gle organism, and a mixed culture is a rare finding [4]. Diphtheroids are not rare as agents of peritonitis in peritoneal dialysis, and *Corynebacterium jeikeium* and *Corynebacterium aquaticum* are the species most commonly reported [1]. However, to the best of our knowledge, this is the first time in which CDC coryneform group A-4 is described as the etiologic agent of peritonitis. Centers for Disease Control group A-4 organisms have been implicated in several infections such as endophthalmitis, endocarditis, and sepsis [5–9], and some of the isolates of this group were recently reclassified using molecular methods as belonging to the genus *Microbacterium* or *Cellulomonas* [9]. Unfortunately, we cannot perform these studies, so definitive identification of the isolate remains as CDC group A-4.

Another significant finding in the present case is the fact that the only microorganism involved in both episodes of peritonitis was the CDC coryneform group A-4 isolate, while *Rothia dentocariosa* was isolated only in the second episode. Because *Rothia dentocariosa* grew only in one sample of peritoneal fluid, we cannot establish the actual role of this bacteria in the second episode of peritonitis (true superinfection or mere contamination due to a human saprophyte organism).

In conclusion, we report here the first case of peritonitis due to an uncommon diphtheroid, CDC coryneform group A-4. Uncommon gram-positive rods isolated in the future cannot, therefore, be interpreted automatically as contaminants, because these bacteria can actually be the cause of infection in humans, mainly when associated with foreign devices.

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Deafness Caused by Didanosine

The main side effects associated with didanosine treatment are polyneuritis and pancreatitis [1]. To our knowledge, deafness caused by didanosine has not been described previously. We report a case of hearing loss in an AIDS patient treated with didanosine. Hearing was regained when didanosine treatment was discontinued.

A 37-year-old Belgian homosexual man was found to be HIV seropositive when he presented in 1989 with a herpes zoster infection. In 1990 he developed *Pneumocystis carinii* pneumonia (PCP), and treatment with zidovudine 400 mg daily was begun. In April 1992 zidovudine was replaced by didanosine, 400 mg/daily. In 1994 the patient experienced a second episode of PCP. At that time he began cotrimoxazole treatment (960 mg/day), for PCP prophylaxis. He also was treated frequently with short courses of fluconazole because of recurrent oral candidiasis.

In February 1996 he was hospitalised because of fever, cough, and anaemia. He also had an herpetic mouth ulcer. He received a blood transfusion and was treated with clarithromycin and acyclovir. The fever and the cough disappeared for a while, but they reappeared in April 1996. Blood cultures revealed a *Mycobacterium avium* complex infection. The patient was treated with azithromycin 1000 mg/day, ciprofloxacin 500 mg/day, myambutol 1200 mg/day, and cotrimoxazole 960 mg every two days. His CD4+ lymphocyte count was 15/mm^3.

In May 1996 he developed bilateral deafness. Nuclear magnetic resonance examination of the brain was normal. A bilateral otoscopic examination was normal. Audiometry showed a bilateral neuro-sensorial hearing deficit of 40–60 dB. Tympanometry and stapedius reflexes were normal. Auditory brainstem response

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showed an increased bilateral latency time of wave V and increased I–V interval. Except for the abnormal audition, the rest of the neurological examination was normal. Didanosine therapy was discontinued, but the azithromycin and myambutol were continued. Clarithromycin treatment was started again in June 1996. As antiviral treatment the patient received zalcitabin, followed by ritonavir, followed by indinavir. All of these antiviral agents were discontinued after a few weeks because of side effects. The patient's audition improved progressively and returned to normal by August 1996. He improved immunologically and virologically after a combination treatment that included a high dose of saquinavir, lamivudine, and stavudine was started. The patient was still in relatively good health in October 1997. Stavudine was stopped after he developed polyneuritis and slightly diminished audition again. His CD4+ lymphocyte count had increased to 237/mm^3 and his viral load was low: 4714 copies per milliliter of plasma.

We do not have proof that our patient's deafness was caused by didanosine because he has not been rechallenged with the drug. However, because didanosine is known to cause neuritis and because the deafness disappeared after stopping the didanosine, it is likely that this drug was the cause of the hearing deficit. Because the hearing deficit occurred at a moment the patient was treated with didanosine and clarithromycin, it may be that this deficit was caused by the prolonged association of these two drugs. Hearing loss has been described previously in AIDS patients treated with clarithromycin for a *Mycobacterium avium* complex infection [2–4]. However, this always occurred in patients who were receiving several other medications, and therefore the effect of clarithromycin on hearing loss remains unclear. It seems prudent to monitor hearing in patients receiving long-term clarithromycin-didanosine treatment.

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Steroid-Induced Invasive Aspergillosis with Thyroid Gland Abscess and Positive Blood Cultures

Leukopenia is the most important risk factor for invasive aspergillosis, but steroid therapy is a recognized predisposing condition as well [1, 2]. We report a case of steroid-induced fatal aspergillosis with an atypical presentation and an uncommon feature of premorbid diagnosis.

On 19 October 1993 a 74-year-old male patient was admitted for malaise and persistent pain in the neck and shoulders. Laboratory tests showed evidence of inflammation (erythrocyte sedimentation rate 126/148, C-reactive protein 2.97 mg/dl). Serological tests for autoimmune diseases were negative and a biopsy of the arteria temporalis was nondiagnostic; thus, autoimmune disease could be neither proven nor excluded. Cultures and serological tests for infectious agents, including mycobacteria and fungi, were negative. Extensive diagnostic tests were performed, but no infection or neoplasm was found; the patient was diagnosed with polymyalgia rheumatica. Treatment with prednisolone was begun (tapered from 60 to 40 mg/day), and symptoms improved and signs of inflammation declined.

After three months of steroid therapy the patient's condition worsened again and he was readmitted to the hospital (1 May 1994). The leukocyte count (under steroid treatment) was 28.4×10^9/l; an intensive search for a possible infectious or neoplastic cause of the patient's complaints was repeated. An examination of the bone marrow showed locally increased granulopoesis. Chronic myelogenous leukemia could not be ruled out morphologically, but the Philadelphia chromosome was not present, and no rearrangement in the *bcr* gene was found. A computed tomographic (CT) scan of the thorax (11 May 1994) showed no pulmonary infiltrations but revealed a hypodense node 1.5 cm in diameter in the left caudal thyroid lobe that had not been present seven months earlier; a fine-needle puncture of the focus (18 May 1994) yielded 5 ml of fluid that was almost free of cells.

Two days later (20 May 1994) the patient became febrile (39.5 °C), and a urinary tract infection due to *Escherichia coli* was diagnosed and treated wich ciprofloxacin. The patient's condition improved initially, but

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