

Silicosis in Dental Laboratory Technicians — Five States, 1994–2000

Silicosis is a debilitating, sometimes fatal, yet preventable occupational lung disease caused by inhaling respirable crystalline silica dust. Although crystalline silica exposure and silicosis have been associated historically with work in mining, quarrying, sandblasting, masonry, founding, and ceramics, certain materials and processes used in dental laboratories also place technicians at risk for silicosis (1–3). During 1994–2000, occupational disease surveillance programs in five states identified nine confirmed cases of silicosis among persons who worked in dental laboratories; four persons resided in Michigan, two in New Jersey, and one each in Massachusetts, New York, and Ohio. This report describes three of the cases and underscores the need for employers of dental laboratory technicians to ensure appropriate control of worker exposure to crystalline silica.

Silicosis cases in Michigan, New Jersey, and Ohio were identified through the Sentinel Event Notification Systems for Occupational Risks (SENSOR) surveillance program (4). The case in New York was identified through the state's Occupational Lung Disease Registry, and the case in Massachusetts was identified through the state's Occupational Disease and Injury Surveillance System; both use the SENSOR model. Although cases were identified during 1994–2000, diagnoses preceded state surveillance system identification. State surveillance programs identify suspected cases of silicosis through various sources, including hospital discharge data, death certificate data, workers' compensation reports, and physician reports. Cases are confirmed on the basis of the silicosis surveillance case definition (Box 1) adopted by these state surveillance programs and information from interviews, medical record review, or chest radiograph classification by a National Institute for Occupational Safety and Health (NIOSH)–certified B-reader*.

Case Reports

Case 1. In November 1992, a man aged 65 years in New Jersey died 4 days after being admitted to a hospital with abdominal pain, nausea, and vomiting. The cause of death was shock, pancreatitis, and respiratory failure. At age 48 years, silicosis had been diagnosed in the patient. He had worked for 46 years in dental laboratories, 27 years as an owner, and had performed routine dental laboratory operations. He reportedly was exposed to dust, cobalt, and chemicals, and

*NIOSH B-reader certification is granted to physicians who demonstrate proficiency in classifying chest radiographs for pneumoconioses by using the International Labour Office Classification System (5).

BOX 1. Silicosis surveillance guidelines for state health departments

Reporting guidelines

State health departments should encourage physicians, including radiologists, pathologists, and other health-care professionals to report all diagnosed or suspected cases of silicosis. These reports should include persons with one or more of the following:

- a physician's provisional or working diagnosis of silicosis
- a chest radiograph interpreted as consistent with silicosis
- pathologic findings consistent with silicosis.

State health departments should collect appropriate clinical, epidemiologic, and workplace information on persons reported with silicosis to set priorities for workplace investigations.

Surveillance case definition

- History of occupational exposure to airborne silica dust* and one or both of the following:
 - chest radiograph or other imaging technique interpreted as consistent with silicosis[†]
 - pathologic findings characteristic of silicosis[§].

Source: National Institute for Occupational Safety and Health.

* The induction period between initial silica exposure and development of radiographically detectable nodular silicosis usually is ≥ 10 years. Shorter induction periods are associated with heavy exposures, and acute silicosis might develop within months after massive silica exposure.

[†] Cases can be classified as nodular or acute. Common radiographic findings of nodular silicosis include multiple, bilateral, rounded opacities in the upper lung zones; other patterns have been described. Because patients might have mixed dust exposure, irregular opacities might be present or even predominant. To be considered consistent with silicosis, radiographs of nodular silicosis classified by National Institute for Occupational Safety and Health–certified B-readers should have small opacity profusion categories of $\geq 1/0$ by the International Labour Office classification system. If the largest opacity is > 1 cm in diameter, progressive massive fibrosis (i.e., “complicated” silicosis) is present. A bilateral alveolar filling pattern is characteristic of acute silicosis and might be followed by rapid development of bilateral small or large nodular opacities.

[§] Characteristic lung tissue pathology in nodular silicosis consists of fibrotic nodules with concentric “onion-skin” arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent particles observed under polarized light. In acute silicosis, microscopic pathology shows a periodic acid-Schiff–positive alveolar exudate (i.e., alveolar lipoproteinosis) and a cellular infiltrate in the alveolar walls.

never wore a respirator. A B-reader classification of a chest radiograph taken 1 day before his death revealed small, rounded opacities involving the upper zones, with a profusion category of 1/2, consistent with silicosis.

Case 2. In September 1995, silicosis was diagnosed in a man aged 66 years in New York. He had worked as a dental technician for 30 years and had been exposed to various min-

"The wisest mind has something yet to learn."

George Santayana

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eral and metallic dusts. A chest radiograph taken at a local hospital revealed category 2/3 profusion of small, nodular opacities in the middle and lower lung zones, consistent with silicosis. Asbestosis and berylliosis[†] also were diagnosed.

Case 3. In November 1992, a man aged 67 years in New Jersey died 3 days after being admitted to a hospital with progressive confusion. The cause of death was urosepsis caused by scleroderma, anemia, and renal failure. He had worked for 28 years in dental laboratories, 24 years as an owner. He reportedly was exposed to various dusts, performed sandblasting in dental laboratories, and never wore a respirator. A chest radiograph taken 10 weeks before his death revealed small, rounded opacities involving the upper zones, with a profusion category of 3/2, consistent with silicosis.

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Editorial Note: The findings in this report suggest that dental laboratory technicians might be at risk for silicosis as a result of uncontrolled exposure to airborne crystalline silica dust. For the patients described in this report, the only identified source of crystalline silica exposure was their work as dental technicians. Exposure to respirable crystalline silica in dental laboratories can occur during procedures that generate airborne dust (e.g., mixing powders, removing castings from molds, grinding and polishing castings and porcelain, and using silica sand for abrasive blasting). The proportion of crystalline silica in mold and porcelain materials, by weight, can range up to 70%. A study of dental technicians in South Korea (6) that described materials and processes similar to those used in the United States found exposures during polishing operations that exceeded the NIOSH recommended exposure limit of 0.05 mg/m³ (7).

The United States has approximately 14,000 dental laboratories (8), and approximately 6,400 dental laboratory technicians are certified by the National Association of Dental Laboratories (NADL) (NADL, unpublished data, 2004). Because dental laboratories are not registered or licensed, the actual number of dental laboratory technicians is unknown.

The findings in this report are subject to at least three limitations. First, data for some variables (e.g., month or year of diagnosis and job history) were not available for all cases. Second, the risk for exposure to crystalline silica could not be

[†] Asbestosis is caused by exposure to asbestos, once commonly used in dental laboratories; berylliosis is caused by exposure to the metal beryllium, a component of some alloys used in dental prostheses.

quantified because data on exposure levels among dental laboratory technicians are limited. Finally, silicosis case ascertainment is not complete (9).

Occupational diseases such as silicosis frequently are not recognized or reported by physicians (10). Health-care providers and employers should be educated on the importance of screening and reporting silicosis to their state-based surveillance systems.

Methods to control exposure to silica are well established. Through industrywide educational outreach, state-based surveillance programs can alert employers to a potential occupational hazard and provide guidance for controlling worker exposure. The Occupational Safety and Health Administration (OSHA) requires employers to identify occupational health hazards and control them by instituting engineering and work-practice controls, issuing personal protective equipment (PPE), and ensuring that PPE is working and used properly. As part of an effective OSHA-compliant hazard communication program, dental technicians should be trained in the hazards of crystalline silica exposure and the methods to control exposure (Box 2). Guidance for controlling silica exposure in dental laboratory settings is available at <http://www.state.nj.us/health/eoh/survweb>. Additional information about silica and silicosis is available at <http://www.cdc.gov/niosh/topics/silica>.

BOX 2. Exposure-control methods for crystalline silica in dental laboratories

- Substitute nonsilica-containing materials for silica-containing materials (e.g., aluminum oxide as an abrasive blasting media instead of silica sand).
- Isolate the source of silica exposure from the dental technician (e.g., perform divestment of castings while materials are immersed in water).
- Remove dust at its point of generation by using engineering controls (e.g., local exhaust ventilation system with dust collector).
- Incorporate work and housekeeping practices that minimize the release of dust into the workroom air (e.g., use high-efficiency particulate aerosol-filtered vacuums for clean-up instead of dry sweeping).
- Use respiratory protection devices (e.g., half-mask air-purifying respirator fitted with type N-100 filters).

References

1. Choudat D. Occupational lung disease among dental technicians. *Tuber Lung Dis* 1994;75:99–104.
2. Rom WN, Lockey JE, Lee JS, et al. Pneumoconiosis and exposures of dental laboratory technicians. *Am J Public Health* 1984;74:1252–7.
3. CDC. Silicosis surveillance—Michigan, New Jersey, Ohio, and Wisconsin, 1987–1990. In: *CDC Surveillance Summaries* (November 19). *MMWR* 1993;42(No. SS-5).

4. Baker EL. Sentinel Event Notification Systems for Occupational Risks (SENSOR): the concept. *Am J Public Health* 1989;79(suppl):18–20.
5. International Labour Office. *Guidelines for the Use of the ILO International Classification of Radiographs of Pneumoconioses*, 2000 ed. Geneva, Switzerland: International Labour Office, 2002 (Occupational Safety and Health Series, No. 22, rev. 2000).
6. Kim TS, Kim HA, Heo Y, Park Y, Park CY, Roh YM. Level of silica in the respirable dust inhaled by dental technicians with demonstration of respirable symptoms. *Ind Health* 2002;40:260–5.
7. National Institute for Occupational Safety and Health. *NIOSH Hazard Review: Health Effects of Occupational Exposure to Respirable Crystalline Silica*, 2002. Cincinnati, Ohio: U.S. Department of Health and Human Services, CDC, National Institute for Occupational Safety and Health, 2002; DHHS publication no. (NIOSH)2002-129.
8. Marketplace [CD-ROM database]. New York, New York: Dun & Bradstreet, April 2002.
9. Rosenman KD, Reilly MJ, Henneberger PK. Estimating the total number of newly-recognized silicosis cases in the United States. *Am J Ind Med* 2003;44:141–7.
10. CDC. Mandatory reporting of occupational diseases by clinicians. *MMWR* 1990;39(No. RR-9).

Brief Report

Azithromycin Treatment Failures in Syphilis Infections — San Francisco, California, 2002–2003

The San Francisco Department of Public Health (SFDPH) is investigating several clinical failures in syphilis patients treated with the macrolide antibiotic azithromycin. This report describes the use of azithromycin for syphilis treatment, recent treatment failures in San Francisco, and CDC recommendations for syphilis treatment. Clinicians should exercise caution in using azithromycin for treating incubating syphilis or syphilis infection until the risk and mechanism of failure are better understood.

Syphilis has been increasing in the United States since 2000 and is of particular concern in San Francisco, which, in 2002, had one of the highest rates of primary and secondary syphilis in the United States. To facilitate treatment of early syphilis patients and their sexual contacts, certain disease-control programs have administered azithromycin as a single oral regimen. Several small studies have documented the efficacy of a single oral dose of azithromycin in the treatment of incubating and early syphilis infection in patients who were not infected with the human immunodeficiency virus (HIV) (1). In addition, the oral dose is more convenient to administer than intramuscular benzathine penicillin, CDC's recommended treatment for sexually transmitted diseases (2).

In April 2003, SFDPH became aware of an azithromycin failure in the treatment of primary syphilis in one patient and subsequently collected case information on this and seven other apparent treatment failures occurring during September 2002–July 2003. The median patient age was 34 years (range: 23–

Use of Carbon Monoxide Alarms to Prevent Poisonings During a Power Outage — North Carolina, December 2002

Each year in the United States, approximately 500 persons die from unintentional carbon monoxide (CO) poisoning (1), often during electric power outages caused by severe storms (2–4). Use of residential CO alarms has been recommended to reduce the incidence of CO poisoning (5,6). In September 2000, Mecklenburg County, North Carolina (2002 population: 722,367), adopted a public health ordinance requiring a CO alarm in the majority of residences; all-electric residences without attached garages (35.4% of all homes) were exempt. The ordinance also permitted use of alarms without battery back-up. On December 4, 2002, an ice storm caused 78.9% of county households to lose power. During the next 9 days, 124 cases of symptomatic CO poisoning were reported. To characterize these poisonings and the effectiveness of the CO alarm ordinance, local emergency physicians, fire department authorities, and CDC conducted an investigation. This report summarizes the results of that investigation, which determined that 96.2% of the severe poisonings occurred in homes with no reported functioning CO alarm. As a result of these findings, on October 8, 2003, Mecklenburg County officials amended the ordinance to require alarms with battery back-ups in all residences (7). Officials in other communities should consider enacting such alarm ordinances to prevent CO poisonings.

Data were extracted from 1) medical records of patients with CO poisoning at all hospitals serving Mecklenburg County, 2) emergency medical service (EMS) and fire department reports, and 3) readings from handheld CO meters operated by members of the Charlotte Fire Department (Figure 1). Cases were included if they occurred from the time electric power was lost on December 4 until full restoration on December 13. Confirmed CO exposure was defined as an elevated CO level in the ambient air of a person's home (>50 ppm) or in a person's blood (carboxyhemoglobin level >10% in smokers

FIGURE 1. A firefighter uses a portable meter to measure the carbon monoxide (CO) level after CO exposure caused by a generator forced evacuation of an apartment building — Charlotte, North Carolina, 2003



Photo/Charlotte Fire Department

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