also in research literature the reports are scant.³⁻⁵ We describe a patient presenting with paraparesis and urinary retention in whom a pontine tumour extending bilaterally was discovered.

A 69 year old woman with hypertension and type II diabetes had complained of weakness in the lower limbs with a fluctuating but progressive course since November 1994. On February 1995 she had an acute deterioration and could not walk or urinate. At admission there was pronounced weakness (MRC 2) in the lower limbs (worse on the right) and slight weakness (MRC 4) of the proximal and distal muscles of the upper limbs. Tendon reflexes were brisk in the upper limbs and hypoactive in the lower limbs. Plantar response was extensor bilaterally. Sensory examination was normal. There was bladder distension at percussion and 1200 ml urine were evacuated by catheterisation. The course was characterised by spontaneous improvement of strength in the lower limbs and reappearance of spontaneous, although incomplete, voiding. After two weeks there was worsening of lower limb weakness, definitive urinary retention, the appearance of left internuclear ophthalmoplegia, and mild dysphagia.

Lumbar puncture showed normal pressure values. Examination of CSF was normal. Urodynamic studies showed a hyporeflexia of detrusor muscle without detrusor-sphincter dyssynergia. Motor conduction velocities and compound muscle potential amplitudes, sensory conduction velocities and sensory nerve potential amplitudes, and late response latencies were normal. Tests of autonomic nervous system function were normal. Echography excluded a pelvic tumour. A complete spinal MRI study was normal. Brain MRI showed a lesion in the pons which was hypointense in T1 weighted images, and non-homogeneously hyperintense in T2. Gadolinium thiopentate infusion profoundly enhanced the lesion, which was bilateral, extended from the pontine base to the tegmentum, and slightly compressed the floor of the fourth ventricle (figure). Some small lesions, which did not show enhancement, were present in the hemispheric periventricular white matter and were ascribed to vascular leucoencephalopathy. The clinical and imaging findings suggested a low grade glioma. Because of family reasons the patient was transferred to a hospital in another country and died after five months.

The patient we report had urinary reten-

tion and a pontine tumour extending bilaterally and involving the pontine tegmentum. Other possible causes of urinary retention were excluded. The pontine micturition centre responsible for urine emptying is localised in the cat in the medial part of the dorsolateral pontine tegmentum.² Physiological studies in experimental animals show that the centre is represented in both sides of the pons and that bilateral lesions are necessary to cause severe micturition disturbances. The occurrence of urinary retention in patients with pontine disease has been rarely reported. Ueki in 1960 and Reiner and Gabreels in 1980 mentioned urinary retention in some patients with pontine tumours.^{3,4} These reports, lacking modern neuroimaging, did not correlate the site and the extension of the lesions with the urinary disturbances. Up to now only one other case with MRI showing a bilateral pontine lesion (thought to be a developmental abnormality) and voiding dysfunction has been reported.⁵ It is noteworthy that urinary retention is not mentioned in circulatory disorders involving the pontine tegmentum. The patient we describe provides further clinical evidence for the importance of the pons in the pathophysiology of human micturition and suggests some points:

(1) In humans, as in experimental animals, the pontine micturition centre seems to be located in the dorsal tegmentum confirming that normal micturition is a brain-stem reflex and not a simple sacral one,

(2) In humans, as in experimental animals, a bilateral lesion in the pontine micturition centre seems to be necessary to cause voiding dysfunction indicating that the integrity of only one pontine micturition centre is sufficient to maintain normal voiding,

(3) The necessity of a bilateral lesion accounts for the rarity of clinical reports and for the fact that urinary retention has been reported essentially in tumours and not in pontine vascular lesions.

A large series of patients correlating site and extension of the lesion demonstrated by MRI with the urodynamic findings would be necessary to ascertain the role of the pons in human voiding disorders and confirm our data. In the meantime, from the practical point of view, neurologists should be aware that urinary retention can be due to bilateral pontine disease involving the tegmentum.

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Chronic inflammatory demyelinating polyneuropathy with multifocal CNS demyelination in an Afriid

The occurrence of a syndrome that combines chronic inflammatory demyelinating polyneuropathy (CIDP) with multifocal CNS demyelination is now well recognised but rare.¹⁻³ The nature of the CNS demyelination is still uncertain. Although the clinical manifestations and MRI findings resemble multiple sclerosis, no pathological studies are yet available. All patients reported so far have been ethnically European. We have recently encountered a case in an Afriid. In view of the rarity of multiple sclerosis in Afriid populations it seems of interest that the patient should be documented.

The patient was an ethnically Afriid Sudanese girl aged 21 years with no European antecedents. Her parents were normal but were remote cousins. When aged between 1 and 2 years she had developed a right lateral rectus muscle weakness which had recovered over the course of some weeks. When aged 9 years she had developed weakness of her right hand so that she was no longer able to hold a pen to write. A cranial CT performed at the time was normal. This disability recovered fully after two to three weeks. Her recent history was of numbness in her right arm, trunk, and leg for the previous two months with weakness of her right arm and leg and poor balance on her feet. She had experienced burning paraesthesiae distally in all four limbs and "crawling" paraesthesiae in her right upper limb.

Both optic discs were pale. Her visual acuity was normal and her visual fields were full. Bilateral palmar-mental reflexes were present. She showed clumsiness for fine finger movements on the right, mild weakness in her right hand, and ataxia on tandem walking. Apart from sluggish brachioradialis and knee jerks, her tendon reflexes were absent, as were her abdominal reflexes. Both plantar responses were extensor. The appreciation of light touch and pinprick was impaired over her right face, arm, trunk, and leg and joint position sense was mildly impaired in the fingers and toes bilaterally. Other sensory modalities were normal. Her peripheral nerves were not thickened. Examination of other systems was negative.

Routine haematological, biochemical, and autoantibody screening was negative, including testing for anti-G_{M1} ganglioside antibodies; her serum proteins and white cell enzymes were normal. Examination of CSF disclosed oligoclonal IgG bands but was

Brain MRI: T1 weighted images after gadolinium thiopentate infusion. Left: coronal section showing abnormal enhancement in the pons more pronounced on the left. Right: sagittal section showing that the lesion extended from the base to the pontine tegmentum.

otherwise normal. Motor nerve conduction velocity (MNCV) in the right ulnar nerve was slowed at 47 m/s and 48 m/s in the forearm and around the elbow respectively, but severely reduced to 10 m/s in the upper arm where there was conduction block. The evoked compound muscle action potential (CMAP) at the wrist, below elbow, above elbow, and in the upper arm was 4.3, 3.3, 2.4, and 1.4 mV respectively. MNCV was moderately reduced, to 37 m/s in the left ulnar nerve in the forearm, but to 16 m/s around the elbow where there was conduction block. The amplitude of the CMAP fell from 3.6 mV at the wrist to 2.5 mV below and 1.2 mV above the elbow. The distal motor latency (DML) was normal in both nerves; F waves were absent. MNCV in the right peroneal and tibial nerves was borderline at 40 and 42 m/s without block and with normal DML values. F wave latency in the tibial nerve was increased at 60–64 ms; no peroneal nerve F responses were obtained. The right median sensory nerve action potential (SAP) was of normal amplitude (22 μ V) and velocity (63 m/s). The left sural SAP was 5.5 μ V with a velocity of 46 m/s; it was absent on the right.

The P100 visual evoked potential latencies were increased bilaterally (117 ms on right, 112 ms on left). Cranial MRI showed lesions of high T2 signal in cerebral white matter (figure), many abutting the walls of

the lateral ventricles, especially their posterior portions. Lesions were also present in the corpus striatum, pons, and middle cerebellar peduncles.

The electrophysiological results indicated a patchy demyelinating disorder with conduction block affecting the peripheral nerves consistent with CIDP with concomitant CNS involvement. Clinical examination also showed CNS abnormalities. The cranial MRI showed clear evidence of multifocal demyelinating pathology. The patient was treated with prednisolone with resolution of her symptoms apart from persistent paraesthesiae distally in the limbs and objective evidence of improvement on examination.

This patient is of interest in relation to the onset of relapsing symptoms in early childhood. It is uncertain whether the two previous episodes represented peripheral or central nerve disease. Cranial nerve lesions are known to occur with either in CIDP with central involvement.⁴ The rarity of multiple sclerosis in Afriid populations suggests that the central demyelinating lesions in this type of case may reflect a different form of demyelination than occurs in multiple sclerosis. Experimental allergic neuritis has been considered to provide a model for the Guillain-Barré syndrome and its chronic variant for CIDP.⁵ Possibly the chronic variant of experimental allergic encephalomyelitis⁶ provides an equivalent for the

central demyelination seen in the present case and other instances of CIDP with central involvement. The question as to whether chronic relapsing experimental allergic encephalomyelitis is a valid model for multiple sclerosis continues to be controversial.

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Neurothekeoma of the cauda equina

Neurothekeoma or nerve sheath myxoma is a rare benign tumour of peripheral nervous tissue. Histogenesis from Schwann cells¹ or undifferentiated nerve sheath precursor cells² has been suggested. Intrathecal location is rare.

An 84 year old woman presented in May 1994 with severe low back pain and pain radiating into both legs. She had fallen on to the floor at home three weeks previously. The pain was exacerbated by coughing, sneezing, and straining. She had increasing difficulty walking, requiring the aid of one person. She had difficulty initiating micturition and there was occasional faecal soiling. Clinical examination disclosed a flexed posture. She was unable to stand erect due to pain. There was pain on femoral stretching and on straight leg raising bilaterally. Tendon reflexes were absent in the lower limbs. There was sensory loss below the right knee and in the left foot. Saddle sensation and anal tone were normal. Power testing was not possible due to pain.

Blood biochemistry and haematology were normal. Magnetic resonance imaging disclosed a 3 × 1.7 × 1.2 cm intrathecal mass displacing the lower conus anteriorly and to the left. The tumour had high signal intensity on T2 weighted images (figure, a). After gadodiamide there was pronounced enhancement of the tumour mass with a small central non-enhancing component (figure, b). No adjacent bone or extradural abnormality was identified.

At operation a thoracolumbar laminectomy was made and the thecal sac was opened longitudinally. The tumour seemed exophytic from the right side of the caudal conus and displaced the roots of the cauda equina. The major exophytic part of the

Cranial MRI showing lesions of high T2 signal in cerebral white matter as detailed in the text.