either relapsing or progressive over a period of six to 12 months by the time of presentation. Two patients had had bilateral disease. 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 decompensation of the nerve helped stabilise the condition.

Optic neuropathy 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 fibroblastic 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 or swollen with perivasculitis. The most complete description of pathological changes showed centric deposition of collagenous fibroconnective tissue in the dural sheath with neovascular 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 intracranial 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 may be to desensitise the patient to the offending drug. This has already been described with carbamazepine. 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. 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 desensitisation to oxcarbazepine. We believe this is the first reported case of successful desensitisation to oxcarbazepine.

This 23 year old single man with mild mental handicap lives with his parents. He had a head injury 1982 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 antipyrctic treatment.

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 14 hours later. No further adverse reactions were seen during desensitisation.

By day 63 he achieved 1200 mg per day. Phenytoin was then tapered 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 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 attempted in select 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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Buprisone in the treatment of levodopa induced dyskinasias

Long-term levodopa treatment of patients with Parkinson’s disease is commonly complicated by on-off fluctuations and dyskinesia. While recent advances have been made in the treatment of fluctuations inter-dose dyskinesias have become an increasing problem in the management of the long-term levodopa syndrome. Dopamine receptor antagonists have not shown benefit in reducing levodopa induced dyskinesias but only at the expense of increased Parkinsonian disability. We have tested the azapirone and buprisone which has shown no effect on dyskinesias and has problems that preclude the use of levodopa.

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 (247–72) years, mean duration of disease 10±2 (6–20) years and 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 625–1250 mg).

In three patients acute challenges with single doses of buprisone (10 and 20 mg) given 30 minutes before an apomorphine injection did not show any effect 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 by any side effects of buprisone.

All patients were then treated with daily doses of 15, 30, 45 and 60 mg of buprisone over three consecutive days. For assessment of inter-dose dyskinesias patients kept a self-scoring diary for three days before starting buprisone and throughout the treatment period. Involuntary movements were scored 1 when mild (not interfering with daily routine), 2 when moderate (with occasional 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 buprisone per day and one patient each at 30 and 60 mg, respectively. In three of them this was, however, accompanied 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...
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D Watts and J Bird

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