ology. I would like to point out that the autopsy has long been a cornerstone of anatomic pathology and has been frequently used for the diagnosis of infectious diseases. The autopsy has proven to be important in the recognition of emerging infectious diseases and in the description of the pathology of these conditions. In recent years, the autopsy has been critical in the recognition and description of AIDS, hantavirus pulmonary syndrome, and West Nile encephalitis [2–4].

An autopsy does not provide information that can be used directly for patient management in the same way as can information derived from a lung biopsy. However, information derived from an autopsy can guide management decisions for subsequent patients. Additionally, an autopsy can diagnose fatal communicable infections, such as tuberculosis [5], meningococcemia [6], and plague [7], that may have a direct bearing on the clinical treatment of the deceased patient’s contacts. Certainly, the frequency of the hospital autopsy has declined substantially in recent years [8]. I worry that the neglect of the autopsy by pathologists might be contributing to its decline.

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References

Reply
Sir—The inference by Dr. Nolte [1], that our article on infectious disease pathology centered primarily on the use of these diagnostic techniques for the living, is correct. Perhaps a more exclusive title or a more inclusive text would have been a better choice on our part. Nevertheless, Dr. Nolte’s remarks afford me an opportunity to comment on the use of autopsy for the diagnosis of infectious diseases.

In the age of MRI and CT-guided biopsy of deep-seated lesions, some may feel the postmortem examination has little to offer. With this I contend. I vividly remember a patient during my residency training who developed an invasive pulmonary infection with the neurotropic fungus *Ochroconis gallopava* after lung transplantation. The fungus disseminated to the brain, and multiple nodules appeared in both lungs as well. Although treated aggressively with antifungal agents, the patient died. To the surprise of all, only a microscopic focus of fungal infection was apparent on dissection of the lungs; the remaining nodules consisted of an undiagnosed Epstein-Barr virus–related posttransplantation lymphoproliferative disorder. Although this example is extreme, it is not uncommon for the postmortem examination to reveal infections undiagnosed in the living patient. In addition, I wholeheartedly agree with Dr. Nolte regarding the usefulness of the autopsy for the diagnosis of communicable and emerging diseases.

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Reference

Kawasaki-like Syndrome: Abacavir Hypersensitivity?

Sir—Johnson et al. [1] described fever, rash, abdominal pain, conjunctivitis, and peripheral edema in 2 patients infected with HIV. The authors attributed these symptoms to a Kawasaki-like syndrome without considering the possibility that they were due to an abacavir hypersensitivity reaction, which can be life-threatening and is more severe when abacavir is reintroduced. There is much overlap in the signs and symptoms associated with these 2 clinical syndromes. Because the syndrome originally described by Kawasaki and colleagues appears to be extraordinarily rare in adults [2–4], these unusual clinical findings will be encountered more often in patients treated with abacavir who are experiencing a hypersensitivity reaction.

Reintroduction of abacavir, as described in the first case report [1], resulted in a syndrome of fever, rash, and abdominal pain. Abacavir was promptly and permanently discontinued. Although the patient in the second case report was not receiving antiretroviral therapy at the time of the event, inadvertent ingestion of abacavir after discontinuing it for reasons other than hypersensitivity has re-
sulted in life-threatening hypersensitivity reactions [5, 6]. In these cases, the possibility of an abacavir hypersensitivity reaction should be considered. Identification of an abacavir hypersensitivity reaction has vital implications for HIV-infected individuals, because reintroduction of abacavir (either ZiaGen or Trizivir [which contains abacavir]; GlaxoSmithKline) may result in a recurrence of abacavir hypersensitivity that is potentially fatal.

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Reply
Sir—Drs. Toerner and Cvetkovich raise important concerns related to the differential diagnosis of Kawasaki-like syndromes in adult patients infected with HIV [1]. It is critical that hypersensitivity reactions to the multitude of HIV-related medications be at the top of the differential diagnosis for patients who present with a rash, fever, and other constitutional complaints. This is especially true for patients receiving abacavir, because of the severe and potentially fatal nature of that hypersensitivity reaction. That said, it is important to clarify several issues related to the clinical presentations of the 2 patients described in our article, because the case reports were simplified to be concise.

At the time of presentation, patient 1 had not received any antiretroviral therapy for 1 year. The regimen chosen ( stavudine, abacavir, ritonavir, and saquinavir) was a new salvage regimen. The patient had never taken abacavir, ritonavir, or saquinavir as components of any previous antiretroviral regimen dating back 7 years. The initial fever of his Kawasaki-like syndrome occurred 48 h after abacavir treatment was initiated. He was evaluated in our clinic the next morning for the possibility of an abacavir hypersensitivity reaction. Treatment with abacavir was continued because it was felt that a hypersensitivity reaction after taking four 300-mg abacavir tablets was unlikely in the absence of any prior exposure to abacavir.

One week later, the patient again presented to our clinic with continued fever, sore throat, rash, abdominal pain, nausea, and other symptoms, and he was initially given the diagnosis of abacavir hypersensitivity reaction. Antiretroviral medications were stopped, methyldprednisolone was started, and the patient was warned never to take abacavir again. Forty-eight hours later, he was given the diagnosis of Kawasaki-like syndrome and treated with intravenous immunoglobulin because features of his clinical presentation were unusual for a drug reaction: the principal atypical feature was markedly swollen and painful hands and feet. To our knowledge, this symptom has not been described in patients with hypersensitivity reactions to abacavir; it is a rare finding in patients with adverse drug reactions [2, 3]. Even though patient 1 was treated for Kawasaki-like syndrome, it was emphasized to him that he should never take abacavir again. He had originally been reluctant to begin abacavir treatment because of the hypersensitivity issue. A thorough history revealed that patient 1 was receiving no antiretroviral therapy of any kind when he relapsed with a Kawasaki-like syndrome 5 months later.

Patient 2 has never been given a prescription for abacavir and, when a thorough history was determined, she also denied taking any medications other than dapsone and clarithromycin. After she recovered from the acute phase of the Kawasaki-like syndrome, treatment with those 2 medications was restarted without any adverse events.

We believe that patient 1 and patient 2 each had an idiopathic syndrome with features similar to pediatric Kawasaki disease, which we referred to as a Kawasaki-like syndrome. Since publication of our report, we have been made aware of 4 additional cases of HIV-infected adult patients with Kawasaki-like syndromes. Three of the 4 patients were treated with dapsone and clarithromycin, and each had a prompt clinical response ([4]; R. B. Porwancher, personal communication). It is important to apply the clinical diagnostic criteria for Kawasaki disease, as discussed in detail in our article, and to exclude drug reactions and opportunistic infections before giving an HIV-infected patient a diagnosis of Kawasaki-like syndrome. We support the call of Drs. Toerner and Cvetkovich for caution in ruling out adverse drug reactions to abacavir before attributing a febrile illness with rash and constitutional symptoms to a Kawasaki-like syndrome.