cular transmission pushed the LEMS above the threshold of clinical manifestation. This case again demonstrates, that even low doses of botulinum toxin affect remote muscles by systemic effects and may act in combination with other neuromuscular disturbance. In mice the interaction between the two presynaptically acting agents LEMS-IgG and botulinum toxin was studied, and it was shown that LEMS-IgG did not prevent the binding and electrophysiological action of botulinum toxin. In conclusion it should be emphasised that patients with an underlying neuromuscular disease have an increased risk of developing generalised muscle-weakening after local botulinum toxin injections. Patients with LEMS or myasthenia gravis and patients receiving substances that alter neuromuscular transmission should therefore be treated with caution if botulinum toxin injections are used to treat focal dys-tonia.

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Electrophysiological improvement after intravenous immunoglobulin in motor neuropathy with multifocal conduction block

Multifocal demyelinating motor neuropathy (MMN) with persistent conduction block⁠¹ may masquerade as multiple mononeuropathy or motor neuron disease.⁠² A careful electrophysiological study in these patients is necessary for diagnosis. Patients with chronic inflammatory demyelinating neuropathies may respond to high-dose intravenous immunoglobulin (IVIg).⁠³ In view of the need for more effective therapy in MMN, we treated a patient with high-dose IVIg. A 53-year-old man had slowly progressive limb weakness over eight years. Weakness, fasciculations started in the right forearm, and left forearm and hand involvement occurred. A year before admission he noticed slight weakness in both lower limbs. No bulbar symptoms were noticed. There was no known previous illness. Examination confirmed weakness in the limbs, 3/5 in the right arm muscles. The weakness was also prominent in extensor muscles of the left forearm (3/5) but grade 4/5 for flexion and extension of the left elbow, hand and shoulder girdle muscles. Foot dorsiflexion was weak on both sides (3/5). There was slight atrophy of all intrinsic hand muscles. Deep tendon reflexes were uniformly depressed. Occipital fasciculations were seen in both arms. Cranial nerves and all modalities of sensation were normal. Myokymia was seen in the right intrinsic hand muscles.

IgG and IgM antineurophil antibodies measured by ELISA were not found. Other laboratory tests, including serological screening were either normal or negative. Sural nerve biopsy was normal. Muscle biopsy of the left peroneus showed signs of mild denervation atrophy.

Conventional electromyography revealed sparse fasciculation and fibrillation potentials, particularly in the right upper limb, and myokymic discharges in the right hand muscles. Motor unit potentials were often polyphasic and were followed by "satellites". Recruitment of the MUPS was considerably reduced. Bilateral distal and proximal sensory conduction along sural, superficial peroneal, median, and ulnar nerves and amplitude of the nerve evoked potentials were normal. Motor conduction studies showed multifocal proximal conduction blocks involving nerves of both upper limbs (figure). A reduction in amplitude of ratio more than 40% at proximal supramaximal stimulation has been accepted as the criterion for conduction block, in the absence of increased duration of compound muscle action potential more than 20%.⁠⁴ The block was not located at the usual sites of compression, and the segment blocked varied from nerve to nerve. More than one segment with blocks was possible in the same nerve (figure). The neuropathy was asymmetrical. Distal motor latencies were normal. There was proximal slowing in motor conduction velocity in the upper limb nerves (median, ulnar, musculocutaneous), but only moderate slowing in peroneal and tibial posterior nerves (knee- to-ankle segment) (figure). Right median and ulnar nerves F waves were absent. Central motor pathways conduction time was calculated by magnetic stimulation of the brain and this was normal (5-0ms). Radial muscle conduction (C- median point) was prolonged.

The patient continued to deteriorate steadily until treatment with high-dose IVIg. He was started on 165 grams immunoglobulin divided into 5 daily doses (400 mg/kg/day). The patient noticed an increase in strength. Improvement started two to three days after infusion of IVIg. Clinical examination showed however, maximal improvement at seven to 10 days after infusion, with normal strength (5) except for extension muscles of left forearm and feet dorsiflexion (4/5). The improvement was, however, short-lived and five weeks later he returned to his initial pre-treatment level due to the delayed dose administration. The patient improved again and regained strength upon restimulation of the dose (165 grams) at four week intervals. After starting treatment with IVIg we have followed the patient for 18 months and no significant side effects were noted.

Electrophysiological study performed one week after the completion of the first IVIg infusion showed absence of myokymic discharges, increased MUPS recruitment, and full interference pattern. Conduction blocks were resolved after the first IVIg administration. Motor conduction block resolution was seen seven to 10 days after IVIg administration (figure) and correlated well with increased strength. After five weeks, conduction blocks were again seen, maximal less marked and observed in a smaller number of sites in each nerve than before IVIg administration. Slowing in motor conduction velocity did not show conclusive changes after treatment.

The present report provides evidence...
that high-dose IVIg may be an effective treatment for MMN. Temporal improvement was related to the administration of immunoglobulin. Repeated infusions induced sustained improvement. Deterioration was observed when the dose administration was delayed, and improvement was sustained upon regular (monthly) administered high-dose. The evolution after IVIg administration in MMN is similar to those reported in chronic inflammatory demyelinating neuropathy.3

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This is a practical, easily read book with a sensible clinical orientation on the epilepsies and seizures. The approach and organisation is logical and follows accepted terminology and classification. The basic mechanisms and causes of epilepsy are comprehensively discussed and the chapter on pathophysiology is particularly clear. The illustrations are well represented, supportive to the text and often excellent.

It is useful in one volume to have the variety of epileptic syndromes outlined; not always in the detail a specialist would demand, but with particular reference to the chosen readership, which is that of physicians in training and practising physicians. The indexing and references make it of additional value to the practising neurologist and neurophysiologist.

It is necessary to turn to more specialised texts for precise indications for surgery and new pharmacological approaches, which will of necessity emphasise the changing scene in therapeutics of epilepsy. There is a pleasant balance achieved by attention to community needs in epilepsy and a historical background. The wise counselling on the place of the electroencephalogram will be appreciated by clinical workers in the field. The statement that the most common cause of unwarranted diagnosis of epilepsy is over clinical interpretation of the EEG, and that epilepsy is not an EEG diagnosis but clinically based deserves quotation. As a contemporary review by an experienced neurologist, it covers in considerable detail the mosaic of the epilepsies. It is instructive, particularly for physicians and neuroscientists in training and to the clinical neurophysiologist.

Seizures and Epilepsy reads agreeably, it is thought provokingly didactic and is recommended.

JR HERON

BOOK REVIEWS

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Dr Smith and Professor Taylor have assembled review articles from the major contributors to the study of neurological complications occurring during heart surgery. They have deliberately concentrated on the cerebral injuries. The informative review of the relevance of carotid artery disease in the prediction of cerebral damage by Professor Harrison concludes that the excess risk is rarely enough to warrant prophylactic or simultaneous endarterectomy. This is followed by data defining the risk of neurological and psychological complications of putting in an artery bypass and heart transplantation.

The middle section consists of a review of investigative techniques used to monitor and assess cerebral damage suggesting that cerebral blood flow monitoring is an important and useful technique but that the present state of cerebral function monitors precludes their usefulness. Retinal fluoroscopy, angiography and digital image analysis of such angiography during bypass is described, though little comment is made about the potential damage to the eye caused by these techniques during surgery.

The final section analyses those interventions in blood gas management, arterial filtration and the use of membrane rather than bubble oxygenators, which techniques have reduced the risk of cerebral damage, and considers the use of excitotoxic amino receptor antagonists and platelet antagonists, concluding that although the former are worthy of study, the latter, complicated as they are by risks of hypotension, appear ineffective in reducing damage.

In the summary Mr Treasure recognises the considerable advances achieved and the consequent problems in assessing future therapies created by this success. The fact that only 1-2% of bypass operations now result in permanent neurological deficits indicates that studies of novel agents and techniques will need to recruit large numbers of patients to show a statistically significant benefit.

The selection of topics and the instructions to authors to review their topic and then to comment on future possibilities has resulted in a comprehensive, up to date and well referenced volume which covers the field of cerebral complications of heart surgery.

DAVID BATES

NOTICE

1993 Meeting of the European Federation of Neurological Societies

The meeting will be held in Berlin on 8-11 December 1993, and will focus on topics in cerebrovascular diseases, epilepsy, and migraine. Further details can be obtained from P & R Kongresse GmbH, Monika Portsmann, Neue Promenade 6, D-10178 Berlin, Germany. Tel. +49-30-2825113; fax +49-30-2827835.