Rhabdomyolysis during interferon-β 1a treatment

Interferon-β (IFN-β) is one of the most effective currently available treatments for multiple sclerosis. It has also been used in the therapy of viral diseases and certain malignancies, as has the other type 1 interferon IFN-α. Most frequent side effects are transient flu-like symptoms such as myalgia, chills, and headaches. We describe a patient with relapsing-remitting multiple sclerosis who developed acute rhabdomyolysis during IFN-β 1a treatment.

After the medication was discontinued, the patient improved rapidly. A 39 year old man with a history of first symptoms in April 2000 was diagnosed as having relapsing-remitting multiple sclerosis, supported by the demonstration of oligoclonal IgG bands in the CSF but not in the blood, and multiple white matter lesions in periventricular localisations on MRI. Treatment with 22 µg IFN-β 1a (Rebif®, Serono, Unterschleißheim, Germany) by subcutaneous injection three times weekly was initiated in October 2000, after three exacerbations with predominant sensory disturbances leading to an expanded disability status score (EDSS) of 1.5. To alleviate potential flu-like symptoms due to IFN-β therapy, the patient was recommended to take 400 mg ibuprofen at least 2 hours before and after the time point of injection. Because he did not recognise any adverse side effects, he first stopped omeprazole medication, which he had taken occasionally, and thereafter the ibuprofen medication. He reassured us that he did not use any other drugs not prescribed by his physicians. Thus, 3 months after initiation of IFN-β treatment the patient was only on this immunomodulatory therapy. One month later, he suddenly developed acute generalised myalgia as well as weakness 1 day after IFN-β application. An MRI of the brain at this time demonstrated haemosiderin deposition from chronic subacute cerebellar ataxia. Apart from bilateral sensory neuritis and spinal cord demonstrated haemosiderin deposition from chronic subacute cerebellar ataxia. Apart from bilateral sensory neuritis and spinal cord...
de sac (fig 1). There were no other abnormalities on the MRI and a carotid and spinal angiogram failed to disclose a source of bleeding within the CNS. The patient declined a lumbar puncture to look for evidence of active haemorrhage. There was no history of CNS trauma or surgery.

Neuropsychological examination showed normal sensory nerve conduction. Motor conduction was essentially normal. Electromyography of the first dorsal interosseous and extensor digitorum communis muscles demonstrated fibrillations and fasciculations with high amplitude units. Somatosensory evoked potentials were normal from the arms but showed delayed latencies in the legs.

A diagnosis of superficial siderosis was made and he was given a trial of subcutaneous desferrioxamine fortnightly for 8 weeks with no benefit. The patient has continued to deteriorate.

Superficial siderosis of the CNS is a clinical syndrome characterised by progressive cerebellar ataxia and sensorineural deafness. Pyramidal signs develop in 76% and other features that may occur include dementia, dystonia, and sensorineural deafness. In the review of Fearnley et al of 63 patients four had lower motor neuron involvement with absent or diminished reflexes thought to be secondary to arachnoiditis or radiculopathy. One patient had muscle wasting with brisk reflexes thought to be due to concurrent lower motor neuron pathology and myopathy. In our patient the duration of the symptoms and the lack of bulbar and pyramidal features were against this being a classic amyotrophic lateral sclerosis. It is more likely that superficial siderosis was the cause of our patient’s anterior horn cell dysfunction and it is recognised that iron pigmentation may be found deep within the spinal cord and other normal observations have been described. The clinical picture of anterior horn cell damage in superficial siderosis is of particular interest as in the review of Fearnley et al they note that although heavy haemosiderin deposition is recognised in the anterior horns of the spinal cord there is little in the way of neuronal fall out.

The predominance of CNS involvement and the paucity of lower motor neuron features in superficial siderosis has been the subject of several novel studies. Koepen and Borke have shown that an intracisternal injection of red cells produces increased synthesis of ferritin in microglia, especially Bergmann glia in the cerebellum, and this binds with iron to form haemosiderin. It is postulated that the glia and astrocytes of the central nervous system respond to the presence of haemoglobin whereas this process does not occur in Schwann cells of the peripheral nervous system. This is supported by the pathological finding that there is a sharp demarcation of haemosiderin deposition in the cranial nerves and spinal roots at the junction of the central glial and perineuronal Schwann cell segments. Koepen and Detinger have also suggested that the formation of haemosiderin is neuroprotective and it is once this protection has been exhausted that tissue damage occurs, thus it is not the haemosiderin which is toxic but the unbound iron. There are no other case reports of superficial siderosis causing an anterior horn cell syndrome, posing the question of why our patient developed this combination. Whether our patient’s presentation was due to anomalous intracellular processing or an unusual source of haemorrhage impacting on the spinal cord remains speculative. It is also possible that in our case the motor root exit zone is a site of iron deposition with resultant lower motor neuron pathology.

We think that our case of superficial siderosis with anterior horn cell dysfunction is unique, and raises interesting questions about pathological mechanisms in this rare disorder.

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References

Use of intrathecal baclofen for treatment of spasticity in amyotrophic lateral sclerosis

Baclofen, an agonist of γ-aminobutyric acid, is one of the most effective drugs in the treatment of spastic movement disorders. However, higher oral dosages required for sufficient spasticity control are related to intolerable central side effects. In this situation, continuous intrathecal application of baclofen in microgram dosages has documented its efficacy in numerous series of patients with spasticity of cerebral or spinal origin. Nevertheless, the use of intrathecal administered baclofen in amyotrophic lateral sclerosis, representing the most common degenerative motor neuron disease in adult life, has been mentioned in only one short communication. In this context our experience with intrathecal baclofen therapy is worth presenting. These two patients are the only ones we have treated in this manner and both experienced a marked improvement in their quality of life.

Patient 1, a 25 year old man, was previously reported in brief; he is still alive and benefiting from intrathecal baclofen therapy. Five years ago he noticed progressive gait disturbance, weakness of his right foot, and painful nocturnal cramps in his legs. At that time he exhibited neurologically mild pareses of his right hand and foot, generalised fasciculations, and spasticity. Amyotrophic lateral sclerosis was diagnosed and oral antispastic medications were supplied with a nasofacial mask for non-invasive intermittent ventilation to alleviate
symptoms of nocturnal hypoventilation. He has been followed up now for 49 months, and no complications related to intrathecal baclofen therapy have been seen.

Patient 2, a 39 year old man, experienced progressive stiffness and weakness of his legs 2 years ago. Amyotrophic lateral sclerosis was diagnosed, and medical treatment consisting of riluzole and baclofen was started. Initially the patient remained ambulatory for 6 months but then he rapidly developed a severe tetraplegia. He was able to stand with help, but confined to a wheelchair otherwise and completely in need of care. The major sources of discomfort were frequent nocturnal pain attacks due to uncontrolled spasms and central side effects related to oral baclofen medication. Intrathecal baclofen therapy was initiated, and at a daily dose of 80 µg painful spasms stopped. Despite preservation of some spasticity on purpose for support and improvement in general ease of care.

None the less, quality of life was improved considerably as the patient was able to sleep of some spasticity on purpose for support and improvement in general ease of care. Although progressive pareses result in increasing debilitation of life. It is a marked reduction of disabling spasticity that helps to achieve these goals and not the influence on prevalent muscle weakness. Our clinical findings show that even in the terminal phase of the disease the patients still benefit by relief of painful spasms, making intrathecal baclofen therapy possible. This form of palliative treatment has proved to be a safe procedure without substantial risks.

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References

No male predominance in α-synuclein Parkinson’s disease but the affected female fetus might be less viable

In their recent article on the clinical phenotype in Greek patients with α-synuclein Parkinson’s disease (α-sPD) Papapetropoulos et al. reported male predominance (60%) in their patients. The authors concluded that the sex ratio in their families does not differ significantly from patients with sporadic idiopathic Parkinson’s disease (3:2). But then he rapidly developed a severe spastic tetraplegia. He was able to stand with help, but confined to a wheelchair otherwise and completely in need of care. The major sources of discomfort were frequent nocturnal pain attacks due to uncontrolled spasms and central side effects related to oral baclofen medication. Intrathecal baclofen therapy was initiated, and at a daily dose of 80 µg painful spasms stopped. Despite preservation of some spasticity on purpose for support and improvement in general ease of care.

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References
As Horstink and Bloem suggest, the segregation ratio for men and women is indeed the most appropriate method to calculate the genetic risk for developing a disease. In our recent publication,1 the sex ratio was calculated from the sample of 15 patients with α-synuclein Parkinson’s disease (α-synPD) included in the study. We now provide additional mathematical data to calculate the segregation ratios and to compare them with the other published series of patients with α-synPD.

In the 10 families examined in our study,190 members were at risk of developing α-synPD. Of the 103 male members at risk, 27 (26.2%) developed Parkinson’s disease (PD), whereas of the 95 women at risk, 27 (28.4%) developed PD (p = 0.73). When our data were combined with the data computed by Horstink and Bloem, the segregation ratio of all known subjects at risk of developing α-synPD was 149/135 = 1.10 (p = 0.22 for the difference from a 1:1 ratio). The ratio of 103/90 = 1.14 (p = 0.08 for the difference from a 1:1 ratio) was observed for the segregation ratio of family members at risk for acquiring α-synPD. This is a very interesting finding, which raises several issues for the diagnosis and management of sporadic adult onset cerebellar degeneration. I make three comments. Firstly, there is another clinical presentation of “Hashimoto’s associated ataxia”, consisting of an acute cerebellar syndrome associated with abnormal behaviour. Protein concentrations are increased in CSF. Brain MRI shows a high intensity signal in T2 weighted images, restricted to the cerebellum. This other presentation should not be overlooked because steroids and thyroid hormonal therapy improve the cerebellar deficits markedly. This ataxic syndrome associated with Hashimoto’s thyroiditis differs from the disorders reported recently1 by (1) the acute onset, (2) distinct MRI findings, and (3) the dramatic clinical/radiological response to treatment which is a strong argument in favour of an immune attack against the cerebellum.

The authors should consider multiple system atrophy (MSA) in the differential diagnosis of sporadic adult onset cerebellar degeneration.2 Various combinations of pyramidal, extrapyramidal, pyramidal, and autonomic features occur in MSA. The disorder having an estimated prevalence ratio of 1.6-8.0/10003 raised the possibility of antithyroid antibodies being a coincidence. Patient 6 exhibited cerebellar deficits associated with autonomic/urinary dysfunction, pyramidal signs (bilateral Babinski’s signs), and parkinsonism (axial rigidity, hyperpronia).4 Multiple system atrophy is likely in this patient. Were splinter EMG studies performed? Were dysautonomic signs significantly looked for in other patients?

In one of our patients exhibiting a chronic and sporadic cerebellar syndrome with atrophy, high concentrations of antinuclear antibodies and presence of a rheumatoid factor were initially considered as markers of an immune disease producing a cerebellar degeneration. However, a subsequent genetic testing disclosed a spinocerebellar ataxia type 6 (SCA-6). Genetic analysis for SCA-1 to 7 was performed in one of the patients reported by Selim and Drachman,1 and was not available in the remaining five patients. Detailed genetic tests should be carried out, even when there is no family history of ataxia. Recent studies show that about 4% of patients with a sporadic ataxia harbour a mutation.5 Negative genetic results would reinforce the appealing concept of “Hashimoto’s associated ataxia”.

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References

1. Selim M, Drachman DA. Ataxia associated with Hashimoto’s disease: progressive non-familial adult onset cerebellar degeneration.1 Raised concentrations of antithyroid antibodies were found. Modest increases in antithyroid antibodies were considered to be the result of longstanding autoimmunized thyroid disease. Analysis of CSF showed increased protein concentrations in one patient. Brain MRI disclosed atrophy of the vermis in four patients and showed a concomitant atrophy of the brain stem in two patients. Treatment with L-thyroxine did not improve cerebellar signs. The authors suggested that ataxia associated with Hashimoto’s disease could be due to an autoimmune cerebellar degeneration.

Table 1 The segregation ratios of all α-synPD cases reported

<table>
<thead>
<tr>
<th>Family members at risk for α-synPD</th>
<th>Family members with α-synPD</th>
<th>Male (segregation ratio)</th>
<th>Female (segregation ratio)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reported α-synPD cases</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Papapetropoulos et al1</td>
<td>103</td>
<td>95</td>
<td>27 (26.2%)</td>
</tr>
<tr>
<td>Papadimitriou et al4</td>
<td>14</td>
<td>17</td>
<td>6 (42.8%)</td>
</tr>
<tr>
<td>Golbe et al5</td>
<td>86</td>
<td>56</td>
<td>39 (45.3%)</td>
</tr>
<tr>
<td>Samii et al1</td>
<td>32</td>
<td>23</td>
<td>10 (31.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>235</td>
<td>191</td>
<td>82 (34.9%)</td>
</tr>
</tbody>
</table>

α-synPD = α-synuclein Parkinson’s disease.
Conversion hysteria: towards a neuropyschological account


It probably would not have pleased Aubrey Lewis to know that one of his lasting legacies to psychiatry is his now often quoted words from his paper The survival of hysteria that the effect of hysteria will outlive its obstinators. It seems that in the past decade, almost in defiance of the mighty DSM (emphasis added), humble hysteria is not only again a popular topic for investigation, but its very name lives on. In this book, the term hysteria is unashamedly used, even in the title, and it is not cloaked by its suit of new invisible clothes dressing up as somatoform disorders or one of their variants.

This small but very readable book is a collection of papers published as a special edition by the Journal of Cognitive Neuropsychiatry. The aim was to bring modern neuropsychological theory to the field, to balance the “traditional overreliance on psychodynamic accounts”. However, it also embraces the paradigm, encapsulated over a century ago by Charcot, that hysteria should be open to investigation as other conditions in medicine, using the same methods and observation techniques. Thus, in the text, several of the chapters touch on the more recent neuroimaging and evoked potential studies. These seem fairly consistent, noting the important association of the frontal and cingulate cortex to symptoms present in hysteria. However, it is unclear whether thyroperoxidase antibodies target Purkinje cells, or whether the increase in antithyroid antibodies reflects a disconnection of these processes. The widening of the field, to balance the “traditional overreliance on psychodynamic accounts”, is not cloaked by its suit of new invisible clothes dressing up as somatoform disorders or one of their variants.

The most interesting contributions, however, are the newer cognitive neuropsychological approaches to the subject. For, and here Lewis would surely have seen, hysteria lingers on in clinical practice, as seen in patients galore, in different forms, especially in settings such as medicolegal practice. Yet, the mechanisms leading to the florid manifestations of hysteria in these patients may be part of a psychological tendency available to a much broader community. This extent perhaps should not be regarded as pathological.

The main thrust of the book, and the most relevant for those engaged with such patients, reflects on this theme. The wider patient social construct is emphasised, the relevance of the concept of the Will is debated (another famous quote, from Paget, repeated here: “The patient says he cannot, it looks like he will not, but the truth is that he cannot say ‘will’”), and the role of consciousness re-examined. For sure, one of the most tedious yet well-intentioned questions that doctors are asked when discussing such patients is “is it conscious, or unconscious”?

The text provides elegant ways of resolving this dilemma, in themes in keeping with modern cognitive psychology. Like a good book reviewer, I will not give away the game away, but encourage the interested to read the texts herein published. One quote will help the temptation: “In an attempt to encapsulate the self-deceptive view of hypnotised subjects, they have been labelled as “Hypnotic liars”. By the same token, hysterics should perhaps be seen as “genuine malingerers” (“Oakley”).

Michael Trimble

Movement disorders in children


This is a truly marvellous book. The authors combine a vast clinical experience with an up to date review of literature scattered throughout neurological and paediatric publications to produce the first textbook on movement disorders in children. A clinical approach to movement disorders in childhood is taken with chapters devoted to the predominant movement disorder. Clinical descriptions and illustrations are given for all the important conditions producing that movement disorder; comprehensive lists of the rarer causes of movement disorders are also provided. The authors start by reviewing general concepts relevant to the diagnosis of movement disorders in childhood. The current model of basal ganglia functioning is discussed and this is followed by a description of the main types of movement abnormalities. This is followed by a brief, but important, guide to specific areas of clinical history taking and examination in the evaluation of movement disorders in children. Subsequent chapters are based on the predominant movement disorder. Each is organised with an initial introduction and classification followed by a discussion of the major disorders producing abnormal movements. Important features of each chapter are a discussion of conditions that may simulate the movement disorder; the discussion of difficult cases according to the authors’ own experience and from the search of the literature; and the search for treatment of the individual movement disorders. Relevant investigations are also presented within the context of each individual movement disorder. A chapter is also devoted to those complex movement disorders where one type does not predominate. The movement disorders covered include the hypokinetic-rigid syndromes, tremor, chorea, dystonia, myoclonus, tics, and complex. There are some important chapters on genetic movement disorders and movement disorders in cerebral palsy. Last is a chapter about ancillary investigations that either have, or may well in the future, prove useful in our understanding of paediatric movement disorders. Throughout the book, additional authors have contributed their own expertise.

This is a comprehensive and up to date textbook about movement disorders in children. All child neurologists and paediatricians with an interest in neurology or neurodisability should have access to this book and I suspect that most will want to own a personal copy. In addition, this book will be extremely useful to adult neurologists evaluating patients with a movement disorder the origins of which are in childhood or adolescence.

R Surtees

Advances in dementia research


This book is a collection of presentations from a symposium “Ageing and Dementia” held at the end of 1999 in Graz. As always with such collections, the book is as good as the presentations were and some of these are excellent and useful, others are worth reading. One or two could just have provided an opportunity to go and have a look around Graz. What the book is not is a systematic review of advances in dementia research and probably books are not good places to turn to for such reviews, as this material is generally best accessed directly from the journals themselves. The book starts well enough with an interesting variety of papers on the relation between vascular damage to the brain and dementia. Some of the articles are non-systematic and short reviews, others are more thoughtful discussions of the important questions in dementia research, and others are straightforward data presentations. I was left with more questions than answers, which is probably healthy. The papers in the book then go on to discuss other important issues in dementia research, including neuroinflammation, apoptosis, mitochon- drial dysfunction, and genetics. Some of the most important advances came just a little bit after this book was published. The discussion of transgenic models of Alzheimer’s disease for
example includes no discussion of transgenic models of the frontal lobe dementias even though that is clearly related and the various papers on immunological approaches do not include any of the amyloid vaccine data.

A more general question arises, to my mind, however, reading this book, as to quite who else is likely to read it. If read by somebody coming new to the dementia field they would have a very unbalanced picture of the field and this is not be recommended to novices to dementia research. On the other hand, those familiar with dementia research are unlikely to treat this book as other than a handbook. Interestingly the final six or so chapters are on a proprietary compound which is being developed for treating Alzheimer’s disease. According to one article, this compound is widely used to relieve symptoms in various neurological disorders, which was new to me. A previous meeting held in 1997 also resulted in a book very similar to this one and is advertised in the back. My other strike is that the book is poorly edited and written.

A review of the 1997 meeting book, published in Acta Psychiatrica Scandinavica and used as a promotional blurb mentions that “The book will be of interest to those following the development of neurotrophic factors for treatment of dementia who need an extensive introduction to the clinical application of this proprietary compound. Things haven’t changed much.

Simon Lovestone

Limbic seizures in children


Limbic seizures in childhood differ from those in adults. They are more likely to be caused by cortical dysplasias, related malformations, and tumours. They are more easily, but not invariably, controlled by drugs. Have new imaging and EEG techniques advanced the cause and effect debate about febrile seizures and mesial temporal sclerosis (MTS)? Such considerations make this monograph timely. Initial chapters on the history of the subject, the evolving definition of what constitutes limbic structures, their functional organisation and the relevance of MTS are clear, instructive, and thought provoking. “Limbus” is a border, in this case the border between the midbrain and the rest of the cerebral hemisphere. In non-primates, primarily concerned with smell, it has decreased in size relative to the elaborating neocortex, but in so doing has acquired multiple connections with association cortices. The hippocampus and perihippocampal cortex are distinguished by several features. Their cell properties are particularly determined by the level of activity—long term potentiation or depression. The learning properties coupling short term memory. Whereas the hippocampus may code memories by semantic association, the hippocampus assigns them a personal context in time and space. The number of possible associations and ways of filing past events is almost infinite. The original roots in olfactory function may linger as the powerful evocation of memories by smell (Madeleine cakes served Proust for his life’s work). It is possible that the flexibility and enhanced activity of certain hippocampal circuits on which its function is contingent make it particularly liable to epileptogenesis.

Many patients with catastrophic epilepsy do not have MTS. Seizures themselves do not cause MTS. Seizures per cent de-patients with MTS have dual pathology, 15% have increased neuronal heterotopias, and 15% have bilateral involvement. Degree of cell loss is not related to duration of epilepsy. Mossy fibre sprouting is not seen in children younger than 2 years, suggesting that this is a secondary progressive lesion. From facts such as these Spencer et al conclude that mesial temporal lobe epilepsy has a probable developmental aetiology. Hippocampal abnormalities pre-exist (and can be demonstrated in unaffected members of familial temporal lobe pedigrees) but convey vulnerability to febrile convulsions and subsequent MTS.

Subsequent chapters treat different aspects of limbic seizures—language disturbances, motor automatisms, impairment of consciousness, autonomic changes, and postural changes. The literature distinguishing frontal from mesial lobe epilepsy in children is easily situated. The complex partial seizures is summarised. There are chapters on structural and functional imaging.

This book arose out of a colloquium. Of the 26 contributors included all but four are from French or Italian centres. The two from America are particularly good and perhaps the standard of the rest might have been higher if the net had been spread wider. Some authors speak from very limited experience. The chapters on treatment are particularly disappointing. That systematic errors in English abound and much information is repeated throughout implies lack of adequately firm editorial grip.

This book will be useful to paediatric epileptologists, but the patchy quality overall precludes a warm recommendation to a wider audience.

Richard Robinson

Spinal cord injury desk reference. Guidelines for life care planning and case management


This is a reference text. It contains information that will be of considerable assistance to those who are involved in the planning of the long term care of those with spinal cord injury in the United States. The authors, all United States spinal cord injury physicians, a behavioural scientist, and a rehabilitation counsellor.

The information in this book will assist pre-dominantly those healthcare professionals who are closely involved in the case management of spinal cord injury. It will also be of interest to all who are involved with spinal cord injury including patients, their families, and all the many groups who work in the area including doctors, nurses, therapists, social workers, healthcare planners, lawyers, and many others. For those who already have wide knowledge of spinal cord injury care the chapter on resources and legislation may be of particular value as it contains numerous addresses and telephone contacts.

Even though the specific information in this book is largely relevant to the United States, many outside that country will find the book of interest, perhaps encouraging them to seek the comparable data relevant to their own countries. The information contained in texts such as this is a prerequisite to ensuring that there is adequate appropriate long term provision for people with spinal cord injury, especially as they age.

Inevitably in a wide ranging book there are weaknesses. For example, the debilitating orthostatic hypotension induced fatigue and coat hanger pain experienced by many persons with higher level spinal cord injury is not mentioned and the 1993 rather than the more complete 1998 United Kingdom life expectancy data are used. These omissions do not detract from the importance of this book’s attempt to fill an important niche that has not been adequately addressed before.

It would be of great interest if similar texts were produced in other countries. Not only would this help improve long term care in these countries but it would also enable comparisons of costs and approaches to care to be made that would assist the improving systems of care for patients with spinal cord injury worldwide.

Head trauma: basic, preclinical, and clinical directions


Miller and Hayes have assembled chapters from 42 expert contributors renowned for their work in investigation of traumatic brain injury. They have divided the text into three main sections, basic science overview, preclinical studies, and clinical directions.

Organising the text in this way the authors have struck a theme which passes from experimental concepts through to preclinical feasibility studies and eventually on to clinical trials. They acknowledge from the outset that the wealth of basic scientific information gathered over the past 3 decades has not led to substantial clinical gain. The reasons for this are debated in a latter chapter.

The work represents a comprehensive review of the information available on traumatic brain injury. The basic science overview I found to be particularly well written and concise, introducing concepts and experimental data in a highly readable way. The main theories of cytotoxicity, inflammatory response, apoptosis, traumatic axonal injury, and haemodynamic dysfunction have separate attention, as do the important vascular aspects of severe head injury.

The final section refers to the clinical efforts of attempting to translate scientific knowledge into clinical work. The preclinical trials organised in the United States, Europe, and Asia are discussed and potential reasons for their failure debated.

In succinct, and for example the work of Miller and Hayes is a valuable addition to the reading of those involved in traumatic brain injury. This is particularly so for those who engage in the experimental and clinical design of novel therapies for the traumatised brain.

Peter J Kirkpatrick
Meeting the challenge of progressive multiple sclerosis


Having been diagnosed in 1982 I have lived for 19 years with a slowly progressive form of multiple sclerosis. I was therefore glad of the opportunity to catch up on recent developments in the understanding of the disease and discussion of some of the latest options for treatment. Although the book states in the opening paragraph that it is written for people with this form of multiple sclerosis, it is also obvious from the first page that it is going to be very hard work for anyone without a scientific or medical background to make sense of the information it contains. I constantly found myself having to reread and struggle to understand the technical language used throughout the book. Such a pity when there is much potentially useful information there.

I was interested to see what the writers would have to say in the section on management and self help since this is an area the medical profession has often overlooked. There is discussion under various headings such as coping with fatigue, bladder dysfunction, tremor, and cognitive dysfunction, followed in each case by a series of bullet points on the management of symptoms. Once again the language defeats the object of the book as these read more like checklists for doctors and multiple sclerosis nurses than clear, accessible summaries that people with multiple sclerosis can make use of.

It is heartening to see that in these days of disability legislation (the Disabilities Act in the United States and the Disability Discrimination Act in the United Kingdom) questions of access to buildings and equipment and discussions of legal rights and financial entitlements are seen as having a place in a book on multiple sclerosis. The past 19 years have taught me that factors such as attitudes towards disabled people, the design of buildings, and the way in which services are delivered may impact on the lives of people with multiple sclerosis and their families just as much as the effects of the disease.

Michele Wates
Hashimoto's associated ataxia

M-U Manto

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