Acute weakness associated with West Nile virus (WNV) infection has previously been attributed to a peripheral demyelinating process (Guillain-Barré syndrome); however, the exact etiology of this acute flaccid paralysis has not been systematically assessed. To thoroughly describe the clinical, laboratory, and electrodiagnostic features of this paralysis syndrome, we evaluated acute flaccid paralysis that developed in seven patients in the setting of acute WNV infection, consecutively identified in four hospitals in St. Tammany Parish and New Orleans, Louisiana, and Jackson, Mississippi. All patients had acute onset of asymmetric weakness and areflexia but no sensory abnormalities. Clinical and electrodiagnostic data suggested the involvement of spinal anterior horn cells, resulting in a poliomyelitis-like syndrome. In areas in which transmission is occurring, WNV infection should be considered in patients with acute flaccid paralysis. Recognition that such weakness may be of spinal origin may prevent inappropriate treatment and diagnostic testing.

Most human infections with West Nile virus (WNV), a flavivirus within the Japanese encephalitis virus antigenic complex, are clinically inapparent (1,2). Mild febrile illness develops in approximately 1 in 5 infected persons; more severe neurologic disease, mostly meningitis or encephalitis, occurs in 1 in 150 (1–4). Less frequently, acute WNV infection has been associated with acute flaccid paralysis, which has been attributed to Guillain-Barré syndrome, motor axonopathy, or axonal polyneuropathy (4–6). However, these reports describe clinical and laboratory features that seem inconsistent with such diagnoses, and the exact cause of acute flaccid paralysis has not been thoroughly assessed with rigorous electrophysiologic, laboratory, and neuroimaging data. Brief descriptions of six patients have suggested that this flaccid paralysis is due to anterior horn cell involvement with a resultant poliomyelitis-like syndrome (7–9). Because understanding the clinical characteristics and underlying etiology of WNV-induced acute flaccid paralysis is critical for therapeutic decisions as well as prognosis, we describe the detailed clinical, laboratory, and electrophysiologic findings from these six patients and from one additional patient.

Patients and Methods

Seven patients were detected through WNV surveillance conducted by the Mississippi Department of Health and the Louisiana Office of Public Health. For each patient, a standardized questionnaire, including demographics, medical history, initial signs and symptoms, risk factors, and treatment, was completed; a standardized neurologic examination was performed by a single neurologist (JJS). Electrodiagnostic studies were performed by neurologists (AAL and JAVG) specializing in electrodiagnostic medicine.

Cerebrospinal fluid (CSF) and acute- or convalescent-phase serum specimens (or both) from each patient were tested for antibody to WNV by immunoglobulin (Ig) M antibody-capture enzyme immunoassay (10) or plaque reduction neutralization assay (11). The initial specimen for one patient (patient 5, Table 1) was tested with a slightly modified IgM antibody assay at a commercial laboratory (12). IgM assays were considered positive if the optical density ratio of the patient and negative control samples (P/N ratio) was greater than three. For patient samples, a P/N ratio for WNV at least three times that for St. Louis encephalitis virus indicated WNV infection (13). A plaque reduction neutralization test result of at least 10 was considered positive.

All seven patients had serologic evidence of WNV infection (Table 1). On the basis of serologic data, three of the patients were classified as confirmed case-patients (patients 4, 6, and 7) and four as probable case-patients (patients 1–3, 5), according to the national case definition (14).

Case 1

On July 1, 2002, a previously healthy, 56-year-old, male Mississippi resident was hospitalized with a 1-week
history of fever, chills, night sweats, myalgias, and acute encephalopathy. Neurologic examination showed profound weakness in both arms, asymmetric weakness in the legs with a right foot drop, and acute respiratory distress (Table 2). Sensory test results were normal. Although computed tomography (CT) and magnetic resonance imaging (MRI) of the brain showed normal results, heparin was administered for suspected evolving stroke. Admission laboratory values (Table 3) showed serum leukocytosis, with elevated protein (Table 3). On day 5, acute respiratory distress developed, and the patient required mechanical ventilation. Upon extubation 2 weeks later, the patient had continued extremity weakness and was aspirating fluids; a modified barium swallow study showed oropharyngeal dysphagia. Upon transfer to a rehabilitation center on day 30, the patient had asymmetric weakness in the legs and right arm and moderate weakness in neck flexors and facial muscles. Hypotonia and areflexia were noted in all limbs; loss of bladder function was evident.

### Case 2

On July 15, 2002, a 57-year-old male Mississippi resident with a remote history of prostate cancer and glucose intolerance was hospitalized with a 3-day history of fever, chills, nausea, vomiting, and headache. Neurologic examination showed encephalopathy and asymmetric weakness in all limbs (Table 2). Results of a brain MRI were normal. Admission laboratory studies showed a CSF pleocytosis with elevated protein (Table 3). On day 5, acute respiratory distress developed, and the patient required mechanical ventilation. Upon extubation 2 weeks later, the patient had continued extremity weakness and was aspirating fluids; a modified barium swallow study showed oropharyngeal dysphagia. Upon transfer to a rehabilitation center on day 30, the patient had asymmetric weakness in the legs and right arm and moderate weakness in neck flexors and facial muscles. Hypotonia and areflexia were noted in all limbs. Sensation was slightly diminished to vibration and proprioception in toes bilaterally but preserved to light touch, pinprick, and temperature. Sensory testing was normal in the upper limbs. Urinary incontinence was noted.

Table 1. Serologic results for West Nile virus (WNV)-specific antibodies in patients with acute flaccid paralysis associated with acute WNV infection

| Case no. | Onset   | Collection | Sample   | IgM-capture enzyme immunoassay | Plaque reduction neutralization assay |
|----------|---------|------------|----------|-------------------------------|-------------------------------------|
|          |         |            |          | SLEV | WNV | SLEV | WNV |
| 1        | 6/24    | 7/12       | Serum    | 3.5  | 22.3 | 320  | 5,120 |
| 2        | 7/16    | 7/16       | Serum    | 8.0  | 22.7 | 80   | 1,280 |
| 3        | 7/26    | 8/1        | Serum    | 2.79 | 24.9 | <10  | 640  |
| 4        | 7/29    | 8/3        | Serum    | 1.1  | 14.1 | <10  | 80   |
| 5        | 7/29    | 8/3        | CSF      | 3.3  | 39.2 | 40   | 2,560 |
| 6        | 7/29    | 8/13       | Serum    | 4.4  | 23.5 | 25.7 | 7.4  |
| 7        | 8/11    | 8/15       | Serum    | 3.4  | 25.5 | 40   | 320  |
| 8        | 8/11    | 8/29       | Serum    | 1.0  | 5.7  | <10  | 40   |
| 9        | 8/13    | 8/16       | CSF      | 6.1  | 23.8 | 10   | 320  |

Table 2. Initial clinical signs and symptoms in patients with acute flaccid paralysis associated with acute West Nile virus infection

| Case no. | Fever (>38.5°C) | Headache | Nuchal rigidity | Altered mental status | Tremor | Distribution of weakness |
|----------|-----------------|----------|-----------------|-----------------------|--------|-------------------------|
| 1        | +               | +        | +               | +                     | +      | Upper and lower limbs, R > L |
| 2        | -               | +        | -               | +                     | -      | Upper and lower limbs, R > L |
| 3        | +               | -        | -               | -                     | +      | Lower limbs, R > L         |
| 4        | +               | +        | -               | +                     | +      | R upper limb              |
| 5        | -               | +        | -               | -                     | -      | R upper limb              |
| 6        | +               | +        | -               | -                     | +      | Lower limbs, R > L         |
| 7        | +               | +        | +               | -                     | -      | Upper and lower limbs, L > R; bulbar muscles |

1IgM, immunoglobulin M; SLEV, Saint Louis encephalitis virus; CSF, cerebrospinal fluid.

2R, right; L, left.
Electrodiagnostic studies showed widespread denervation, reduced CMAP amplitudes in all nerves of the lower limbs and right upper limb, and normal SNAP responses, consistent with a severe, asymmetric process affecting anterior horn cells or motor axons. Myopathy, demyelinating polyneuropathy, and diffuse axonal polyneuropathy were not apparent.

**Case 3**

On July 24, 2002, a low-grade fever, nausea, and vomiting, followed by shaking chills and sweats, developed in a 56-year-old male Louisiana resident with a history of hypertension and coronary artery disease. The next day, asymmetric weakness developed in the lower extremities, with no pain or numbness. Upper extremities were normal. No bowel or bladder dysfunction was present. The patient was hospitalized on July 29, and neurologic examination showed a flaccid, areflexic right lower extremity and a weak left lower extremity with diminished reflexes. Results of strength and reflex testing of the upper extremities were normal. Sensory examination results were normal except for a mild decrease in sensitivity to pinprick, temperature, touch, and vibration in a stocking-and-glove distribution (i.e., distal arms and legs). A coarse bilateral upper extremity action tremor was noted. The patient had no headache, neck stiffness, or alteration of mental status (Table 2). Admission laboratory values showed leukocytosis and CSF pleocytosis (Table 3). Results of other diagnostic tests were unremarkable. Postviral demyelination syndrome and viral-induced polyradiculitis were considered, and IVIG, dexamethasone, and antibacterial and antiviral medications were administered without patient improvement. On day 15, the patient was discharged to a skilled nursing facility for rehabilitation.

MRI of the cervical, thoracic, and lumbosacral spine obtained during rehabilitation was notable for showing mild cervical and lumbosacral spinal stenosis and foraminal restrictions from C3 through C7 and homogeneous enhancement of the nerve roots of the cauda equina consistent with meningitis. Electrodiagnostic studies showed denervation in thoracic and lumbosacral myotomes, with no muscle activation in the right leg and reduced muscle activation in the left leg. CMAPs in the right leg were absent; SNAPs were normal. Electrodiagnostic findings suggested a severe, asymmetric process affecting anterior horn cells or motor axons. Diffuse axonal polyneuropathy was not evident, despite a slight sensory loss in the distal extremities.

**Case 4**

On August 2, 2002, fever, headache, and neck stiffness developed in a 69-year-old female Louisiana resident with a history of diabetes and degenerative disc disease; the next day acute weakness occurred in the right arm without pain, numbness, or paresthesias. She was hospitalized on August 4. On admission, physical examination documented fever, vomiting, encephalopathy, nuchal rigidity, and a bilateral rash on the lower extremities. Neurologic examination displayed a flaccid and areflexic right arm. Her legs and left arm exhibited normal strength, reflexes, and coordination, with normal sensation in all limbs. A coarse tremor was noted in the chin, left arm, and legs (Table 2). Laboratory findings included CSF pleocytosis (Table 3). Differential diagnoses included meningoencephalitis with associated motor polyradiculopathy and monoplegia secondary to stroke. The patient was treated with antibacterial and antiviral medications. Results of CT and MRI of the brain were normal. MRI of the cervical spine showed multilevel degenerative disc disease. The patient remained lethargic until day 13, when mental status abruptly improved; right arm weakness persisted. On day 19, she was transferred to a rehabilitation facility. Electrodiagnostic studies showed absent CMAPs and profound denervation with no voluntary activation in muscles of the right arm. Scattered denervation was also seen in the other three limbs. SNAPs had borderline amplitudes and conduction velocities bilaterally. The results were most consistent with a severe, asymmetric process affecting anterior horn cells or motor axons. The patient was subsequently transferred back to intensive care because her respiratory function was deteriorating, but she was not intubated. After Guillain-Barré syndrome was diagnosed, she was started on IVIG but had no improvement in weakness.
Case 5
On August 16, 2002, a 46-year-old male Louisiana resident with a history of coronary artery disease was hospitalized with fever, headache, fatigue, and leg weakness of 3 days’ duration. He reported no nuchal rigidity or mental status changes, although family members described him as intermittently confused. Neurologic examination showed a plegic and areflexic right leg and mild left leg weakness; sensation was intact throughout. A bilateral tremor of the upper extremities and jaw was noted (Table 2). Laboratory abnormalities included a CSF pleocytosis (Table 3). He was diagnosed with Guillain-Barré syndrome and started on IVIG. Brain CT and MRI results were normal. Results of an enhanced MRI of the spine suggested meningitis involving the conus medullaris and cauda equina. Electrodiagnostic studies performed on day 4 demonstrated early denervation and absent activation in muscles of the right leg and reduced activation of muscles in the right arm. CMAPs and SNAPs in the right arm and leg were normal. These findings were consistent with a severe, asymmetric process affecting anterior horn cells. The patient was transferred to a rehabilitation facility on day 6 with no improvement of weakness.

Case 6
On August 16, 2002, a 46-year-old male Louisiana resident with a history of coronary artery disease was hospitalized with fever, headache, fatigue, and leg weakness of 3 days’ duration. He reported no nuchal rigidity or mental status changes, although family members described him as intermittently confused. Neurologic examination showed a plegic and areflexic right leg and mild left leg weakness; sensation was intact throughout. A bilateral tremor of the upper extremities and jaw was noted (Table 2). Laboratory abnormalities included a CSF pleocytosis (Table 3). He was diagnosed with Guillain-Barré syndrome and started on IVIG. Brain CT and MRI results were normal. Results of an enhanced MRI of the spine suggested meningitis involving the conus medullaris and cauda equina. Electrodiagnostic studies performed on day 4 demonstrated early denervation and absent activation in muscles of the right leg and reduced activation of muscles in the right arm. CMAPs and SNAPs in the right arm and leg were normal. These findings were consistent with a severe, asymmetric process affecting anterior horn cells or motor axons. He was transferred to a rehabilitation facility on day 6 with no improvement of weakness.

Case 7
On September 1, 2002, a previously healthy, 39-year-old male Louisiana resident had onset of fever, headache, and nuchal rigidity followed the next day by dysphagia and bilateral arm and leg weakness that was worse on the left. He was hospitalized on September 6 for acute respiratory failure and intubated. Neurologic examination showed normal cognition, asymmetric flaccid paralysis of the left arm and leg with absent reflexes, hyporeflexic weakness of the right arm and leg, and weakness of bulbar muscles (Table 2). A partial supranuclear gaze palsy, cogwheel rigidity, and bilateral Babinski signs were also evident. Admission laboratory findings showed peripheral leukocytosis and CSF pleocytosis (Table 3). Brain MRI showed increased T2 signal in the periaqueductal gray matter, substantia nigra, and trigeminal motor nuclei. Electrodiagnostic studies performed on day 15 showed diffuse denervation in all myotomes, reduced CMAPs (worse on the left), and preserved SNAPs. On day 25, he was transferred to a long-term care facility with no improvement of limb weakness.

Discussion
The clinical and electrodiagnostic findings in these patients with WNV infection suggest involvement of spinal cord gray matter, specifically anterior horn cells, and a resulting acute poliomyelitis-like syndrome. All patients exhibited features typical for polio, including acute flaccid paralysis without paresthesias or sensory loss, marked asymmetric weakness, diminished or absent deep tendon reflexes in the affected limbs, and weakness that developed during an acute infectious process. Other typical features of poliomyelitis included CSF pleocytosis in five of six patients with CSF examination, acute respiratory distress in four, and acute changes in bowel or bladder function in two. In addition, electrodiagnostic findings showed asymmetric muscle denervation, reduced CMAPs, and preserved SNAPs. No patients had evidence of demyelinating polyneuropathy or myopathy. The absence of new sensory abnormalities localizes the disease process to the anterior horn cells or motor axons. Although muscle denervation and reduced CMAP amplitudes do not distinguish loss of anterior horn cells from loss of motor axons (15), these patients’ clinical features can be explained only by anterior horn cell disease, since no known infectious processes limited to motor axons produce widespread, asymmetric paralysis without sensory involvement. While MRI signal abnormalities in the anterior spinal cord have been noted in patients with poliomyelitis (16,17), these findings are inconsistent (18,19), and the absence of such changes in our four patients in which imaging was performed does not preclude a diagnosis of a poliomyelitis-like syndrome.

Since immunization has eradicated wild-type poliovirus from the developed world, most cases of paralytic polio-like conditions in the United States have been linked to other RNA viruses, including echoviruses, enteroviruses, and coxsackieviruses (20). Case reports have documented a poliomyelitis-type syndrome associat-
ed with other flaviviruses (21–23), as well as anterior myelitis associated with WNV infection (24).

The assertion that WNV infection involves anterior horn cells and causes a polio-like syndrome has a pathologic basis. The neuropathology of experimental WNV infection in monkeys was most pronounced in the cerebellum, medulla, and the cervical and lumbar regions of the spinal cord (25). Anterior horn cells showed degeneration and neuronal cell death; conversely, no changes were seen in the oligodendroglia or peripheral nerves. Similarly, WNV-infected horses displayed multifocal polioencephalomyelitis, with involvement of the ventral and lateral horns of the thoracic and lumbar spinal cord (26,27). WNV antigen was mainly localized within the gray matter of the spinal cord, with no lesions apparent in peripheral nerves or ganglia. In WNV-infected birds, lesions and viral antigen were most prominent in the cerebellum and the gray matter of the spinal cord (28).

Previous case studies have attributed WNV-associated acute flaccid paralysis to Guillain-Barré syndrome, motor axonopathy, or severe axonal polyneuropathy (4–6). The clinical signs and symptoms and electrodiagnostic findings reported in those cases, and those described here, are most consistent with a polio-like condition, and would be atypical for Guillain-Barré syndrome or other peripheral nerve disorders. Although acute poliomyelitis and polio-like conditions may occasionally simulate Guillain-Barré syndrome (29), our cases had several clinical, laboratory, and electrodiagnostic features that differed from typical Guillain-Barré syndrome (30–32; Table 4).

In Guillain-Barré syndrome, electrodiagnostic findings generally suggest peripheral nerve demyelination or, less commonly, a combined demyelinating and axonal process (30,31). The cases reported here displayed reduced or absent CMAPs with preserved SNAPs, no evidence of demyelination, a neurogenic pattern of recruitment, and widespread denervation; combined with the clinical picture of an asymmetric paralysis, these findings are typical for a polio-like condition and uncommon for Guillain-Barré syndrome. A pure axonal variant of Guillain-Barré syndrome has been described (33) and may be confused with poliomyelitis and polio-like conditions; however, such cases are generally characterized by distally prominent weakness and show subclinical sensory nerve involvement on electrodiagnostic testing. Thus, in the context of WNV infection, electrodiagnostic studies previously interpreted as motor axonal polyneuropathy or motor axonopathy without sensory nerve involvement (4–6) are more suggestive of anterior horn cell loss than of Guillain-Barré syndrome.

Three of the seven patients had acute flaccid paralysis without other findings, suggestive of severe central nervous system involvement caused by WNV infection. Physicians should suspect WNV infection in patients from areas where WNV is being transmitted and who have acute, painless, asymmetric weakness, even if unaccompanied by fever or apparent meningoencephalitis. Diagnostic studies should include testing for WNV-specific IgM antibody in CSF or acute- and convalescent-phase serum samples. In patients from such areas who have acute flaccid paralysis, CSF analysis, thorough electrodiagnostic studies, and spinal imaging should be considered before initiating diagnostic evaluations or therapies directed at Guillain-Barré syndrome, stroke, inflammatory myopathies, or other peripheral inflammatory processes. These therapies are ineffective for polio-like syndromes and can produce serious sequelae (34–37).

Continued surveillance and investigation of WNV-infected patients are needed to fully define the scope of clinical illness and determine the incidence of acute flaccid paralysis. In addition to assessing clinical outcome, the identification of risk factors and the pathologic confirmation of anterior horn cell involvement in patients with WNV-associated acute flaccid paralysis remain important public health goals.

**Table 4. Clinical characteristics of patients with West Nile virus–associated acute flaccid paralysis compared with patients with typical Guillain-Barré syndrome (25–27)**

| Characteristic                   | West Nile virus–associated flaccid paralysis | Guillain-Barré syndrome |
|---------------------------------|---------------------------------------------|-------------------------|
| Timing of onset                 | Acute phase of infection                    | 1–8 weeks after acute infection |
| Fever and leukocytosis          | Present                                     | Absent                  |
| Weakness distribution           | Asymmetric; occasional monoplegia           | Generally symmetric; proximal and distal muscles |
| Sensory symptoms                | Absence of numbness, paresthesias, or sensory loss; occasional myalgias | Painful distal paresthesias and sensory loss |
| Bowel/bladder involvement       | Often present                               | Rare                    |
| Concurrent encephalopathy       | Often present                               | Absent                  |
| CSF profile                     | Pleocytosis and elevated protein            | No pleocytosis; elevated protein (albuminocytologic dissociation) |
| Electrodiagnostic features      | Anterior horn cell/motor axon: reduced/absent CMAPs, preserved SNAPs; asymmetric denervation | Demyelination: marked slowing of conduction velocity; conduction block, temporal dispersion; reduced SNAPs |

*CSF, cerebrospinal fluid; CMAPs, compound muscle action potentials; SNAPs, sensory nerve action potentials.
Acknowledgments

We are grateful to Stanley W. Chapman, the Wilson Research Foundation, Raoul Ratard, Andrea Vicari, Grant L. Campbell, Michael Bunning, and Susan P. Montgomery for their important contributions to this investigation.

Dr. Sejvar is a neurologist and epidemiologist with the Centers for Disease Control and Prevention’s Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases. His current areas of research include the epidemiology of encephalitis, prion diseases, and other infections of the nervous system.

References

1. Tsai T, Popovici F, Cernescu C, Campbell G, Nedelcu N. West Nile encephalitis epidemic in southeastern Romania. Lancet 1998; 352:767–71.
2. Mostashari F, Bunning M, Kitsutani P, Singer D, Nash D, Cooper M, et al. Epidemic West Nile encephalitis, New York, 1999: results of a household-based seroepidemiological survey. Lancet 2001; 358:261–4.
3. Chowers M, Lang R, Nasser F, Giladi D, Rubinstein E, Itzhaki A, et al. Clinical characteristics of the West Nile fever outbreak, Israel, 2000. Emerg Infect Dis 2001;7:675–8.
4. Nash D, Mostashari F, Fine A, Miller J, O’Leary D, Murray K, et al. The outbreak of West Nile virus infection in the New York City area in 1999. N Engl J Med 2001;344:1807–14.
5. Sampson B, Ambrosi C, Charlot A, Reiber K, Veress J, Armbrustmacher V, et al. The pathology of human West Nile virus infection. Hum Pathol 2000;31:527–31.
6. Asnis D, Conetta R, Teixiera A, Waldman G, Sampson B. The West Nile virus outbreak of 1999 in New York City: the Flushing Hospital experience. Clin Infect Dis 2000;30:413–8.
7. Leis A, Stokic D, Polk J, Dostrov V, Winkelmann M. A poliomyelitis-like syndrome from West Nile virus infection. N Engl J Med 2002;347:1279–80.
8. Glass I, Samuel O, Rich M. Poliomyelitis due to West Nile virus. N Engl J Med 2002;347:1280–1.
9. Centers for Disease Control and Prevention. Acute flaccid paralysis syndrome associated with West Nile virus infection—Mississippi and Louisiana, July–August 2002. MMWR Morb Mortal Wkly Rep 2002;51:825–7.
10. Martin D, Muth D, Brown T, Johnson A, Karabatsos N, Roehrig J. Standardization of immunoglobulin M capture enzyme-linked immunosorbent assays for routine diagnosis of arboviral infections. J Clin Microbiol 2000;38:1823–6.
11. Beatty B, Calisher C, Shope R. Arboviruses. In: Lennette E, Lennette D, and Lennette E, editors. Diagnostic procedures for viral, rickettsial, and chlamydial infections. Washington: American Public Health Association; 1995. p. 89–212.
12. Prince H, Hogrefe W. Performance characteristics of an in-house assay system used to detect West Nile virus (WNV)-specific immunoglobulin M during the 2001 WNV season in the United States. Clin Diagn Lab Immunol 2003;10:66–9.
13. Martin D, Biggerstaff B, Allen B, Johnson A, Lanciotti R, Roehrig J. Use of immunoglobulin M cross-reactions in differential diagnosis of human flaviviral encephalitis infections in the United States. Clin Diagn Lab Immunol 2002;9:544–9.
14. Petersen L, Marfin A. West Nile virus: a primer for the clinician. Ann Intern Med 2002;137:173–9.
15. Kimura J. Electrodiagnosis in diseases of nerve and muscle: principles and practice. 2nd ed. Philadelphia: F.A. Davis Co.; 1989. p. 249–74.
16. Kornreich L, Dagan O, Grunebaum M. MRI in acute poliomyelitis. Neuroradiology 1996;38:371–2.
17. Rao D, Bateman D. Hyperintensities of the anterior horn cells on MRI due to poliomyelitis. Journal of Neurology, Neurosurgery, and Psychiatry 1997;63:720.
18. Huang P, Liu C, Chang Y, Chen C, Wang S, Yeh T, et al. Neurologic complications in children with enterovirus 71 infection. N Engl J Med 1999;341:936–42.
19. Ohry A, Karpin H, Yooli D, Lazari A, Lerman Y. West Nile virus myelitis. Spinal Cord 2001;39:662–3.
20. Rotbart H. Viral meningoencephalitis and the aseptic meningitis syndrome. In: Scheld W, Whitley R, Durack D, editors. Infections of the central nervous system. Philadelphia: Lippincott-Raven Publishers; 1997. p. 239–63.
21. Solomon T, Kneen R, Dung N, Khan V, Thuy T, Ha D, et al. Poliomyelitis-like illness due to Japanese encephalitis virus. Lancet 1998;351:1094–7.
22. Kho L, Sumarwo W, Jahja E, Gubler D. Dengue hemorrhagic fever accompanied by encephalopathy in Jakarta. Southeast Asian J Trop Med Public Health 1981;12:83–6.
23. Sumarwo W, Wuhr L, Jahja E, Gubler D, Suharyono W, Sorensen K. Clinical observations on virologically confirmed fatal dengue infections in Jakarta, Indonesia. Bull World Health Organ 1983;61:693–701.
24. Gadoth N, Weitzman S, Lehmene E. Acute anterior myelitis complicating West Nile fever. Arch Neurol 1979;36:172–3.
25. Manuelidis EE. Neuropathology of experimental West Nile virus infection in monkeys. J Neuropathol Exp Neurol 1956;15:484–60.
26. Cantile C, Di Guarro G, Eleni C, Arispeci M. Clinical and neuropathological features of West Nile virus equine encephalomyelitis in Italy. Equine Vet J 2000;32:31–5.
27. Cantile C, Del Piero F, Di Guarro G, Arispeci M. Pathologic and immunohistochemical findings in naturally occurring West Nile virus infection in horses. Vet Pathol 2001;38:414–21.
28. Steele K, Linn M, Schoepp R, Komar N, Geisbier T, Manduca R, et al. Pathology of fatal West Nile virus infection in native and exotic birds during the 1999 outbreak in New York City, New York. Vet Pathol 2000;37:208–24.
29. Gorson K, Ropper A. Nonpoliovirus poliomyelitis simulating Guillain-Barré syndrome. Arch Neurol 2001;58:1460–4.
30. Ashby A, Arnason B, Karp H. Criteria for diagnosis of Guillain-Barré syndrome. Ann Neurol 1978;3:565–6.
31. Weinberg D. AAEM case report #4: Guillain-Barré syndrome. Muscle Nerve 1999;22:271–81.
32. Hurwitz E, Holman R, Nelson D, Schonberger L, Breman D, Kaslow R, et al. National surveillance for Guillain-Barré syndrome. Journal of Neurology 1978–March 1979. Neurology 1983;33:150–7.
33. Visser L, Van der Meche F, Van Doorn P, Meulstee J, Jacobs B, Oomes P, et al. Guillain-Barré syndrome without sensory loss (acute motor neuronopathy): a subgroup with specific clinical, electrodiagnostic, and laboratory features. Brain 1995;118:841–7.
34. Norda R, Berseus O, Stemayr B. Adverse events and problems in therapeutic apheresis. A report from the Swedish registry. Transfusion and Apheresis Sciences 2001;25:33–41.
35. Stangel M, Muller M, Marx P. Adverse events during treatment with high-dose intravenous immunoglobulins for neurological disorders. Eur Neurol 1998;40:173–4.
36. Gottlieb S. Intravenous immunoglobulin increases the risk of thrombotic events. BMJ 2002;324:1056.
37. Struble E, Dice Y. Intravenous immune globulin (IVIG) precipitating acute myocardial infarction. J Miss State Med Assoc 2002;43:115.

Address for correspondence: James J. Sejvar, Medical Epidemiologist, Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, 1600 Clifton Road, Mailstop A39, Atlanta, GA 30333, USA; fax: 404-639-3163; email: zeas3@cdc.gov