MRI of the lumbar spine at L3 level (left); enhancement of the cauda equina roots after gadolinium (right). The roots remain discrete.

clotting function which was corrected with fresh frozen plasma. The CSF was turbid with 6000 white cells per mm³, 90% of which were polymorphonuclear cells, and a protein concentration of 0.6 g/l. Antibiotic treatment with cefotaxime and benzylpenicillin was instituted with the benzylpenicillin alone being continued when both blood and CSF cultures showed *Neisseria meningitidis*.

By the fifth day he had persistent headache but was oriented. He complained of lumbar backache and leg pain with unsteadiness, and failed a trial without a urinary catheter because of urinary retention. He was unable to stand for more than a few seconds and could not walk at all; he was also faecally incontinent. Examination on the 12th day of his illness showed him to be confined to bed, afebrile, alert, and fully oriented without signs of meningism. He had no papilloedema and no cranial nerve signs. Apart from a depressed left triceps reflex he had no signs in the arms. There was a severe flaccid asymmetric leg weakness, worse on the left, with all tendon reflexes, except for that at the right knee, being absent even with reinforcement.

Both plantar responses were flexor. There was impaired pinprick sensation from L4 to S5 bilaterally. Proprioception was impaired in the right toes and in the right ankle. Vibration sensation was intact. A urinary catheter remained in situ.

Further investigations showed an erythrocyte sedimentation rate of 85 mm/hour with a C reactive protein concentration of 36 mg/l. Full blood count, urea, electrolytes, clotting screen, liver function tests, calcium, phosphate, complement and HIV-1 antibodies were normal or negative. A repeat CSF examination showed 66 white cells per mm³, of which 58 were monocytes, a protein concentration of 3.8 g/l, and a normal CSF/blood glucose ratio. No oligoclonal IgG was detected in either the serum or CSF. A polymerase chain reaction performed on this CSF was negative for cytomegalovirus, herpes simplex, and varicella zoster. Magnetic resonance imaging of the lumbar spinal canal including the conus showed a minor L4/5 disc protrusion and gadolinium enhancement of the cauda

equina roots which, however, remained discrete (figure). Nerve conduction studies showed reduced compound muscle action potentials distally in the legs with normal conduction velocities, normal F wave latencies, and normal sensory action potentials. Needle EMG showed denervation changes in the extensor digitorum brevis, tibialis anterior and rectus femoris muscles bilaterally. During the next six weeks a gradual improvement occurred on no treatment, and three months later he was continent and walking outdoors.

This patient developed a lower motor weakness of the legs within five days of a meningococcal meningitis, which continued to progress for a week thereafter before plateauing and improving. On both clinical and neurophysiological grounds it seemed likely that this was the result of a lumbosacral radiculopathy. Various neurological sequelae are described after meningococcal infection, occurring in about 10% of cases.¹ The commonest are single palsies of the sixth, seventh, and eighth cranial nerves with either unilateral or bilateral involvement. Third and fourth nerve palsies have been described. More rarely a transient hemiparesis, often with seizures, has been reported to occur in the convalescent phase when the CSF has cleared. Mention is made of flaccid weakness of single muscles or groups of muscles in the review by Banks, but no cases were seen in his series.² There is a single report of lower motor neuron leg weakness which occurred between 8 to 10 days after the onset of the meningococcal meningitis and when the patient had recovered from the acute infection.³ This weakness was attributed to a conus lesion on the grounds of symmetry of both motor and sensory involvement and the combination of tendon areflexia with extensor plantars. Myelography in this case was normal. This patient was treated with a two week course of prednisolone and made an almost total recovery.

The pathogenesis of these sequelae remains uncertain. As most occur in the convalescent phase when the purulent CSF has cleared it is doubtful that they represent a direct infective pathology or the result of the pressure of meningeal exudate on nerve

roots as suggested by Banks.²

Magnetic resonance imaging in the present case showed gadolinium enhancement of the cauda equina roots, which has not been reported before. The roots remained discrete, unlike the appearance in arachnoiditis. Gotshall³ suggested an intramedullary vasculitis with secondary parenchymal damage as the pathological basis for a presumed conus medullaris lesion, by analogy with the brain pathology findings in an 11 month old infant who died 36 hours into the illness.⁴ The time course of the onset and the recovery of the neurological syndrome in both Gotshall's case and the present one seems to be more suggestive of a focal postinfectious inflammatory polyradiculopathy.

An infective or postinfective cauda equina syndrome is an unusual clinical phenomenon. A cauda equinitis can occur as a result of cytomegalovirus infection in HIV positive patients and usually leads to death within a few weeks.⁵ In our patient, the negative serology for HIV antibodies, the absence of cytomegalovirus in the CSF on polymerase chain reaction analysis, and the patient's spontaneous recovery make this unlikely to be the diagnosis. Appearances on MRI, showing gadolinium enhancement of the cauda equina roots, seem to indicate that a postinfectious cauda equinitis may occur after meningococcal meningitis.

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External anal sphincter electromyography in the differential diagnosis of parkinsonism

Multiple system atrophy may account for up to 10% of patients with parkinsonism.¹ By contrast with idiopathic Parkinson's disease, patients with multiple system atrophy tend to have pronounced sphincter disturbance, which commonly appears early in the course of the illness.² A great part of this is due to impairment of the motor nucleus of the urethral and anal striated sphincters (Onuf's nucleus).²⁻⁴ Electromyography of either of those two muscles may establish such a neurogenic lesion.^{2,4} We evaluated the usefulness of EMG assessment of the external anal sphincter in disclosing the neurogenic lesion, thus helping to differentiate multiple system atrophy from idiopathic Parkinson's disease.

Twenty four patients presenting with parkinsonian features underwent clinical assessment and external anal sphincter EMG. They had no other disease that could lead to neuropathy. A clinical diagnosis of

Motor unit potential polyphasicity (A) and fibre density (B) in patients with idiopathic Parkinson's disease (IPD) and multiple system atrophy (MSA).

*A woman with four vaginal deliveries

probable multiple system atrophy¹ was made in 10 (eight men, two women) and of idiopathic Parkinson's disease in 14 (12 men, two women) patients. Median age was 57 (range 41–73) years in the multiple system atrophy group and 56 (range 35–76) years in the idiopathic Parkinson's disease group. Median disease duration was 3.5 (range 1–11) years in the multiple system atrophy group and 4.5 (range 1–10) years in the idiopathic Parkinson's disease group. A concentric needle EMG and a single fibre EMG⁵ of the external anal sphincter were performed. In concentric needle EMG 10 to 20 different motor unit potentials (MUPs) were recorded. Mean MUP duration, mean MUP amplitude, and MUP polyphasicity (percentage of polyphasic MUPs) were expressed for each patient. Mean muscle fibre density⁵ per insertion site was determined by single fibre EMG. A non-parametric (Mann-Whitney) test was used to analyse the results.

The difference in mean MUP duration was close to significance (6.7 ms and 5.1 ms, $P = 0.09$), being longer in the multiple system atrophy group, whereas there was no difference in the mean MUP amplitude (0.50 mV and 0.45 mV, $P = 0.79$). The MUP polyphasicity was significantly higher in patients with multiple system atrophy (68% and 40%, $P = 0.0002$), as was fibre density (3.3 and 2.1, $P = 0.0003$). No patient with multiple system atrophy had MUP polyphasicity below 50% and fibre density below 2 (figure). On the other hand no patient with idiopathic Parkinson's disease had MUP polyphasicity above 60% and fibre density above 3. Thus MUP polyphasicity of 60% and/or a fibre density of 3 could be a "cut off" level to distinguish patients with multiple system atrophy from those with idiopathic Parkinson's disease. In this respect, the sensitivity and specificity of concentric needle EMG would be 80% and 93%, respectively, and for single fibre EMG these estimates would be 80% and 100%. The diagnostic value of the two tests is similar.

Our neurophysiological data were obtained from small groups of patients, but they had similar ages, male to female ratios,

and durations of disease. Whether the results from patients with idiopathic Parkinson's disease deviate from normal or not will have to be ascertained in future studies. Importantly, however, the results from the patients with multiple system atrophy showed a distinctly more abnormal pattern, particularly much increased polyphasicity of MUPs on concentric needle EMG and fibre density on single fibre EMG, compared with patients with idiopathic Parkinson's disease.

In conclusion, a polyphasicity of 60% or above and/or fibre density of 3.0 or above in the anal sphincter in a patient with parkinsonian symptoms should raise the possibility of multiple system atrophy.

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Ependymal cyst and psychiatric symptoms

Many reports have documented the relation between intracranial cysts and psychiatric symptoms.^{1–3} Ependymal cysts are rare, non-invasive, benign tumours.⁴ We report a patient with an ependymal cyst and psychiatric symptoms which improved with cyst resection.

A 22 year old right handed blue collar worker, with no personal or family history of psychiatric disorders, had had general fatigue, poor concentration, and insomnia since the age of 16. Gradually, psychiatric symptoms such as depressed mood, agitation, depersonalisation, ideas of observation, and suicidal ideation, without any disturbances of consciousness, appeared. These symptoms were continuous and severe, so he was unable to work. The patient was first diagnosed with depression at a psychiatric clinic, and was given a 20 mg dose of methylphenidate hydrochloride, which failed to alleviate his symptoms. At the age of 21 his pronounced agitation caused him to extinguish a cigarette on his own hand, so he was admitted to the psychiatric ward at our hospital. A neurological examination, an EEG, and SSF were normal. Brain MRI showed a cystic tumour (6 × 4 cm) in the posterior horn of his left lateral ventricle (figure A). This proved to be an ependymal cyst on subsequent pathological examination. We determined that the cyst did not require resection, because of the absence of abnormal neurological findings. The Wechsler adult intelligence scale-revised (WAIS-R) yielded a verbal intelligence quotient (VIQ) of 67, a practical IQ (PIQ) of 70, and a total IQ of 63. His score on Bender's gestalt test was 18 points. Major tranquilizers and mianserin (60 mg) were partially effective in treating his psychiatric symptoms, and he was discharged after 99 days in hospital. However, he soon experienced a relapse, and in accordance with his wishes, cyst resection was performed. Using an endoscope, the wall and contents of the cyst were partially removed, and communication was established between the cyst and the body of the lateral ventricle anteriorly; this resulted in a reduction in cyst size (figure B). His psychiatric symptoms subsequently improved and had almost resolved within three months of the operation. His psychological test scores also greatly improved (WAIS-R total IQ 96 (VIQ 92, PIQ 105) and Bender's gestalt test 10 points). Postoperatively, he has worked as a blue collar worker for the past six months without a relapse to his previous psychiatric symptoms.

Ependymal cysts are usually seen in the fourth ventricle, but sometimes occur in the

MRI (axial view: T2 weighted image) (A) showing an ependymal cyst in the posterior horn of left lateral ventricle before surgery; (B) postoperatively, the cyst appears smaller.