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## Cyclosporin neurotoxicity in cardiac transplant recipient

Sir: We read with interest the report of Lane RJM *et al*[1] explaining the toxicity of cyclosporin on the central nervous system through changes of the blood-brain barrier. However, we feel that their explanation concerning the abnormal MRI seems hazardous. Indeed patient 1 and patient 3 described in the report experienced status epilepticus a few days before the MRI. The occurrence of CT scan hypodensities slightly enhanced by contrast injection following severe epileptic seizures is well described.[2-4] These hypodensities spontaneously regressive within months and are thought to be due to alterations of the blood brain-barrier produced by local hypoxia, lactic acidosis and loss of vascular autoregulation.

Furthermore, MRI T2-weighted abnormalities were described by Stone *et al*[5] in one patient and Lesser *et al*[6] in three patients with intractable focal seizure. These abnormalities were not correlated with the findings on subsequent pathologic examination.

Thus, it seems that the MRI features in Lane's report are probably due to the seizures. The sensitivity of MRI will probably lead to the description of more transient abnormalities secondary to severe epileptic seizures.

In consequence, the discovery of abnormal signal on MRI following severe epileptic seizures cannot be considered as an unequivocal proof of toxic effects of cyclosporin on the blood-brain barrier.

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*Lane and Langes reply:*

We thank Dr Mavroudakis and Dr Zegers de Beyl for their interesting suggestion regarding the cause of the reversible MRI signals in our cases, but suspect their hypothesis may be equally hazardous.

First, the transient CT scan abnormalities in the papers cited were single hemispheric lesions; Sethi *et al* provide further examples.[1] Our patients had multiple MRI abnormalities, including one cerebellar hemisphere lesion, and CT was normal in all cases. Secondly, MRI abnormalities have been reported in patients with cyclosporin neurotoxicity in the absence of seizures.[2,3] The distribution of the MRI (and CT) abnormalities in these cases of liver transplantation was somewhat different from our cardiac transplant patients, being strikingly occipital and largely confined to the white matter, but this may be a reflection of differing aggravating factors in the two situations; low blood cholesterol in liver transplant patients[2,4] and possibly hypomagnesaemia in the cardiac patients. Thus, while we cannot entirely discount a contribution from the seizures to the MRI appearances we still feel that they are more likely to be related to the neurotoxic effects of the drug.

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## Frozen shoulder and other shoulder disturbancies in Parkinson's disease

Sir: We read with interest the article by Riley *et al*[1] on the high incidence of shoulder pain in Parkinson's disease. We ourselves have made a similar observation.

As part of a survey to investigate a possible aetiological relationship between Parkinson's disease and essential tremor[2] we interviewed 100 consecutive Parkinson's disease patients attending the clinic. We were particularly concerned to establish the nature of the first symptom experienced and our interest was aroused when five consecutive patients gave shoulder pain as the initial symptom. On completion of the survey, we found a total of 15 patients (eight female) giving upper limb pain as the first symptom. Pain occurred in the shoulder (12 patients),