the diagnosis. Treatment lacks proof by controlled trials; isoniazid, rifampicin, pyrazinamide and either streptomycin or ethambutol are used in the first 2 months; isoniazid and rifampicin in the next 7-10 months; and in patients not suffering from HIV, dexamethasone is advised. Steroids improve survival but may not prevent disability. *M tuberculosis* resistant to antituberculosis drugs is an increasingly common clinical problem, and the use of WHO recommended alternative treatment with fluoroquinolones is restricted to case reports.

Of interest regarding the increasing importance of infectious disease in neurology, during 2004 one quarter of the case reports in The Lancet were patients with neurological infections. (Solomon T, Love R. **Lancet Neurol** 2005;4:139).

NEUROMUSCULAR DISORDERS

STEROIDS FOR CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY

The efficacy and safety of high-dose, intermittent IV methylprednisolone (IVMP) as initial and long-term maintenance therapy for chronic inflammatory demyelinating polyneuropathy (CIDP) were analyzed by a retrospective review of outcome data derived from patients' medical records between 1992 and 2003 at Washington University School of Medicine, St Louis, MO. Of 57 patients with clinical and electrophysiologic evidence of CIDP, 39 had sufficient data to classify and compare patients in 3 cohorts according to their primary treatment with IVMP, IVIg, or oral immunosuppression with prednisone or cyclosporine. There was no significant difference in mean improvement of quantitative muscle testing (hand dynamometer) at 6 months or at the last clinic visit (average 4.5 years later) among the 3 groups. At the last visit, 81% to 88% improved in all groups. Weight gain and cushingoid features were less frequent in patients treated with IVMP (19%) than in those receiving oral prednisone (58%). (Lopate G, Pestronk A, Al-Lozi M. Treatment of chronic inflammatory demyelinating polyneuropathy with high-dose intermittent intravenous methylprednisolone. **Arch Neurol** Feb 2005;62:249-254). (Respond: Glenn Lopate MD, Department of Neurology, Washington University School of Medicine, 660 S Euclid Ave, Box 8111, St Louis, MO 63110).

COMMENT. IVMP is as effective in improving and maintaining strength in patients with CIDP as is IVIg or oral prednisone, and has fewer adverse effects. The authors recommend IVMP as initial and maintenance therapy in CIDP patients with weakness or disability.

PUFFER FISH POISONING

The effects of puffer fish poisoning on peripheral nerve were investigated in 4 of 9 patients (7 adults and 2 children) treated at the Prince of Wales Hospital, Sydney, Australia. The patients had consumed soup made from 30 puffer fish. They experienced numbness of the lips approximately one hour later, and the numbness spread to the tongue, throat, and then hands and feet. Symptoms progressed rapidly, the gait became ataxic, and the reflexes were normal or depressed. Full recovery occurred within one week. The urine of each patient

tested positive for tetrodotoxin. Excitability measurements of sensory and motor nerves showed that, compared with controls, axons were of higher threshold, compound muscle and sensory action potentials were reduced in amplitude, latency was prolonged, and strength-duration time constant was reduced. Threshold electrotonus of motor axons showed less threshold decline than normal on depolarization and greater threshold increase on hyperpolarization. The changes in excitability were reproduced in a mathematical model by reducing sodium permeabilities by a factor of 2. (Kiernan MC, Isbister GK, Lin CS-Y et al. Acute tetrodotoxin-induced neurotoxicity after ingestion of puffer fish. **Ann Neurol** March 2005;57:339-348). (Respond: Dr Kiernan, Prince of Wales Medical Research Institute, Barker Street, Randwick, Sydney, NSW 2031, Australia).

COMMENT. The neurotoxic effects of puffer-fish poisoning are due to tetrodotoxin blockade of Na^+ channels. In an editorial, Kaji R and Nodera H, Tokushima University, Japan (**Ann Neurol** 2005;57:309) discuss the differentiation of puffer fish poisoning and Guillain-Barre syndrome (GBS) with reference to persistent sodium channels. GBS occurs in a demyelinative form (acute inflammatory demyelinating polyneuropathy [AIDP]) and the axonal form (acute motor axonal neuropathy [AMAN]). In AMAN, anti-GM1 antibodies may bind specifically to motor nerves and interfere with axonal sodium channels. Nerve excitability changes in AMAN are different from those in puffer fish poisoning, sensory fibers are spared, and a neurotoxin is not involved. Inflammatory mediators in AMAN are the likely explanation for reduced excitability of axons through sodium channels.

SEIZURE DISORDERS

MIDAZOLAM IN REFRACTORY NEONATAL SEIZURES

The outcome of 45 neonates with EEG-confirmed seizures (ESZ) was analyzed at the University Hospital of the Canary Islands, La Laguna, Spain. Of 32 neonates treated with phenobarbital/phenytoin, ESZ persisted in 17; of these, 13 had a poor outcome and 4 died. Of 13 nonresponders to phenobarbital/phenytoin who were treated with midazolam early, within 1 hr, ESZ were rapidly controlled in 13, only 4 had a poor outcome and 2 died. Neonates treated with midazolam had significantly better neurodevelopment than those receiving phenobarbital (53.9% vs 11.8%). (Conde JRC, Borges AAH, Martinez ED et al. Midazolam in neonatal seizures with no response to phenobarbital. **Neurology** March (1 of 2) 2005;64:876-879). (Reprints: Dr JR Castro Conde, Neonatology Service, University Hospital of the Canary Islands, Ofra S/N, La Laguna 28230, Spain).

COMMENT. Electrographic seizures in neonates correlate with poor neurodevelopmental outcome. (McBride MC et al. **Neurology** 2000;55:506-513). In the above retrospective, nonrandomized study of neonates with refractory EEG-confirmed seizures, midazolam, a GABA agonist, is proven effective as a third-line treatment in patients who have failed to respond to phenobarbital and phenytoin. Patients who respond to midazolam and those whose electrographic seizures are controlled by conventional first line treatments show improved neurodevelopmental outcome when compared to a group with refractory neonatal seizures. Midazolam is currently employed in the treatment of status