increasing the likelihood of apyrexial presentation. They discussed possible mechanisms but state that carbamazepine is not known to affect dopaminergic systems.

The hypodopaminergic state thought to be responsible for NMS may arise: from direct interference with dopamine (DA) transmission by neuroleptics or withdrawal of anti-parkinsonian drugs (Toru *et al*, 1981); by imbalance of serotonin (5-HT) and DA, as has been suggested for a case induced by fluoxetine (Halman & Goldbloom, 1990); or by acetylcholine:DA imbalance (Corrigan & Coulter, 1987).

As the hyperthermia of NMS may be attributable to DA receptor blockade in the hypothalamus, it may be relevant that carbamazepine has been shown to enhance the growth hormone response to apomorphine in humans (Elphick *et al*, 1990). Although studies in rats suggest that carbamazepine has no affinity for DA receptors (Marangos *et al*, 1983), the growth-hormone response to apomorphine in humans may be modified by a direct action on dopamine receptors, perhaps supersensitivity of post-synaptic receptors secondary to reduced DA transmission. A selective increase in dopamine activity in the hypothalamus but not in the nitrostriatal pathways would account for the absence of pyrexia in the case described.

FIONA COULTER
Department of Psychological Medicine
Gartnavel General Hospital
Glasgow G12 0YN

F. M. CORRIGAN
Argyll and Bute Hospital
Lochgilphead
Argyll

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Benzodiazepine withdrawal syndrome

SIR: Ashton and her colleagues (*Journal*, August 1990, **157**, 232–238) showed that buspirone is unhelpful in the management of the withdrawal syndrome that commonly follows the discontinuation of long-term benzodiazepines given at therapeutic doses. This disorder may persist for months, occasioning great distress and despair. Treatment of these symptoms remains unsatisfactory (Higgitt *et al*, 1985) and relapse with resumption of drug-taking is only too common (Golombok *et al*, 1987).

The biochemical basis for the dependence remains unclear, although alterations in benzodiazepine receptor function have been sought (Miller *et al*, 1988). In animals, administration of the benzodiazepine antagonist, flumazenil (Whitwam, 1988), results in reversal of receptor changes (Gonsalves & Gallager, 1985) and obviation of benzodiazepine withdrawal symptoms (Gallager *et al*, 1986). Based on this research we have attempted to treat protracted benzodiazepine withdrawal symptoms by administration of intravenous flumazenil.

With informed consent, we have so far tested 11 patients who have been benzodiazepine-free for at least three weeks. In the first five patients a total dose of 0.5 mg or less was used with little effect. In most of the other six patients a larger total intravenous dose of 2 mg of flumazenil divided into three doses over a few hours was found to have promising effects. Long-standing symptoms were reported by several patients to be dramatically relieved. These included clouded thinking, tiredness, muscular symptoms such as neck tension, jerks and shaking, and the perceptual symptoms occurring as a characteristic component of benzodiazepine withdrawal: pins and needles, pain and subjective sensations of bodily distortion. Mood disorder, when present, also improved but the changes in anxiety and depression may have been a response to relief of physical symptoms. Some patients reported the maximum response delayed by as much as a day but in most the onset of effect was noted soon after the injections. Side-effects were reported to be either absent or typically described as light-headedness or dizziness lasting only a few minutes and usually well tolerated.

The benefits were reported to last between a few hours and several days which is noteworthy considering flumazenil's half-life is less than one hour. Those patients receiving a second or third dose tended to report subsequent longer relief. However, symptoms did return to varying degrees in most cases, suggesting that a course of treatment may be required. The parameters of such therapy are currently being explored.

MALCOLM LADER
SALLY MORTON
Institute of Psychiatry
De Crespigny Park
London SE5 8AF

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Dystonia – a potential psychiatric pitfall

SIR: We welcome the article by D. G. Cunningham Owens (*Journal*, May 1990, **156**, 620–634) since it is most important for the psychiatric community to be familiar with the recent developments surrounding dystonia (Fahn *et al*, 1987; Marsden & Quinn, 1990). Recently, a 42-year-old woman admitted to our psychiatric unit with the diagnosis of conversion hysteria was, after a comprehensive evaluation (including computerised tomography, magnetic resonance imaging, and cerebral angiography), given the diagnosis of dystonia, secondary to an arteriovenous malformation occupying the right basal ganglia. The patient presented with flexion of the fourth and fifth fingers and sustained contraction of the left hand and forearm. She also complained of pain and stiffness in the affected area. Her condition had started five years before admission, while she was going through significant life stress, and during that period she sought the help of several neurologists, neurosurgeons, psychiatrists and orthopaedists. However, she was first seen by a neurologist who, considering her disorder primarily psychogenic, referred her to a psychiatrist.

We agree with the author's opinion that psychiatrists should be cautious in attributing any dystonic abnormality to a purely psychogenic causation. As our case shows, however, we would like to extend this advice to other clinicians, in particular neurologists.

M. H. PINTO DE AZEVEDO
MARGARIDA MONIZ

Day Hospital, Clinica Psiquiatrica
University Hospital of Coimbra
3049 Coimbra Codex
Portugal

References

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Primitive reflexes in Alzheimer's disease

SIR: Girling & Berrios (*Journal*, December 1990, **157**, 888–893) report on an uncontrolled study of 146 elderly patients with clinical evidence of Alzheimer's disease and find a correlation between cognitive impairment, frontal lobe signs, including primitive reflexes, and extrapyramidal signs.

We have carried out a pilot study of 133 female patients above the age of 55 in a large psychiatric hospital in order to see if primitive reflexes were associated with cognitive impairment.

The mean age of our population was 78.6 years, similar to the psychiatric ward population of Girling & Berrios, which had a mean age of 80.0 years. We found the following frequencies of primitive reflexes: glabellar tap 81.2%, grasp reflex 52.6%, sucking reflex 30.8%, forced grasping 33%, palmomental reflex 23.3%, snout reflex 26.3%. These reflexes were found more frequently in patients with severe global dementia. When reflex frequency was plotted against age in the severely demented group, there appeared to be a bimodal distribution with a dip in frequency in the 75–79 age range.

These preliminary findings are compatible with those of Girling & Berrios and with the existence of two subtypes of Alzheimer's disease, as postulated by several authors.

However, the experiment should be replicated in a controlled study of a much larger population of patients with clinical evidence of Alzheimer's disease to minimise the possibility of an age effect.

PAUL CRICHTON

Charing Cross Hospital
Fulham Palace Road
London

CARRICK MCDONALD

Warlingham Park Hospital
Warlingham
Surrey CR3 9YR

Panic attacks in schizophrenia

SIR: Argyle (*Journal*, September 1990, **157**, 430–433) studies the occurrence of regular panic attacks in 20 chronic schizophrenic patients, and finds seven cases, a far from negligible amount. The author also reports that neuroleptics may increase panic attacks in some psychotic patients.