after admission, a week before the appearance of the cutaneous eruption.

The pathogenesis of HSV-2 myelitis in this case remains ill defined. Although IgM antibodies were detected in the first serum sample we are not sure that the patient's myeloradiculitis reflected true HSV-2 primary infection because reappearance of IgM antibodies can be detected in herpes virus reactivation. All but one previously reported case of HSV-2 myelitis were fatal and occurred in immunocompromised hosts. This patient was neither diabetic nor HIV-infected, but despite normal CT examinations of the thorax and abdomen, a hidden malignancy cannot be excluded. Reactivation of a latent infection within dorsal root ganglia neurons with a contiguous spread via sacral ganglia to the spinal cord has already been proposed in HSV-2 ascending myelitis.³ It is of note that despite antiviral treatment, our patient died within six weeks. Thus an immunologically mediated injury triggered by the herpes infection could also be involved here, as in another patient with HSV-1 myelitis whose necropsy examination showed patchy demyelination of the spinal cord.⁷

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Peduncular hallucinosis and right hemiparkinsonism caused by left mesencephalic infarction

Since the original description of Lhermitte¹ several causes of peduncular hallucinosis have been reported, but always in relation to a bilateral mesencephalic lesion. We describe here a patient with prolonged visual hallucinations and right hemiparkinson-

ism. An MRI showed a unilateral infarction involving the left cerebral peduncle.

A previously healthy 70 year old right handed woman presented with visual hallucinations. Two months previously, she began, one night, to see objects (motorbikes), animals (dogs, horses), and people (Japanese) entering and driving silently round her room, across the entire visual field. Although the images were of normal colours and sizes, she was aware that they were not real, and never described "déjà vu" or "jamais vu" phenomena, tactile, or auditory hallucinosis. The hallucinatory events became progressively longer and more frequent, lasting from minutes to hours, during both day and night. Her medical history included mild hypertension, normofunctional multinodular goitre, and surgery for left breast carcinoma six years earlier, without subsequent evidence of recurrence. There were no other remarkable personal or familial antecedents. On examination, she was alert, oriented, and cooperative. She remembered four of five words after five minutes, and the mini mental state test was 32/35. She remained in a left tilted posture, and showed severe impairment of postural reflexes, mild bradykinesia, cogwheel rigidity, and intermittent resting tremor in the right extremities (mainly in the lower limb). Tendon reflexes were brisk and increased on the right side, but there was no clonus and the plantar responses were flexor. There were no other remarkable findings on general or neurological examination. Laboratory investigations, including ESR, routine haematological, biochemical, and immunological studies, thyroid function tests, serological tests for syphilis, cerebrospinal fluid examination, EEG, and cranial CT were normal or negative. An MRI showed an abnormal high intensity signal in the left cerebral peduncle on T2-weighted images (figure). Multiple foci of T2-weighted high signal intensity were also seen throughout the periventricular white matter. Such findings were consistent with ischaemic damage. Despite treatment with haloperidol and phenytoin the patient became more impaired within the next several weeks, showing continuous visual hallucinations, with frequent episodes of agitation and disorientation.

Vascular lesion of the upper brainstem is the most often cause of peduncular hallucinosis. A case of peduncular hallucinosis due to bilateral mesencephalic infarction diagnosed by MRI has been recently reported.² The present one is the first report, however, to our knowledge, of peduncular hallucin-

Axial T2-weighted MR image obtained with a 0.5-T unit shows an area of hyperintensity in the left cerebral peduncle, consistent with infarction.

nosis due to unilateral lesion. Although the precise anatomical basis for peduncular hallucinosis remains unclear, it seems that the substantia nigra pars reticulata (SNpr) is directly implicated.³ Little is known about the pathogenesis of peduncular hallucinosis. A relation with the rapid eye movement (REM) phase of sleep has been proposed. In this sense, it is known that the SNpr may play an important part in the regulation of the different phases of sleep through its connections with centromedian/parafascicular nuclei of the thalamus, superior colliculus, and reticular formation.⁴

Finally, although we cannot rule out the presence of brainstem Lewy bodies, it is probable that the right hemiparkinsonism found in our patient was related to ischaemic damage of the left substantia nigra pars compacta.⁵

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Intraneural ganglion of the sciatic nerve: detection by ultrasound

Intraneural ganglia are a rare cause of peripheral nerve lesions most often affecting the peroneal nerve. Their origin is unknown. Some 50 cases were reported up to 1979.¹ Sciatic nerve ganglia are very rare. We report a sciatic nerve lesion caused by a giant ganglion situated at the level of the distal thigh and damaging the tibial portion only.

A 36 year old male right handed and right footed state officer complained of pain in his right calf for about six years especially when jogging and walking for more than 30 minutes. He was treated for a lumbar disc hernia, but a lumbar CT was unremarkable. Simultaneously he noted discomfort in his right toes when wearing shoes; this was relieved by wearing orthopaedic sandals.

On first neurological examination he was found to have Lasègue's sign with his right leg at an angle of about 85°. Reflexes and sensation in the lower extremities were normal. There was moderate paresis of the toe flexors. Plain radiographs of the lower spine showed six lumbar vertebrae and a fissured vertebral arch of the first sacral segment.

On electromyographic examination fibrillation potentials and positive sharp waves were recorded from the abductor hallucis and flexor digitorum brevis muscles. Motor conduction velocity of the right tibial nerve was slow on the right (38 m/s) and normal on the left (55 m/s) side; sensory nerve conduction velocity of the right sural nerve and H-reflexes bilaterally were normal.

Eleven months later the pain was localised to the outer aspect of the right calf and foot, and the ankle jerk was diminished on the right side. Weakness of the toe flexors was unchanged, Hoffmann-Tinel's sign was positive over the posterior tarsal tunnel and palpation of the popliteal fossa yielded neither a mass nor tenderness. Sensory nerve conduction velocity of the right medial plantar nerve could not be determined. The distal motor latency of the right tibial nerve was prolonged (7.8 ms).

As the findings were not typical of a tarsal tunnel syndrome, surgery was discouraged.

Thirteen months later MRI of the lower leg up to the popliteal fossa showed no abnormalities other than atrophy of the popliteal and posterior tibial muscles. Electromyography yielded equivocal evidence of neurogenic damage to the posterior tibial muscle prompting examination of the proximal tibial and sciatic nerve by B-scan ultrasound. Here a cystic formation of

about $7 \times 3 \times 3$ cm was found at the bifurcation of the sciatic nerve at the level of the distal thigh (figure). A subsequent transaxial CT confirmed this result.

The patient was operated on one week later. A spindle like multiloculated cyst measuring roughly $15 \times 3 \times 2$ cm was found between the biceps and semitendinosus/semimembranosus muscles within the sciatic nerve, producing a gelatinous mass on incision. The cyst was excised by a microsurgical technique leaving the fascicles of both the tibial and peroneal compartment intact. Histologically, a benign ganglion with pseudocystic walls was found. After the operation paresis of the short and long toe flexors was more pronounced and the ankle jerk and tibialis posterior reflex were absent, accompanied by mild trophic changes. One year later the pain had disappeared and walking for two hours was possible without complaint. The ankle jerk was still absent, flexion and distention of the toes paretic, the tips of the toes hypesthetic, and the sole of the foot anhidrotic.

Having first sought the location of the lesion too far cephalic (lumbar disc hernia) it subsequently was suspected too far caudal (tarsal tunnel) because of clinical involvement of the toe flexors only. Sciatic nerve lesions are more prone to involve the peroneal than the tibial fascicles and the cyst was missed initially. It was the ultrasound

technique which gave the first clue as to the true level of the sciatic ganglion.

Although the ganglion in this patient was seated proximal to the bifurcation of the sciatic nerve into the tibial and peroneal portion, only tibial fibres were affected. The origin of the cyst is not clear. A mucoid degeneration of collagen fibres, metaplasia of irritated connective tissue, ectopia of embryonal synovial tissue, or a mucoid degeneration of a formerly solid tumour for example, a Schwannoma—are possibilities.

After Mahaley's² first report of a ganglion of the (posterior) tibial nerve at the popliteal fossa other ganglia or (Baker's) cysts were also found at the tarsal tunnel.³ Only two intraneural synovial cysts of the sciatic nerve were reported between 1980 and 1991.⁴

However rare, a ganglion such as this must be considered in the differential diagnosis of L5 and S1 root, sciatic, tibial and peroneal nerve lesions, as it is amenable to surgery and results are usually favourable. Like other authors² we strongly recommend nerve ultrasonography in any case of progressive peripheral nerve damage of unexplained origin.

The MRI was done by Dr V Buchholz and colleagues, Erlangen.

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Familial Parkinson's disease and polymorphism at the CYP2D6 locus

Because 10% of patients with Parkinson's disease have an affected relative¹ and kindred with a pattern of disease transmission compatible with autosomal dominant inheritance have been described,² genetic factors may play a part in the pathogenesis of the disease. Defective 4-hydroxylation of debrisoquine by CYP2D6, a member of the cytochrome P-450 family, has been found in more than 50% of patients with idiopathic Parkinson's disease, but in less than 20% of controls.³ Four alleles of the CYP2D6 gene, containing point mutations or deletions which inactivate the gene, result in a poor metaboliser phenotype.⁴ The mutant allele CYP2D6B in particular, occurs twice as often in the patients than in the controls, with a relative risk ratio of 2.7.⁵ The risk of idiopathic Parkinson's disease in those with the CYP2D6 mutant

(A) Longitudinal (left) and transaxial (right) section of the distal thigh by B-scan ultrasound (Siemens Sonoline SL-2). Septated echofree space occupying mass (black and white solid bold arrows) measuring 2 cm in diameter distending and displacing the sciatic nerve (+, - and x), which measures $7.0 \times 4.9 \times 9.2$ mm. (B) Transaxial CT through the distal thigh (Siemens Somatom DR) confirmed a septated cystic space occupying hypodense mass of 2.8 cm in diameter (slim black arrows) less dense (11 to 12 Hounsfield units) than the surrounding muscles (M semimembranosus, semitendinosus, and biceps femoris).