trations of phenytoin, phenobarbital, and diazepam in the high therapeutic ranges, there was continuous bilateral facial twitching and asynchronous clonic movement of the muscles of the left neck and shoulder, and right arm. Nitrous oxide (60%) or halothane (1.25%) alone slowed but did not eliminate convulsions (Figure). Together they suppressed seizures for 30 minutes. Isoflurane (0.25 to 1.5%) alone reduced seizures and paroxysmal EEG activity approximately in proportion to the administered concentration. Isoflurane (2%) caused EEG inactivity (less than 3 μV) and convulsions ceased. On day 40, with serum concentrations of pentobarbital and lorazepam in the high therapeutic ranges, isoflurane (1%) stopped convulsions and caused virtual EEG inactivity for 90 minutes. With isoflurane (1.5%) for 3 hours there were two brief facial convulsions arising from an isoelectric EEG. Continuous anesthesia was tolerated without hypotension for 5 hours. He died on the 52nd hospital day with no cause for the encephalitis found.

General anesthetics have not been systematically studied in status epilepticus. Barbiturates in anesthetic doses, particularly pentobarbital [5], will make the EEG isoelectric but may cause hypotension before seizures are controlled. Enflurane, an isomer of isoflurane, reportedly suppressed seizure activity in a patient with psychomotor epilepsy but has also caused convulsions. In our patient, high concentrations of halothane produced unsustained seizure control, and nitrous oxide in maximum-tolerated concentrations was ineffective. There may have been a synergistic effect when both agents were used. However, isoflurane (1.5 to 2%) produced an isoelectric EEG with rare convulsions, suppressing the EEG background and seizure discharges. Unlike halothane and enflurane, isoflurane has no known toxic metabolites and less hemodynamic effect. All of these inhalation agents may raise intracranial pressure; however, monitoring in our patient showed no elevation. Isoflurane can be used safely for several hours in critically ill patients with status epilepticus and may be preferable to other anesthetics because of EEG suppression at well-tolerated inspired concentrations.

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Amantadine May Be Useful in Essential Tremor

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The therapeutic efficacy of amantadine in essential tremor [3] has been challenged recently by Koller [2], who failed to detect any improvement in 6 patients previously unresponsive to propranolol. The following case description illustrates that amantadine can be useful in essential tremor.

A 69-year-old woman was admitted for evaluation and treatment of severe tremor. There was no family history of neurological disease. She had not taken any drug known to provoke or enhance tremor. Examination showed no tremor in the limbs at rest. On upright position, a head tremor was evident. Holding the arms outstretched disclosed a severe postural tremor, which did not disappear during movement. Eating, drinking, or writing was impossible. Electromyographic recording showed a mean tremor frequency of 4.2 Hz. The rhythm of the tremor was changed by rapidly stretching the wrist or applying a rhythmic sinusoidal force to the wrist at a frequency of 2 Hz which provoked "beating" of the wrist. These physiological studies indicated the existence of a central generator for the tremor in addition to peripheral sensitivity [4]. Treatment with propranolol (240 mg daily) resulted in abatement of the tremor, but the patient was unable to carry out motor activities of daily life. Treatment with amantadine alone (300 mg daily) produced a significant reduction of the tremor of the head and arms. The maximal benefit was achieved when both propranolol (200 mg daily) and amantadine (300 mg daily) were used together. Under this regimen the patient was able to perform all types of manual activities. Attempts to withdraw either drug led to an increase in the severity of the tremor.

This patient represents an example of severe essential tremor (type III of Marsden and associates [4]), which is probably produced by some central generator, although peripheral influences may also play some role. The biochemical basis of essential tremor is not yet known. Recent evidence indicates that β-adrenoreceptor antagonists improve tremor by virtue of peripheral β-2 receptor mechanisms [1]. The mechanism by which amantadine results in improvement in some patients with essential tremor is not understood. However, we agree with Manyam [3] that it is worth considering amantadine among the available drugs for the treatment of essential tremor. Detailed clinical, neurophysiological, and pharmacological analysis of a large number of patients treated with amantadine is needed to clarify what type of patients are likely to respond favorably.

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Guillain-Barré Syndrome Associated with Hepatitis A

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Guillain-Barré Syndrome (GBS) is an acute polyradiculoneuropathy of uncertain cause. It is generally benign, and numerous infectious agents and immunological mechanisms may play a role in its pathogenesis. Sixty-six percent of patients experience an antecedent illness within 8 weeks before onset of GBS [5]. Although there are numerous reports linking acute hepatitis and GBS, only a few specify the serological markers, with hepatitis B virus accounting for 10 cases, hepatitis A (HA) virus for 5 cases [1–4], and non-A, non-B hepatitis virus for only 1 case. We report the clinical and serological findings for a case of GBS associated with hepatitis A infection.

A 34-year-old woman, whose son had acute viral hepatitis, presented with asthenia, anorexia, jaundice, and elevated transaminase levels (serum glutamic oxaloacetic transaminase [SGOT], 301 IU; serum glutamic pyruvic transaminase [SGPT], 314 IU); a diagnosis of acute hepatitis was established. Seven days later she developed generalized paresthesias and weakness and was referred for neurological examination. Findings included generalized muscle weakness which was most pronounced in the facial and pelvic girdle muscles, hypotonia, areflexia and impaired proprioception in all limbs, and bilateral Lasègue's sign. The liver was slightly enlarged, soft, and nontender on palpation. Transaminase levels were still elevated (SGOT, 200 IU; SGPT, 550 IU). Cerebrospinal fluid (CSF) analysis showed protein, 156 mg/dl, glucose, 61 mg/dl, and a cell count of 5 red blood cells. Results of serological tests for recent cytomegalovirus and Epstein-Barr virus infection were negative. Hepatitis markers measured by enzyme immunoassays were positive for HA IgM antibody, HBsAb, and HBcAb, and negative for HBsAg. Determination of HA IgM antibody in CSF was negative 33 days later. Neurophysiological studies demonstrated generalized alteration in nerve conduction velocity and an increased F-response conduction time; all findings suggest polyradiculoneuropathy.

Three weeks after onset, neurological improvement began and transaminase levels returned to normal. Three months later, the patient was symptom free with the exception of persistent bilateral facial weakness.

In common with other reported cases, the appearance of neurological symptoms in our patient was preceded by acute hepatitis, with both disorders following a favorable course. The presence of HA IgM antibody points to recent infection with hepatitis A virus, to which both the acute hepatitis and acute polyradiculoneuropathy could be attributed. The negative HBsAg titer and positive HBsAb and HBcAb titers suggest previous hepatitis B virus infection. The negative finding of HA IgM antibody in the CSF may be due to the delay in determination. This is consistent with the findings of Igarashi and associates [3] who described a similar patient in whom HA IgM antibody was demonstrated in CSF at onset but was undetectable two weeks later, while the antibody persisted in serum as late as 45 days after onset. Because viral hepatitis infections remain subclinical and undetected in a majority of cases, it is quite possible that hepatitis virus infection plays a more important role in the pathogenesis of GBS than has been suspected so far.

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Incubation Period and Severity of Experimental Allergic Encephalomyelitis: Analogy with Swine-Flu-Vaccine-Induced Guillain-Barré Syndrome

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The letter by Asbury [2] calling attention to the paper by Langmuir and colleagues [7] prompted review of our records concerning experimental allergic encephalomyelitis (EAE) in guinea pigs (Fig 1) and rabbits (Fig 2).