have been reported [6, 8, 10]. Factors that may predispose a person to septic bursitis include trauma, chronic mechanical irritation of the affected bursa, gout, rheumatoid arthritis, steroid therapy, alcoholism, renal insufficiency, and diabetes mellitus [4–6, 9]. In our case, septic bursitis occurred in an elderly alcoholic man who had an underlying malignancy, heart disease, and lung disease. Direct inoculation from an environmental source seems to have been the most probable cause of *S. maltophilia* bursitis in this patient because the bursae are poorly vascularized; hematogenous seeding from a distant focus of infection occurs infrequently [5].

Roschmann and Bell [5] compared cases of septic bursitis in nonimmunocompromised patients with cases in those whose immune systems were compromised because of alcoholism or steroid therapy. They found no differences in clinical presentation, bacteriologic spectrum, or response to treatment between the two groups of patients. The only notable differences were that sterilization of the bursae took three times longer in the patients who were immunocompromised and that higher WBC counts were seen in this group. It is of interest that no cases of gram-negative septic bursitis were identified among the immunocompromised patients.

Generally, antibiotic therapy is effective in the treatment of septic bursitis, although surgical drainage may be required in some cases where bursal fluid reaccumulates rapidly [6, 8, 9]. Hospitalization and parenteral administration of antibiotics may be preferable in treating patients who have severe infections, especially if the prepatellar bursa is involved [6]. However, some patients can be treated successfully as outpatients with oral antibiotics [10], as was the patient in this report. Our experience with this patient leads us to conclude that *S. maltophilia* should be considered as a potential cause when cases of unusual gram-negative septic bursitis are encountered.

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A Pseudoepidemic of Recent Tuberculin Test Conversions Caused by a Dosing Error

The resurgence of tuberculosis and regulatory demands from the United States Occupational Safety and Health Administration have made tuberculin testing a major recurring task for hospitals. Although tuberculin testing remains the best screening tool for identifying individuals infected by *Mycobacterium tuberculosis,* tuberculin testing is not without pitfalls. During a 6-week interval from February to April 1992, the employee health unit of a 400-bed hospital (University of Utah Medical Center, Salt Lake City) identified an unusually high proportion of employees with new positive tuberculin tests (reaction size, >10 mm of induration [range, 10–50 mm; median, 18 mm]). During the apparent epidemic period, eight (36%) of 22 employees had positive tuberculin tests, and two employees had indeterminate tuberculin tests (i.e., erythema of >10 mm in diameter and indistinct borders of induration that approached 10 mm in diameter). This high incidence of positive tuberculin tests was alarming because the proportion of newly discovered positive tuberculin tests was expected to be 0.36% per year (6/1,658). The known prevalence of positive tuberculin tests among employees was 8.9% (161/1,813). None of the employees whose tuberculin tests were found to be positive during the apparent epidemic period had knowingly been exposed to active tuberculosis cases, and none had common work assignments.

A review of testing procedures identified an error in the dosing of PPD. During the apparent epidemic period, the employees were mistakenly tested with 250 intradermal TU of PPD (Tubersol [5 TU or 250 TU], Connaught Laboratories, Swiftwater, PA) instead of the correct dose of 5 TU.

The dosing error necessitated retesting these employees with the correct dose to determine their true tuberculin reactivity. This situation provided an unusual opportunity to address two issues regarding the use of tuberculin for skin testing: (1) the rate of false-positive tuberculin tests associated with the use of 250 TU of PPD—as compared with the correct 5-TU dose—in a population with a low incidence and prevalence of tuberculosis, and (2) whether 250 TU of PPD enhanced the booster effect when employees were retested with 5 TU. A minimum interval of 3 weeks (range, 3 weeks to 3 months) elapsed before tuberculin skin testing was repeated. When seven of the eight PPD-positive employees were retested, six were found to have negative tests, and one had a test with 6 mm of induration. One employee was lost to follow-up (table 1). The two employees with

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Table 1. Results of initial and repeated tuberculin tests for 10 employees at the University of Utah Medical Center during a pseudoepidemic of skin test conversions caused by a dosing error.

Employee no.	Size (mm) of induration on initial testing with 250 TU*	Size (mm) of induration on repeated testing with 5 TU*
1	10	No induration
2	11	No induration
3	14	No induration
4	18	No induration
5	18	6
6	20	...‡
7	21	No induration
8	50	No induration
9	Indeterminate†	No induration
10	Indeterminate†	No induration

NOTE. The interval between initial testing and repeated testing ranged from >3 weeks to 3 months.

* The solution (0.1 mL) was administered intradermally on the volar aspect of the forearm, and the test was read 72 hours later.

† The induration, whose margins were indistinct, approached 10 mm in diameter, and the erythematous area was >10 mm in diameter.

‡ Employee was lost to follow-up.

initially indeterminate tests were negative on retesting. Two of the employees with false-positive skin tests had chest roentgenograms that appeared normal; one of these persons received chemoprophylaxis before the results of repeated skin testing were known. None of the employees who were negative on testing with 250 TU of PPD were positive on retesting.

The lowest estimate of a false-positive rate associated with administration of 250 TU of PPD, expressed as an OR, was 5.9 (95% CI = 2.4–14.9; P = .0005, Fisher's exact test). That is, administration of 250 TU was 5.9 times more likely to produce a positive result (8 of 22 employees) than was administration of the correct dose of 5 TU (161 of 1,813 employees). When we compared the incidence of positive tuberculin skin tests (8/22) during the apparent epidemic period with the expected incidence in our hospital (6/1,658), we found that administration of 250 TU was 157 times more likely to produce a positive result than was the administration of 5 TU (OR = 157; 95% CI = 42–604; $P < .000005$, Fisher's exact test).

Mycobacterial pseudoepidemics have typically been associated with contamination of solutions (such as stain solutions or growth media), leading to false-positive acid-fast stains or cultures [1–3]. Testing errors that cause pseudoepidemics of mycobacterial infection have been reported infrequently. Inadvertent administration of 10,000 TU of PPD has been reported [4]. False-positive tuberculin tests are unusual [5]. Two recent reports indicate that potency differences between PPD preparations from different manufacturers have led to false-positive tuberculin tests [6, 7]. A dosing error involving the erroneous administration of tetanus toxoid instead of PPD occurred at a health agency in Salt Lake City and resulted in a large number of false-positive tuberculin tests (B. Mooney, unpublished data). Conversely, a large number of false-negative tuberculin tests may have occurred at a local institution when patients were mistakenly given 1 TU of PPD instead of 5 TU (R. P. Rose, unpublished data).

The projected cost associated with this pseudoepidemic was $247 per patient. In contrast to the minimal medical consequences of this PPD dosing error, dosing errors with other medications have had serious sequelae (e.g., fatal neonatal pseudosepsis syndrome caused by oral administration of epinephrine instead of vitamin K) [8]; similar labeling of the two products, which came from the same manufacturer, may have facilitated that dosing error [8].

Failure to carefully examine the PPD vial label facilitated the occurrence of our pseudoepidemic. Labeling similarities between the 5-TU vials (green and white label) and 250-TU vials (red and white label) could not have contributed to the error unless red-green color blindness or decreased visual acuity played a role (the numerals are 1 mm in height). Because skin testing with PPD has been standardized at 5 TU, one might question the reasons for ever having 1 TU or 250 TU per 0.1 mL of solution available in clinical situations; discovery of a dosing error of the type we have described could easily be delayed or even overlooked in health care settings with a high prevalence of positive tuberculin tests among employees. In fact, it would be highly unusual to retest individuals with positive tuberculin tests—particularly those with large zones of induration, as occurred in this pseudoepidemic—unless a dosing error was suspected. Evaluation of data collected from the investigation of the dosing error suggests that interpretations of skin test reactions to 250 TU are unreliable in predicting true tuberculin reactivity. The consequences of the false-positive tuberculin tests caused by the dosing error included wasted time, unnecessary expense, and unnecessary exposure of patients to the potentially harmful effects of chemoprophylaxis.

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