that each can be supported by one hemis-
phere or the other.

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Pure sensory stroke due to midbrain haemorrhage

After reading the interesting case reported by Azouvi et al., we would like to report a similar case, but in our patient the underlying disease was a cavernous angioma.

A 42 year-old woman was admitted to our hospital because of a mild occipital pain, and dysesthesia in the left part of her body. Neurological examination showed a fully oriented patient, loss of touch and pain sensations, and a medullary left hemihypalgesia. She was unable to find her way without help. She had brisk reflexes and no other neurological sign.

Extraocular movements were normal, but with subtle nystagmus on the left side. The patient was a rather hypokinetic Parkinsonian-like patient.

The neurological disorder resolved within three months. An MRI performed three months later revealed a hypodensity, of 0.8 x 1.8 cm diameter, in the right dorsal and lateral aspect of thepons, suggesting a cavernous angioma (fig).

Since the first description in 1977, only eight cases of haemorrhagic pure sensory stroke (PSS) have been described. All of them were secondary to small haematomas in the thalamus, internal capsule or pons. As far as we know, this is the first case of haemorrhagic PSS secondary to cavernous angioma located in the pons. In our patient, as in the one reported by Azouvi et al., the damage was restricted to the right dorsal spinothalamic tract without involving the medial lemniscus. We agree with the authors that small haematomas located on the sensory pathways, before they reach the thalamus, can produce partial PSS. MRI is useful in detecting vascular malformations, even when, as in our case, angiographic studies are normal.

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Parkinson syndrome and defects of praxis following methanol poisoning

The neurological sequelae of methanol intoxication shows marked individual variation, but Parkinsonism, optic atrophy and focal cranial nerve deficits have been described.1 We report a patient with Parkinsonism and dyspraxia who recovered from acute severe methanol poisoning. The MRI scan showed damage of the deep grey nuclei.

A 28 year-old woman with a history of depression and recurrent alcohol abuse experienced nausea, vomiting, ataxia and blurred vision for 24 hours before she was admitted to hospital with dilated pupils and altered mental state. She was incoherent, although able to communicate, so that she tried to take advantage of the staff. She did not react to light. The optic fundi were normal, and there were no localising neurological signs. The liver was enlarged. She had a leucocytosis of 12900/mm3 and a severe metabolic acidosis (pH 7.15; bicarbonate 2.8 mmol/l). The patient was treated with haemodialysis for 48 hours. She recovered from acute severe methanol poisoning. The MRI scan showed damage of the deep grey nuclei.

The patient had a history of methanol poisoning in mind, she was given intravenous infusions of sodium bicarbonate and ethanol, and haemodialysis. The level of methanol in the blood was 2.85 g/l. After three hours, the acidosis was controlled. When the patient’s level of consciousness improved, she admitted to drinking some 200 ml of methanol in the past 48 hours. She left the hospital after four days without apparent neurological or ocular abnormalities.

She was examined two years later, complaining of motor slowness and loss of memory. She had been working at the same factory at which she was employed before taking the methanol. The work required sequential and repetitive movements of both hands and feet. The employers stated that her productivity was reduced by 50%.

Neurological examination showed a mild dysarthria, a Parkinsonian-like syndrome, with an expressionless face, limb bradykin-
esia, and abnormal postural reflexes. Rapid alternating finger movements were poorly performed. The gait was wide-based and the patient had a tendency to fall forward. The vision was normal. Cerebral radiographs, EEG and ECG were normal. An unenhanced CT scan showed bilateral areas of decreased density, especially in the putamen. An MRI scan (fig) showed bilateral lesions involving the claustrum.
Pure sensory stroke due to midbrain haemorrhage.

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