affected the left hand, then she had some dysarthria. The association of the lateralisation of a migrainous aura with aphasia if her right hand was affected and with dysarthria if her left hand was affected is predictable, if extremely unusual, and has not been previously described.

In December 1977 she developed herpes zoster affecting the right sacral 3–5 segments. She continued to produce crops of cutaneous sarcoid. In July 1982 there was deterioration in her mental state due to inappropriate ADH secretion requiring fluid restriction. In March 1984 she developed avascular necrosis of the left shoulder due to steroids and in January 1985 she had a left shoulder prosthesis. In September 1985 she developed a febrile illness with rapid deterioration, she lapsed into coma and died.

A necropsy carried out by Dr WL Brander showed that the cause of death was acute pylonephritis with septicema. No evidence of pulmonary sarcoidosis was found. The brain weighed 1272 g. The leptomeninges over the frontal convexity were mildly opaque and thickened. There was a small old contusion in the orbital surface of the right frontal lobe. The old biopsy site in the left hemisphere was marked by a 1.5 cm diameter defect with underlying scarring (fig 1A). The lateral ventricles, including the temporal horns, were enlarged, as was the third ventricle. In the white matter, there were numerous irregular grey, slightly depressed lesions in both hemispheres (fig 1B), the largest measuring 1.8 x 1.1 cm. In the left frontal lobe the lesions were shrunken and cavitated.

The corpus callosum and periventricular regions were well preserved. Similar lesions were also seen in the right putamen and left thalamus. The cortex was relatively well preserved. The gyri and sulci, and the spinal cord appeared normal. Histological examination showed that there were large and small poorly demarcated pale lesions in the white matter of burst out progressive multifocal leukoencephalopathy (fig 2A). However, immunocytochemistry for JC virus failed to reveal the Papova virus antigen. Sections from the patient's earlier biopsy demonstrated the presence of the viral antigen when stained with the same antibody. No viral particles were seen in postmortem tissue taken for electron microscopy as had been found in the biopsy.1 The lesions involving the deep grey matter looked similar.

In addition there was widespread meningoencephalitis (fig 2B). Loose granulomata composed of epithelioid cells, lymphocytes, plasma cells, and multinucleated giant cells were present in the leptomeninges and in the walls of meningeal veins and arteries. The inflammation was more severe in the region of the biopsy site and extended into the underlying scarred brain. In the cerebellum, there was patchy cortical scarring related to the meningeal sarcoaid. There was no degeneration of the long tracts of the brainstem and spinal cord. Re-examination of the earlier biopsy showed mildly thickened leptomeninges but no evidence of sarcoidosis.

Progressive multifocal leukoencephalopathy typically has a fatal outcome, usually within six months.4 Rare exceptions include a patient also with coeliac disease, who died 10 years after the onset of the illness,6 and another with lymphosarcoma and progressive multifocal leukoencephalopathy for five years who seemed to have periods of clinical remission.6

There has been considerable interest in the management of patients with this condition in recent years because of the AIDS epidemic. The response to cytosine arabinoside is usually disappointing. Most reported patients have only received one or two courses of five days. This patient was given an unusual drug regime with five day courses of intravenous cytosine arabinoside at three weekly intervals for a year and the intervals were slowly extended to two months over a second year and to three months for the third year. She had her last course of treatment in May 1978, just over three years from the first treatment. This programme was devised because it did not seem likely that cytarabine would eliminate the virus completely from the nervous system and her immunosuppressed state persisted throughout this time.

The pathological evidence indicates cure from progressive multifocal leukoencephalopathy, which has not been claimed before. Cytosine arabinoside seems to have effected this cure, as her condition deteriorated progressively until treatment was started and improvement occurred soon afterwards.

We are grateful to Dr WL Brander, who kindly sent the brain to us for examination and to Dr Herbert Budka who performed the immunocyto-pathology.

MD O'BRIEN
Department of Neurology
Guy's Hospital, London
TM HONAYAN
Department of Neuropathology,
Institute of Psychiatry, Denmark Hill, London

Correspondence to: Dr MD O'Brien, Department of Neurology, Guy's Hospital, London SE1 9RT, UK.

1 Marriott PJ, O'Brien MD, Mackenzie ICK,
2 Euri MM, Kennedy PGE. In: Hume Adams J,
Duchen LW, eds. Virus diseases, in Greenfield's

MATTERS ARISING

Brain and spinal cord MRI in motor neuron disease

In your September issue, Thorpe et al describe their MRI findings in 11 patients with motor neuron disease. They found symmetric areas of high signal intensity on T2 weighted images within the corticospinal tracts, as previously reported by other investigators. We7 reported a case of amyotrophic lateral sclerosis with further high signal intensity in fibres of the corpus callosum on proton density and T2 weighted images, closely matching findings of earlier pathological reports.8 We would be interested to know if Thorpe et al found similar callosal signal abnormalities in some of their patients. These findings would enhance the diagnostic role of MRI in patients with suspected motor neuron disease, as suggested by the authors.

MICHEL VAN ZANDIJCKE
Department of Neurology
JAN CASSELMAN
Department of Medical Imaging
AZ St-Jan,
Rudershove 10,
B-8000 Brugge, Belgium.

Correspondence to: Dr M V Zandijcke.