the work area, all workers recovered. The metalworking fluid was cultured for bacteria and fungi. Isolates from broth cultures were sonicated to obtain antigen extracts. Serum precipitins to one or more of the microbial isolates were identified in all six workers but not in eight of nine nonexposed control subjects. The most frequent precipitin response (six of six workers) was against antigens of *Pseudomonas fluorescens*, which was cultured from the metalworking fluid. In all workers, precipitins to at least one other cultured or-

ganism were detected; these included *Aspergillus niger*, *Staphylococcus capitis*, an acid-fast *Rhodococcus* sp, and *Bacillus pumilus*. This represents the first report of hypersensitivity pneumonitis associated with industrial exposure to aerosolized metalworking fluid. Observed precipitin responses to a variety of microbial contaminants in metalworking fluid strongly suggest a causative role for microbial antigens in the induction and elicitation of this manifestation of hypersensitivity pneumonitis. (CHEST 1995; 108:636-41)

BHI=brain heart infusion; Dco/VA=diffusing capacity for CO and alveolar gas volume ratio; TLC=total lung capacity

Three classes of metalworking fluids are commonly used as coolants and lubricants for metal grinding and cutting procedures: insoluble lower viscosity petroleum oils, soluble emulsified oils diluted in water,

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and synthetic or semisynthetic fluids. Typically, synthetic fluids are alkaline solutions of hydrocarbons, esters, polyglycols, and water. Synthetic fluids also contain alkanolamines, such as triethanolamine and diethanolamine. Borates and phosphates are added for use as corrosion inhibitors. Surfactants are added to these fluids for their desired lubricant properties.<sup>1</sup> Microbiological analyses of these fluids have identified bacterial contaminants including *Pseudomonas aeruginosa*, *Proteus mirabilis*, *Enterobacter cloacae*, *Escherichia coli*, and *Klebsiella pneumoniae*. Saprophytic fungi, such as *Fusarium*, *Candida*, and *Cephalosporium* also have been cultured from metalworking flu-

ids.<sup>2</sup> For this reason, a variety of biocidal agents (eg, bromopol, morpholine, or triazine compounds) are incorporated into metalworking fluids to control growth of bacteria and fungi.

Little is known about respiratory effects associated with inhalational exposure to machining fluid aerosols. In a Danish survey of machine shop workers, symptoms of cough and sputum production were positively associated with high concentrations (0.1 to 2.0 mg/m<sup>3</sup>) of aerosolized particulates of a metalworking fluid.<sup>3</sup> In 1992, we evaluated a group of machine tool workers who presented with acute respiratory impairment, pulmonary infiltrates, and decrements in lung function associated with exposure to aerosols from a synthetic metalworking fluid which had been introduced into a common work environment 6 to 8 months prior to the onset of symptoms. Serum-precipitating antibody responses to microbial antigens isolated from the synthetic metalworking fluid were demonstrated. These cases represent a new form of hypersensitivity pneumonitis which has been coined "machine operator's lung."

### METHODS

#### Patients and Exposure

Six male machining workers (5 operators and 1 supervisor) from a single work area were evaluated and treated for unusual respira-

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tory illnesses which occurred between the months of April and September of 1992. They shared a common work area located in an auto parts manufacturing facility. The 6 workers were part of a larger group of 16 workers who collectively operated 12 machine tool devices which cut and ground metal parts. A large open pit was used to store 16,000 gallons of water-based metalworking fluid which was recirculated and pumped 7.6 m through hoses to the machines. Upon reaching the machines, the fluid was sprayed on the surface of metal during cutting and grinding, thereby generating aerosols. All 16 workers performed the same tasks and all experienced similar exposure to the aerosolized fluid. This metalworking fluid was an aqueous synthetic mixture that had been introduced into the work area in October 1991. The concentrated solution supplied by the manufacturer contained phosphate ester (9%), a biocidal triazine compound (3%), high molecular weight polybutene (10%), amine borate (10%), benzisothiazine (2%), water (52%), alkanol amide (10%), and nonionic surfactants (2.5%).

#### Microbiological Methods

Two samples from the metalworking fluid in use during occurrence of the illnesses were submitted to Pathcon Laboratories (Norcross, Ga) for microbiological analysis. The laboratory determined that the fluid did not contain *Legionella* and estimated that the fluid contained less than 10 colony forming units (CFU) of fungi and  $1.3 \times 10^6$ /mL (sample 1) and  $1.1 \times 10^6$  (sample 2) CFUs/mL of bacteria. Our laboratory obtained two 8-oz metalworking fluid samples from the plant. An aliquot of each sample was dialyzed against three changes of physiologic saline solution with a membrane cutoff of 6,000 to 8,000 kd. Dialyzed and undialyzed samples of fluid (0.1 mL) were plated separately on blood, Sabouraud-dextrose, YT (5 g yeast, 8 g tryptone, 5 g NaCl, 15 g agar), AK sporulating agar (Whittaker MA Bioproducts; Walkersville, Md), and brain heart infusion (BHI) agar plates under aerobic conditions. Plates were incubated overnight at 37° C in a 5% CO<sub>2</sub> incubator or at room temperature and subsequently observed 2 weeks for growth. Six colony types were isolated from the undialyzed fluid samples and two from the dialyzed samples. The colonies were then

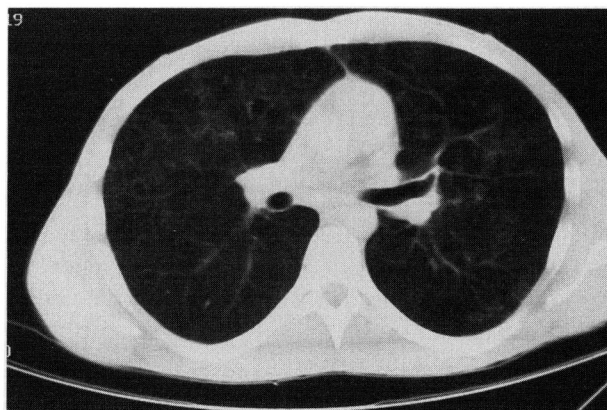


FIGURE 1. Chest CT scan (normal resolution) in worker 2 showing evidence of diffuse interstitial infiltrates.

subcultured on agar plates and in BHI broth. Gram and acid-fast bacilli staining were performed on all isolates. Organisms also were tested for optimal growth temperature at 24, 37, 50 and 56° C but no thermophiles were detected. Species identification of bacterial isolates was performed by Biolog or fatty acid analysis (FAME; Analytical Services; Essex Junction, Vt). Endotoxin was quantitated in metalworking fluid samples using the Limulus Amebocyte Lysate Assay (Whittaker MA Bioproducts).

#### Preparation of Antigens and Gel Diffusion

Isolates were maintained as pure cultures in BHI agar. These were grown in 10 mL BHI broth, centrifuged, washed 3 times in sterile saline solution. The cell pellet was resuspended in 1 mL ice-cold saline solution and sonicated for 30 s at 50 W with a Sonifier Cell Disrupter (Heat Systems-Ultrasonic; Plainview, NY) in order to obtain somatic antigen extracts. Using slides coated with 1.5% agarose in physiologic saline solution (0.15 mol/L), fivefold

**Table 1—Summary of Clinical Symptoms, Chest Radiographic Findings, and Lung Function Studies in Six Machine Workers\***

| Patient | Symptoms                                  | Smoking Status, Pack-years | Time Exposed Prior to Onset of Symptoms | X-ray Film Findings            | Spirometric Studies, % Predicted |                 |                             | Diffusion Dco/VA Ratio       | TLC, % Predicted            |
|---------|-------------------------------------------|----------------------------|-----------------------------------------|--------------------------------|----------------------------------|-----------------|-----------------------------|------------------------------|-----------------------------|
|         |                                           |                            |                                         |                                | FEV <sub>1</sub>                 | FVC             | FEV <sub>1</sub> /FVC Ratio |                              |                             |
| 1       | Cough, dyspnea, 5.9 kg weight loss        | 11                         | 6 mo                                    | Interstitial/air-space disease | 36                               | 35<br>(5/29/92) | 84                          | 60%<br>(7/9/92) <sup>†</sup> | 95%<br>(7/9/92)<br>FVC:92%  |
| 2       | Dyspnea, fever                            | 61                         | 7 mo                                    | Interstitial                   | 58                               | 66<br>(6/9/92)  | 72                          | 57%<br>(6/26/92)             | 88%<br>(6/26/92)<br>FVC:86% |
| 3       | Cough, dyspnea                            | 0                          | 7 mo                                    | Interstitial                   | 66                               | 62              | 87                          | Not done                     | Not done                    |
| 4       | Cough, dyspnea, fatigue                   | 19                         | 8 mo                                    | Normal                         | 61                               | 59<br>(6/26/92) | 84                          | 42%<br>(7/8/92)              | 93%<br>(7/8/92)<br>FVC:73%  |
| 5       | Cough, dyspnea                            | 4                          | 7 mo                                    | Normal                         | 90                               | 94              | 79                          | Not done                     | Not done                    |
| 6       | Cough, dyspnea, fever, 6.9 kg weight loss | 39                         | 8 mo                                    | Interstitial                   | 52                               | 63              | 67                          | 55% <sup>‡</sup>             | 84%                         |

\*Spirometric studies are those measured at initial presentation during active exposure at work. In workers 1, 2 and 4, TLC and Dco/VA ratio studies were not performed until at least 2 weeks after removal (dates are shown) from the work environment; concurrent FVC values reflect physiologic improvement.

<sup>†</sup>Dco/VA improved to 88% of predicted after avoidance and 1 month of daily prednisone (20 to 50 mg) doses.

<sup>‡</sup>Dco/VA improved following clinical improvement to 63% of predicted.

concentrated workers' sera were tested. Gel diffusion studies to all microbial antigen extracts also were evaluated in nine sera obtained from University of Cincinnati laboratory workers who had no prior exposure to metalworking fluids.

RESULTS

Clinical Evaluations

The index case was a 30-year-old man (worker 1) who presented with a 2-month history of dyspnea on exertion, chest tightness, and cough. Symptoms began on the second day of the work week and diminished 2 to 3 days after leaving the workplace. There was a 5.9 kg weight loss over 2 months, increased fatiguability, and diminished appetite. A chest radiograph and chest CT scan revealed bilateral diffuse pulmonary interstitial infiltrates (Fig 1). The FVC was 1.95 L (35%), the FEV<sub>1</sub> was 1.65 (36%), and the FEV<sub>1</sub>/FVC ratio was 84%. The ratio of diffusing capacity for CO and alveolar gas volume (Dco/V<sub>A</sub>) was 60% and the TLC was 93% of predicted (Table 1). Symptoms resolved completely following reassignment to a work area where there was no ongoing use of metalworking fluids. The Dco improved to 88% predicted after subsequent treatment with daily doses of prednisone for a month, and the patient remained asymptomatic after finishing treatment.

As shown in Table 1, five other workers had work-related dyspnea and four reported a dry cough. Other symptoms included fever, fatigue, and weight loss. All these workers improved with complete avoidance of the workplace. Upon return to work, worker 6 also reported the onset of symptoms on the second workday. Workers 2, 3, 4, and 5 noted the onset of symptoms within 3 to 10 h on the first day of the workweek. Four workers presented with pulmonary interstitial infiltrates, and two had normal radiographs. A representative chest radiograph in one of these workers is shown in Figure 2. As shown in Table 1, five workers exhibited a restrictive ventilatory pattern based on the spirometric data alone. However, a restrictive abnormality could not be confirmed based on TLC measurements performed either during (worker 6) or at 2 to 5 weeks after the initial spirometric tests (workers 1,

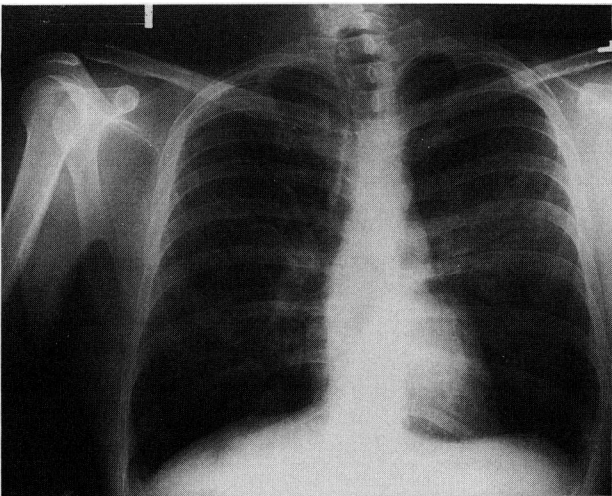


FIGURE 2. Chest radiograph (posteroanterior view) performed in an acutely symptomatic worker (worker 1) which exhibited diffuse interstitial infiltrates.

2, and 4). By the time TLCs were obtained, the FVC values had already improved after the workers had left the work environment (Table 1). Although it appears that in workers 1, 2, and 4 the early spirometric studies are inconsistent with the TLC data obtained at a later date, it is also possible that the TLC may have been decreased initially had it been measured. In 4 of the 5 workers (workers 1, 2, 4, and 6) in whom complete lung function testing was performed, abnormal diffusing capacities (Dco/V<sub>A</sub>) confirmed the presence of interstitial lung disease. Based on pulmonary function testing and chest x-ray film results, objective evidence of pneumonitis was present in all but worker 5 who had normal spirometry and a normal chest x-ray film. In worker 6, a diagnostic bronchoscopy with bronchial brushings was performed to exclude infectious causes. Sputum cultures yielded normal bacterial flora and no fungal growth. Stains for acid-fast bacilli and silver stain for *Pneumocystis* were negative. The fluorescent antibody test and cultures for *Legionella* were negative. Blood and urine cultures were negative. An acid-fast bacilli sputum culture from worker 6 grew *Mycobacterium chelonae*, which was not considered a clinically

Table 2—Follow-up Lung Function Studies in Four Symptomatic Workers Following Cessation of Exposure to Metalworking Fluid Aerosols in Whom Radiographic Infiltrates Were Detected at Initial Presentation

| Patient | Intervention/Treatment                      | FVC      |                                          | FEV <sub>1</sub> |                                          |
|---------|---------------------------------------------|----------|------------------------------------------|------------------|------------------------------------------|
|         |                                             | Baseline | After Cessation of Exposure,<br>% Change | Baseline         | After Cessation of Exposure,<br>% Change |
| 1*      | Avoidance (1 mo)                            | 2.0      | 4.5 (125%)                               | 1.7              | 3.4 (100%)                               |
| 2       | Avoidance (2 wk)                            | 3.5      | 4.6 (31%)                                | 2.5              | 3.5 (40%)                                |
| 3       | Avoidance (2 wk)                            | 3.4      | 4.2 (24%)                                | 2.9              | 2.8 (0%)                                 |
| 6       | Avoidance (2 mo), prednisone therapy (1 mo) | 3.3      | 4.6 (39%)                                | 2.2              | 3.6 (64%)                                |

\*After 1 month of cessation of exposure to contaminated metalworking fluid, prednisone therapy was initiated.

**Table 3—Serum Precipitating Antibodies to Antigens of Eight Microbial Isolates With Colony Morphology, Characteristics, and Identification**

| Organism | No. Workers<br>With Precipitins/<br>Total No. | Morphology                     | Characteristics           | Species Identified         |
|----------|-----------------------------------------------|--------------------------------|---------------------------|----------------------------|
| 1        | 1/6                                           | Gram-negative rod              | No pigment                | <i>Pseudomonas</i> species |
| 2        | 6/6                                           | Gram-negative rod              | Fluorescein Gelatinolytic | <i>P fluorescens</i>       |
| 3        | 2/6                                           | Conidiophores                  | Black                     | <i>A niger</i>             |
| 4        | 3/6                                           | Gram-positive rod              | Acid-fast                 | <i>Rhodococcus</i> species |
| 5        | 0/6                                           | Gram-negative diplococcus      | Yellow pigment            | <i>Neisseria</i> species   |
| 6        | 0/6                                           | Gram-positive rods, endospores | Rhizoid colonies          | <i>Bacillus</i> species    |
| 7        | 2/6                                           | Gram-positive coccus           | Clumps                    | <i>S capitis</i>           |
| 8        | 2/6                                           | Gram-positive rods, endospores | Yellow/black motile       | <i>B pumilus</i>           |

significant pathogen and therefore was not treated. Five of six workers had been initially treated by their physicians with orally administered antibiotics with no apparent clinical improvement.

Symptoms improved and eventually resolved in all six workers following removal from the work area. Two of the workers were also treated with orally administered corticosteroids. As shown in Table 2, follow-up lung function studies performed in 4 workers demonstrated a mean improvement in FVC of 1.4 L (57% increase). In 2 workers (workers 1 and 6) in whom clinical improvement was apparent, Dco/V<sub>A</sub> was repeated and had improved (Table 1). Resolution of pulmonary infiltrates was documented in the three workers for whom follow-up chest radiographs were obtained. None of the workers presented with skin eruptions or infections.

#### Microbiological and Serologic Studies

Eight microbial isolates were subcultured from metalworking fluid samples. Six isolates were cultured from undialyzed fluid. Two Gram-negative rods were isolated from dialyzed fluid. The characteristics of these eight bacterial isolates are listed in Table 3. Ouchterlony gel diffusion studies were performed with eight antigen extracts prepared from the eight isolates. Precipitating antibody to 6 of the 8 microbial isolates was found in workers' sera. The most frequent precipitin responses were against the *P fluorescens* antigen extract (6 workers). In all 6 workers precipitins were

also detected to a cell-free filtrate obtained from the *P fluorescens* culture. In worker 6, precipitins were also demonstrated to antigen extracts of another nonpigmented *Pseudomonas* species. As shown in Tables 3 and 4, precipitins were detected against 4 other microbial antigens (*Aspergillus niger*, a *Rhodococcus* species; *Staphylococcus capitis* [subspecies ureolyticus], and *Bacillus pumilus*) in 2 or more workers. The levels of endotoxin in the 2 dialyzed fluid samples were 0.04 and 0.17 µg/mL, respectively.

Precipitin responses to the 6 antigens for each individual worker are listed in Table 4. Only worker 6 exhibited precipitins to all 6 antigens. Worker 4 reacted to 4 antigens; workers 1 and 3 reacted to 2 antigens; and workers 2 and 5 reacted to only 1 antigen. Repeat serum samples were obtained in 3 of the workers (workers 2, 3, and 6) at 19 months after diagnosis and after cessation of exposure to metalworking fluid aerosols. At follow-up, microbial precipitins persisted in workers 3 and 6 but were no longer present in worker 2. Precipitin responses were not detected to pseudomonad antigens among the nine nonexposed control subjects.

#### DISCUSSION

Exposure to microbial organisms in the workplace has been associated with a variety of occupational respiratory disorders including hypersensitivity pneumonitis. Hypersensitivity pneumonitis is an immune-mediated disorder which is induced by specific cellu-

**Table 4—Serum Precipitin Responses to Six Antigens in Six Workers Exposed to Metalworking Fluid**

| Organism                    | Workers |   |   |   |   |   | Control Subjects<br>(n=9) |
|-----------------------------|---------|---|---|---|---|---|---------------------------|
|                             | 1       | 2 | 3 | 4 | 5 | 6 |                           |
| <i>Pseudomonas</i> species* | —       | — | — | — | — | + | —                         |
| <i>P fluorescens</i> *      | +       | + | + | + | + | + | —                         |
| <i>A niger</i>              | —       | — | — | + | — | + | —                         |
| <i>Rhodococcus</i> species  | +       | — | + | — | — | + | —                         |
| <i>S capitis</i>            | —       | — | — | + | — | + | + <sup>†</sup>            |
| <i>B pumilus</i>            | —       | — | — | + | — | + | + <sup>†</sup>            |

\*Cultured from dialyzed metalworking fluid.

<sup>†</sup>Positive in only 1 of 9 control subjects with no exposure to metalworking fluid.

lar immune reactions to bacterial or fungal antigens contained in organic dusts.<sup>4,5</sup> Among these, thermophilic bacteria and mold spore species (*ie*, *Aspergillus*, *Cladosporium*, *Penicillium*, and *Alternaria* genera) have been well-documented as important causative antigens.<sup>6</sup> Dyspnea, cough, and fever beginning within hours after inhalation of antigen and resolution following cessation of exposure are characteristic symptoms of acute hypersensitivity pneumonitis. Other features include pulmonary interstitial infiltrates, restrictive ventilatory defects, and diffusion abnormalities. Serum immune precipitins to causative antigens are detectable in affected patients as well as in similarly exposed asymptomatic individuals.<sup>4,6</sup>

Rarely, nonthermophilic bacteria have been identified as causative antigens. One such organism, *B subtilis*, was linked to a familial outbreak of hypersensitivity pneumonitis caused by exposure to microbial dust dispersed during home reconstruction.<sup>7</sup> Flaherty et al<sup>8</sup> identified a group of workers who presented with hypersensitivity pneumonitis or humidifier lung disease associated with exposure to a biomass of bacteria in a water spray air humidification system. Symptomatic workers exhibited serum precipitins to endotoxin purified from a *Cytophaga* bacterial species which was subcultured from the humidifier water. *Klebsiella oxytoca* and *Bacillus cereus* also have been identified as causes of humidifier lung disease.<sup>9,10</sup>

In this paper, we have described an endemic case of acute hypersensitivity pneumonitis in a small group of machine operators who had common exposure to an aerosolized, water-based metalworking fluid at work. In addition to serum precipitins, the diagnosis of hypersensitivity pneumonitis was established based on work-related respiratory and systemic symptoms, radiographic pulmonary infiltrates, abnormalities in diffusing capacity, all of which improved following cessation of work exposure. Thus, this collection of clinical findings was sufficient to establish a clinical diagnosis of hypersensitivity pneumonitis in each of five workers based on previously recommended diagnostic guidelines.<sup>4</sup> In this study, it was not possible to obtain lung biopsies or cellular studies of bronchoalveolar lavage fluid which might have provided pathologic data further supporting this diagnosis. However, Richerson et al<sup>4</sup> has published diagnostic guidelines which advise that bronchoalveolar lavage and lung biopsy studies are not absolutely necessary if other essential clinical diagnostic criteria are satisfied.

In the differential diagnosis, infectious pneumonitides (*eg*, *Legionella*) as well as toxic inflammatory responses caused by inhalation of microbial products or chemicals were considered. Because negative culture studies were documented in one worker who underwent diagnostic bronchoalveolar lavage and other workers failed to respond to antibiotics, an infectious

etiology was considered unlikely.

Respiratory disorders related to exposure to contaminated humidifier water have been recognized. Rylander and Haglund<sup>11</sup> described an outbreak of "humidifier disease" in a print shop where 20% of workers reported work-related fever, chills, dyspnea, and cough. This illness was traced to high levels of aerosolized endotoxin produced by *Pseudomonas* bacteria in humidifier water. "Humidifier fever" is a term which has been used to describe this syndrome, but it must be distinguished from humidifier lung disease which is a form of hypersensitivity pneumonitis.<sup>12</sup> The organic dust toxic syndrome is very similar to humidifier fever in that it occurs in agricultural settings among grain workers exposed to endotoxins or mycotoxins in grain dust (*ie*, grain fever).<sup>13,14</sup> In sharp contrast to hypersensitivity pneumonitis, humidifier fever and the organic dust toxic syndrome are toxic, nonimmunologic reactions which are often associated with relatively benign clinical courses and characterized by the absence of pulmonary infiltrative disease. Furthermore, the concentrations of endotoxin measured in metalworking fluid samples in the present study (0.04, 0.17 µg/mL) were 1 to 2 logs lower than the concentration reported (3.91 µg/mL) in the aforementioned study of Rylander and Haglund.<sup>11</sup>

Friend and coworkers<sup>15</sup> previously have described hypersensitivity pneumonitis among workers in a large stationary factory. The point source of this illness was traced to water which was used to cool vacuum pumps and air compressors that had been found to be contaminated with Gram-negative bacteria and a variety of fungi. Serum precipitins to the contaminated water but not to extracts of cultured microorganisms were demonstrated. Restrictive impairment in lung function and diffusion abnormalities were demonstrated in some workers; the illness was confirmed by decreases in FVC and Dco after controlled challenge with the water extract. The latter disorder resembles clinical features in our group with the exception that their workers did not exhibit pulmonary infiltrates.

A diverse array of microorganisms have been cultured from metalworking fluids.<sup>1,2,16</sup> In the present study, culturing of metalworking fluid yielded a number of distinct isolates from which antigens were extracted. Interestingly, Gram-negative *P fluorescens* isolates could be cultured from dialyzed but not from undialyzed fluid which may have been due to the presence of biocidal small molecular weight chemicals present in the fluid prior to dialysis. Despite the fact that there were no data which quantified ambient microbial exposure in this study, the presence of *Pseudomonas* precipitins in all six workers and their absence in nonexposed control subjects suggested that there was sufficient exposure to *Pseudomonas* via metalworking fluid aerosols in the workplace to elicit

specific humoral responses.

Precipitins can be demonstrated in 10 to 50% of asymptomatic individuals with adequate exposure to antigens known to elicit hypersensitivity pneumonitis.<sup>4-6</sup> Thus, precipitin responses observed in these workers could merely reflect exposure to microbes rather than represent specific markers of immunologic lung disease. Unfortunately, sera of similarly exposed asymptomatic workers in the plant could not be obtained. Thus, it is not possible to make inferences or conclusions with regard to the pathologic significance of precipitin responses observed in symptomatic employees. Nevertheless, the association of *Pseudomonas* precipitins with characteristic clinical findings of hypersensitivity pneumonitis was striking.

In conclusion, we have described an outbreak of hypersensitivity pneumonitis which occurred in a novel occupational setting. The finding of serum precipitins in all six symptomatic workers suggested that this disorder was associated with immunologic responses to aerosolized microbial antigens from metalworking fluid. Further epidemiologic studies are required to determine the exact prevalence of this condition in industries where metalworking fluids are utilized. Environmental monitoring and medical surveillance studies are needed to further define exposure conditions and the relative risks of hypersensitivity pneumonitis in workers exposed to metalworking fluids so that specific preventative measures can be initiated.

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