Sera of 20 patients without neurological disorders were screened for borrelia infection and used as normal control cases. In two of GBS cases (15%) antibodies of IgG class were detected in the serum (at titres of 1:128 and 1:256 respectively), but not in the CSF. In the same sera sampled two months later antibodies to borrelia were no more detectable. The clinical course of the disease in the two positive cases did not differ from other negative GBS patients and they recovered in 6–8 months. Only one of the normal control cases showed antibodies to borrelia at the titre of 1:16, which is not considered as significant of infection.[9,10]

At least half of the patients with GBS suffer a few weeks before the onset of the disease an infective illness which can trigger an immune-mediated reaction involving the peripheral nervous system.[1,11] Viral infections of the respiratory or gastrointestinal tract, surgical procedures, vaccinations, non-viral agents like *Mycoplasma pneumoniae* or *Campylobacter jejeuni* or gram-negative bacteria infections have been described as antecedent diseases.[1,12] The presence of antibodies in the serum but not in the CSF of typical cases of GBS, together with their disappearance in the following months, suggests that in a few cases a borrelia infection can be the antecedent illness precipitating the immune-mediate disorder of acute inflammatory demyelinating polyneuropathy. Borreliosis should be added in the numerous preceding infections of GBS.

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Winer and Hughes reply:

We were interested to read the report of Mancardi and his colleagues on the serological identification of borrelia infection on the basis of a transiently raised IgG titre among two out of 13 patients with the Guillain-Barré syndrome. In our own study of one hundred patients with acute idiopathic neuropathy we were able to identify a specific antecedent infection in 31% serologically. A further 24% could recall symptoms of unexplained gastrointestinal or respiratory infection in the month preceding the neuropathy. None of these 24 patients had arthalgia or skin rash. Three had more than 5 white cells/μl in the CSF.

A case controlled study of borrelia serology using both IgM and IgG assays of serum and CSF would be of interest. Controls with such a study should include patients with inflammatory conditions which might cause a non-specific rise in total serum IgG.

Book reviews

Neuromuscular Disease, 2nd ed. By M Swash, MS Schwartz. (Pp 456; £99.00.) London: Springer, 1988.

The first edition of this book was widely acclaimed and it achieved a secure place as a "bench book" for those involved in the management of children and adults with disorders of the neuromuscular apparatus. The second and revised edition (with 202 illustrations) is a larger and more comprehensive account of these disorders, particularly the peripheral neuropathies and metabolic muscle disease. It is a welcome addition to the literature on peripheral neuropathology, the photomicrographs of muscle biopsy material particularly being measurably superior to those in the first edition (although the authors have retained a few illustrations of *longitudinal* cryostat sections which are pictorially unpleasing, technically unsatisfactory and correspondingly difficult to interpret). However, the question of balance arises when considering the chapters dealing with peripheral neuropathies. There is a profusion of (good) photo- and electronmicrographs of myopathology but only a few illustrations of the commoner peripheral nerve disorders, genetically determined and acquired. Presumably this is a reflection of the special interest of the senior author but it does make for a rather large "bare area" in the middle of an otherwise well-arranged and presented volume. Illustrations of cyclical demyelination/remyelination and axonal degeneration in teased fibre preparations and of some of the more dramatic peripheral neuropathic changes would have been welcome, for examples "tornaculous" neuropathy and "glue sniffer's" neuropathy. Contrariwise, one wonders why the authors felt it necessary to include references to SMON, lathyrism and syringomyelia in a consideration of predominantly or exclusively lower motor neuron dysfunction. Some of the references in the chapters on peripheral neuropathy appear to be rather antediluvian.

The chapter dealing with disorders of neuromuscular transmission is an admirable summary of an area in which breathtaking progress has been made since the publication of the first edition. Any criticism here inevitably seems carping but the lighting of the patient in Fig 12.2a is poor and a CT scan of the chest might have been more impressive than the lateral chest radiograph illustrated in Fig 12.2b.

It is a pleasure to welcome this new edition of an old friend to the neuromuscular literary