Febrile Hashimoto's encephalopathy

Hashimoto's encephalopathy is a rare association of Hashimoto's thyroiditis. The clinical course is often characterised by relapses.1,2 Persistent fever has not been previously recorded in the literature.

A 48 year old white woman was referred in April 1996 from a psychiatric clinic, where she had presented with psychotic episodes with gradual deterioration and been diagnosed as possibly having Creutzfeldt-Jakob disease. She was confused and had brisk tendon reflexes, an external left plantar response, bilateral moderate cogwheeling, action myoclonus, and nuchal rigidity. The gait was unstable and uncoordinated.

Several psychosis occurred at the age of 22 and from the age of 30 she had periodic mild confusional states of about a week long. Hashimoto's thyroiditis was diagnosed 10 years before the neurological admission and since then had been on thyroxine replacement, although confusional states appeared periodically, roughly every 3–4 months.

A few days before admission the clinical features changed, with variable stupor, generalised seizures and myoclonus, incontinence, and nuchal rigidity. The gait was unstable and uncoordinated.

Her recent past medical history included psychosis at the age of 22 and from the age of 30 she had periodic mild confusional states of about a week long. Hashimoto's thyroiditis was diagnosed 10 years before the neurological admission and since then had been on thyroxine replacement, although confusional states appeared periodically, roughly every 3–4 months.

After admission the clinical features changed, with variable stupor, generalised seizures and myoclonus, incontinence, and anorexia. There was a persistent fever which varied between 37.7 and 39°C, with variable stupor, generalised seizures and myoclonus, incontinence, and nuchal rigidity. The gait was unstable and uncoordinated.

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After admission the clinical features changed, with variable stupor, generalised seizures and myoclonus, incontinence, and anorexia. There was a persistent fever which varied between 37.7 and 39°C, with little or no response to antipyretic drugs. Antiepileptic drugs achieved some control of the seizures.

Normal or negative laboratory tests included blood count, CSF analysis, erythrocyte sedimentation rate, liver and kidney function tests, coagulation screen, serum and CSF electrophoresis, immunoelectrophoresis, ANCA, anti-DNA, ASMA, antimitochondrial, antiphospholipid, and antinuclear antibodies, C reactive protein, tumour markers, serum copper, ceruloplasmin, serum electrolytes, vitamin B12 and folic acid, serological tests for HIV, HSV, HBV, CMV, syphilis, Widal, Wright, and PPD. Blood, CSF, and urine cultures were negative. The EEG showed diffuse slow activity. Brain CT and MRI were normal. T3, T4, TSH, FT3, and FT4 were normal but the titre of autoantibodies to thyroid peroxidase (Anti-TPO) was increased at 7000 mIU/L (normal value<60 mIU/L).

Treatment with prednisolone (70 mg/day) led to gradual improvement and the fever ceased promptly. Within 2 months the patient had good recovery on 40 mg/day prednisolone and had no relapses during 3 subsequent years (maintenance prednisolone dose 15 mg every other day, Anti-TPO titre within normal range).

The clinical features in this case are compatible with a diagnosis of Hashimoto's encephalopathy with an associated fever. Both fever and Anti-TPO concentrations were suppressed with prednisolone treatment. Increase of TNF-α concentration is seen in the serum of patients with autoimmune thyroiditis3 and this is known to act as an endogenous pyrogen.4 This mechanism may explain the persistent fever as a prominent symptom in our case.

Devic's neuromyelitis optica and HIV-1 infection

Neuromyelitis optica (Devic's syndrome) can be defined as a severe transverse myelitis, an acute unilateral or bilateral optic neuritis, no clinical involvement beyond the spinal cord or optic nerves, and a monophasic or, rarely, a multiphasic illness.5 Optic neuritis precedes transverse myelitis in 80% of cases, by less than 3 months in most cases. Neuromyelitis optica is often a result of demyelination from multiple sclerosis, but many differences have to be noted between neuromyelitis optica and multiple sclerosis. Neuromyelitis optica is not predominantly seen in the white ethnic groups. Severe deficits after the acute episode are more frequent in neuromyelitis optica. Multiple sclerosis presenting as transverse myelitis is rare.6 Oligoclonal bands in the CSF and white matter lesions on brain MRI are rare in neuromyelitis optica compared with over 90% of patients with definite multiple sclerosis. These two abnormalities can resolve in neuromyelitis optica but very rarely in multiple sclerosis. The high CSF protein concentration and pleocytosis seen in 60% of cases are often more important in neuromyelitis optica. Swelling and signal change on MRI extending beyond a single segment are more frequent in neuromyelitis optica and cavitation can be seen. Most authors consider that neuromyelitis optica is a variant of postviral acute disseminated encephalomyelitis7 mostly due to varicella zoster virus, but other causes have been identified — for example, systemic lupus erythematosus, antiphospholipid syndrome, and pulmonary tuberculosis. To the best of our knowledge, only one case of neuromyelitis optica (folowed by a bilateral acute retinal necrosis) due to varicella zoster virus has been described in a patient with AIDS.8 We report the case of an HIV infected African woman developing neuromyelitis optica. A 41 year old woman from the Congo Democratic Republic was admitted to hospital in March 1999 because of a 10 day progressive blindness of the left eye with optic disc oedema. There were left leg dysaesthesiae of 1 month duration and a T6 left sensory level was noted within the next few days. Progressive ascending paralysis of both legs without bowel or bladder dysfunction developed. HIV infection had been diagnosed 11 years earlier. Her CD4 lymphocyte count was 499/mm3 and her plasma viral load was 6.0 log (1150 RNA/ml). She received no treatment. Examination of CSF showed 57 white blood cells/mm3 (94% lymphocytes). Total protein concentration was 48 mg/dl. No oligoclonal bands were detected. Serology for syphilis and Borrelia burgdorferi and Cryptococcus neoformans antigen were negative in blood and CSF. Search for herpes simplex virus type 1 and 2, varicella zoster virus, cytomegalovirus, and Epstein-Barr virus DNA polymerase chain reaction remained negative in CSF. Serology for HTLV-1 was negative. Interferon and angiotensin converting enzyme concentrations were normal in blood and CSF. Cultures of CSF were negative. Antinuclear and anti-cardiolipin antibodies were not detected. Brain MRI with and without gadolinium DPTA was normal. Spinal cord MRI disclosed four high signal intensity lesions on the T2 weighted images within the spinal cord (80 mg/day) for 12 days and treatment with lamivudine (300 mg/day), zidovudine (600 mg/day), and nevirapin (2250 mg/day) was started. Symptoms dramatically improved. The patient was able to walk alone without help after 10 days. Visual acuity returned to normal. Dysesthesias were absent 1 month later. Three months later, plasmatic viral load was under 50 RNA/ml. Examination of CSF and MRI of the spinal cord gave normal results. There was no recurrence after a 5 month follow up.

Optic neuritis occurring in patients infected with HIV9 is usually due to syphilis or to opportunistic organisms such as varicella zoster virus, cytomegalovirus, Cryptococcus, Histoplasma, Bartonella, Toxoplasma, or Mycobacterium tuberculosis. The role of HIV itself is now well established. Intramedullar involvement in the course of HIV infection may result from HIV itself (vacular myelopathy), coinfection with HTLV1 in endemic areas, or transverse myelitis mostly due to varicella zoster virus, cytomegalovirus, or cryptococcus. Such infections were excluded in our patients. There are only few reports of multiple sclerosis-like illness occurring with HIV infection.10 Cases of sarcoidosis associated with HIV infection remain exceptional even when highly active antiretroviral therapy (HAART) is used11 (optic neuritis is rare in sarcoidosis and no case of neurosarcoidosis has been described with AIDS). Primary CNS lymphoma associated with HIV is rare12.13,14,15 Associated with Epstein-Barr virus and may cause optic neuritis or spinal cord involvement. We think that multiple sclerosis, sarcoidosis, and lymphoma were excluded in our patient. Neuromyelitis optica without a known cause can be seen in the course of HIV infection, even at an early stage of the disease, before immunodepression occurs.

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because there are so few studies of taste in multiple sclerosis plaque at the Hemiageusia from an ipsilateral reflex was normal on stimulation on the right, nerve stimulation were normal. The blink evoked potentials, brainstem auditory evoked intrathecal IgG, and oligoclonal bands. Visual mm 3 Merle H, Smadja D, Cordoba A. Neuromyélite 1 Mandler RN, Davis LE, Jefferies RD, et al. Devic’s neuromyelitis optica: a clinicopathological study of eight patients. Ann Neurol 1993;33:162–8.

Hemiageusia from an ipsilateral multiple sclerosis plaque at the midpontine tegmentum

The exact location of the pontine gustatory pathway has not yet been clarified, probably because there are so few studies of taste in patients with well localized brainstem lesions. Here we report on a patient with isolated hemiageusia and trigeminal sensory neuropathy from a single pontine lesion.

A 46 year old woman experienced a burning sensation on the left side of the tongue. The next day she discovered a loss of taste on the entire left half of her tongue and numbness on the left side of her face. Neurological examination was normal except for numbness on the left side of her face. Norgren et al described a patient who presented with ageusia and ipsilateral trunci ataxia presumably due to bra- chium conjunctivum involvement, and, therefore, it was suggested that the adjacent parabulbar nucleus might constitute a pontine taste area. Our patient developed left sided hemiageusia and trigeminal sensory disturbance, and electrical stimulation on the left elicited no R1 response and absence of masseter reflex. These electrophysiological abnormalities imply ipsilateral brainstem lesions at the trigeminal principal sensory and motor nuclei, respectively. Moreover, MRI confirmed the existence of a new gadolinium-enhanced demyelinating lesion in the left midpontine tegmentum. The precise correlation between our patient’s symptoms and the electrophysiological and MRI abnormalities indicates that the involvement of the central tegmental tract, which is the anatomical structure adjacent to the sensory and motor trigeminal nucleus at the midpontine level (fig C), is probably important in causing taste disturbance. According to this hypothesis, Norgren et al showed in primates that axons of neurons located in the solitary nucleus ascend in the central tegmental tract to the ventroposteromedial nucleus of the thalamus without terminating first in the pontine parabulbar nucleus.

Use of lamotrigine to treat paroxysmal kinesigenic choreoathetosis

We report the use of lamotrigine to treat paroxysmal kinesigenic choreoathetosis. Paroxysmal cases of kinesigenic choreoathetosis were first described in 1962 by Lishman et al and the term “paroxysmal kinesigenic choreoathetosis” was first coined by Kertesz in 1967. Recently, a more universal and potentially useful classification of these disorders has been proposed amending the terminology to “paroxysmal kinesigenic dyskinesia”. However, we have used the more familiar terminology for our patient as it is more precise. The clinical features of paroxysmal kinesigenic choreoathetosis have been reviewed by Marsden and Luders.

There is a male preponderance of the condition with about 50% of cases inherited as an autosomal dominant trait, the rest being sporadic. Attacks are precipitated by sudden movements or startle and may be unilateral, bilateral, or affect alternate sides. Often the cause is idiopathic but a few cases have been attributed to multiple sclerosis. There has been no clear evidence for seizure activity in this disorder even though the condition is very responsive to antiepileptic drugs such as phenytoin and carbamazepine.

A 13 year old boy of unrelated parents and with no family history of neurological disorder presented with a 6 month history of muscle spasms affecting the left side of his body. Close questioning indicated that his symptoms were brought on during the initiation of sudden movements (such as starting
to run) and manifested as spasm of the left side of the face and flexor spasm of the left arm and left leg. There were no premonitory symptoms and consciousness was normal during the attack. Attacks lasted for a few seconds only and he felt perfectly well afterwards. Neurological examination was normal. Brain MRI was normal and three EEGs performed over the course of the next year were all normal. There were no biochemical or immunological abnormalities; copper studies were normal. A diagnosis of paroxysmal kinesigenic choreoathetosis was made. He was allergic to carbamazepine and did not respond to sodium valproate.

Lamotrigine at a dose of 50 mg twice daily, completely abolished the involuntary movements. On two occasions, these returned on stopping lamotrigine and were abolished by reinstituting treatment. Our patient has taken lamotrigine for 8 years without any side effects. He now only experiences symptoms of paroxysmal kinesigenic choreoathetosis if he omits his medication. The response to lamotrigine may provide insight into the pathogenesis of the disease, suggesting that it may be caused by an ion channel defect; these are known to be responsible for some paroxysmal neurological conditions. As far as we are aware, this is the first report of the successful use of lamotrigine to treat paroxysmal kinesigenic choreoathetosis.

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6 Hanna MG, Wood NW, Kullmann DM. Ion channels and neurological disease: DNA based diagnosis is now possible, and ion channels may be important in common paroxysmal disorders. J Neurol Neurosurg Psychiatry 1998; 65: 427–31.

Chronic inflammatory demyelinating polyneuropathy as a complication of cutaneous scratch disease

Cat scratch disease (CSD) was first described in 1950 as a benign regional lymphadenitis. This infection is caused by Bartonella henselae. The clinical range of CSD has expanded beyond the classic presentation. In 5%–20% of the infected patients the disease may spread to other organs. However, neurologi-
peripheral nerves. Ten patients had neuroretinitis, two children had paresis of the facial nerve, and three adult women complained of neuralgia. One case study presented a peripheral facial nerve paralysis as a complication of CSD.

Up until now CIDP has never been reported as a neurological complication of CSD.

Given the history and clinical course, the electrophysiological and nerve biopsy findings, coupled with the strongly positive serology to B. henselae, we think that the CIDP in this patient is a direct complication of CSD. CIDP is an autoimmune process in which this patient is a direct complication of CSD.

In our patient a delayed myelin destruction is induced by sensitised macrophages, originally activated by the Bartonella infection. Therefore, we hypothesise that the pathophysiology of both central and peripheral nervous system complications following CSD infection shares a similar immunological mechanism.

The authors reply:

We thank Derakhshian for his comments on our case report of iatrogenic disseminated encephalomyelitis after use of the suckling mouse brain postrabies exposure vaccination. We were not responsible for the postexposure vaccination, which was administered in a provincial hospital in central VietNam. However, in the circumstances (and in the absence of the human diploid cell tissue culture vaccine) we think that it was appropriate to use the vaccine in this case. The dog had previously been well behaved and it was highly uncharacteristic for it to bite its owner. After the event the dog disappeared into the forest and was not seen again. Hence, it was not possible to retrieve the brain for analysis, as is usual in most cases.

The mortality from rabies is essentially 100%, a figure that can be reduced dramatically by the expeditious use of the suckling mouse brain vaccine after exposure. We agree with Derakhshian’s comments on excessive enthusiasm for any medication, and obviously the relative risks and potential benefits must always be balanced. In a disease with a 100% mortality, where a potentially effective treatment is associated with a severe side effect in only 1:27 000 cases it would seem reasonable to use the treatment. At this centre we vaccinate 2000 people every year after a dog bite, we see about 30 people a year die of rabies. We would therefore anticipate seeing a case of iatrogenic disseminated encephalomyelitis after use of the suckling mouse brain postrabies exposure vaccination once every 13.5 years. In the same period we would see 675 people die of the disease. Hence, it was not possible to retrieve the brain for analysis, as is usual in most cases.

In the second sample of the study, 113 patients completed an ADL questionnaire at three points in time (at waking, best, and worst) before any drug intake. This was done at baseline. The authors determined that sleep benefit was present when the mean ADL score difference between best and worst was more than 12—that is, when strong variations occurred in baseline score before medication.

The authors state that no objective sleep benefit was measured. Unfortunately they do not indicate if the correlation was found at any point in time or if all evaluations were lumped together, as no correlation index or graph is given.
Finally, we would like to add a word of caution and remind the authors that it would be wise to avoid deriving the measure of “objective duration” of sleep benefit from three scales filled in at home by patients without any further instructions than to fill them at waking, and during best and worst before drug intake.

As the only dopamine agonist mentioned in this study is bromocriptine, we would be grateful to know what year the study was conducted in.

In any case, although “little is known about sleep benefit” any study concerning this phenomenon should certainly attempt to increase knowledge and avoid a further increment of confusion.

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Intracranial dural fistula as a cause of diffuse MRI enhancement of the cervical spinal cord

We read the recent short report by Bousson et al. on spinal MR findings in a patient with progressive myelopathy and intracranial dural arteriovenous fistula with great interest.

We recently had a 42 year old man admitted as an emergency with a 3 week history of stepwise altered sensation in both lower limbs ascending to the torso which progressed to weakness involving his legs and hands. Two years before this he had an episode of severe backache associated with a tight band of pain around the waist and significant bilateral leg weakness. Resolution occurred only after 4 months, when he was able to walk normally. On the current admission examination showed a spastic tetraparesis; there was minimal increased tone in the upper limbs, mild weakness of the small muscles of both hands, and marked pyramidal weakness of the legs with extensor plantar responses. He was unable to support his weight and in urinary retention. He had a sensory level at T5 although dorsal column function was preserved.

Brain and spinal cord MRI showed increased signal in the medulla extending into the upper cervical cord down to C4 (figure). Slightly prominent vessels were seen overlying the right cerebellar hemisphere and a varix was visible close to the torcula. There was no enhancement in the cord or medulla and no abnormal flow void in the spinal veins. The changes were thought to represent a spinal cord infarct and in view of the “stuttering” course in his history we proceeded to cerebral angiography. This showed an arteriovenous fistula supplied by the left middle and posterior meningeal artery and both occipital arteries. Venous drainage was into prominent varices lying just to the left of the midline and in front of the transverse sinus and then on the transverse sinus itself.

After an unsuccessful attempt at embolisation via the arterial route, the fistula was occluded by packing the varix with Guglielmi detachable coils.

He made an uneventful recovery; after 2 months of intensive neurorehabilitation he recovered full function in his upper limbs and now has sufficient power in his legs to be able to walk with the aid of crutches.

This case emphasises that the intracranial arterovenous fistula should be included in the differential diagnosis of increased signal on MRI of the cervical cord, even when dilated veins are not, as in this case, very apparent. Procedural symptoms can occur and a careful history in a patient with ascending paraparesis and tetraparesis is essential. Endovascular occlusion at these fistulae can lead to useful improvement in neurological function.

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Inversve relation between Braak stage and cerebrovascular pathology in Alzheimer predominant dementia

Goulding et al. carried out a preliminary retrospective postmortem analysis of 25 patients (13 men, 12 women, mean age 80.7 years) with the clinical diagnosis of Alzheimer-type dementia (only one with suspected multi-infarct dementia) and a 36.4% frequency of the ApoE4 allele. Eighteen brains (89%) with neuritic Braak stage ≤4 had either additional cerebrovascular lesions (n=14), or Lewy bodies (n=3), or both (n=6), with a significant inverse correlation between cerebrovascular lesions and Braak stage. Forty eight per cent of the brains showed small focal in- farcts, and only 20% disclosed “pure” Alzheimer’s disease pathology. No association between the E4 allele and any pathological variable was found. Based on these data, the authors emphasised the importance of screening for concomitant pathology in Alzheimer’s disease, in which a cerebrovascular component has been suggested as an

MRI of lower brain and spinal cord.
additional pathogenic factor. These data can only in part be confirmed by personal experience in a cohort of 27 necropsy cases (13 men, 14 women) aged 77 to 91 (mean 85.9) years with the clinical diagnosis of degenerative dementia (possible or probable Alzheimer’s disease) in two, of vascular dementia in two, and Parkinson’s Alzheimer’s disease in one, studied between 1989 and 1998. Mnini mental stages (n=14) ranged from 6 to 15 (mean 8.0). Apolipoprotein performed from paraffin blocks after a polymerase chain reaction (PCR) method disclosed the ε4 allele in 33%. In addition to the neuropsychological evaluation criteria by Gourley and McKeith ‘NIA’ and CERAD criteria for Alzheimer’s disease were used.

The following data were obtained.

The NIA criteria for Alzheimer’s disease were positive in all but three cases which diagnosed a senile dementia of the tangle type. 44% staged CERAD A and 28% staged each CERAD B and C. Only 16/27 brains (59%) showed additional cerebrovascular lesions, either white matter changes alone (n=3) or in combination with lacunar state or small vessel infarcts in the basal ganglia (n=13), or an old infarct in the area of the left posterior cerebral artery (n=1). Six of 27 (22%) showed additional subcortical (n=4) or both subcortical and cerebral white matter lesions (n=2). Association with cerebrovascular lesions was seen in eight cases staged Braak 4 and 5 (or 5 and 6). When looking at the severity of the associated cerebrovascular lesions, severe ones (combined cerebrovascular disease II or III) were seen in seven brains with Braak stage 4 and in six staged Braak 5 (or 6), mild ones (cerebrovascular disease I) in one brain staged Braak 4, and in two with Braak stages 5 or 6. Carriers of the ApoE ε4 allele staged Braak 4 in five cases and Braak 5 in three, with additional cerebrovascular lesions in three brains each. Although in our small necropsy series similar to that of Gourley et al., “pure” Alzheimer’s disease was seen only in 22% (three cases each with Braak stages 4 and 5), in view of our data, the inverse relation between cerebrovascular disease and Alzheimer’s disease, suggested by these authors, requires further confirmation in larger and possibly prospective clinicopathological case series.

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Link between the CSF shunt and achievement in adults with spina bifida

We read with interest the results of Hunt et al in their long term follow up of spina bifida survivors with intraventricular shunts.1 In 48 shunt treated adult patients, they find that 27.1% (13 patients) live independently, 35.4% (17 patients) drive a car and 25% (12 patients) are in open employment. They also report that those requiring shunt revision, particularly those who have had more than one revision, have lower levels of achievement as defined by these three criteria. Overall, 22.9% (11 patients) are “community walkers”.

The clinic for adult spina bifida and hydrocephalus (CASBAH) in Belfast receives referrals on a regional and non-selective basis from paediatric services after the age of 16 years. Of those currently attending the clinic, 95 are shunt treated survivors. There are 30 men and 45 women, average age 25.6 years (range 16–39 years). Fifty three patients (55.8%) are wheelchair dependent, 7 (7.4%) are largely wheelchair dependent but retain some ambulatory capacity, 4 (4.2%) are ambulatory with aid, and 17 (17.9%) are independently ambulatory. Overall 35/95 (36.8%) can be considered “community walkers”.

Of 61 patients in whom complete data are available, 22 (36%) are in full or part-time employment, 10 (16.4%) are students (six of these in higher/further education), three (4.9%) are in fully fledged training schemes, two receive sheltered training, and 21 (35%) are employed. Seventy patients (32.7%) are regular drivers and a further two (3.3%) are actively learning to drive. Eight patients are married (13.1%) and three are parents (4.9%). In five patients (8.2%), epilepsy is an active problem.

Our data therefore suggest a rather more optimistic outcome in terms of mobility and employment for long term shunt treated survivors of spina bifida. Interestingly, and again by contrast with the data of Hunt et al, these figures differ little from those for our whole clinic population, shunt and non-shunt treated survivors, where 34% are in employment, 33% are employed, 32% are regular drivers, 17% are married or engaged, 8% are parents, and 8% have epilepsy. Although it is possible that the more severely affected shunt treated patients simply did not survive into adulthood, the lack of this confounding bias, these figures would indicate that the presence of a shunt and the potential for subsequent revision has rather less of an impact on prognosis than is suggested by their paper.

We would, however, agree with their conclusion that ease of access to a neurosurgical unit is essential for infants and children with spina bifida. We would further recommend that this be continued in adulthood as seven of our patients have required surgical intervention for a Chiari/hydrocephromyelographic complex at that stage, another shunt related complication.1

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BOOK REVIEWS


It was a pleasure to review this CD Rom. It is certainly a sign of changing times that the book review section now also contains reviews of CD Rom material. This is basically a review of the use of botulinum toxin in the treatment of cerebral palsy built and edited by the staff in the Department of Neurology in Innsbruck in May 1997. The CD Rom contains the talks of eight speakers recorded at the time. While listening to the speaker’s voice the slides come on and off the screen at the right moment. Thus, it is simply like being at the talk itself in Innsbruck. If this idea catches on then we may begin to see the end of international medical travel! The final section contains two of the speakers (not in the Department of Neurology) and this is thus a waste of time. I suspect this is a technical fault as the quality of the video pictures was very good but obviously needed some verbal context to imparting information. I suspect that we shall see a lot more of this methodology in the coming years and this is to be welcomed.

Finally, it is worth pointing out that the computer system requirements are quite high to run the video adequately. Preferably a modern Pentium machine is required with at least 16 Mb of Ram and, obviously, a multimedia configuration with a high quality sound card.

Overall, a novel approach to an important topic and one to be recommended.

MICHAEL P BARNES


Those familiar with previous incarnations of McAlpine’s Multiple Sclerosis will know that authors have changed from one edition to the next and with that the emphasis has changed as well. This third edition is no exception and it is almost entirely a new book. It has six outstanding authors, each bringing formidable authority to bear on their own and their colleagues’ contributions. The book is big and aims at a more comprehensive coverage of the subject than previous editions. It is, however, no mere indiscriminate assemblage of facts, but very much reflects the views, indeed the vision (individual and collective), which the authors have of the pathogenesis and natural history and epidemiology of multiple sclerosis.

Professor Alastair Compston has the lion’s share of the writing, and is in his element on epidemiology and genetics and the cellular biology of neuronal and glial cells. He starts the book with an absorbing and scholarly historical piece.

Far from resting on his laurels Professor Bryan Matthews has extensively rewritten and updated his chapters on symptoms and signs and differential diagnosis.

Magisterial is a word Compston is fond of using to describe the accomplishments of luminaries from the past and it could reasonably be applied to Professor Ian McDonald’s professional lifetime of laboratory and clinical research which is reflected in his chapters on pathophysiological and diagnostic methods and investigations.

There are stimulating and provocative chapters on the natural history and on neuropathology. Arguably we all need to try to understand the immunology of multiple sclerosis, particularly in the current era of therapeutic strategies aimed at modifying the course of the disease. Clinicians may think that a chapter on animal models of multiple sclerosis will not be of immediate relevance to their day to day work, and may think that more space could have been apportioned to some aspects of symptomatic treatment and the management and rehabilitation of patients with moderate or severe disability.

This is a very fine book; instructive, edifying, and enjoyable, chiefly because of the writing but also because of the quality of the print and the abundance of excellently reproduced illustrations.

RODNEY WALKER


This book is a good description of the normal anatomy and imaging findings of some of the most common clinical conditions affecting the brachial plexus. I am sure that this will be useful for neuroradiologists and general radiologists confronted with clinical questions about brachial plexus pathology.

The book summarises the author’s own experience during 5 years at Addenbrooke’s Hospital. This involved 230 MRI studies performed in patients with suspected brachial plexus pathology. The book is overall well written, in simple and concise language. Tables and good quality figures are well explained, and contain interesting information. The number and quality of the references are adequate. The book is divided into six chapters. This division results in a well balanced book in general, although some chapters may be too extensive when one considers the more limited author’s own experience. The anatomical description of the brachial plexus in chapter 2 is one of the best depicted parts of the book, and I am sure that it will be very useful for radiologists. On the contrary, the section dealing with imaging techniques is somewhat long and confusing. Some studies comparing the efficacy of different techniques are missing, something which I am sure would have enhanced the work significantly, and at the end of this section, the reader does not really know which imaging techniques to choose for a simple examination of the brachial plexus. I was more than pleased that in the chapter on trauma and the thoracic arlet syndrome that the author’s experience agrees with my own of MRI not being very helpful here. There is a chapter on radiation induced brachial plexopathy, not an infrequent disorder in hospitals with large oncology units, but only three cases are presented. The last chapter deals with inflammatory conditions such as multifocal motor neuropathy and chronic inflammatory demyelinating polyradiculoneuropathy, and the ability of MRI to differentiate them from others having a similar clinical presentation (lower motor neuron disease). In my opinion, this is probably the most interesting chapter of the book, as it covers a novel subject.

BEATRIZ GOMEZ-ANSON


The editors claim to provide a “state of the art review of the role of SPECT in neurology and psychiatry”. The 64 chapters are divided into 11 sections, covering dementia, stereotopsychology, psychiatry, movement disorders, epilepsy, paediatrics, cerebrovascular disease, tumours, trauma, specific applications, and physical techniques. Although the emphasis is on SPECT, the role of PET is also mentioned. Most chapters represent the results of the authors’ research and experience; unfortunately this piecemeal approach does not lend itself to a cohesive text. Although most sections commence with a brief review it would have been conducive for the reader if these reviews were more comprehensive, this could then have provided the entree for the subsequent detailed studies.
It is unfortunate that the first section on dementia, most probably the major indication for SPECT at present, lacks an overview. Furthermore, there is little mention of the OPTIMA project (Oxford prospective investigation into memory and ageing)—the largest ongoing study in the world with histological confirmation. Presumably, this is partially a reflection of the inherent delay in the production of such a wide ranging text.

In essence this is a reference text, and despite the foregoing criticisms, this book does fulfil a niche in the literature. Neuroradiologists, psychologists, and other relevant clinical specialists will find their appropriate sections to be extremely useful in demonstrating the contribution of PET/SPECT investigations and also showing the new areas of development. Trainees in nuclear medicine and neuroradiology would benefit by studying the review chapters. Certainly, for the nuclear medicine specialists with an interest in neuroimaging, this text is an essential acquisition.

**PAUL KEMP**


This multiauthor review is based on a symposium on new antiepileptic drugs (AEDs). The book suffers from the usual difficulties with symposia publications including too many chapters which are often too short to usefully summarise the topic in detail but with much repetition and a lack of consistency and style. Nevertheless, excellent practical guidance is given in several chapters, including those on monotherapy, combination therapy, and the management of infants and children. For most patients with epilepsy none of the new AEDs are more effective than standard current first line treatment, with which we have many patient-years of experience. Marketing of the new drugs against each other as add on or treatment resistant group and as practical experience with the drugs in clinical practice is obtained it would be appropriate to design a randomised study comparing several of the new drugs against each other as add on or replacement therapy.

Further toxic effects of the new drugs are almost certain to be discovered. For example, at the time of the symposium the effect on visual fields of treatment with vigabatrin was not widely recognised and this is not covered in the text.

**STEVE WROE**


This is an interesting, well written, and useful book—but will not sell many copies. Regrettably it seems pitched at various different audiences and thus I suspect it will not greatly appeal to any particular group. Nevertheless I would thoroughly recommend this book. It contains some excellent descriptions of complex subjects and should be of considerable interest to all practitioners working within the field of multiple sclerosis.

The first part provides an overview of genetics. The first chapter is entitled “What the specialist in multiple sclerosis needs to know about genetics”. It is an excellent overview and easily understandable by the non-specialist. The second part of the book covers the field of immunology. Some of the chapters in this section are rather short and of somewhat patchy quality but nevertheless still provide the non-specialist with a good summary of our present state of knowledge. The chapter, for example, by Neil Scolding on oligodendrocyte injury and the role of complement provides a good update on the subject. The next chapter on cytokines is a little less clear to the non-specialist but nevertheless is useful. The following chapters in this section then become rather too specific to be of use to the non-specialist and probably too superficial to provide any new information to the specialist in the field. The third section covers MRI and once again the same pattern predominates: good updates in the subject but I am uncertain as to whether they are directed to the specialist or non-specialist. The chapter on diffusion MRI is an exception and is beautifully written. The fourth section discusses therapy. John Noseworthy provides his usual excellent contribution on emerging therapeutic options in multiple sclerosis but the chapters on gene therapy and the rationale for antiviral therapies are of somewhat less interest to the clinician.

In the final part(3) the book broadens out even further and discusses various aspects of the organisation of multiple sclerosis care. Personally I found this section of considerable interest but I suspect that those at the more scientific end of the multiple sclerosis spectrum will not find this section very enthralling. However, more scientific colleagues should certainly read the two excellent chapters by Alan Thompson and Jeremy Hobart on advances in multiple sclerosis rehabilitation and an update on outcome measurements.

Overall this book tries to be everything to everybody and thus probably fails in the market place. However, it would be a pity if some of the excellent chapters were lost to a wider audience—so buy it anyway.

**MICHAEL BARNES**

**CORRECTION**


Page 292, para 2. During the editorial process, the sentence “The limit of detection on microscopy is 100 mycobacteria/ml” was altered. It should read “The limit of detection on microscopy is 10 000 mycobacteria/ml.”
Febrile Hashimoto's encephalopathy

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