developed without difficulty at a dose of 20 mg/day. A year later the patient developed a depressive syndrome and fluoxetine was indicated at a dose of 20 mg/day. Previously the patient had been on 115 mg/day. Ten days later she developed vomiting, difficulty with getting up and sitting and vertigo. The neurological examination showed trunk ataxia, limb dystonia, and multidirectional nystagmus. Phenytion plasma level was 47 μg/ml.

The fluoxetine was suspended and there was a progressive recovery of the signs and symptoms with a complete recovery in approximately three weeks. Four weeks after suspension of fluoxetine, the phenytion plasma level was 20 μg/ml for the same described dose.

In human studies an alteration of the pharmacokinetics of fluoxetine was not found when administered simultaneously with other drugs (such as, ethanol, diazepam, chlorothiazide, tolbutamide and warfarin) and hydrocortisone. In studies it was shown that fluoxetine is a potent inhibitor of hepatic microsomal metabolism, this could be responsible for the increase of the phenytion plasma level in these cases. The interaction between the first administration of fluoxetine and the beginning of the phenytion overdose symptoms, also suggests a mechanism of the metabolic alteration in the degradation of the anticonvulsant drug.

Correspondence to: Dr Jalil, Avda Salvador 2194, Santiago, Chile


1 Crowson AR. A hypothesis on the pathophysiological mechanisms that underlie levodopa or dopamine agonist-induced dyskinesia in Parkinson's disease. Implications for future strategies in treatment. Mov Disord 1990;5:100.

Gamma vinyl GABA in the treatment of Levodopa-induced dyskinesias in Parkinson's disease

In non-human primates blockade of the GABAergic inhibitory strio-pallidal pathways to the lateral segment of the globus pallidus causes chorea, whereas stimulation causes a Parkinsonian syndrome. This has led to renewed interest in the potential value of gabaminergic agents in the treatment of Parkinson's disease and the complications of levodopa therapy.

We have investigated the irreversible inhibitor of GABAtransaminase, gamma vinyl GABA (GVG) in the treatment of disabling levodopa-induced chorea in 5 patients with Parkinson's disease. The patients had a mean age of 54 (41-74) years, a mean duration of disease of 11 (7-21) years, a mean duration of levodopa therapy 9 (6-18) years and a mean levodopa dose of 760 (450-1400) mg/day. All the patients were also receiving subcutaneous apomorphine, 4 selegiline and 1 bro-mocriptine.

The patients' dyskinesias were assessed over a one week baseline period on optimum anti-Parkinsonian therapy. They were then given 2 Gm/day of GVG for one week and 3 Gm GVG for a second week. Assessment of dyskinesia severity was carried out using a 4-point scale after a standard therapeutic dose of SC apomorphine.

The patients kept self-scoring diaries for three days of each week to assess the number of hours “on” with and without dyskinesias and the number of hours “off.” Baseline assessments showed that dyskinesias were more severe later in the day in all patients. On GVG no change in dyskinesia severity occurred as judged by either the apomorphine challenge or the self-scoring diaries, but there was an increase in off hours from 3 to 4 hours.

Four patients were unable to tolerate more than 2 Gm GVG due to increased severity of Parkinsonian symptoms. The other patient also noticed worsening of Parkinsonism on 3 Gm GVG.

Contradicting results with probalide, a gabaminergic agonist, have been reported in the levodopa-induced dyskinesias in Parkinson's disease. GVG was reported to aggravate Parkinsonism without improving tardive dyskinesias in psychotic patients on sustained neuroleptic therapy. GABA mimetic drugs therefore appear to have complex and contradictory actions in patients with movement disorders. This study is of interest in that aggravation of Parkinsonism occurred without significant reduction in dyskinesias suggesting that these two phenomena may not be inextricable.

Hypochromia iridis in acquired Horner's syndrome

The uncommon condition of congenital Horner's syndrome consists of ptosis, miosis, facial anhidrosis and hypochromia of the affected iris. This condition commonly results from injury to the brachial plexus at birth. The mechanism of the hypochromia iridis is generally thought to be that of failure of pigment development rather than loss of pigment that has already formed. Hypochromia iridis is a rare condition. Hypochromia iridis in the iris following acquired Horner's syndrome has been reported but is rare. We report a case of this rare but interesting manifestation of damage to the sympathetic nervous system to the eye.

A 17 year old man was involved in a motorcycle accident and suffered brachial plexus trauma, with loss of power and sensation in the right arm followed by pain. Examination 23 years later revealed complete loss of vision in the right eye. There was no photophobia and no hyphema. There was a left ptosis with corresponding sensory loss. He had post-traumatic brachial plexopathy pain for which he was seeking advice. Examination also revealed a right Horner's syndrome with loss of pigment in the right eye, his left being coloured grey/green.

Several mechanisms by which alteration in sympathetic function may influence iris pigmentation have been proposed. There may be failure of delivery of noradrenaline or other melanin precursors to the melanocytes in the iris, perhaps mediated via cyclic adenosine monophosphate. There may be loss of activation of prostaglandins, or their precursors, or some melanotropic moiety, that are involved in melanin synthesis. Several cases of depigmentation of the iris or horizontal iridis or horizontal iridis or hypopigmentation of the iris have been reported following injury to the sympathetic nervous system, but this condition in the acquired state appears to be rare although it may often be unrecognized.

Shoulder pain from glomus tumour

Localised pain in the shoulder often suggests a brachial plexus neuropathy or cervical radiculopathy. Pain limited to a small area with sensory loss suggests a focal nerve lesion such as a neuroma. Glomus tumours are usually small and may cause arm pain distally; they rarely occur proximally.

A 41 year old woman presented with point tenderness in the right upper arm and shoulder that localized to the lateral suprascapular area. The tender area, which had been present for several years, was less than 1 cm in diameter and located lateral to the spine of the right scapula. A friendly 'touch on the shoulder' would cause an
Acute dystonia due to amitriptyline

Acute dystonic reactions are most frequently seen in patients receiving neuroleptic medication or metoclopramide but have also been observed in association with phenytoin and carbamazepine. Although it is generally believed that the anticholinergic and dopaminergic properties of these drugs cause such reactions the precise mechanism is unclear although it has been suggested that they may be due to enhanced dopaminergic neuronal release on supersensitive post-synaptic receptors. However, most explanations cannot fully explain why only a small proportion of patients develop acute dystonia or why it may occur durin delayed chemotherapy. A 20 year old man was admitted with severe muscular spasm. He had first become aware of stiffness in his lower limbs whilst jogging on the day of admission. He then developed spontaneous arching of his back and involuntary tongue protrusion. His past medical history was unremarkable but because of a depressive illness he had been taking amitriptyline 50 mg daily for three months prior to his admission. He denied taking any other medication. A subsequent examination of his tablets confirmed they were amitriptyline. Examination revealed marked opisthotonus, retrocollis and orofacial contortion with spontaneous tongue protrusion. Intravenous prochloride (10 mg) terminated the attack. Routine haematological and biochemical indices were normal.

Amitriptyline and other tricyclic antidepressants only rarely cause extrapyramidal side-effects although tremor, dystardia and akathisia have been observed. There are at least two previous reports of acute dystonia due to amitriptyline and we believe this patient is a further case. Whilst tricyclic antidepressants have anti-cholinergic properties and potentiate the actions of biological amines in the central nervous system the mechanism of this reaction seems unclear. However the drugs are widely used and it is a side effect that prescribers should be aware of.

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Paradoxical akathisia response to apomorphine in Parkinsonism

Apomorphine is a direct D1 and D2 dopaminergic receptor agonist, and its efficacy in Parkinson's disease (PD) depends on intact post-synaptic receptors. Administration by subcutaneous injection and intranasally, has been beneficial in Parkinsonian patients with declining motor response, intractable on/off fluctuations which are commonly accompanied by dyskinesia. In addition, these patients may present with choreoatetoid and dystonic symptoms. We describe studies in a patient who showed a hitherto unreported profound akathisic response to the drug. A 60 year old man presented in 1988 with three months of lethargy, slowness of movements and slurred speech. Examination showed an extrapyramidal type of dystarhia, facial hypomimia, reduced spontaneous and automatic movements, symmetrical bradykinesia of both upper limbs, micrographia and a shuffling, short stepped gait. There was no tremor, no supranuclear palsy and no signs of autonomic denervation; rigidity was minimal in axial and appendicular. Dystonia was minimal and treatment was withheld.

Over the next year he deteriorated with increasing gait disorder, difficulty with stairs and reduced arm-swing, but no tremor. In July 1989 he was given Sinemet plus, three times daily, without improvement. He was admitted for further investigation and treatment. Examination confirmed the previous signs there was symmetrically diminished arm-swing, slight postural flexion and masked facies; tremor was absent, rigidity minimal.

Routine haematological, biochemical, intravenous edrophonium tests and CT head scan were normal. EMG showed no myasthenic reaction.

All drugs were withdrawn for 24 hours. An oral dose of 2 tablets of Madopar (levodopa 400 mg, benzarzide 100 mg) given at 9.00 am produced no significant change in the Webster rating 4 hours for 3 hours (table). On a separate day, on dopirone 20 mg, 4 hours, apomorphine 2 mg, 4 mg and 6 mg were administered subcutaneously at 8 hour intervals. Serial Webster scores recorded over 2 hours.

Apomorphine 4 mg produced no change in score at 5 and 10 minutes. At 15 minutes he became totally immobile and mute, lying on his bed, conscious but apparently drowsy and sweating. There was no voluntary movement to commands, muscle tone was not obviously altered from his pre-treatment state. Eyes were closed, mouth slightly open, no abnormal movements were seen. Webster scores are shown in the table. This state continued until 90 minutes when he walked to the office door and his Webster score had returned to basal values. Identical episodes, with profound akinesia, resembling a very severe "off" period occurred with both 2 mg and 6 mg doses. On the latter dose there was a short period of pre-syncope, BP 90/60 mm Hg, pulse 52/min.

The batch of apomorphine was assayed by the manufacturers and its potency and freedom from contaminants were confirmed.

The diagnosis of idiopathic PD is excluded by bilateral signs at presentation, lack of tremor and lack of response to anticholinergic drugs. The probable diagnosis is striatonigral degeneration, with no current evidence

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<tr>
<th>Table</th>
<th>Total Webster scores (10 items maximum score 10)</th>
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<tr>
<td></td>
<td>Time (mins)</td>
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<tr>
<td>Oral Madopar × 2 (400 mgms Levodopa)</td>
<td>6</td>
</tr>
<tr>
<td>Apomorphine</td>
<td>2 mg</td>
</tr>
<tr>
<td>Apomorphine (Subcutaneous)</td>
<td>4 mg</td>
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<td>6 mg</td>
<td>3</td>
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Shoulder pain from glomus tumour.

E W Massey

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