of buspirone (15 and 30 mg) did not lead to increased Parkinsonian disability and a beneficial effect was maintained over two weeks of treatment. Subsequent cessation of buspirone for 48 hours led to immediate deterioration of dyskinesias. Both patients requested reintroduction of buspirone and have now been treated with constant benefit for two months.

The drug was generally well tolerated: three patients reported occasional lightheadedness during the first days of dose increment and in one patient pre-existing benign visual hallucinations present with levodopa and apomorphine became more intense with 15 mg of buspirone. All patients experienced a heightened degree of relaxation and tranquillity.

These preliminary results provide some evidence that in a dose of 15–30 mg a day buspirone may be useful in producing selective anti-dyskinetic effects. Whether this occurs through its D2 antagonist actions or a non-specific anxiolytic action is unclear.

We thank Bristol-Meyers Pharmaceuticals, UK, for supplies of drugs.

B KLEEDORFER
AJ LEES
GM STERN
Department of Neurology, The Middlesex Hospital, London W1N, UK

Corrspondence to: Dr Lees

1 Quinn N. The modern management of Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1990;53:93–5.
2 Price P, Parkes JD, Marsden CD. Tiapride in Parkinson's disease. *Lancet* 1978;ii:1006.
3 Tarsy D, Parkes JD, Marsden CD. Metoclopramide and pimozide in Parkinson's disease and levodopa-induced dyskinesias. *J Neurol Neurosurg Psychiatry* 1975;38:331–5.
4 Temple DL, Yevich JP, New JS. Buspirone: chemical profile of a new class of anxioselective agents. *J Clin Psychiatry* 1982;43:4–9.

Exacerbation of myasthenia by propafenone

We report generalised myasthenic symptoms developing in a patient with longstanding ocular myasthenia (OMG) treated with propafenone for cardiac dysrhthymias complicating ischaemic heart disease.

A 68 year old man had a 14 year history of ocular myasthenia. His ptosis and diplopia were well controlled on pyridostigmine 660 mg daily. He had angina and had suffered three episodes of myocardial infarction. Two years before admission he developed a sudden right hemiplegia. This was thought to be of embolic origin and he was anticoagulated with warfarin.

He was admitted with palpitation and episodes of presyncope. He was found to be in atrial fibrillation with frequent ventricular ectopics and runs of ventricular tachycardia (VT). In addition to pyridostigmine and warfarin, he was receiving frusemide 40 mg and captopril 37·5 mg daily. Three days later propafenone 450 mg daily was added.

Within a few hours of starting the propafenone, the patient and his relatives noted a marked increase in ptosis and diplopia. For the first time, he developed features of generalised myasthenia with dysarthria, dysphagia and generalised limb weakness. He also developed dyspnoea although this may have been partly of cardiac origin. Two weeks after admission a permanent cardiac pacemaker was inserted because of severe sinus bradycardia and the propafenone was stopped. There was prompt improvement in the diplopia and dyspnoea and the patient reported marked improvement in well-being. Seven days later he was readmitted with an infected haematoma in the pacemaker site. Tachydysrhythmias were briefly treated with amiodarone and disopyramide. He then collapsed with VT and required ventilation. There was deterioration in his myasthenia and prednisolone 60 mg daily was started. He subsequently made a good recovery. His myasthenia is well controlled on prednisolone 25 mg on alternate days and the pyridostigmine has been withdrawn.

Although, by definition, the clinical disturbance in OMG is confined to the external ocular muscles, there is strong evidence for a widespread sub-clinical disturbance of the neuromuscular junction in other striated muscles.[1] Propafenone is a class Ic antiarrhythmic drug, blocking fast sodium channels in cardiac conducting tissue, with weaker β-blocking activity.[2] It is suggested that the former of these actions may interfere with the generation or propagation of the motor endplate potential. This effect is presumably insignificant in the normal subject but important if neuromuscular transmission is already compromised. The drug has been in clinical use since 1977 and we are not aware of any published reports of adverse effects of this type. The manufacturers are aware of five instances of myasthenic-like reactions which are not well documented (E Chong, personal communication, 1990). A case of extraocular muscle palsy has been reported in a patient receiving the related drug etafonone but this disturbance was not edrophonium-responsive.[3]

BRF LECKY
Mersey Regional Department of Neurology, Walton Hospital, Liverpool L9 1AE, UK
D WEIR
Regional Department of Cardiology, Broadgreen Hospital, Liverpool L14, UK
E CHONG
Knoll Limited, Maidenhead SL6 1DU, UK

1 Lecky BRF, Morgan-Hughes JA, *et al.* Intercostal muscle acetylcholine receptors in long-standing ocular myasthenia. *Lancet* 1980; ii:1311.
2 Dukes ID, Vaughan Williams EM. The multiple modes of action of propafenone. *Eur Heart J* 1984;5:115–25.
3 Fierro B, Castiglione MG, *et al.* Myasthenia-like syndrome induced by cardiovascular agents. *Ital J Neurol Sci* 1987;8:167–70.

Unsuccessful treatment of subacute sclerosing panencephalitis treated with transfusion of peripheral blood lymphocytes from an identical twin

Subacute sclerosing panencephalitis (SSPE) is a rare and invariably fatal illness of childhood. Because of its association with various immunological abnormalities immunotherapy has been attempted in the past. We report our experience with a case of SSPE in one of two identical twins in whom treatment with transfusion of lymphocytes from the healthy twin was unsuccessful.

The patient, a 19 year old girl, presented with a ten months history of involuntary movements, mental deterioration and personality change. The onset of her symptoms was insidious and there was no fever, headaches or other symptoms at any stage of her illness. Her initial symptoms were brief, sudden jerky movements of the limbs. A few weeks later she became forgetful, emotionally labile and incontinent of urine. The involuntary movements also increased in frequency and severity and spread to involve the face. Over the following few months her mental function progressively deteriorated and she became childish and also increasingly dependent on others for daily living activities.

She had uncomplicated measles in early childhood but she was otherwise healthy. Her family history was non-contributory. The patient had an identical twin sister.

When seen at our hospital 10 months after her initial symptoms she had marked intellectual deterioration to the extent that a formal assessment of mental function was not possible. There were repetitive cries and utterances and stereotyped myoclonic jerks. Her tendon reflexes were symmetrically brisk in all limbs and both plantar responses were extensor. There were also multiple spider naevi. The rest of the physical examination was unremarkable.

Routine investigations were normal. EEG demonstrated repetitive stereotyped high voltage delta activity which occurred every 6–7s and was synchronous with the involuntary movements, findings which are typical of SSPE. A brain CT scan was normal. CSF protein was 0·26 g/l with normal cells and glucose. Oligoclonal bands were detected in the CSF. The CSF IgG percentage of total protein was 28·2% and IgG/albumin index was 2·48 (normal values are $<10\%$ and 0·22–0·66, respectively). Measles antibody titre was 1:2048 in CSF and 1:32 in serum. Peripheral blood lymphocyte (PBL) subsets were normal (table).

The patient was transfused with 300 ml of PBL from her healthy ABO and HLA identical twin sister. This procedure was repeated three days later. Serial EEGs following transfusion were similar to that at presentation. Repeated CSF examinations following transfusion were also similar to the pre-treatment values but PBL subsets measurements showed a significant reduction in total T lymphocytes and also in T helper cells (table). Despite treatment, the patient's condition continued to deteriorate and she died five weeks later.

Life-long persistence of the measles virus

Table Peripheral blood lymphocyte subsets before and after treatment

PBL						*CSF*	
Total T 71 + 6·4	*T(H) 48 + 3·9*	*T(S) 23 + 5·4*	*B 11 + 3·9*	*NK 10 + 5·3*	*Protein*	*Ig% <10*	*Ig/albumin 0·22–0·66*
74	43	29	9	8	0·26	21·7	1·67
54	31	24	10	16	0·16	28·2	2·48
—	—	—	—	—	0·20	25·3	2·08