<table>
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<th>Title</th>
<th>MRI in vertebral artery dissection (multiple letters) [9]</th>
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<tr>
<td>Author(s)</td>
<td>Cheung, RTF; Mak, W; Tsang, KL; Auer, A; Felber, S</td>
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MRI in vertebral artery dissection
R T F CHEUNG, W MAK, K L TSANG; A AUER and S FELBER

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Facial diplegia with paraesthesias: facial nerve enhancement in three dimensional MRI

Facial diplegia (bilateral facial paralysis) is a rare clinical finding that can be the presenting symptom in a wide range of diseases. It occurs in about 50% of patients with Guillain-Barré syndrome (GBS). Guillain-Barré syndrome causes regional and functional variants with unusual features. Ropper described four patients with facial diplegia and distal limb paraesthesias, and he defined them as having a rare variant form of GBS because of shared clinical, electrophysiologic, and CSF features. The aetiology and nosological position of facial diplegia presenting in this variant form is still controversial. We experienced a patient who had bilateral facial paralysis, distal limb paraesthesias, and diminished reflexes whose contrast enhanced three dimensional MRI (3-D MRI) showed enhancing lesions in the bilateral facial nerves.

A 27 year old woman had nasal discharge and coughing. One week later she noticed paraesthesias in her fingers and toes. Nine days after the onset of her neurological symptom, she developed bilateral facial weakness. On admission (day 12) she showed moderate, bilateral facial paralysis that caused her difficulty in moving her forehead, in approximating her eyelids, and in lifting the corners of her mouth and masticatory movements. The other cranial nerves were normal. A motor examination showed normal strength in her limbs. Superficial and deep senses were normal even though she had distal limb paraesthesias. Deep tendon reflexes were absent in all her limbs and her plantar responses were flexor type. Cerebellar ataxia and autonomic nervous dysfunction were excluded. Chest radiography was normal. Laboratory studies of the identifiable causes of facial diplegia (sarcoidosis, Lyme disease, syphilis, infectious mononucleosis, herpes simplex virus, diabetes mellitus, and connective tissue disease) were all negative. On day 12, the CSF examination detected mild increases in protein concentration (57 mg/dl) without pleocytosis. The blink reflex was elicited and both the R1 and R2 components were reduced, but their latencies were not prolonged on day 15. Motor and sensory nerve conduction velocities, and median and tibial nerve frespersions were all normal on day 18. Auditory brainstem responses were normal.

On Day 19, when her facial diplegia was moderate, conventional brain MRI detected no abnormality. A contrast enhanced 3-D MRI, which was obtained by spoiled gradient recalled acquisition in the steady state sequence using a 1.5 Tesla system after injection of gadolinium-diethylenetriamine penta-acetic acid (Gd-DTPA), was performed. The intracanalicular segments of the bilateral facial nerves were remarkably enhanced by Gd-DTPA (figure). On Day 45, when her symptoms were no longer present, there were no abnormal enhancements of her facial nerves.

After showing symptoms of upper respiratory infection, the patient experienced the acute onset of facial diplegia, distal limb paraesthesias, and areflexia but no other neurological deficits. The CSF examination showed albuminocytological dissociation, and clinical and laboratory examinations excluded the possibility of viral or bacterial infection, Lyme disease, tumour, sarcoidosis, cerebrovascular disease, diabetes mellitus, bilateral Bell's palsy, and congenital and familial disorders. The patient's illness followed a monophasic course. We therefore diagnosed this case as having "facial diplegia with paraesthesias", which should be included for the differential diagnosis whenever sudden bilateral facial paresis occurs. Routine brain MRI showed no abnormalities, whereas contrast enhanced 3-D MRI showed Gd enhancement of the bilateral facial nerves. The MRI findings indicate the involvement of the peripheral facial nerves in our patient.

It is widely understood; however, it is widely understood that the aetiology and nosological position of facial diplegia presenting in this variant form is still controversial. We experienced a patient who had bilateral facial paralysis, distal limb paraesthesias, and diminished reflexes whose contrast enhanced three dimensional MRI (3-D MRI) showed enhancing lesions in the bilateral facial nerves. The mechanism of abnormal enhancement of the cranial nerves in the patients with GBS is not entirely understood; however, it is widely regarded as disruption of the blood nerve barrier by the inflammatory infiltrate. Ramsey et al. evaluated the MRI findings obtained with Gd contrast enhancement in five patients who had classical facial paralysis: GBS (n=1), herpes simplex polyneuritis (n=1), meningeval lymphoma (n=1), and bilateral Bell's palsy (n=2). Gd enhanced MRI has been shown to be the procedure of choice for diagnosing inflammatory lesions of the facial nerves. Nagaoka et al. showed oculomotor nerve enhancement on 3-D MRI in Fisher's syndrome, the best known variant of GBS. Ours is the first report of facial nerve enhancement in "facial diplegia with paraesthesias". These findings suggest that 3-D MRI with Gd-DTPA can be used to identify inflammatory conditions that produce peripheral lesion of the cranial nerves in GBS variants.

Restless legs syndrome associated with spinal cord lesions

Restless legs syndrome may be either a primary or a secondary disorder. The primary form of the syndrome is often familial whereas the secondary form is mainly associated with iron deficiency or pregnancy. Almost all patients with restless legs syndrome show periodic leg movements during sleep. The pathogenesis of both restless legs syndrome and periodic leg movements is still speculative. Yokota et al. have reported an association of periodic leg movements with spinal cord lesions. However, none of these patients had the typical clinical features of restless legs syndrome. Restless legs syndrome associated with myelopathy is documented in one patient with a Borrelia induced myelitis. We report three patients who developed a restless legs syndrome in close temporal association with spinal cord compression.

Case 1 was a 35 year old woman who presented with a 3 week history of painless restlessness of her left lower leg that occurred only at rest, particularly in the evening and at night. The restlessness was partially relieved by walking. With the onset of these symptoms, the patient had noticed a numbness of her left hemibody below the breast. Nine months previously, a numbness of the right leg had subsided spontaneously within 1 week. On examination, the patient had decreased senses for touch, pain, and temperature over the left hemibody below the T6 dermatome. Examination of the trapezius revealed normal cell count, normal protein content, increased CNS synthesis of IgG, and positive oligoclonal bands. In MRI studies of the spinal cord, no abnormalities were detected. 3-D MRI was not performed. Transcranial magnetic stimulation showed a slightly prolonged central conduction time of motor evoked potentials recorded over the left abductor hallucis muscle. Otherwise, multifocality and evoked potentials were normal. A myelitis due to multiple sclerosis accompanied by a symptomatic unilateral restless legs syndrome was diagnosed. The patient was treated with 500 mg prednisolone intravenously over 5 days without any clinical effect. However, a single dose of 100 mg levodopa plus benserazide led to a dramatic improvement of the restless legs syndrome. The levodopa treatment was continued and resulted in complete relief.

Case 2 was a 49 year old man who had a traumatic atlantoaxial dislocation that necessitated operative stabilization of the cervical spine. Preoperative MRI studies had shown a compression of the medulla and the cervical cord. When we saw the patient 3 years later, he complained of a sensation of cold, pain, and restlessness in both lower limbs that was present only at rest, particularly in the
evening, and was relieved by walking around and rubbing the legs with cold water. The onset of these symptoms was only a few weeks after the accident. Clinical examination disclosed a mild dysarthria and atrophic paralyses of the left sternomastoid and the left rectus muscle. Tendon reflexes were hyperactive and plantar responses were extensor bilaterally. The patient’s gait was spastic, but he was able to walk unassisted. In both legs, pain and temperature sensation were markedly reduced. The diagnosis of a restless legs syndrome secondary to a traumatic lesion of the medulla and the cervical cord was made. Treatment with 100 mg levodopa plus benzerazide and 100 mg tramadol resulted in a satisfactory relief of the restless legs syndrome.

Case 3 was a 65 year old man who developed slowly progressive spastic tetraparesis and ascending sensory disturbances in both legs. A MRI study showed a cervical spondylyotic myelopathy at the level C3-C6 and the patient underwent spinal cord decompression. Five years later, he was referred to our hospital because of an intense sensation of restlessness of both legs located in the feet and calves. The restlessness occurred when sitting and lying for more than 20 minutes. It was pronounced at night and was relieved by walking around. The symptoms had started simultaneously with the motor and sensory disturbances due to the cervical spondylyotic myelopathy and did not improve postoperatively. On examination, the patient was mildly impaired in carrying out motor tasks and his gait was moderately spastic. He had reduced touch and vibration sensibility in both upper limbs. A restless legs syndrome due to a cervical spondylyotic myelopathy was diagnosed. Treatment with pergolide resulted in an excellent control of the restless legs syndrome.

Our patients meet the criteria for the diagnosis of restless legs syndrome.1 Over a follow up period of at least 6 months, restless legs syndrome symptoms were sufficiently relieved by dopaminergic treatment. The association of myelopathy and restless legs syndrome may be merely coincidental. However, the close temporal relation between the onset of myelopathy and restless legs syndrome strongly suggests that restless legs syndrome was secondary to the spinal cord lesion. The pathogenesis of restless legs syndrome and periodic leg movements is still speculative. In patients with myelopathy and periodic leg movements, it is hypothesised that a spinal cord lesion may permit the expression of a restless legs syndrome due to myelopathy which may respond well to dopaminergic drugs.

**Coma in thrombotic thrombocytopenic purpura**

Patients with thrombotic thrombocytopenic purpura (TTP) can present with devastating neurological abnormalities.1 Mortality may be as high as 95%, but current treatment has reduced this to about 10% and early treatment improves the rate of recovery.2 We describe two patients who presented with predominantly neurological symptoms and signs who, because of a delay in making a diagnosis of TTP, were referred for treatment at a late stage. Both patients were reviewed by neurologists and haematologists, who considered that the prognosis was poor.

The first case was a 49 year old woman with a longstanding diagnosis of schizophrenia and a previous left sided cerebrovascular accident. She was admitted to her local hospital with a 3 day history of drowsiness, confusion, epistaxes, and spontaneous bruising, having been noted to be increasingly agitated and disoriented over the preceding 6 weeks. Her only medication was trifluoperazine and paroxetine. The second case was a 58 year old man, previously fit and well, who presented to his local hospital with a 3 week history of confusion, drowsiness, jaundice, and right upper quadrant pain. He was taking no medication. The initial findings in both patients are summarised in the table. In both a diagnosis of TTP was made, although this was not confirmed by laboratory tests until admission in the first case, and both patients were transferred to the intensive care unit for plasma exchange and further management.

Treatment was started in both cases with five cycles of plasma exchange, each cycle consisting of using 31 cryoedeployed fresh frozen plasma, and in the first patient this was followed by a course of oral prednisolone and azathioprine. Both made an excellent recovery, with an improvement in consciousness level, a rise in platelet count, disappearance of red cell fragments, a fall in LDH and bilirubin concentrations, and normalisation of renal function. The first patient was self ventilating with no neurological deficit at time of transfer back to the referring hospital. The second patient had a Glasgow coma score of 15 by the fifth day of treatment, the only focal neurological being a bilateral internuclear ophthalmoplegia (INO). Three months later the ophthalmoplegia had resolved and the patient was self caring with minimal disability. Both patients were extensively investigated to look for an underlying cause for TTP, but none was found.

Thrombotic thrombocytopenic purpura is a syndrome comprising a pentad of features—fever, thrombocytopenia, microangiopathic haemolytic anaemia, neurological abnormalities, and renal dysfunction. Not all five features are required to make the

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**Table: Summary of patients**

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>Patient 2</th>
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<tbody>
<tr>
<td>Age (y)</td>
<td>49</td>
</tr>
<tr>
<td>Sex</td>
<td>F</td>
</tr>
<tr>
<td>Platelet count (x10³/µl)</td>
<td>19</td>
</tr>
<tr>
<td>Peripheral blood film</td>
<td>RBC fragments +++</td>
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<tr>
<td>Clotting screen+fibrinogen</td>
<td>Normal</td>
</tr>
<tr>
<td>Bilirubin (µmol/l)</td>
<td>67</td>
</tr>
<tr>
<td>Lactate dehydrogenase (U/l)</td>
<td>931</td>
</tr>
<tr>
<td>Creatinine (µmol/l)</td>
<td>121</td>
</tr>
<tr>
<td>Glasgow coma score</td>
<td>3/5</td>
</tr>
<tr>
<td>Midposition, non-reactive pupils, disconjugate gaze in all directions; extension of all four limbs to painful stimuli; plantars equivocal</td>
<td></td>
</tr>
<tr>
<td>Brain CT</td>
<td>Multiple foci of high intensity on the T2 weighted images in white matter of both cerebral hemispheres and poles of lateral ventricles</td>
</tr>
<tr>
<td>Brain MRI</td>
<td>Raised ICP, nil else.</td>
</tr>
</tbody>
</table>
diagnosis—often fewer are present—and there is no pathognomonic test, so diagnosis may be difficult. It is often considered along with haemolytic uraemic syndrome (HUS) to form part of a range of diseases called the thrombotic microangiopathies. In these disorders, intravascular platelet aggregation (there is minimal fibrin deposition) leads to obstruction of arterioles and capillaries, causing local ischaemia. Thus TTP is seen when the cerebral microcirculation is affected, and HUS when the renal microcirculation is affected. An episode of TTP may present as a one off illness, may be recurring, 1 or may arise in association with drugs, neoplasia, pregnancy, or HIV infection.

Thrombotic thrombocytopenic purpura presents with neurological manifestations in over 50% of episodes, with headache, confusion, and somnolence being most common, leading to focal neurological deficit, convulsions, and eventually coma and death. 2 These clinical features are often fleeting and fluctuating and several important points regarding investigation should be made. Firstly, brain CT may be normal or may show multiple hypodense areas indicative of generalised cerebral oedema. 3 Secondly, brain MRI may also be normal, although it is likely to show minimal changes on T2-weighted images. 4 Coma has been shown to be a bad prognostic indicator. Of importance is the finding that despite the presence of subclinical dysfunction, normal findings on brain CT strongly suggest the potential for full clinical recovery. 5

Plasmapheresis is now the treatment of choice: plasma infusion alone should not be regarded as an acceptable alternative but as a short term measure only. 6 Fresh frozen plasma is the usual replacement fluid, although it remains to be determined whether cryopreseruant or solvent/detergent fresh frozen plasma is more effective. These plasmas lack von Willebrand factor, and since ultralarge von Willebrand factor multimers have been demonstrated in TTP, it is postulated that additional factor exacerbates the disease. Platelet transfusions should be avoided (unless there is life threatening bleeding) as they may worsen the condition. These two cases illustrate that patients with TTP may present to the intensive care unit with profound coma, such that many clinicians would consider the prognosis so poor that further active management would be inappropriate. In addition, the case shows that patients can survive and even make a full recovery despite a delay in diagnosis and appropriate treatment.

FIONA E KELLY DAVID F TREACHER Department of Intensive Care FRANCES M K WILLIAMS BEVERLEY J HUNT Departments of Haematology and Rheumatology ROBIN S HOWARD Department of Neurology, St Thomas’ Hospital, Lambeth Palace Road, London SE1 7EH, UK


Anaphylactoid reaction to methyl prednisolone developing after starting treatment with interferon β-1b

Courses of intravenous methyl prednisolone are a routine treatment for disabling relapses in relapsing-remitting multiple sclerosis. The Interferon β-1b Multiple Sclerosis Study Group’s research published in 1993 showed that interferon β-1b reduces the frequency of relapses in patients with multiple sclerosis. 8 We present the case of a 35 year old man with multiple sclerosis who became allergic to intravenous methyl prednisolone after the initiation of treatment with interferon β-1b and discuss what part the drug could have played in this.

A 35 year old man with asthma presented early in 1994 with “dizziness”, double vision, and paraesthesia in the right leg extending into the trunk. He was found to have bilateral pyramidal signs in the legs with extensor plantar responses and gait ataxia. His CSF contained oligoclonal bands. Visual evoked potentials were normal. Brain MRI showed multiple periventricular high density lesions with a similar lesion identified on imaging of the cervical cord, all consistent with demyelination. A 3 day course of intravenous methyl prednisolone in May, 1994 was associated with improvement in his initial symptoms. Four months later he presented with a Vth nerve palsy which again responded to a 3 day course of intravenous methyl prednisolone.

Between September 1995 and February 1996 he had a further four uneventful 3 day courses of intravenous methyl prednisolone for various symptoms related to his multiple sclerosis. In March 1996 he was started on interferon β-1b (8 MIU subcutaneously on alternate days).

In June 1996 he was admitted with pyramidal weakness of the left limbs, altered sensation in the left leg and urgency of micturition. Soon after starting his first dose of intravenous methyl prednisolone he felt a “lump” in his throat, developed an urticarial rash. Again treatment was stopped and his peak expiratory flow rate (PEFR) measured as 485 l/min. Chlorpheniramine (10 mg) was given intravenously and after 5 minutes his PEFR had returned to 625 l/min. Further methyl prednisolone was not given on this occasion.

In August 1996 he was admitted with symptoms similar to those at his admission in June 1996. Ten minutes after starting his first dose of intravenous methyl prednisolone his chest felt tight and he started developing a similar urticarial rash. Again treatment was stopped. Fifteen minutes later the rash had worsened and he felt swelling in his mouth.

His symptoms settled after 10 mg intravenous chlorpheniramine. Further courses of methyl prednisolone were not given. After this episode he chose to stop interferon β-1b.

In September 1996 he developed wheeze and a rash after the first dose in a course of intravenous methyl prednisolone. Four subsequent doses in that course were preceded by chlorpheniramine. In November 1996 he developed nasal congestion and a rash after 500 mg methyl prednisolone. Again subsequent doses were preceded by a dose of chlorpheniramine. Since then he has been given chlorpheniramine before each dose of intravenous methyl prednisolone, which he has tolerated well.

Allergic reaction to steroids is rare and anaphylactoid reaction to methyl prednisolone is rarer still with only three reports in the literature. 9, 10 One of these reactions occurred in the course of treatment for multiple sclerosis. The allergic reactions are more likely to be to the carrier than to the steroid itself. Pathology in multiple sclerosis is thought to be due to a delayed type hypersensitivity reaction. The mechanism of action of interferon β-1b in multiple sclerosis is unknown, although several mechanisms are postulated. There is evidence that, among its many effects on the immune system, interferon β can increase interleukin-2, and that interleukin-2 can stimulate a Th2 response (found in allergic type responses). These effects would explain the sequence of events in this man. However, evidence suggests that interferon β is less likely to suppress both Th1 (found in delayed type hypersensitivity reactions) and Th2 responses. 11 It remains uncertain whether the sequence of events here is due to an effect of interferon β-1b or to an effect of methyl prednisolone. However, clinicians should be aware that the complexity of the effects of interferon β-1b on the immune system may lead to unexpected clinical outcomes.

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Poststreptococcal neuropathy

Streptococcal infection can lead to a wide range of sequelae. Peripheral nervous system injury is not well known as one of its possible complications. We report a case with a typical history for a poststreptococcal vasculitis causing an axonal neuropathy of the right common peroneal nerve.
A 17 year old girl was referred by her general practitioner due to the sudden onset of numbness on the dorsum of her right foot associated with stamping her foot on walking. She had been well until 6 weeks previously when she developed tinnitus for which she received a 1 week course of oral penicillin V. She was also taking minocycline for mild acne. As her throat recovered she developed symmetric polyarthralgia and night sweats which persisted as the numbness developed. There was no history of trauma or compression of the common peroneal nerve at the neck of the fibula.

On examination she had a right foot drop with weakness of ankle dorsiflexion (Medical Research Council grade 3/5). There was sensory loss in the distribution of the common peroneal nerve. She was otherwise neurologically intact with normal reflexes. There were no skin lesions and her joints were quiescent.

She had an erythrocyte sedimentation rate of 87 mm/h, a C reactive protein concentration of 112 mg/l and an antistreptolysin-O titre (ASOT) of 1600 units/ml. Autoimmune screen, antineutrophil cytoplasmic autoantibody (p-ANCA) IgG titre of 160. Renal function was normal and there were no casts on urine microscopy.

Nerve conduction studies showed uniform reduction of compound motor action potential amplitude from all sites of stimulation of the right peroneal nerve with mild slowing of conduction velocity. Sensory studies disclosed an absent response from the right superficial peroneal nerve (table). F wave late responses were normal in the right tibial (with true H response) and left peroneal nerves, but delayed and inconstant in the right peroneal nerve. Needle EMG was normal in the right tibialis posterior muscle nerves, but delayed and inconstant in the right peroneal nerve. Needle EMG was normal in the right tibialis posterior muscle; however, there were only normal responses in the right tibial and superficial peroneal nerves. Nerve conduction studies showed uniform reduction in amplitude and normal (with true H response) and left peroneal responses were normal in the right tibial superficial peroneal nerve. (table). F Wave late responses were normal in the right tibial (with true H response) and left peroneal nerves, but delayed and inconstant in the right peroneal nerve. Needle EMG was normal in the right tibialis posterior muscle nerves, but delayed and inconstant in the right peroneal nerve. Needle EMG was normal in the right tibialis posterior muscle; however, there were only normal responses in the right tibial and superficial peroneal nerves.

### Table 1: Nerve conduction study results confirming a mainly axonal neuropathy of the right common peroneal nerve

<table>
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<th>CMAP Amplitude</th>
<th>Terminal latency</th>
<th>Velocity</th>
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</thead>
<tbody>
<tr>
<td>Right tibial</td>
<td>32.0 µV</td>
<td>3.3 ms</td>
<td>36.4 m/s</td>
</tr>
<tr>
<td>Ankle</td>
<td>6.7 µV</td>
<td>5.0 ms</td>
<td>44.8 m/s</td>
</tr>
<tr>
<td>Behind knee</td>
<td>9.1 µV</td>
<td>3.3 ms</td>
<td>39.5 m/s</td>
</tr>
<tr>
<td>Right peroneal</td>
<td>3.7 µV</td>
<td>6.5 m/s</td>
<td>90.0 m/s</td>
</tr>
<tr>
<td>Ankle</td>
<td>3.4 µV</td>
<td>3.6 ms</td>
<td>30.0 m/s</td>
</tr>
<tr>
<td>Neck of fibula</td>
<td>3.2 µV</td>
<td>3.4 ms</td>
<td>44.8 m/s</td>
</tr>
<tr>
<td>Behind knee</td>
<td>3.2 µV</td>
<td>2.3 ms</td>
<td>36.4 m/s</td>
</tr>
<tr>
<td>Left peroneal</td>
<td>3.2 µV</td>
<td>2.3 ms</td>
<td>24.5 m/s</td>
</tr>
<tr>
<td>Ankle</td>
<td>6.5 µV</td>
<td>3.6 ms</td>
<td>44.8 m/s</td>
</tr>
<tr>
<td>Neck of fibula</td>
<td>6.7 µV</td>
<td>3.6 ms</td>
<td>50.0 m/s</td>
</tr>
<tr>
<td>Behind knee</td>
<td>9.1 µV</td>
<td>2.3 ms</td>
<td>36.4 m/s</td>
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<tr>
<td>Sensory conductions:</td>
<td></td>
<td></td>
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<tr>
<td>Nerve</td>
<td>Amplitude</td>
<td>Onset latency</td>
<td>Velocity</td>
</tr>
<tr>
<td>Right sural</td>
<td>3.0 µV</td>
<td>2.3 ms</td>
<td>36.4 m/s</td>
</tr>
<tr>
<td>Right sup peroneal</td>
<td>Absent</td>
<td>2.3 ms</td>
<td>36.4 m/s</td>
</tr>
<tr>
<td>Left sural</td>
<td>9.6 µV</td>
<td>5.3 ms</td>
<td>44.8 m/s</td>
</tr>
<tr>
<td>Left sup peroneal</td>
<td>3.0 µV</td>
<td>3.6 ms</td>
<td>39.4 m/s</td>
</tr>
</tbody>
</table>

CMAP = Compound muscle action potential.

**Correspondence to:** Dr Richard B Godwin-Austen (emeritus consultant), Department of Neurology, University Hospital, Queen’s Medical Centre, Derby Road, Nottingham NG7 2UH, UK.

cycline-induced autoimmune hepatitis and sys
2. Lucas SB, Moxham J. Recurrent vasculitis asso
ciated with β-haemolytic streptococcal infec
4. Sommer C, Schroder JM. Immune-mediated neuropathy and myopathy in post

**A sensory level on the trunk and sparing the face from vertebral artery dissection: how much more subtle can we get?**

We read with interest the short report on sen
sory loss in lateral medullary infarction by Vuadens and Bogousslavsky.1 Unusual senso
ratory variants include contralateral leg and lower trunk with ipsilateral lower face hyp
gasia; or contralateral upper trunk, arm, and true hypogasia; or contralateral hypagal
sia with facial sparing; or hemibody sensory loss.2,3 We recently encountered a patient with sensory loss of the spinohalamic type involving only the contralateral leg and lower trunk from vertebral artery dissection. The sensory level in our patient with facial sparing differs from those in the literature; it suggests a thoracic hemisensory lesion and is false loca
lisation.

A 44 year old, right handed man with no relevant history presented with sudden onset of vertigo and left eye pain. There was no his
tory of trauma or neck manipulation. How
ever, the patient had had a dental abscess involving the lower incisors requiring drain
age 4 weeks previously. He has a 30 pack-year history of smoking. Vertigo developed while he was changing his car tyre. He noted that the vertigo was worse when he put his head between his knees. The vertigo lasted 15 minutes and was associated with profuse sweating in the upper half of his body. There was no nausea or vomiting. This recurred twice that day each time lasting 15 minutes.

Examination disclosed normal visual acuity and fundoscopy. There was scleral injection in the left eye. The left pupil was 4 mm compared with 5 mm on the right. Both reacted briskly to light. There was counter
clockwise rotary nystagmus in the primary position. The eye movements were normal. Cranial nerve reflexes were brisk and normal. The sensation examination was normal for all modalities. There was no upper limb ataxia. He tilted to the left on sitting and on stand
ing he fell to the left. The next day he noted loss of pinprick and appreciation of tempera
ture on the right leg and trunk with a sensory level at T9 with preservation of touch, vibra
tion, and joint position sense in all limbs (figure).

Brain MRI showed a small infarct in the lateral medulla and left cerebellum in the
distribution of the lateral branches of the left posterior inferior cerebellar artery. There was a crescent sign involving the left vertebral artery from the skull base to the basilar artery suggesting vertebral artery dissection (figure).

Four types of lateral medullary infarct are recognised: small medial infarct, inferolateral infarct, and a large inf redoorsilateral and dorsolateral infarct. The topography of the lesion in our patient corresponds to inferolateral infarct, and a large inferodorsolateral artery from the skull base to the basilar artery.

A sensory level to the trunk may point to a lateral brainstem lesion in the presence of other features suggesting brainstem disease. In our patient these signs were transient and sensory loss predominated. This new pattern of sensory loss should be recognised as symptomatic of lateral medullary infarction in addition to other sensory variants.  

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criteria for remission (no seizures for 5 years or more with or without medication) and on follow up are 111 from an original 792 who had definite or probable epilepsy. On its own, therefore, it does not provide a true indication of the incidence of SUDEP but it is nevertheless an interesting finding on the prognosis of epilepsy in a large, community based cohort.

Financial support and sources of funding for the NGHSE included an NHS Executive funded pilot study in the Departments of Neurology and Neurosurgery, Brain Research Trust, and the National Society for Epilepsy.

Opportunities for improving the quality of care in malignant cerebral glioma

There is scope for improving the services offered to patients with malignant glioma. Clinical audit has highlighted several important issues including some variation in the management of patients aged over 60, delays in beginning treatment, and problems with communication between different departments involved in patient care. A multidisciplinary Working Group, funded by the NHS Executive, recently developed evidence based guidelines for the management of these patients by surgery, radiotherapy, and chemotherapy. The group also considered the views of patients and their relatives about follow up and psychosocial aspects of care.

We have derived a package of audit measures from these guidelines that allow treatment centres to assess the care that they provide. Proformas within the package cover various topics—for example, technical aspects of treatment, breaking the news of the diagnosis, the support of patients and relatives, and palliative care while in the community. Information is drawn from case records, feedback from patients, relatives and general practitioners, and a review of the policy a centre has already developed.

We piloted the proforma by reviewing the case records of 60 patients diagnosed at two treatment centres in London between 1992 and 1994. The table shows some results using one proforma which could be tweaking the news of the diagnosis. We found, for example, that overall most case records (67%, 40/60) did not record what the patient and their relative had initially been told about the prognosis. However, there could be a difference between centres. At one, clinicians rarely recorded what they had said to patients and relatives whereas at the other this was recorded in just over 50% of cases. Patients at one centre were also more likely to be seen subsequently by counselling or palliative care services. Neither centre had the benefit of a dedicated specialist nurse in neuro-oncology.

The lack of a record does not, of course, mean that the diagnosis and prognosis were not actually discussed in some depth with the patient and their relatives. However, it is clearly likely to be helpful for others involved in the care of the patient to have sight of such a record. It is also possibly relevant that an earlier study found that only a quarter of a sample of 75 patients drawn from different centres seemed to be fully aware of the likely prognosis for their disease as they began treatment.

The aim of the guidelines developed by the Working Group has been to suggest methods which will help decision making in general terms rather than provide firm guidance on how particular patients should be treated. For example, an initial assessment of patient disability is recommended. Ten of the 60 case records we audited included some assessment of disability, but none formally recorded the patient’s performance status, an important prognostic factor, using either the WHO clinical performance status or the Karnofsky score.

The current review of cancer services after the Calman-Hine report represents an opportunity for the development of neuro-oncology services in Great Britain. A few centres have made progress towards the ideal of neuro-oncology clinics with specialist nurse support and well developed links with rehabilitation and palliative care. The guidelines and audit measures developed by the Working Group will need to be adapted for local circumstances, but treatment centres and purchasers may find them a useful tool in assessing and developing their services.

MrI in vertebral artery dissection

In a recent report, Auer et al described the clinical and imaging findings in 19 cases of extracranial vertebral artery dissection retrospectively.1 We make the following comments.

Firstly, the authors described the “sensitivity” and “specificity” of digital subtraction angiography (DSA), magnetic resonance imaging/angiography (MRI/A), and duplex sonography for diagnosing extracranial vertebral artery dissection.1 These figures were based on the percentage of probable and definite features among the 19 patients. Nevertheless, sensitivity of a test is the number of cases with true positive results divided by the total number of positive results (including both true and false positives), and specificity is the number of cases with true negative results over the sum of true and false negatives. The authors misquoted the terms “sensitivity” and “specificity” in their report, as the diagnostic criteria of the various tests have not been applied to a control group to disclose the false positive cases and true negative cases. Secondly, the criteria for case inclusion were not defined. Apparently, extracranial vertebral artery dissection was diagnosed by either radiological features on MRI/A (which may be “pathognomonic” or “suggestive”) in the appropriate clinical context or confirmatory radiological features on DSA (which may be “specific” or “indirect”). The accuracy and usefulness of DSA, MRI/A, and duplex sonography cannot be compared directly, as no single “gold standard” diagnostic method was used and because results of the present study simply reflected the proportion of cases diagnosed by the authors.

Dissection of neck arteries was thought to be an uncommon cause of ischaemic stroke. The true incidence of this condition remains unknown as angiography is not performed in every patient during the acute or subacute phase. Younger patients are more likely to undergo early angiography when there is a history of recent neck trauma1 or pain, or when no other causes of stroke are apparent. This selection bias may underestimate the...
incidence of stroke due to arterial dissection in older patients and those without neck trauma or pain.

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Auer and Felber reply:

Cheung et al state in their comment, that dissections of the neck arteries are an underestimated cause of stroke, because angiography is not performed in every patient during the acute and subacute phase. This bias is even more important in the case of vertebral artery dissection if the initial symptoms are non-specific. Non-invasive diagnostic methods are likely to be performed earlier in these patients and this was our motivation to report on the magnetic resonance angiography of vertebral artery dissection.

The diagnosis of vertebral artery dissection is often based on the consensus of clinical and neuroradiological features. We agree with Cheung that there is no single “gold standard” test for a dissection exists. Imaging procedures more often show indirect signs which have to be interpreted in the appropriate clinical context. Therefore, the “inclusion criterion” we used for this retrospective analysis was the clinical and neuroradiological consensus on the diagnosis of vertebral artery dissection. The sensitivity of DSA, ultrasound, and MRI/angiography was assumed. The MRI findings of the affected and the contralateral normal vertebral arteries, there were no false positive results. The term specificity could have been misleading, because it did not refer to the overall specificity of a test but to the frequency of findings that reached a level of specificity sufficient to establish the diagnosis of vertebral artery dissection. 1

Further prospective studies on the sensitivity and specificity of magnetic resonance imaging for the diagnosis of vertebral artery dissections are certainly necessary, but our retrospective evaluation already showed that MRI and MR angiography will have a major contribution in future. As a non-invasive means, magnetic resonance can be employed without risk in patients with non-specific symptoms and may provide specific findings that are not accessible with other methods. This will lead to a better estimation of the true incidence of dissections and will improve the early diagnosis and management of dissections in individual patients.

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Clinical usefulness of MRI in multisystem atrophy

Schrag et al suggest that certain putaminal and infratentorial changes on MRI are useful in distinguishing between patients with multisystem atrophy (MSA) and patients with idiopathic Parkinson’s disease. 1 The specificity and positive predictive value of these changes were both about 90%. However, whether these changes will be useful in clinical practice or epidemiological research is unclear for several reasons.

The number of patients included was small and so the confidence intervals were wide. For example, the specificity of the MRI changes for MSA could be as low as 80%. Moreover, only patients with clinically probable MSA were included. In this group of patients the clinical diagnosis alone had a positive predictive value as high as that of MRI and so there would seem to be little added value of MRI (14/15 (93%) patients with probable MSA had the diagnosis confirmed at postmortem). A more relevant question is whether the MRI changes are equally specific in those with possible MSA in whom the clinical diagnosis is much less certain. Indeed it is also unclear from this study whether the MRI changes are specific to MSA as patients with other conditions that enter into the differential diagnosis were not included. It may therefore be more correct to state that the MRI changes are helpful in excluding Parkinson’s disease rather than in confirming MSA.

Finally, the positive predictive value of MRI quoted in this study is likely to be an overestimate compared with its routine use in most movement disorder clinics. Schrag et al included a very high proportion of patients with MSA (nearly 50%) compared with Parkinson’s disease. As the positive predictive value is directly related to the prevalence of the disease in a given population, this resulted in a high positive predictive value. In a typical movement disorder clinic, fewer than 10% of patients will have MSA, in which case, even if the specificity of MRI is 90%, the positive predictive value would only be about 50%—that is, only half of those with the MRI changes would turn out to have the disease.

It is, therefore, too early to include specific MRI changes as part of the diagnostic criteria for MSA.

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A dubious therapy for patients with multiple sclerosis

Plohmman et al investigated the effects of computer training of attentional deficits in...
patients with multiple sclerosis. They conclude that “significant improvements of performance could almost exclusively be achieved by the specific training programmes”. The validity of this conclusion is called into question by severe methodological shortcomings of their study.

Before training, three baseline measurements of attentional functions were administered with 3 week intervals. For evaluation of training effects the median value of the three baseline measurements was compared with the values obtained after training. This statistical approach manifests a curious misunderstanding of the purpose of repeated baseline measurements. They serve to determine a baseline level, that is, a rate of change during training without any therapy. The critical value is therefore not the mean (or median) of the baseline measures but the difference between them. If therapy is efficient, the difference between pretherapy and post-therapy measurements should be greater than that between two consecutive baseline measurements. This crucial comparison is not presented.

The selection of the median of the three baseline measurements as starting point for calculation of improvements during the first training period poses further problems. If there was any improvement from the first to the third measurement, the median is lower than the third measurement after which training began. This difference inflates apparent improvement in the first training period. It may feign specific training effects if the pretraining group had a steeper baseline than the control group. A possible reason for different baselines are different severities of initial impairment. We (Motz, Grömminger, Göttert, Goldenberg, unpublished data) have administered the PASAT, another test of attentional capacities, four times with weekly intervals to 30 patients with chronic brain damage from different aetiologies. During intervals these patients did not receive any training of attentional functions. Thus, the repeated measurements determined a baseline without therapy. None the less, performance on PASAT improved from test to test. There was a negative correlation between initial performance and improvement. Patients with poor initial performance improved more than those with better performance.

The allocation of patients to treatment groups in the study of Plohmann et al was not randomised. Patients were trained in those two functions that were affected most, and group comparisons were made between patients who had been trained in a function and those who were not. Thus the training group tended to start from a lower level of performance than the control group. Figures 2 and 3 of their paper illustrate this effect impressively. If, as suggested by our results with the PASAT, initial level of impairment has a systematic influence on improvement independently of any therapy, the allegedly specific training effect may be accounted for by differences in initial level.

Whether or not the results of Plohmann et al study are reliable has clinical and ethical implications. Multiple sclerosis is one of the most common neurological diseases, and I have the suspicion that no other neurological disease has given rise to a comparable number of scientifically unfounded therapies and advice. The above critique raises the problem that whatever computer assisted retraining of attention is one of them. It may be relatively harmless in that it has no organic side effects. None the less, if its efficacy cannot be proved, it would be a waste of money, time, and patients’ hopes.

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Plohmann and Kappos reply:

We thank Goldenberg for his interest in our paper. After having been actively involved in planning and conducting controlled trials in multiple sclerosis in the past 15 years, we can only agree that the risk of drawing wrong conclusions from unreliable data cannot be overstressed in this area. All the same we cannot follow Goldenberg’s reasoning. His critique is probably based on his own unpublished observations but is neither supported by the available literature nor by our own data.

His main critique is that the effect described in our paper may only reflect non-specific practice effects. These effects are dependent on the interval between test presentations and the population studied, and differ from test to test according to their respective stability and reliability. In our study we assessed patients with multiple sclerosis in a stable or eventually slightly progressive phase of their disease. For cognitively impaired patients with multiple sclerosis it has been shown in longitudinal studies that they lack practice effects compared to cognitively intact patients. 2 In our data a possible but in no way significant practice effect was found between the first (T1) and second (T2) baseline measurement and improvement and— with two exceptions (“divided attention” and “flexibility”)—not at all between T2 and T3. Clearly significant improvement of performance could only be seen between all three baseline measurements and the posttraining test (Friedman two way analysis of variance, Wilcoxon signed ranks test). Also the assumption that the median might be lower than the third measurement and therefore might not be an adequate starting point for statistical comparisons is not supported by our data. In most of the control tests the median is equivalent to T3 or even larger.

Goldenberg’s second point is that the initial level of performance has a systematic influence on improvement independently of any therapy. Furthermore, on the basis of his results concerning the PASAT, he assumes a negative correlation between initial performance and improvement. As our patients had received specific training in those two functions that were severely impaired and thus started from a lower level of performance, Goldenberg claims that the specific training effects may be accounted for by the differences in initial level of impairment. For this reason we reanalysed our data. For each training group we built subgroups of patients according to their initial capability (low, medium, high) and printed the course of performance during the baseline measurement (T1–T3). Our results do not confirm Goldenberg’s assumption that initial level of performance has a systematic or inverse influence on improvement.

Nevertheless, as already stated in our original paper, due to the small sample size of our study the results do not allow a definite assessment of the training and its effects in patients with multiple sclerosis but they provide enough evidence to encourage further studies in a larger population of patients. Thus, we have started a prospective multicentre study in comparison with three Swiss neurological rehabilitation centres to further assess and possibly improve this kind of computer assisted retraining of attentional functions.

The unpublished additional data mentioned in our letter have been submitted with this letter and are available on request (tables 1a–1e, figures 1 and 2).

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Neurology and the gastrointestinal system

Neurology and the gastrointestinal system,1 or an analysis of the “brain-gut” axis would be incomplete without allusion to the neuroendocrine system, and its mediation, via somatostatin, in the regulation of splanchnic blood flow and gastric acid secretion. 2 These actions could account for the established haemostatic action of somatostatin in oesophageal variceal bleeding, 3 and for the perception, derived from meta- analysis, that similar benefits might occur in non-variceal upper gastrointestinal haemorrhage. 4 On the basis of the involvement of somatostatin in the regulation of gastric blood flow and acid secretion, it also seems reasonable to attribute gastric erosive bleeding, so-called Cushing’s ulcers, which occur in CNS disorders, 5 to derangements in neuroendocrine pathways.

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“All we really understand about neurological disease we have learned from pathological studies.”

Self evidently not a perfect truth, but it is a fair approximation, particularly if “understand” is properly weighted. Take multiple sclerosis as a random example. We have been taught a great deal about the course and dynamics of the disease—for example, by new imaging techniques, but MRI has come closest to contributing to our understanding of the disorder when married to pathological studies, or when used as a surrogate marker of pathology. The huge power of the new genetics has now set its sights on multiple sclerosis, and although proceeding apace and well in to the genome screen approach, it too has yet to contribute major insights to our understanding of multiple sclerosis. Animal models, even the aged EAE, can only offer suggestions which live or die according to correlative studies of multiple sclerosis tissue or patients. And my own area of interest, the cell biology of oligodendrocytes studied—their culture by pathology.

It follows that good neurology must depend on a decent grounding in pathology, and a neurological training which fails to include a wide and deep and thick and even across the whole area of the human neurology and neuropathology. There is no other way to learn about the normal peripheral nerve, an outstanding chapter, fifth is devoted exclusively to a description of the normal peripheral nerve, an outstanding chapter, and there are instructive and useful accounts of other important areas: familial tumours, metastatic disease, etc. Again, the narrative is as contemporary as a new edition, and there are very few outright omissions. I could find no typo (though quite a howler—missing a letter).

How to move on, to provide anything remotely resembling a useful review of the remaining 25 chapters? Several merit particular attention. “Prion diseases” (DeArmond and Prusiner) and “tumours of the nervous system” (Lantos, Vanden Berg, and Kleihard) are both new to Greenfield’s book. Both represent topics whose biology and pathology have changed at a breathtaking pace over the past five years, a sure challenge to any textbook harbouring ambitions of eternity. Typically, both rise to the call with apparent ease. The chapter on prion disease is only 35 pages long, but this is nevertheless a comprehensive and fine account of an extraordinary area of the human neurology and neuropathology. There are excellent descriptions not only of conventional dementing prionopathies, but also of rarer, more recently recognised entities such as familial insomnia. The key to cat independence and molecular dissection of the disease are amply covered, and space is even found for speculation concerning the possible involvement of prions, in yet another evasive and tantalising disease, inclusion body myositis.

The tumour chapter—an all embracing 200 pages with more than 3000 references (I lost count)—is again quite masterly. The bread and butter tumours are capably described, and there are instructive and useful accounts of other important areas: familial tumours, metastatic disease, etc. Again, the narrative is as contemporary as a large text can be, and more up to date than most, with succinct descriptions of the NF syndromes plus the basic management of their biology, as far as is known. Surprisingly, in this genre the chapter, paraneoplasia is perhaps a little brief.

It would be unforgivable also to omit mention of the chapter on peripheral neuropathy (Thomas, Landon, and King). Just 100 pages long, this yet again is a joy to read. The first fifth is devoted exclusively to a description of the normal peripheral neuropathy, an outstanding account. The whole chapter is (predictably) beautifully illustrated, with authority spread deep and thick and even across the whole landscape of peripheral nerve disease, from new immunological concepts in relation to inflammatory neuropathies, to the molecular genetic advances in inherited nerve disease.

So, it is not easy to criticise. I managed to amass a perfectly miserable haul of just one typo (though quite a howler—BAI for BALO—in a bold, italicised, large font header). The editing is lightly but highly effectively addressed and there are very few outright omissions. I could find no account of Hashimoto’s encephalopathy, which is a shame; I suspect many years of further use might fail to add appreciably to this one omission.

This is such a good book. Do buy one. It is well worth the investment, and will stand by you and repay you all the days of your working life.

NEIL J SCOLDING
neurovascular clinic and stroke unit, how to
overcome resistance to change, how to partici-
pate in or set up large multicentre trials etc.
Whether you read this book will largely
depend on your point of view.

LIZ WARBURTON

Disorders of the Brain and Mind. Edited
by MARIA A RON and ANTHONY S DAVID. (Pp
375, £55.00). Published by Cambridge
0-521-47306-3.

In choosing the title for this book, the editors
have wisely avoided the use of the term “neu-
ropsychiatry”, which in Britain, at least,
implies a primarily psychiatric audience. I
think that this book should be read by a much
wider audience, including neurologists inter-
ested in behaviour and cognition. There are
relatively few books available that bridge this
important divide. The editors have assembled
an impressive international cast who cover
most of the hot topics at the interface of neu-
rology and psychiatry.

The first section is dedicated to the frontal
lobes with contributions from neuropsychol-
yogy and frontal lobe abnormalities on struc-
tural scanning in schizophrenia. The second
section deals with basal ganglia disorders
with excellent overviews of neuropsychologi-
cal findings and behavioural psychopharma-
cology. The third section is dedicated to
memory and its disorders, with extremely
readable overviews of advances and contro-
versies in the neuropsychology of memory
and clinical disorders. The fourth section
deals with psychiatric manifestations of pa-
tients with a known brain pathology and
structural imaging in the psychoses. Stricter
editorial intervention could have avoided
some redundancy and overlap with an earlier
chapter. Section five covers for what is for
many people the central ground of
neuropsychiatry—namely, epilepsy—with ex-
cellent accounts of the behavioural and
psychiatric changes seen in the context of
chronic epilepsy. The sixth section takes a
developmental perspective, particularly re-
lated to schizophrenia, and the final two
chapters of the book deal with advances in
brain imaging, namely magnetic resonance
spectroscopy and imaging of patients with
hallucinations.

The editors have deliberately decided not
to write a comprehensive textbook, but rather
to choose areas of advance and controversy,
and in doing so have produced a very
readable text. The book is in many ways a
celebration of the immense contributions of
Professor Alwyn Lishman to the study of the
brain and mind. I can thoroughly recommend
it to everyone working in this exciting area.

JOHN HODGES