symptoms, becoming completely normal after 4 weeks. Fluoxetine was reintroduced at the same dose and the depressive syndrome disappeared without recurrence of toxic effects.

A second case, a 57 year old woman, developed a generalised secondary partial epilepsy 3 months after an embolic cerebral infarct. Phenytoin was introduced at 400 mg/day. A year later the patient developed a depressive syndrome and fluoxetine was indicated at a dose of 20 mg/day. Previously the phenytoin plasma level had been 11.5 µg/ml. Ten days later she developed vomiting, difficulty with getting up and sitting and vertigo. The neurological examination showed trunk ataxia, limb dysmetria, and multidirectional nystagmus. Phenytoin 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 phenytoin 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 there was no toxicity. In animal studies it was shown that fluoxetine is a potent inhibitor of hepatic microsomal metabolism, this could be responsible for the increase of the phenytoin plasma level in these cases. The interaction between the first administration of fluoxetine and the beginning of the phenytoin overdose symptoms, also suggests a mechanism of the metabolic alteration in the degradation of the anticonvulsant drug.

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

In non-human primates blockade of the GABAergic inhibitory striatopallidal 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 gabamergic 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 bromocriptine.

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 then 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' 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 challenges or the self-scoring diaries, but there was a mean 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.

Confronting results with probabide, a gabamergic agonist, have been reported in 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 effects 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.

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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 are 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 partial ptosis C7 and complete C8 and T1 paresis with corresponding sensory loss. He had posttraumatic 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 activity may influence iris pigmentation have been proposed. There may be failure of delivery of noradrenaline or other melamin precursors to the melanocytes in the iris, perhaps mediated via cyclic adenosine monophosphate. There may therefore 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 heterochromia iridis and the development of pigment dispersion syndrome, or secondary irises, 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 unrecognised.

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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 neurona. Glomus tumours are occasionally found in the lateral supraclavicular 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 "touche on the shoulder" would cause an
electric shock-like sensation into the shoulder and sometimes the upper arm. Non-steroidal anti-inflammatory medications produced no relief. She denied neck pain or weakness of the extremity. Cortisone injection into the area of pain gave only minimal relief.

She had had papulofollicular thyroid carcinoma 20 years previously which required a modified radical neck dissection. There had been no recurrence. A left parotid tumour had been excised 10 years previously when she also received 5 600 rads to the left parotid.

Cranial nerves were intact and there was an obvious large scar on the right retrorgetown-area of the neck. There were no palpable nodes except for a small 1 cm tender nodule along the dorsolateral scapular border on the right. When compressed it reproduced severe pain. There were no motor signs. Sensory examination was normal, except for a small area on the top of the right shoulder. There was no loss of range of motion of the shoulder.

A neuroma was suspected. Local injection with 1% xylocaine and epinephrine at the point of tenderness over the nodule produced total relief of pain. She had resection of the nodule with relief of pain and histology revealed a glomus tumour.

Glomus tumour is rare, constituting 1–5% of all hand tumours occurring in the third to fifth decade of life.1 Over 50% of glomus tumours are subungual. However, they occur on many body surfaces, but rarely include the trunk.1,2 They usually present with a triad of severe pain, tenderness, and cold sensitivity. Paroxysms of this triad are pathognomonic.3

Glomus tumours are usually less than one centimeter in diameter and histological examination shows polyhedral cells, fibrotruma and small blood vessels. It may represent hyperplasia of a normal glomus body around arterioles.4 Prognosis is excellent and the relief spectacular, unless the tumour is incompletely removed.

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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 phenytin1 and carbamazepine. Although it is generally believed that all 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 receptor 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 during chronic therapy. A 20 year old man was admitted with severe muscular spasm. He had first been aware of stiffness in his lower limbs whilst jogging on the day of admission. He then developed spontaneous arching of his back and involuntory 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 opisthotonos, retrocolic and orofacial contortion with spontaneous tongue protrusion. Intravenous procyclidine (10 mg) terminated the attack. Routine haematological and biochemical indices were normal.

Amitriptyline and other tricyclic antidepresants only rarely cause extrapyramidal side-effects although tremor, dystarthis and akathisia have been observed. There are two previous reports of acute dystonia due to amitriptyline1,2 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 akinetic response to ampophrine in Parkinsonism

Apopomphine 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 dyskinesias, bradykinesia and other extrapyramidal symptoms. We describe studies in a patient who showed a hitherto unreported profound akinetic 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 dystarthis, facial hypomimiasis, reduced spontaneous and automatic movements, symmetrical bradykinesia of both upper limbs, micrographia and a shuffling short stepping gait. There was no tremor, no supranuclear palsy and no signs of autonomic denervation; rigidity was minimal in axial and appendicular areas. Haloperidol 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 symmetrical arm-dimensioned 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, benserazide 100 mg) given at 9.00 am produced no significant change in the Webster rating measured 1 hour for 3 hours (table). On a separate day, on dopamide 20 mg, 8 hourly, ampophrine 2 mg, 4 mg and 6 mg were administered subcutaneously at 8 hour intervals. Serial Webster scores recorded over 2 hours. Ampophrine 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 ampophrine 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 dopaminergic drugs.1 The probable diagnosis is striatogniral degeneration, with no current evidence

### Table: Total Webster scores (10 items maximum score=30)

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>120</th>
<th>150</th>
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<tbody>
<tr>
<td>Oral Madopar × 2 (400 mgs Levodopa)</td>
<td>6</td>
<td>4</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apomorphine (Subcutaneous)</td>
<td>2 mg</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>20</td>
<td>23</td>
<td>22</td>
<td>15</td>
</tr>
<tr>
<td>4 mg</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>19</td>
<td>19</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>6 mg</td>
<td>3</td>
<td>3</td>
<td>19</td>
<td>21</td>
<td>21</td>
<td>19</td>
<td>19</td>
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</tbody>
</table>
Shoulder pain from glomus tumour.

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