A Cluster of Acinetobacter Pneumonia in Foundry Workers

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In a 3-month period, three men who had worked for 5 to 19 years as welders or grinders of steel castings in a foundry acquired pneumonia caused by *Acinetobacter calcoaceticus* variety *anitratus* serotype 7J. Two of the men died, and postmortem examination showed mixed-dust pneumoconiosis with iron particles in the lungs. *A. calcoaceticus* variety *anitratus* serotype 7J was isolated from the air in the foundry but the source was not found. The prevalence of antibody titers of 64 or greater to the 7J strain was significantly higher among foundry workers (15%) than among community controls (2%) ($p < 0.01$). Sampling showed that the concentrations of total and metallic particles (especially iron) and of free silica in air inhaled by welders and grinders at the foundry frequently exceeded acceptable levels. These findings suggest that chronic exposure to such particles may increase susceptibility to infection by this organism, which rarely affects healthy people.

SPORADIC CASES of non-nosocomial infection caused by *Acinetobacter calcoaceticus* variety *anitratus* have been infrequently reported in the medical literature (1-12), and there have been no reports of outbreaks or clusters of infection acquired outside the hospital. In contrast, nosocomial infections caused by *A. calcoaceticus* variety *anitratus* occur both sporadically and in clusters (13, 14) and are believed to be opportunistic infections primarily affecting patients who are elderly, debilitated, or exposed to predisposing surgical or medical procedures or therapy (14). The few reported cases of infection outside the hospital usually have occurred in persons with chronic or debilitating illnesses such as alcoholism or chronic renal failure, although cases have occurred in persons without underlying illnesses (1).

We investigated a cluster of cases of pneumonia and sepsis caused by *A. calcoaceticus* variety *anitratus* in three workers in a steel-casting foundry (Foundry A) in Hartford, Connecticut, from April to June 1979. Foundry A, which produces steel castings, employs 116 manual laborers and 24 supervisors. Our evidence indicates that chronic exposure to metallic dust in the foundry may have predisposed workers to infection and disease caused by this organism, which seems to have spread through the air.

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Materials and Methods

SURVEY OF WORKERS

In May 1979, workers from Foundry A filled out a questionnaire on basic demographic, medical, and job-related information. Serum specimens were obtained from the workers. We also questioned selected supervisors and manual laborers about the work patterns of welders and grinders of castings. In September 1979, we obtained demographic and job-related information from workers at a control foundry (Foundry B) by personal interviews using a standardized questionnaire. We also obtained serum specimens from these workers. Foundry B, 100 km south of Hartford, employs 100 persons and also produces steel castings.

In May and June 1979, we obtained throat and right antecubital skin cultures from available workers at Foundry A. For the workers tested in May, premoistened calcium alginate swabs were applied to the throat and skin and then streaked onto both blood and MacConkey agars (BBL Microbiology Systems, Cockeysville, Maryland) and incubated aerobically at 35 °C for 48 h. Suspicious colonies from these and subsequent cultures were streaked on triple sugar iron slants and incubated at 35 °C for 18 h. Colonies on triple sugar iron slants with typical reactions (alkaline slant and negative H_2S butt) for *A. calcoaceticus* variety *anitratus* were further identified. For the workers tested in June, cotton swabs were applied to the throat and skin and then inoculated into two preparations of brain-heart (calf brains, beef heart) infusion broth (Difco Laboratories, Detroit, Michigan), one with and one without chloramphenicol, 8 μg/mL. These infusions were incubated at 35 °C. After 18 h, the broths were subcultured onto MacConkey agar and again incubated at 35 °C for 18 h.

A reader certified by the National Institute for Occupational

Safety and Health, Centers for Disease Control, reviewed posteroanterior chest roentgenograms of workers at Foundry A taken in 1979 for evidence of pneumoconiosis. A positive interpretation was category 1/0 profusion or greater, based on the 1971 international standard classification (15).

ENVIRONMENTAL SAMPLING

Cultures of the foundry air, water, and environmental surfaces were obtained from May to July 1979. We used a microban or Ross air sampler for 5- to 15-minute intervals to collect samples of air directly onto blood or MacConkey agar. After incubation at 35 °C for 24 h, representative colonies were streaked onto MacConkey agar, incubated at 35 °C for 24 h, and further identified. We cultured the water samples in three ways. 1) One- to 5-mL aliquots of water were directly added to Trypticase soy broth (BBL Microbiology Systems), incubated overnight at 35 °C for 24 to 48 h. 2) Fifty- to 100-mL samples of water were collected in sterile plastic bottles containing sodium thiosulfate. These samples were passed through 0.45-μm filters (Millipore Corporation, Bedford, Massachussetts) and the filters were placed on MacConkey agar and incubated at 35 °C for 48 h. 3) Fifty- to 100-mL water samples obtained in sterile plastic containers were filtered through 0.45-μm filters and the divided filters were placed into two preparations of brain-heart infusion broth, one with and one without chloramphenicol, 8 μg/mL. After incubation at 35 °C for 18 h, aliquots of brain-heart broth with chloramphenicol were inoculated onto MacConkey agar and incubated at 35 °C for 18 h. After incubation at 35 °C for 72 h, aliquots of broth without chloramphenicol were subcultured to broths with cephalothin (20 μg/mL), nitrofurantoin (25 μg/mL), or chloramphenicol (8 μg/mL). Further processing was carried out as described above for the throat and skin samples. Environmental surfaces were cultured using sterile cotton swabs that were placed in Trypticase soy broth and incubated overnight at 35 °C. Aliquots of this broth were inoculated onto blood and MacConkey agar and incubated at 35 °C for 48 h.

Air was sampled on 2 and 3 July 1979 at Foundry A. Personal breathing zone samples for respirable silica were taken at a sampling rate of 1.7 L/min with Model G MSA pumps (Mine Safety Appliance, Pittsburgh, Pennsylvania) using 10-mm cyclones. All samples were collected on preweighed FWSB filters (Millipore Corporation) to measure the total weight of respirable particles. The samples were analyzed for quartz and cristobalite according to previously described methods (16). Samples for total particulates and metallic dusts (including nickel, chromium, iron, lead, manganese, and copper) were collected on preweighed DM 800 filters (Millipore Corporation) and analyzed for metals as previously described (17). Concentrations are expressed as milligrams of material per cubic meter of air (mg/m^3) and exposure limits are those recommended by the National Institute for Occupational Safety and Health.

PROCESSING OF CULTURES

We used the API-20E (Analytab, Inc., Plainview, New York) system to identify isolates of *A. calcoaceticus* variety *anitratus* obtained from human and environmental specimens. Antibiotic susceptibility patterns of these isolates were determined by the method of Thornsberry and associates (18). Serotypes of the isolates were determined as previously described by direct fluorescent antibody examination (19).

SEROLOGY

We obtained serum specimens from workers at Foundry A and Foundry B and from another group of controls who were selected from consecutive persons who had requested premarital VDRL testing in January and February 1980 at the state health department in Hartford. To assay these samples, we used the indirect fluorescent antibody method modified in the following way from that described for *Legionella pneumophila* (20). The four antigens used were whole, heat-killed *A. calcoaceticus* variety *anitratus* that had been grown on blood agar: a pooled preparation composed of the three strains of 7J serotype that had been isolated from blood of patients and separate preparations of three environmental isolates (7J, 1F, 10F serotypes) from Foundry A. Before testing, all serum samples were absorbed with blood-agar-grown organisms of the 1F serotype to remove non-type-specific seroreactivity. A 1-mL milky suspension of organisms in sterile water was centrifuged, the supernatant was decanted, and the packed cells were overlayed with 1 mL of test serum, which was then mixed with a Pasteur pipette and refrigerated overnight at 5 °C. The serum was decanted and the procedure repeated. Twofold dilutions of test serum from 1/64 to 1/1024 were assayed and read blindly. All titers are expressed as reciprocals.

POSTMORTEM TISSUE

Postmortem lung specimens were reviewed by F.H.Y. Green, M.D., Pathology Section, Laboratory Investigations Branch, Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health. Particle analysis of pulmonary dust deposits was done by back-scattered electron imaging and energy dispersion X-ray analysis with a scanning electron microscope as previously described (21).

Case Reports

PATIENT 1

A 54-year-old man who had been a castings' grinder and chipper at Foundry A for 5 years became ill on 4 April 1979, with shortness of breath and cough that produced red-brown sputum. A chest roentgenogram taken the same day showed a right middle and lower lobe infiltrate; penicillin was administered intramuscularly and orally. On 9 April he was hospitalized because of increasing fever, diaphoresis, and left pleuritic chest pain. The patient said he did not drink alcohol and had never smoked cigarettes. In 1974 he had been hospitalized for pneumonia. When he was admitted to the hospital in 1979, he was alert but tachypneic and had cyanotic nail beds. Bloods pressure was 149/80 mm Hg; pulse, 160; respiratory rate, 48; and temperature, 38.9 °C. Bilateral rhonchi and crepitations were noted. A chest roentgenogram showed diffuse bilateral infiltrates suggestive of pneumonia, as well as q opacities, profusion 1/1, involving both upper and mid-lung fields and the lower left zone (15). The leukocyte count was $5300/mm^3$ with 33% polymorphonuclear leukocytes and 57% band forms. Arterial blood obtained while the patient was breathing room air showed Po_2, 28 mm Hg; Pco_2, 36 mm Hg; and pH, 7.42. He required immediate mechanical ventilation because of acute respiratory failure and was given penicillin G, 1 million units intravenously every 4 h. Within 24 hours of admission he had an episode of bradycardia and hypotension associated with endotracheal intubation and died from irreversible cardiac arrest. Antemortem sputum and blood and postmortem lung tissue grew *A. calcoaceticus* variety *anitratus*. Examination of postmortem lung tissue showed widespread bronchopneumonia with consolidation. Alveoli were filled with fibrin, erythrocytes, polymorphonuclear leukocytes, numerous macrophages containing hemosiderin and dust, and large numbers of gram-negative bacilli. Perivascular connective tissue and fibrotic nodules contained large quantities of dust. Marked perivascular reticulin fibrosis was present. A dense, haphazardly arranged, collagenous stroma contained 1- to 4-mm nodules composed of dust-laden macrophages. A single fibrotic nodule with whirling of collagenous fibers was seen.

Comment: Overwhelming *Acinetobacter* pneumonia in a reasonably healthy person is distinctly unusual. Chest radiograph and postmortem examination showed evidence of mixed-dust pneumoconiosis with one nodule suggestive of silicosis.

PATIENT 2

A 63-year-old man who had been a castings' grinder and chipper at Foundry A for 13 years became ill on 30 April 1979, with chills, fever, and cough. On 3 May he was hospitalized when he developed left pleuritic chest pain and cough that produced blood-streaked sputum. He said he smoked 1½ packs of

Table 1. Antibiotic Susceptibility Patterns of Human and Environmental Isolates of *Acinetobacter calcoaceticus* variety *anitratus**

Antibiotic	Source of Isolation and Serotype						
	Patient 1 (7J serotype)	Patient 2 (7J serotype)	Patient 3 (7J serotype)	Cafeteria Air Sample (7J serotype)	Recirculating Furnace Water (10F serotype)	Crosswalk Air Sample (1F serotype)	Lavatory Swab (3F serotype)
Ampicillin	32	32	16	32	32	32	≥ 32
Cephalothin	≥ 32	≥ 32	≥ 32	≥ 32	≥ 32	≥ 32	≥ 32
Carbenicillin	8	8	8	8	32	32	32
Chloramphenicol	≥ 32	≥ 32	≥ 32	≥ 32	≥ 32	≥ 32	≥ 32
Gentamicin	≤ 0.06	≤ 0.06	≤ 0.06	≤ 0.06	0.25	0.25	2
Tobramycin	≤ 0.06	0.125	0.125	≤ 0.06	0.5	1	1
Amikacin	1	1	1	1	2	2	2

* Minimal inhibitory concentrations in μg/mL.

cigarettes per day, drank 1 pint of liquor per day, and had hypertension requiring medication. When he was admitted, his blood pressure was 190/110 mm Hg; pulse, 112; respiratory rate, 20; and temperature, 38.4 °C. Crepitations were present in the left side of the chest and the liver was enlarged. A chest roentgenogram showed an infiltrate in the lingular segment of the left upper lobe. The leukocyte count was 14 500/mm^3 with 65% polymorphonuclear leukocytes and 29% band forms. Serum concentrations of liver enzymes were normal. Arterial blood obtained while the patient breathed room air showed Po_2, 69 mm Hg; Pco_2, 34 mm Hg; and pH, 7.42. Penicillin G, 600 000 units, was given intravenously every 4 h. Within 24 hours, infiltrates were throughout the patient's left lung and he developed respiratory failure requiring mechanical ventilation. Gentamicin therapy (120 g intravenously every 8 h) was begun, and the penicillin dosage was increased to 1 million units intravenously every 4 h. On 5 May cultures of blood and sputum grew *A. calcoaceticus* variety *anitratus*; the penicillin was replaced with 5 g of carbenicillin intravenously every 4 h. On the same day, the patient's leukocyte count fell to 1100/mm^3, his left lung became densely consolidated, and he had an episode of bradycardia and hypotension. On 7 May infiltrates in the left lung had decreased and mechanical ventilation was discontinued. On 20 May the patient became afebrile, and on 26 May gentamicin and carbenicillin treatment was discontinued. He was discharged from the hospital on 28 May.

Comment: Pneumonia rapidly progressed during therapy with penicillin but responded to the combination of gentamicin and carbenicillin, drugs to which his *Acinetobacter* isolate was sensitive (Table 1).

PATIENT 3

A 56-year-old man who had been a welder at Foundry A for 19 years became ill on 11 June 1979. Weakness, dyspnea, and cough that produced bloody sputum led to his hospitalization on 13 June. He said that he had never smoked cigarettes and that he drank alcoholic beverages twice weekly. He had been hospitalized in 1977 for pneumonia. When he was admitted to the hospital in 1979, his vital signs were blood pressure, 90/54 mm Hg; pulse, 130; respiratory rate, 44; and temperature, 38.9 °C. Bilateral lung crepitations were heard. A chest roentgenogram showed a left lower lobe infiltrate and a left pleural effusion. The leukocyte count was 1900/mm^3 with 4% polymorphonuclear leukocytes, 4% band forms, 18% metamyelocytes, 6% myelocytes, 66% lymphocytes, and 2% monocytes. Blood urea nitrogen was 42 mg/dL, and serum creatinine was 4.9 mg/dL. Serum concentrations of liver enzymes were elevated, bilirubin was 2.5 mg/dL, and prothrombin time was prolonged. Arterial blood obtained while the patient breathed room air showed Po_2, 44 mm Hg; Pco_2, 23 mm Hg; and pH, 7.43. He required immediate mechanical ventilation and was given ticarcillin, 3 g intravenously at 4-h interval for two doses, followed by 4 g every 8 h; and tobramycin, 120 mg intravenously once, followed by periodic administration to maintain therapeutic blood levels. Within 24 hours, the leukocyte count fell to 1000/mm^3, and his temperature was 41.1 °C. Septic shock was complicated by metabolic acidosis and renal failure requiring peritoneal dialysis. Cultures of blood and sputum obtained from a transtracheal aspirate grew *A. calcoaceticus* variety *anitratus*. In spite of granulocyte transfusions, cardiac pacemaker insertion on 17 June to control arrhythmias, and therapeutic bronchoscopies on 18 and 20 June, the patient's condition worsened and the pulmonary infiltrates became bilateral. He died on 20 June with massive hemoptysis. Postmortem examination of lung tissue showed widespread bronchopneumonic consolidation with multiple abscesses, primarily in the right lung, and organizing pneumonia in the left lung. Focal areas of suppurative necrosis containing a branching fungus and gram-negative bacilli were seen in the right lung. One of these areas contained a cavitating abscess adjacent to and involving the wall of a bronchus and a pulmonary artery, with fresh hemorrhage noted in the bronchus. The lungs showed widespread hemosiderin and dust deposition. The dust was brown and was located in alveolar macrophages and in the perivascular and subpleural connective tissues. Mild perivascular reticulin fibrosis was associated with the deposited dust. No large nodules were seen. Cultures of postmortem lung tissue grew *Aspergillus* species.

Comment: This patient, like Patient 1, had had another episode of pneumonia in the previous 5 years. His acinetobacter pneumonia and septicemia progressed despite appropriate antibiotic therapy. Postmortem examination showed mixed-dust pneumoconiosis. The associated aspergillus infection may have complicated therapy.

Results

EPIDEMIOLOGIC FINDINGS

Review of medical records for personnel in Foundry A and illness questionnaires found no other persons with pneumonia in the January to June 1979 period. The only common exposure of the three workers with acinetobacter pneumonia was in the foundry, as they did not associate or visit the same places outside work. All three worked within 5 to 10 m of each other in one section of the finishing department of the foundry, a 30-by-16-m area where welding (by arc-air and acetylene gas) and chipping and grinding of large castings were done. Two men were grinders, who used air-driven instruments to grind, chip, and polish castings and removed debris from castings with compressed air. The other man was a gas welder who used acetylene as the gas source. Face masks with disposable filters were provided at the foundry but there was no training program in respirator use, and most workers, including the three patients, usually did not wear respirators.

CULTURES

Acinetobacter calcoaceticus variety *anitratus* was isolated from sputum and blood of the three men with pneumonia. The blood isolates were of the 7J serotype. Antibiotic susceptibility patterns were similar for all isolates of 7J serotype (Table 1). One of 107 arm and none of 106 throat cultures obtained in May 1979 was positive for *A. calcoaceticus* variety *anitratus*; the one positive culture was serotype 1F. Two of 27 arm and none of 27 throat cultures obtained in June 1979 were positive for *A. calcoaceticus* variety *anitratus*; one was serotype 3F and the other nontypable.

One of 27 samples of Foundry A environmental surfaces was positive for *A. calcoaceticus* variety *anitratus* (serotype 3F); this sample was a swab of the lavatory sink in the men's dressing room. One of 20 water samples (from a recirculating furnace) was positive for the organism (serotype 10F). Three of 24 air samples were positive, including organisms of 7J serotype from the foundry cafeteria, and 1F serotype from areas near the welding and grinding booths and near a shake-out pit.

SEROLOGIC STUDIES

In Patient 3, rising reciprocal indirect fluorescent antibody titers to whole-cell antigen from the 7J serotype were seen after absorption. In Patient 2, titers of 64 and 128 were found in absorbed convalescent-phase serum samples (Table 2). In other Foundry A workers, the prevalence of titers of 64 or greater to the 7J serotype was 15% after absorption (Table 2) and did not vary significantly according to work location or job classification (supervisors, grinders, welders, and core makers).

We also determined seroreactivity to *A. calcoaceticus* variety *anitratus* in workers of Foundry B and Hartford residents (Table 3). Foundry A workers tested were all men and averaged 42.2 years of age and 7.7 years of employment; the two women and 45 men tested at Foundry B averaged 44.8 years of age and 9.1 years of employment. Hartford residents tested included 48 women averaging 29.7 years of age and 45 men averaging 30.7 years of age. Rates of seropositivity to the human 7J strains

Table 2. Serum Antibody Titers in Patients 2 and 3 Against Four Preparations of Heat-Killed Strains of *Acinetobacter calcoaceticus* variety *anitratus*

Source of Serum	Reciprocal Indirect Fluorescent Antibody Titers (After Absorption) Against Four Strains with Designated Serotypes			
	7J*	7J†	10F‡	1F§
Patient 2				
(6/27/79)	64	<64	<64	<64
(8/30/79)	128	<64	64	<64
Patient 3				
(6/13/79)	<64	<64	<64	<64
(6/21/79)	512	512	64	64

* Pool of three strains isolated from blood of three men with *Acinetobacter* pneumonia.
† Isolated from air sample from cafeteria.
‡ Isolated from water from a recirculating furnace.
§ Isolated from air sample taken near a shake-out pit.

Table 3. Foundry Workers and Hartford Residents with Elevated* Absorbed Serum Antibody Titers to *Acinetobacter calcoaceticus* variety *anitratus*

Group	Antigen Preparation			
	7J†	7J‡	10F§	1F‖
	no. (%)			
Foundry A workers ($n = 120$)	18(15.0)	2(1.7)	6(5.0)	2(1.7)
Foundry B workers ($n = 47$)	7(14.9)	2(4.3)	17(36.2)	1(2.1)
Hartford residents ($n = 93$)	2(2.2)	0	0	0

* Reciprocal titer ≥ 64 by indirect immunofluorescence using heat-killed strains and after absorption of serum with 1F strain (see Methods).
† Pool of three strains isolated from blood of three men with *Acinetobacter* pneumonia.
‡ Isolated from air sample from cafeteria.
§ Isolated from water from a recirculating furnace.
‖ Isolated from air sample taken near a shake-out pit.

(after absorption) were similar in workers at the two foundries. However, Hartford resident had significantly lower rates than foundry workers ($\chi^2(1) = 10.54$, $p < 0.01$). In Foundry A and Foundry B workers, titers were unrelated to age and duration of employment.

ENVIRONMENTAL DUST EXPOSURE

Particle analysis in postmortem lung tissue from Patients 1 and 3 showed particles with the following elemental composition, in order of decreasing frequency: iron alone; silicon alone; aluminum and silicon; aluminum, silicon and iron; and iron, chromium, nickel, and zinc. These results suggest that iron oxides, silica, and silicates were present in the tissue. Both persons had occupationally related pneumoconiosis of the mixed-dust type. Patient 1 had more severe pulmonary changes with a single lesion compatible with nodular silicosis. He, but not the other two persons with acinetobacter pneumonia, had interstitial densities. Two of 116 readable radiographs from other Foundry A workers had evidence of pneumoconiosis.

When we analyzed the personal breathing zone samples collected on 46 workers, we found that eight of the samples exceeded the standard of the National Institute for Occupational Safety and Health for free silica of 0.05 mg/m^3. Free silica exposure varied by job category; six of 25 molders and shake-out workers, none of 13 coreroom workers, and two of eight welders and grinders had exposures exceeding the standard. Exposures of six of 18 chippers and grinders tested exceeded the American Conference of Governmental Industrial Hygienists threshold limit value for total particulates ($\geq$ 10 mg/m^3), and five exceeded American Conference of Governmental Industrial Hygienists threshold limit value for iron oxide ($\geq$ 5 mg/m^3). Standards of the National Institute for Occupational Safety and Health for chromium and nickel concentrations were exceeded in 17. Eight of 21 welders tested (including arc-air operators and burners) exceeded the American Conference of Governmental Industrial Hygienists threshold limit value for total particulates, eight exceeded the American Conference of Governmental Industrial Hygienists threshold limit value for iron

oxide, and 20 and 19 exceeded standards of the National Institute for Occupational Safety and Health for nickel and chromium, respectively. The above figures for chippers, grinders, and welders contrast with those of 16 workers who pour casting, none of whom had excess exposure to total particulates ($p = 0.005$) and five of whom had excessive exposure to nickel ($p < 0.0001$) and chromium ($p < 0.0001$) (Fisher's exact test, two-tailed).

Discussion

It is striking that the first documented cluster of non-nosocomial *A. calcoaceticus* variety *anitratus* pneumonia occurred in foundry workers who were exposed to unacceptably high levels of total particulates, free silica, and metallic dusts. *Acinetobacter calcoaceticus* variety *anitratus* rarely causes illness in healthy people; only 18 cases of non-nosocomial pneumonia caused by this bacterium have been reported in the English literature (1-12). We have no evidence to support, but have not directly tested, the alternative possibilities that the cluster resulted from increased virulence of the infecting strain or decreased resistance of the workers because of simultaneous viral infections.

Two of the cases in this cluster were in grinders, who have intense exposure to metallic dusts. The attack rate among grinders was statistically significantly higher than among persons with other jobs in the factory ($p < 0.02$, Fisher's exact test, one-tailed). The third case was in a welder who worked within a few metres of the other two. Two of the three cases occurred in workers who had documented mixed-dust pneumoconiosis with deposits of iron (probably iron oxide) predominating in postmortem lung tissue.

Mortality records in Great Britain have shown that welders are twice as likely to die from pneumonia (indicated by the underlying causes of death listed on death certificates) as are others in the general population (22). However, there is little information about the agents responsible for this excess of pneumonia, the places where the infections are acquired, or the prevalence or type of pneumoconiosis or other chronic lung disease among the workers affected. In-vivo and in-vitro studies have shown that the growth and infectivity of many bacteria are enhanced by increased iron concentrations (23). Whether this is true for *Acinetobacter* has not been tested, but, if so, the intense exposure of these workers to iron particles may have predisposed them to develop pneumonia after exposure to the bacterium.

The tight clustering in time and space of the cases in our study and the fact that all three infecting strains were of serotype 7J suggests that these men were exposed to a common source of *A. calcoaceticus* variety *anitratus*. A previous study showed that only one (0.5%) of 182 isolates (primarily from blood, respiratory secretions, and urine) of *A. calcoaceticus* variety *anitratus* was of 7J serotype (19). The commonest serotypes in that study were 10F (14.3%), 3F (7.7%), and 1F (7.1%), types found in environmental cultures in Foundry A. Our recovery of serotype 7J with a similar antibiogram from the air in Foundry A suggests the possibility of airborne spread. This is consistent with the finding that the prevalence of indirect fluorescent antibody titers of 64 or greater to the 7J strain was higher in Foundry A workers than in community controls and that the rate of seropositivity was not affected by work location or activities within the foundry. We could not explain the equally high seroprevalence in Foundry B, although it is possible that intense exposure to *A. calcoaceticus* variety *anitratus* is not limited to one foundry or that the serologic test is not sufficiently specific. We found no evidence to support other modes of spread.

Hospital outbreaks of pneumonia caused by *A. calcoaceticus* variety *anitratus* have been traced to person-to-person spread as a result of skin colonization (24). The three workers in this outbreak were not social acquaintances and apparently had little skin contact with each other. Furthermore, surveys of skin cultures of foundry workers showed a *lower* prevalence of the organism than has been seen in studies of other groups (24-26).

The problem of airborne bacterial infections in the work place has recently received attention with studies of *L. pneumophila* (27-29). Organisms such as *A. calcoaceticus* variety *anitratus* and *L. pneumophila*, which seem to adapt to aqueous environments, could be spread in industrial aerosols and pose special risks to susceptible workers. Additional work is needed to define the source of these organisms, how they become airborne, how exposure of workers can be limited, and to what degree industrial exposures to dust increase workers' susceptibility to infections caused by these organisms.

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References

1. Rudin ML, Michael JR, Huxley EJ. Community-acquired acinetobacter pneumonia. *Am J Med.* 1979;**67**:39-43.
2. Glick LM, Moran GP, Coleman JM, O'Brien GF. Lobar pneumonia with bacteremia caused by Bacterium anitratum. *Am J Med.* 1959; **27**:183-6.
3. Gardner DL, Pines A, Stewart SM. Fulminating and fatal pneumonia and septicaemia due to Achromobacter anitratus. *Br Med J.* 1960;**1**: 1108-9.
4. Reynolds RC, Cluff LE. Infection of man with *Mimeae*. *Ann Intern Med.* 1963;**58**:759-67.
5. Stockwell BA, Whitaker AN, Cheong M. Fatal Achromobacter anitratus pneumonia with bacteriaemia and neutropenia. *Med J Aust.* 1964;**2**:370-3.
6. Hammett JB. Death from pneumonia with bacteremia due to Mimeae tribe bacterium. *JAMA.* 1968;**206**:641-2.
7. Wands JR, Mann RB, Jackson D, Butler T. Fatal community-acquired *Herellea* pneumonia in chronic renal disease. *Am Rev Respir Dis.* 1973;**108**:964-7.
8. Wallace RJ Jr, Awe RJ, Martin RR. Bacteremic acinetobacter (herellea) pneumonia with survival. *Am Rev Respir Dis.* 1976;**113**:695-9.
9. Goodhart GL, Abrutyn E, Watson R, Root RK, Egert J. Community-acquired *Acinetobacter calcoaceticus* var *anitratus* pneumonia. *JAMA.* 1977;**238**:1516-8.
10. Buscaglia AJ. Acinetobacter pneumonia. *Ann Intern Med.* 1978;**89**: 1010. Letter.
11. Altman KA, Sacks F. Bronchopneumonia and endocarditis: caused by Acinetobacter anitratum (Herellea vaginicola). NY St J Med. 1979; **79**:1434-5.

12. Rosen JH, Muren O, Gander GW, Irby SK, Cashion CF. Bacteremic *Acinetobacter* pneumonia with survival in a normal host. *Virginia Medical.* 1979;**106:**660-2.
13. Abrutyn E, Goodhart GL, Roos K, Anderson R, Buxton A. *Acinetobacter calcoaceticus* outbreak associated with peritoneal dialysis. *Am J Epidemiol.* 1978;**107:**328-35.
14. Glew RH, Moellering RC Jr, Kunz LJ. Infections with *Acinetobacter calcoaceticus (Herellea vaginicola):* clinical and laboratory studies. *Medicine.* 1977;**56:**79-97.
15. Jacobsen G, Lainhart W., eds. ILO U/C 1971 international classification of radiographs of the pneumoconiosis. *Med Radiogr Photogr.* 1972;**48:**67-110.
16. National Institute for Occupational Safety and Health. Physical and chemical analyses branch analytical method 109. In: *National Institute for Occupational Safety and Health Manual of Analytical Methods.* Washington, DC: U.S. Government Printing Office; 1977; DHEW publication no. 77-157-A.
17. National Institute for Occupational Safety and Health. Physical and chemical analyses branch analytical method 173. In: *National Institute for Occupational Safety and Health Manual of Analytical Methods.* Washington, DC: U.S. Government Printing Office; 1977; DHEW publication no. 77-157-A.
18. Thornsberry C, Gavan TL, Gerlach EH. New developments in antimicrobial agent susceptibility testing. *Am Soc Microbiol Cumitech.* 1977;**6:**6-7.
19. Marcus BB, Samuels SB, Pittman B, Cherry WB. A serologic study of *Herellea vaginicola* and its identification by immunofluorescent staining. *Am J Clin Pathol.* 1969;**52:**309-19.
20. Wilkinson HM, Fikes BJ, Cruce DD. Indirect immunofluorescence test for serodiagnosis of Legionnaires' disease: evidence for serogroup diversity of Legionnaires' disease bacterial antigens and for multiple specificity for human antibodies. *J Clin Microbiol.* 1979;**9:**379-83.
21. DeNee PB. Identification and analysis of particles in biological tissue using SEM and related techniques. In: Johari O, ed. *Scanning Electron Microscopy.* Part 3. Chicago, Illinois: Chicago Press Corp.; 1976:461-8.
22. Employment Medical Advisory Service. *Chief Employment Medical Adviser's Notes of Guidance: Welding.* London, Department of Employment; 1972.
23. Weinberg ED. Iron and infection. *Microbiol Rev.* 1978;**42:**45-66.
24. Buxton AE, Anderson RL, Werdegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus:* epidemiologic characteristics. *Am J Med.* 1978;**65:**507-13.
25. Taplin D, Rebell G, Zaisa N. The human skin as a source of Mima herellea infections. *JAMA.* 1963;**186:**952-5.
26. Rosenthal S, Tager IB. Prevalence of gram-negative rods in the normal pharyngeal flora. *Ann Intern Med.* 1975;**83:**355-7.
27. Cordes LG, Goldman WD, Marr JS, et al. Legionnaires' disease in New York City, August-September 1978. *Bull NY Acad Med.* 1980; **56:**467-82.
28. Fraser DW, Deubner DC, Hill DC, Gilliam DK. Nonpneumonic, short-incubation-period legionellosis (Pontiac fever) in men who cleaned a steam turbine condenser. *Science.* 1979;**205:**690-1.
29. Glick TH, Gregg MB, Berman B, Mallison G, Rhodes WW Jr, Kassanoff I. Pontiac fever: an epidemic of unknown etiology in a health department. I. Clinical and epidemiologic aspects. *Am J Epidemiol.* 1978;**107:**149-60.