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 retromandible 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.[2] Over 50% of glomus tumours are subungual. However, they occur on many body surfaces, but rarely include the trunk.[3 4] 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, fibrostroma 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

Acute dystonic reactions are most frequently seen in patients receiving neuroleptic medication or metoclopramide but have also been observed in association with phenytoin[1] and carbamazepine.[2] Although it is generally believed that the anti-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 compensatory dopamine 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 drug 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 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 opisthotonos, retrocollis 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 antidepressants only rarely cause extrapyramidal side-effects although tremor, dysarthria and akathesia have been observed.[3] There are two previous reports of acute dystonia due to amitriptyline[4 5] 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 apomorphine in Parkinsonism

Apomorphine is a direct D1 and D2 dopamine receptor agonist,[1] and its efficacy in Parkinson's disease (PD) depends on intact post-synaptic receptors. Administration by subcutaneous injection and intranasally,[2] has been beneficial in Parkinsonian patients with declining motor response, intractable on/off fluctuations which are commonly accompanied by dyskinesias, dystonias and psychiatric 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 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 denervation; rigidity was minimal in axial musculature. Disability 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 bradykinesia, 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, benserazide 100 mg) given at 9.00 am produced no significant change in the Webster rating measured ½ hourly for 3 hours (table). On a separate day, on domperidone 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 levodopa drugs.[3] The probable diagnosis is striatonigral degeneration, with no current evidence

Table Total Webster scores (10 items maximum score/30)

	Time (mins)								
	0	*5*	*10*	*15*	*30*	*60*	*90*	*120*	*150*
Oral Madopar × 2 (400 mgms Levodopa)	6				4	6		6	6
Apomorphine (Subcutaneous)									
2 mg	6	6	5	20	23	22	15	5	
4 mg	3	3	3	19	19		3		
6 mg	4	3	19	21	21	20	19	5	