The incidence of pneumococcal bacteremia or meningitis in the period 1991–1994 was 8.3–9.2 cases per 100,000 person-years (table 1). In 1996, the incidence increased significantly to 14.8 cases per 100,000 person-years, and in 1997 to 13.3 ($P = .0001$); but in 1998 it returned to 8.9 cases per 100,000 person-years. The incidence for the age group $\geq65$ years was approximately 4-fold higher and showed the same trend. The average number of pneumococcal isolates per 1000 blood cultures was 8.7 in the 2 years with the highest incidence of invasive pneumococcal disease, in contrast to 6.1 in the other years (table 1). The gradual increase in the number of blood cultures in our study did not explain the sudden increase in invasive pneumococcal isolates in 1996 and 1997 [3].

The mean incidence of 23 cases per 100,000 person-years for the age category 0–1 year (table 1) was much lower than the incidence of 136 cases per 100,000 person-years reported by Pastor et al. [4]. An explanation may be that children with severe illness in the Netherlands are preferably admitted to specialized children’s hospitals that are not served by the laboratories participating in our surveillance.

No trends in the distribution of pneumococcal serotypes [1], changes in the occurrence of influenza viruses (table 1), or other respiratory viruses were noted. However, the higher incidence of invasive pneumococcal infections in 1996 and 1997 for the age group $\geq65$ years coincided with relatively severe winters, as shown by the winter severity index (table 1).

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Cunninghamella bertholletiae Infection Mimicking Myocardial Infarction

Clinical infection with Cunninghamella bertholletiae is extremely rare, and in immunocompromised hosts it is almost always devastating and usually fatal. Here we report the first example of a patient with acute leukemia who developed heart failure as a consequence of massive cardiac involvement with this fungus. Our case illustrates the difficulties of diagnosing C. bertholletiae and intervening with appropriate therapy.

A 35-year-old man was admitted to our hospital with a history of night sweats, weakness, petechial bleeding, and fever. After the diagnosis of acute myelogenous leukemia (French-American-British classification M2) was established, chemotherapy with a double-induction protocol was initiated. Neutropenic fever was treated with empirical antibiotics with initial success, but during persisting neutropenia (33 days after chemotherapy), the patient developed signs and symptoms (radiographic and CT) compatible with aspergillosis of the right lung. Amphotericin B therapy was added to the regimen (total dose, 225 mg) and replaced after 5 days by voriconazole (total dose, 25,000 mg; duration, 9 weeks), but on bronchoscopy, the etiology could not be ascertained. Two weeks later, the fever retreated and there were signs of improvement of the pulmonary lesions on CT scans. When the patient developed fever again (neutropenia lasting $\geq70$ days), CTs documented further improvement of the previous lesions. Ten days later, signs of pericarditis led to the observation of a minor pericardial effusion on echocardiography and ST-segment alterations in the electrocardiogram. Culture of sputum yielded mucormycosis once, and the fungus was identified as Cunninghamella bertholletiae. The identification was confirmed by Dr. D. W. Warnock (Public Health Laboratory Service, Mycology Reference Laboratory, Bristol, UK). Results of bronchoscopy (with biopsy) and bronchoalveolar lavage remained inconclusive. During the next days, symptoms evolved to severe substernal pain with a pericardial friction rub and elevated ST segments in the precordial ($V_1$ through $V_6$) and limb leads (I, II, $aV_1$) of the electrocardiogram.

A bone-marrow aspirate was hypocellular and without evidence of leukemia. Because of the severe infection, it was decided that an allogeneic stem cell transplantation with a fast restoration of normal neutrophil counts was the only potentially curative treatment. Immunosuppressive conditioning with fludarabine and antithymocyte globulin was initiated. CTs now showed a new infiltration in the lingula adjacent to the pericardium; enzyme studies and an electrocardiogram were con-
On autopsy, a disseminated fungal infection was documented. Mycotic hemorrhagic pneumonia of the left upper lobe had penetrated per continuatatem from the lingula to the pericardium and the left ventricle, with concreto pericardii measuring 5 cm on the level of the left ventricular apical wall. A massive mycotic myocarditis with acute necrosis was predominant in the apex of the heart. Fungal infection appeared as mycotic mural endocarditis with the formation of a 3-cm massive ventricular parietal thrombus. There was no evidence of persistent leukemia. Fluorescence microscopy of wet mounts from sections of lung, heart, spleen, and kidney, made with 20% potassium hydroxide containing 0.4% blankophore under UV light, yielded ribbon-like, rarely septated hyphae. Cultures yielded *C. bertholletiae* only from lung specimens.

This fungus, which is one of the confirmed facultative pathogenic genera of the Mucorales [1], has been increasingly identified as a cause of opportunistic infection in patients with underlying chronic disease, with AIDS, or who are receiving immunosuppressive or cytotoxic therapy [2]. Patients with a *Cunninghamella* infection, which is rarely diagnosed before death, have a very high mortality rate. In leukemia, these infections have been uniformly fatal [3]. To our knowledge, only a few patients with localized infections have survived after therapy [4].

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