hyponatremia played a role in the deterioration in this patient’s neurologic condition. Since this is not the diagnostic issue that Dr. Podolsky was asked to address in the Clinicopathological Conference, physicians involved in the care of the patient provided the following assessment.

Fourteen hours before the seizure, the serum sodium level was 131 mmol per liter, and the glucose level was 212 mg per deciliter. After correction for the elevated glucose level, the effective serum sodium level was 133 mmol per liter. Twelve hours before the seizure, the patient vomited several times, and an infusion of half-normal saline was begun. Immediately after the seizure, the sodium serum level was 127 mmol per liter, and the blood glucose level was 492 mg per deciliter; the corrected sodium level thus remained 133 mmol per liter. After the seizure, boluses and an infusion of normal saline were administered, and the serum sodium level rose to 135 mmol per deciliter. During the remainder of the patient’s hospital stay, the serum sodium level remained between 127 and 133 mmol per deciliter, despite infusions of normal saline and correction of blood glucose levels.

The patient’s physicians agree that isotonic saline is appropriate fluid replacement for patients with brain edema. However, this patient’s serum sodium level does not appear to have been low enough to cause a seizure. Cerebral edema attributable to the large B-cell lymphoma of the brain likely contributed to the seizure. A syndrome of inappropriate antidiuretic hormone secretion may have caused persistent mild hyponatremia.

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The presence of two different fungal components in the antibiotic provides strong evidence of a fungal origin of the cross-reactive components in the drugs. Given the difficulties encountered in the diagnosis of invasive fungal disease, it would be desirable to eliminate the fungal material from antibiotic agents.

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Imipenem in Patients with Immediate Hypersensitivity to Penicillins

TO THE EDITOR: It is considered potentially harmful to administer imipenem–cilastatin to patients with IgE-mediated hypersensitivity to penicillins1 because of a 47.4 percent rate of cross-reactivity (9 of 19 subjects) found in a single study2 on the basis of positive skin tests involving imipenem reagents.

Between 1997 and 2005, we studied 112 consecutive patients with such hypersensitivity, diagnosed as previously described,3 in order to assess the cross-reactivity with imipenem–cilastatin and to evaluate the allergic responses to imipenem–cilastatin in patients who had negative skin tests. Our patients had had a total of 143 immediate reactions to penicillins. All patients had positive skin tests for at least one of the penicillin