developed a syndrome consisting of 11-72 years, 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 amphetamine, 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 levodopa 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 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.

Contradicting results with probugide, a gabamimergic agonist, have been reported in the use of 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 alone may 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.

Correspondence to: Mr Paul Byrne

1 Hyodo T, Kare M, Shintomi Y. Two cases of congenital Horner's syndrome. Folia Ophtalmol 1983;4:387-90.
2 Ogule JW. The influence of the cervical portion of the sympathetic nerve and spinal cord upon the eye and its appendages, illustrated by clinical cases, with observations. Medicochirurg Trans 1850;41:397-440.

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 on the affected area cause arm pain distally; rarely they occur proximally.

A 41 year old woman presented with point tenderness in the right shoulder which increased 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 "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 rad to the left parotid.

Cranial nerves were intact and there was an obvious large scar on the right retromandibular area of the neck. There were no palpable nodes except for a small 1 cm tender node 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 neurona 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. 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.

Glomus tumours are usually less than one centimeter in diameter and histological examination shows polyhedral cells, fibroblasts and small blood vessels. It may represent hyperplasia of a normal glomus body around arterioles. 3 Prognosis is excellent and the relief spectacular, unless the tumour is incompletely removed.

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Acute dystonia due to amitriptyline

Amitriptyline 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, 3 has been beneficial in Parkinsonian patients with declining motor response, intractable on/off fluctuations which are commonly accompanied by dystonic choreoathetotic and pyramidal dystonic symptoms. We describe studies in a patient who showed a hitherto unreported profound akinesic 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 dysarthria, 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 derangement; rigidity was minimal in axial muscles. 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 symmetrical 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 4 hours for 3 hours (table). On a separate day, on doperside 20 mg, 8 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-syncpe, 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 loss of responsive to dopaminergic drugs. 4 The probable diagnosis is striatoniqral degeneration, with no current evidence

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**Table**

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

E W Massey

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