either relapsing or progressive over a period of six to 12 months by the time of presentation. Two patients have had bilateral disease.[4 7] Some patients have responded to and stabilised on steroids and in a number, surgical biopsy has been performed to confirm the diagnosis. Dutton felt that surgical decompression of the nerve helped stabilise the condition.

Optic perineuritis is best confirmed histologically, because other causes of optic nerve sheath lesions, particularly meningioma cannot always be excluded by neuroimaging alone. Most reports have described thickening of the optic nerve sheath from fibrotic changes with varying amounts of chronic lymphocytic or plasma cell infiltrate or granulomatous changes. The intracranial nerve has been found to be pale and atrophic with chronic inflammatory infiltration as in our own case and that of Zhang[2] or swollen with perivasculitis.[3] The most complete description of pathological changes[4] showed concentric deposition of collagenous fibroconnective tissue in the dural sheath with necrobiotic granulomas and a chronic inflammatory infiltrate causing a compressive optic neuropathy with ischaemic infarction. The inflammatory reaction did not extend beneath the pia mater. Electron microscopy showed exuberant fibroplasia, collagenosis and elastogenesis associated with focal extracellular collagen degeneration. The histological changes are non-specific. In suspected cases, biopsy is better taken from the intraorbital nerve rather than intracranially so that detailed examination of the optic nerve sheath can be performed.

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Oxcarbazepine sensitivity treated by desensitisation

Allergic reactions to anticonvulsant drugs may require a change of therapy. An alternative strategy is to desensitise the patient to the offending drug. This has already been described with carbamazepine.[1]

Oxcarbazepine is a new anticonvulsant drug developed as an alternative for patients unable to tolerate carbamazepine and supposedly causing fewer side-effects and allergic reactions.[2] As yet, it is only available on a named-patient basis. Sensitivity to both carbamazepine and oxcarbazepine does occur. We describe a patient successfully managed by densensitisation to oxcarbazepine. We believe this is the first reported case of successful densensitisation to oxcarbazepine.

This 23 year old single man with mild mental handicap lives with his parents. He had a head injury in 1979 resulting in a post-traumatic amnesia of 24 hours. He developed complex partial seizures in 1982, but treatment was not started until 1983 when he had his first generalised seizure. A resting EEG was then normal. Phenytoin greatly reduced his seizure frequency but resulted in problems of slowed cognition and mild toxicity.

In 1987 he developed a psychotic illness characterised by persecutory delusions, delusions of reference and third person derogatory auditory hallucinations. He described the hallucinations as occurring episodically. He had three admissions to a local psychiatric hospital over the next 18 months but the psychotic phenomena continued despite high-dose antipsychotic medication.

In view of his problems on phenytoin and the possibility that the hallucinations may have been related to epileptic phenomena rather than schizophrenia, he was started on carbamazepine in August 1988. Within one week he had developed a fever, generalised erythematous rash and lymphadenopathy. His leucocyte count was raised with an eosinophilia. All these symptoms and signs resolved over the next eight days after carbamazepine was withdrawn.

He was then referred to this hospital for further assessment. Continuous EEG monitoring for five days was unhelpful as he had no hallucinations during this time and the EEG remained normal. Oxcarbazepine was prescribed as an alternative to carbamazepine. Within 12 hours of the first dose (300 mg) he developed a generalised itchy erythematous rash and a fever. Leucocyte count was within the normal range but with a mild eosinophilia. Oxcarbazepine was stopped and his symptoms subsided within 36 hours with antipyretic treatment alone.

Desensitisation was then attempted using low dose oxcarbazepine capsules prepared by the local pharmacy. Starting at 0·1 mg daily, the dose was doubled every two days. On day 2, however, he developed mild itching and erythema on his hands and abdomen, but remained systemically well. The next dose increase was withheld until the symptoms disappeared 24 hours later. On day 19 (50 mg/day) he developed mild itching and erythema confined to his hands. Again the dosage increase was withheld until the symptoms subsided 144 hours later. No further adverse experiences were seen during desensitisation. By day 63 he achieved 1200 mg per day. Phenytoin was then tailed off. One month later he was well established on this dose of oxcarbazepine, with no adverse effects, and with some reduction in the hallucinations. He remains on regular antipsychotic medication and lithium carbonate.

When allergic drug reactions occur, desensitisation can be a useful alternative to changing anticonvulsant therapy. In view of the risk of a severe drug reaction (blood dyscrasia, renal or hepatic toxicity) desensitisation should only be undertaken in selected cases where there are no other satisfactory alternatives. Oxcarbazepine is a promising new anticonvulsant and should not necessarily be abandoned when such sensitivity occurs, but a desensitisation regime considered instead.

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Buspirone in the treatment of levodopa induced dyskinesias

Long-term levodopa treatment of patients with Parkinson's disease is commonly complicated by on-off fluctuations and dyskinesias. While recent advances have been made in the treatment of fluctuations[1] inter-dose dyskinesias have become an increasing problem in the management of the long-term levodopa syndrome. Dopamine receptor antagonists have been shown to be effective in reducing levodopa induced dyskinesias but only at the expense of increased Parkinsonian disability.[2 3] We have tested the azapirone drug buspirone which has mixed dopamine-2 receptor agonist-antagonist properties and main serotonin-1A agonist activity.[4]

Five patients (one female, four male) with idiopathic Parkinson's disease gave their informed consent to participate in the trial. Their mean age was 56·2 (47–72) years, mean duration of disease 10·2 (6–20) years and of levodopa treatment 8·2 (4–15) years. All had on-off fluctuations as well as disabling peak-dose dyskinesias and all but one were treated with intermittent subcutaneous injections of apomorphine (mean daily dose 14 mg, range 4–30 mg) in addition to their oral levodopa (mean daily dose 865 mg, range 625–1·250 mg).

In three patients acute challenges with single doses of buspirone (10 and 20 mg) given 30 minutes before an apomorphine injection did not show any influence on the severity of involuntary movements (AIM scale) when compared with that seen after apomorphine alone. The "on" quality of motor response (Webster scale) was not affected and no side effects occurred.

All patients were then treated with daily doses of 15, 30, 45 and 60 mg of buspirone over three consecutive days. For assessment of inter-dose dyskinesias patients kept a self-scoring diary for three days before starting buspirone and throughout the treatment period. Involuntary movements were scored 1 when mild (not interfering with daily routine), 2 when moderate (interfering with daily routine, but able to continue) and 3 when severe (unable to continue with daily routine).

All five patients experienced a 10 to 40 (mean 20)% reduction of disabling (score 2 and 3) involuntary movements, three at 15 mg of buspirone per day and one patient each at 30 and 60 mg, respectively. In three of them this was, however, associated with an increased frequency of "off" periods. Although compensated by an increased number of apomorphine injections without increasing dyskinesias in two patients this form of "titration" treatment was considered too complicated to embark on long-term therapy. In two patients the anti-dyskinetic daily dose

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.

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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]

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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