critically significant groups × 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 danglers. 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.

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

In their letter [1], Zifko et al described a patient with neurosyphilis who developed fluctuating consciousness, disorientation, restlessness, and fever 10 hours after intravenous penicillin G. He had tachycardia and hyperhydrosis. An EEG showed generalised 3/H 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. Periodic lateralised epileptiform discharges are one of the EEG correlates of non-convulsive status. As stated by the authors, patients with neurosyphilis are susceptible to epiletic seizures, both due to the condition itself and due to a Jarisch-Herxheimer reaction. In addition, penicillin itself may precipitate seizures. 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.

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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.

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 D2 and serotonin 5HT, 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 5HT 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.

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CORRECTION


During production, the figure was incorrectly printed. The correct version is given here.
Risperidone in Parkinson's disease.

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