eased tissue, it would be worthwhile to test the pathogenic properties of these strains in experimental animals. Until the pathogenicity of these *Hartmannella* strains is proven, species of this genus should be treated as harmless commensals.

We were surprised to read that a species, called ''*H. varini,*'' was used as a reference strain in the investigation of Aimard et al. [2]. To our knowledge, this species has not been described in the literature, and it is not listed in any of the definitive naked ameba identification keys reported by Page [4–6]. In addition, it does not correspond to either of the two new *Hartmannella* species that have recently been described [7, 8]. Furthermore, there is no reference strain of a species of this name held by either the Culture Collection of Algae and Protozoa (Ambleside, United Kingdom) or the American Type Culture Collection (Rockville, MD). The use of ''*H. varini*'' as a reference strain appears to have been invalid. We would be interested to know from where Aimard et al. [2] obtained this strain and where (if at all) it was described in the literature.

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Reply

SIR—We read with great interest the letter of De Jonckheere and Brown about our recent brief report [1]. We agree with De Jonckheere and Brown that *Hartmannella* should not be considered a pathogen without testing its pathogenic properties in experimental animals. However, culture of a corneal biopsy specimen yielded *Hartmannella* cysts and *Acanthamoeba* cysts and confirmed results of a histological microscopic examination. The conditions used to process the sample excluded the possibility of contamination with *Hartmannella.* Moreover, this ameba was not present on the superficial layers of the corneal biopsy specimen since scrapings were negative. In addition, the *Hartmannella* isolate found in the cornea was related to failure of hexamidine therapy, which may raise the question about the pathogenicity of *Hartmannella* as was previously suggested [2, 3].

''*H. varini,*'' which was used as a reference strain, corresponded to *Hartmannella vermiformis* and was provided by the Culture Collection of Algae and Protozoa (Ambleside, United Kingdom; reference 1534/7).

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Pneumococcal Pericarditis Since 1980

SIR—Saenz et al. [1] reported a case of purulent pericarditis caused by a highly resistant strain of *Streptococcus pneumoniae* that was successfully treated with vancomycin. With the prevalence of penicillin-resistant *S. pneumoniae* isolates approaching 16% in a New York City Department of Health survey from January to September 1997 (of which 10% had intermediate-level resistance and 6% had high-level resistance), one may expect more of such cases (written communication, New York City Department of Health).

A 75-year-old woman with cirrhosis and breast cancer who was being treated with tamoxifen presented with fever, chills, cough, and pleuritic chest pain. Physical examination revealed a pulsus paradoxus and a pericardial friction rub. A chest radiograph demonstrated right-lower-lobe pneumonia; a two-dimensional echocardiogram showed a large pericardial effusion, and cardiac tamponade was revealed with use of a Swan-Ganz catheter. Pericardiocentesis was done, and 300 mL of fluid was aspirated; the WBC count in the pericardial fluid was 265,000/mm^3. A pericardial window was formed, and an external drain was placed.

Table 1. Summary of data on 15 cases of pneumococcal pericarditis from 1980 to 1998.

[Reference]	Age (y)/sex	Culture specimen(s)	Risk factor(s)	Source	Medical treatment	Surgical treatment	Outcome
[1]	78/F	Blood, PCF	Diabetes	Lung	Vancomycin	Pericardiocentesis	Recovered
[3]	64/M	Blood, PLF, CIE* PCF	Felty's syndrome	Lung	Cephapirin sodium	Chest tube, pericardial window, pleural decortication	Recovered
[3]	60/M	Blood, PLF, sputum, CIE* PCF	Renal insufficiency, cardiac disease, alcoholism	Lung	Penicillin	Chest tube, pericardiocentesis, pericardial window, pleural decortication	Recovered
[4]	31/M	Blood	Alcoholism	None	Penicillin	Pericardial drainage (surgical)	Died
[5]	19/F	PCF	Common variable immunodeficiency	Lung	Penicillin	Pericardial catheter drainage, chest tube	Recovered
[6]	88/F	Blood	None	Lung	Penicillin	Pericardial catheter drainage	Recovered
[7]	32/F	Blood, PLF	HIV infection, CNS lymphoma, TB	Lung	Penicillin	Subxiphoid pericardiotomy, chest tube	Recovered
[7]	27/F	PLF	HIV infection, TB	Lung	Penicillin	Subxiphoid pericardiotomy, chest tube	Recovered
[8]	47/M	PCF	None	Lung	Penicillin	Pericardial window, pericardiocentesis	Recovered
[9]	78/F	PCF	NA	NA	Antibiotics	Intrapericardial streptokinase, pericardiocentesis	Recovered
[10]	29/M	PCF	None	Lung	Antibiotics	Pericardiocentesis	Died
[11]	3.5/M	Blood, PCF, peritoneal, middle ear exudate	None	Otitis media	Penicillin	Pericardiocentesis, pericardial window, laparotomy	Recovered
[12]	57/F	Blood, PCF	Sarcoidosis, steroid therapy	Lung	Penicillin, clarithromycin	Pericardiocentesis	Recovered
[13]	46/M	Blood, PCF	Alcoholism	Shoulder injury	Penicillin	Pericardiocentesis, pericardial window, chest tube, I & D of shoulder	Recovered
[PR]	75/F	Blood, PCF	Breast cancer, cirrhosis	Lung	Penicillin	Pericardiocentesis, pericardial window	Died

NOTE. CIE = counterimmunoelectrophoresis; I & D = incision and drainage; NA = not available; PCF = pericardial fluid; PLF = pleural fluid; PR = present report; TB = tuberculosis.

* Organism detected using CIE; cultures were nondiagnostic, probably secondary to prior antibiotic therapy.

Cultures of both blood and pericardial fluid yielded *S. pneumoniae* susceptible to penicillin. Despite therapy, she died after 4 weeks.

In an autopsy series of cases of pericarditis before 1943 [2], pneumococcal pericarditis occurred in about 51% of cases, followed by staphylococcal pericarditis (19%), pericarditis due to other streptococci (10%), and pericarditis due to gram-negative bacilli (2%). These cases occurred primarily in children and young adults secondary to pneumonia. After 1943, there was a significant change in the etiology of pericarditis, with gram-negative bacilli, staphylococci, other streptococci, and pneumococci accounting for 32%, 22%, 13%, and 9% of the cases, respectively. The change in the microbiological spectrum was attributed to the advent of antibiotics, thoracic surgery, renal dialysis, and chemotherapy. The average age of a patient with pericarditis was 49 years; pericarditis was more likely to be seen in a debilitated adult with chronic disease, malignancy, or a history of thoracic surgery.

We reviewed cases of pneumococcal pericarditis since 1980 to see if age, risk factors, and prognosis were any different in the past 2 decades. A MEDLINE search revealed an additional 14 cases (table 1). The average age of the patients was 49 years (range, 3.5 to 88 years). Risk factors included alcoholism, HIV infection, diabetes, renal insufficiency, common variable immunodeficiency, sarcoidosis, Felty's syndrome, and steroid therapy. The source of pericarditis was as follows: pneumonia or empyema, 11 cases; otitis media, 1; shoulder injury, 1; primary pericarditis (none), 1; and not available, 1. All the patients received antibiotics: penicillin, 10 cases; broad-spectrum antibiotics, 2; cephapirin sodium, 1; penicillin plus clarithromycin, 1; and vancomycin, 1. All the patients had some form of surgical intervention: chest tubes, 6 cases; pericardial window, 6; pericardial drainage with catheter, 2; surgical pericardial drainage, 1; subxiphoid pericardiotomy, 2; pericardiocentesis, 9; and intrapericardial instillation of streptokinase, 1.

Three patients died and 12 recovered. Ten blood cultures and nine pericardial fluid cultures yielded *S. pneumoniae.* Two pericardial cultures were negative, but counterimmunoelectrophoresis was positive for *S. pneumoniae* antigen.

There has been a dramatic decrease in the number cases of purulent pericarditis. The diagnosis is often delayed because classic signs of pulsus paradoxus, friction rub, distended neck veins, and distant heart

sounds may be absent or missed at initial presentation. Thus, a high index of suspicion is needed for patients who present with a history that predisposes them to purulent pericarditis. It is recognized only in about 18% of patients before death, and the mortality rate has approached 35%–40% [2]. Pneumococcal pericarditis is rare, and only 15 cases have been reported since 1980. In these cases, the mortality rate was 20% (three of 15); surprisingly, four (27%) of 15 patients had no risk factors, and only one of these patients would have qualified for receipt of the pneumococcal vaccine. The initial management of pneumococcal pericarditis includes pericardial drainage and therapy with vancomycin and a third-generation cephalosporin until results of susceptibility testing are available [1].

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Endemic Lepromatous Leprosy

SIR—Although it was quite interesting to read the report of lepromatous leprosy in a renal transplant recipient [1], I was surprised that Mushatt et al. were permitted to speculate that the patient might represent a case of contact with armadillos, since he apparently grew up and lived in an area where leprosy is well known to be endemic—New Orleans and Baton Rouge in south Louisiana. In contrast, northern Louisiana has not been associated with the transmission of endemic leprosy until recently [2]; there have been virtually no other cases in these parishes where lifelong residents—with no contact with patients with leprosy—developed leprosy. In northern Louisiana, the argument is that armadillos are the only obvious source of *Mycobacterium leprae.* In contrast, it has been known for >200 years that living in south Louisiana poses some risk for the acquisition of leprosy without travel and without contact with patients with diagnosed cases. The risk predates armadillos in Louisiana [2, 3].

In addition, the case report by Mushatt et al. nicely included HLAs (human leukocyte antigens) and showed once again that the risk of developing leprosy is associated with class II antigens. The analysis that we conducted, which included the six cases previously reported from northern Louisiana and a meta-analysis of data in the literature at that time, showed an association between lepromatous leprosy and HLA-DR2 and HLA-DQw1 [4]. Concerns about the pathogenesis, epidemiology, endemic transmission, and diagnosis of leprosy (including the acquisition of endemic disease) should continue to receive educational and research efforts, particularly in countries where it is endemic, such as the United States [5].

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Reply

SIR—In his letter responding to our recent case report of leprosy in a renal transplant recipient [1], West expresses surprise that we were ''permitted to speculate that the patient might represent a case of contact with armadillos. . . .'' On the contrary, we stated that there was no direct contact with armadillos and that exposure