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 retromandibular area of the neck. There were no palpable nodes except for a small 1 cm tender nodule along the dorsolateral scalpular border on the right. When compressed it reproduced pain, severe tenderness, and cold sensitivity. Paroxysms of a polyhedral cell tumour were elicited.

Glomus tumours are 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.

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 polygonal cells, fibroblasts, and small blood vessels. It may represent hyperplasia of a normal glomus body around arteries. 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 dopamine 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 dyskinetic and dystonic chiasmic symptoms. We describe studies in a patient who showed a hitherto unreported profound akinetiic response to the drug.

A 69 year old man presented in 1988 with three months of lethargy, slowness of movements and slurred speech. Examination showed an extrapyramidal type of dystasia, facial hypomimia, reduced spontaneous and automatic movements, symmetrical bradykinesia of both upper limbs, micrographia and a shuffling left stepped gait. There was no tremor, no supranuclear palsy and no signs of autonomic denervation; rigidity was minimal in axial muscle. Dystasia was minimal and treatment was withdheld.

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 adenosine 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, benzerazide 100 mg) given at 9.00 am produced no significant change in the Webster rating measured hourly for 3 hours (table). On a separate day, on dopiridine 20 mg, 8 hourly, 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 both dopamine agonists and levodopa. The probable diagnosis is striatogniral degeneration, with no current evidence

<table>
<thead>
<tr>
<th>Table</th>
<th>Total Webster scores (10 items maximum score=30)</th>
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<tbody>
<tr>
<td>Time (min)</td>
<td>0</td>
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<tr>
<td>Oral Madopar × 2 (400 mgms Levodopa)</td>
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</tr>
<tr>
<td>Apomorphine (Subcutaneous)</td>
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</tr>
<tr>
<td>2 mg</td>
<td>6</td>
</tr>
<tr>
<td>4 mg</td>
<td>3</td>
</tr>
<tr>
<td>6 mg</td>
<td>3</td>
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</tbody>
</table>

Correspondence to: Dr Dick.


Paraclinical akinetiic response to apomorphine in Parkinsonism

Apomorphine is a direct D1 and D2 dopamine 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 dyskinetic and dystonic chiasmic symptoms. We describe studies in a patient who showed a hitherto unreported profound akinetiic response to the drug.

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