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 diethylthiocarbamate. 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 interval 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.

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


**Gamma viny 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 gabapentinergic agents in the treatment of Parkinson’s disease and the complications of levodopa therapy.

We have investigated the irreversible inhibit of GABATransaminase, gamma viny 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 subsequent 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 an initial test dose of GVG for one week, followed by a 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.

Concluding results with probabide, a gabamaminergic 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 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

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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 neurina. Glomus tumours in this region cause arm pain distally; they rarely occur proximally.

A 41 year old woman presented with point tenderness in the right shoulder over 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 action of these anti-convulsant 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 tone.

Clinical symptoms include marked hyperpyrexia of a normal glomus body around arterioles. Paroxysms of this triad are pathognomonic.

Glotmus tumours are usually less than one centimeter in diameter and histological examination shows polyhedral cells, fibrotruma and small blood vessels. It may resemble hyperplasia of a normal glomus body but is usually less than 0.5 cm in diameter. Sometimes they are encountered with a triad of severe pain, tenderness, and cold sensitivity. The paroxysms of this triad are pathognomonic.

Glotmus 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 other 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.

Acute dystonia occurs when patients develop severe dystonia or involuntary movements, which are dose-related. The symptoms can be severe and may include tremors, movements, and stiffness in the upper and lower limbs.

The symptoms are usually caused by a drug that blocks dopamine receptors, such as neuroleptics. The symptoms can be treated with drugs that stimulate dopamine receptors, such as levodopa or amantadine.

Dr. Dick

Norfolk and Norwich Hospital,
Norwich, Norfolk, UK

Correspondence to: Dr. Dick.


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Paradoxic akinesic 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 dyskinesias and other parkinsonian symptoms.

We describe 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 movement and slurred speech. Examination showed an extrapyramidal type of dystharia, 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 truncal muscle. 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 but there was symmetrically diminished arm-swing, slight postural flexion and masked facies; tremor was absent, rigidity minimal.

Routine haematological, biochemical, intravenous electrophoresis 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 three hours (table). On a separate day, on doproidide 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 woke 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 manufacturer 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 and other dopaminergic drugs. The probable diagnosis is striatogniral degeneration, with no current evidence that would suggest a diagnosis of Parkinson’s disease.