critically significant groups $\times$ measures interaction.

The effect that Rosser and Hodges claim may well be present in their data and, if so, would help to contribute to the understanding of the relation between allegedly cortical and subcortical dementias. Unfortunately the analyses actually reported do not properly permit the suggested conclusion to be drawn and it would therefore be useful to know if the relevant interaction really is significant.

E MILLER
Department of Psychology
(Clinical Section),
University of Leicester,
Leicester LE1 7RH, UK

- 1 Rosser A, Hodges JR. Initial letter and semantic category fluency in Alzheimer's disease, Huntington's disease, and progressive supranuclear palsy. *J Neurol Neurosurg Psychiatry* 1994;57:1389-1394.
- 2 Winer JB. *Statistical principles in experimental design*. 3rd ed. New York: McGraw Hill, 1991.

Hodges replies:

We are very grateful for Miller's comments on our paper. We have now performed a split plot analysis of variance with both within subject and between subject effects, which confirmed the presence of a significant interaction (F (df 51,3) = 19.6, $P < 0.001$). This highly significant group conditions interaction confirms the differential effect of Alzheimer's disease *v* Huntington's disease and progressive supranuclear palsy on category and letter based fluency tests, which we hope will convince Professor Miller.

J R HODGES
Department of Neurology,
Addenbrooke's Hospital,
Hills Road, Cambridge CB2 2QQ, UK.

Jarisch-Herxheimer reaction in a patient with neurosyphilis: non-convulsive status epilepticus?

In their lesson of the month Zifko *et al*¹ described a patient with neurosyphilis who developed fluctuating consciousness, disorientation, restlessness, and fever 10 hours after starting intravenous penicillin G. He had tachycardia and hyperhydrosis. An EEG showed generalised 3/s rhythmic activity (periodic lateralised epileptiform discharges). These findings were attributed to a Jarisch-Herxheimer reaction.

We suggest that non-convulsive status epilepticus is an alternative explanation. Altered consciousness and prolonged confusion are the central clinical findings of non-convulsive status, which may occur without preceding or accompanying generalised tonic clonic seizures, may resolve spontaneously, and may be associated with the autonomic symptoms of fever and tachycardia.^{2,3} Periodic lateralised epileptiform discharges are one of the EEG correlates of non-convulsive status.⁴ As stated by the authors, patients with neurosyphilis are susceptible to epileptic seizures, both due to the condition itself⁵ and due to a Jarisch-Herxheimer reaction. In addition, penicillin

itself may precipitate seizures.⁶ In their patient, non-convulsive status may have been evoked by one of these mechanisms. It would be of value to know whether patients such as this respond to antiepileptic medication.

N HEYE
J W DUNNE
Royal Perth Hospital,
GPO Box X2213, Perth,
Western Australia, Australia

- 1 Zifko U, Lindner K, Wimberger D, Volc B, Grisold W. Jarisch-Herxheimer reaction in a patient with neurosyphilis. *J Neurol Neurosurg Psychiatry* 1994;57:865-7.
- 2 Dunne JW, Summers QA, Stewart-Wynne EG. Non-convulsive status epilepticus. A prospective study in an adult general hospital. *Q J Med* 1987;62:117-26.
- 3 Cascino GD. Non convulsive status epilepticus in adults and children. *Epilepsia* 1993;34 (suppl 1):S21-28.
- 4 Grand Maison F, Reiher J, Leduc CP. Retrospective inventory of EEG abnormalities in partial status epilepticus. *Electroencephalogr Clin Neurophysiol* 1991;79: 264-70.
- 5 Radhakrishnan K, Ashok PP, Sridharan R, El-Mangoush M. Periodic pattern in meningovascular syphilis. *J Neurol Neurosurg Psychiatry* 1984;47:1360-1.
- 6 Davies DM. *Textbook of adverse drug reactions. Chapter 18. Neurological disorders*. 3rd ed. Oxford: Oxford University Press, 1985:495.

Zifko *et al* reply:

We appreciated the interesting response of Dune and Heye to our article. Although we cannot completely exclude the possibility of non-convulsive status epilepticus in our patient, we have several arguments that make their theory unlikely.

Firstly, autonomic symptoms of fever and tachycardia in our patient occurred before onset of confusion and altered consciousness, and were not present during the period of disorientation and psychomotor restlessness, as mentioned in the text. Secondly, although periodic lateralised epileptiform discharges may be a sign of non-convulsive status, this abnormality is non-specific. Thirdly, during CT, psychomotor restlessness was treated with 70 mg intravenous diazepam, which did not affect the confusion. Non-convulsive status of complex partial type usually responds well to diazepam.

U ZIFKO
K LINDNER
W GRISOLD
Department of Neurology,
Kaiser Franz Josef Spital,
1100 Vienna, Austria

Risperidone in Parkinson's disease

A recent, excellent review of the management of Parkinson's disease called attention to the atypical neuroleptic drug clozapine (an antagonist of dopamine D₁ and serotonin 5HT₂ receptors) for amelioration of psychotic symptoms derived from dopaminergic treatments, when temporary withdrawal of antiparkinsonian drugs fails.¹ Small doses of the atypical neuroleptic drug risperidone (0.25-1.25 mg/day) can also be used for ameliorating hallucinations induced by levodopa without worsening motor symptoms in Parkinson's disease.² Risperidone has a strong affinity for 5HT₂ receptors and only moderate affinity for D₂ receptors. For parkinsonian patients in

whom the 1-2% risk of agranulocytosis with clozapine is unacceptable, risperidone is another option.

ALMIR R TAVARES, JR
UFMG School of Medicine,
Belo Horizonte, Brazil

- 1 Marsden CD. Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1994;57:672-81.
- 2 Meco G, Alessandria A, Bonifati V, Giustini P. Risperidone for hallucinations in levodopa-treated Parkinson's disease patients. *Lancet* 1994;343:1370-1.

CORRECTION

Rajsheshkar V. Benign thalamic cyst presenting with contralateral postural tremor. *J Neurol Neurosurg Psychiatry* 1994;57:1139-40.

During production, the figure was incorrectly printed. The correct version is given here.

T1 weighted axial (A) and sagittal (B) MRI of the patient showing the hypointense cystic lesion in the right thalamus and basal ganglia. Compression of the upper midbrain by the mass is evident in the sagittal scan.