submaximal ECT, perhaps due to residual effects of his carbamazepine. However, when multiple ECT has been given therapeutically in one session, it has not led to improved clinical efficacy. In a study of 38 patients receiving multiple ECT, only one patient improved after the first session (Abrams & Fink, 1972). It is also unlikely that the patient's ECT was submaximal since all 11 ECTs produced bilateral seizures lasting from 25 seconds to 100 seconds. It is also unlikely that carbamazepine would have affected seizure generation up to eight weeks after the last dose. We are thus uncertain as to the mechanism underlying this patient's differential response to spontaneous seizures and ECT, and would be very interested to hear of any other similar cases.

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Nifedipine-induced depression

SIR: Eccleston & Cole (1990) recently reported a case of treatment-resistant depression associated with nifedipine. They also cited Hullett *et al* (1988) who described four cases of depression associated with nifedipine, including one case of treatment resistance. A computer-based literature search failed to produce any further similar case reports.

The following is a description of a case of major depression with melancholia and mood-congruent psychotic features, further characterised by a positive dexamethasone suppression test, associated with nifedipine:

Case report. Mr A., a 66-year-old property developer, presented with a two-month history of severe agitated depression associated with delusions of poverty. Symptoms included total anhedonia, loss of energy and interest, hopelessness, guilt, suicidal ideas and nihilism for the future. He was unable to concentrate or make decisions and had completely lost confidence. He was restless and agitated, had a high level of anxiety and had experienced several panic attacks. He could not stop worrying about his financial state, believed that he was bankrupt and 'ruined' and could not be reassured by evidence to the contrary. Associated symptoms included middle and terminal insomnia, anorexia with weight loss of 7 kg and marked diurnal mood variation.

He had no past personal or family history of psychiatric illness. Medically, Mr A. had never had any serious illnesses. Mild hypertension had been discovered three months before the onset of his depression, initially treated with alphamethyl dopa, but this was changed to nifedipine 20 mg b.d. after two months. He felt quite well when nifedipine was introduced. About two to three weeks after nifedipine was commenced, the symptoms of depression appeared. Other psychosocial stresses were the death of his mother three months earlier, and some genuine but not excessive economic losses.

Mr A.'s usual alcohol intake was 40–50 g per day and he was taking no other medication. Premorbidly, he was described by his wife as an active, energetic and interested man who had been successful in business, was sociable and well-liked.

On admission, the Hamilton Rating Scale Depression (HRSD) score (21 item) was 39. Physical examination was normal and his blood pressure was 140/80. The nifedipine was ceased and he was commenced on dothiepin (50 mg increasing gradually over five days to 150 mg nocte) and haloperidol (5 mg twice daily). Biochemistry screen, full blood count, thyroid function tests, B12 and folate levels were all normal. The dexamethasone suppression test showed non-suppression at 17 hours following dexamethasone (1 mg orally) (baseline cortisol 344 nmol/l, 9-hour level 74 nmol/l, 17-hour level 189 nmol/l. Dexamethasone levels at nine hours and 17 hours were 5.2 and 2.3 nmol/l respectively).

Mr A. showed significant improvement within 48 hours of ceasing nifedipine and at one week his HRSD score had fallen to 10. The haloperidol was rapidly reduced. At discharge after two weeks he was virtually asymptomatic, with only some preoccupation with financial matters persisting. At follow-up two weeks later he was completely recovered, with a HRSD score of zero.

The major features which implicate nifedipine in the aetiology of Mr A.'s depression are temporal. The symptoms began within one month of commencing nifedipine and improved dramatically within days of its cessation. The negative past history and family history also weigh against a non-organic aetiology. However, significant psychosocial stresses were present, i.e. a recent bereavement and concurrent financial difficulties, which are also of aetiological significance. In addition, an antidepressant and an antipsychotic were commenced on the same day as the nifedipine was ceased and even though it would be most unusual for such a severe depression with psychotic features to respond to a low dose of tricyclic within 48 hours, such a response cannot be discounted.

The most striking similarity between this case and those reported by Eccleston & Cole (1990), and Hullett *et al* (1988) is the rapidity with which recovery

occurred after withdrawal of the nifedipine. In each case, significant improvement occurred almost immediately and recovery within one week was usual. The temporal association alone is insufficient to prove a causal relationship between nifedipine and depression, but in view of the widespread use of nifedipine, particularly by the elderly, clinicians should be alert to the possibility of depression occurring as an adverse effect.

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Bizarre delusion and post-hemiplegic hemidystonia

SIR: Owens (*Journal*, May 1990, **156**, 620–634) recently underlined the importance of being familiar with the manifold presentations of dystonia, as they are a potential psychiatric pitfall. In the absence of clearly identifiable clinical causes, dystonic symptoms are often considered psychogenic (Pinto de Azevedo, *Journal*, March 1991, **158**, 436). However, we are unaware of any reports of psychiatric changes in patients with symptomatic dystonia. To illustrate the need for a more systematic psychiatric investigation, we describe a case of a post-hemiplegic hemidystonia associated with bizarre delusions, which is the first report of an organic delusional disorder in this rare neurological disturbance.

Case report. The patient was a 65-year-old man, who had been struck by a car three years earlier, leading to a traumatic hemorrhage in the left basal ganglia with a right hemiplegia and an organic personality syndrome. Five months later he developed a bizarre delusion: he said artificial tubes had been implanted into his body, beginning under his tongue, running down his chest and stomach to his back, and forming two intestinal outlets. Furthermore, a metal stabilisation system had been extracted from his left leg. One year later, slow involuntary right-rotating muscle contractions of his right hand appeared, later extending to his right arm, shoulder, neck, mouth, tongue, and facial muscles. A computerised tomography scan showed a hypodense zone 11 mm × 3 mm in size, beginning in the left medio-dorsal part of the lentiform nucleus and extending to the fronto-parietal white matter.

Pettigrew & Jankovic (1985) provided evidence that contra-lateral basal ganglia damage and a history of hemiparesis due to head trauma and cerebral haemorrhage can be found in a number of hemidystonic patients. A considerable delay of months or years before the onset of dystonic symptoms and a subsequent progression of symptoms has also been reported (Grimes *et al*, 1982). A disconnection between the striatum and the thalamus with relative preservation of the cortico-spinal pathway is discussed as the underlying pathomechanism (Dooling & Adams, 1975). Although in our case the possible role of an intermittent unsuccessful neuroleptic therapy in the triggering of the dystonic symptoms (Myslobodsky *et al*, 1984) remains obscure, the topography of the brain damage and the patient's history support a diagnosis of post-traumatic hemidystonia.

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CORRIGENDUM

Journal, August 1991, **159**, 294. The references to Szabadi & Cashman should read Burrell *et al*.