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 plasma level had been 11.5 ug/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 ug/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 ug/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 diuretics. In animal studies it was shown that fluoxetine is a potent inhibitor of hepatic microsomal metabolism, this could be responsible for the decrease 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.

PATRICIO JALIL
Servicio de Neurología, Hospital De Siervo del Rio, UDM Neurología, Pontificia Universidad Católica de Chile, Santiago, Chile

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


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

In non-human primates blockade of the GABAergic inhibitory striato-pallidal pathways to the lateral segment of the globus pallidus causes chorea, whereas stimulation causes a Parkinsonian syndrome.¹ This has led to renewed interest in the potential value of gabamnergic 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 (GVA) 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 selegeline and 1 brocomiptine.

The patients’ dyskinesias were assessed over a one week baseline period on optimum anti-Parkinsonian therapy. They were then given 2 Gm/day of GVA for one week and 3 Gm GVA 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 started 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 GVA 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 GVA due to increased severity of Parkinsonian symptoms. The other patient also noticed worsening of Parkinsonism on 3 Gm GVA.

Concontrary results with probabide, a gabamnergic agonist, have been reported in levodopa induced dyskinesias in Parkinson’s disease.²³ GVA was reported to aggravate Parkinsonism without improving tardive dyskinesias in psychotic patients on sustained neuroleptic therapy.³ 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.

NURJANISRA
t J LEES
Department of Neurology, The Middlesex Hospital, Montevideo Street, London W1N 8AA, UK

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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.¹ This condition commonly results from injury to the brachial plexus at birth.² 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 is 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 only C7 and complete C8 and T1 paresis with corresponding sensory loss. He had post-traumatic 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.² There may be failure of delivery of noradrenaline or other melanin precursors to the melanocytes in the iris, perhaps mediated via cyclic adenosine monophosphate. There may 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 have been reported in patients with Parkinson’s disease, with loss of pigment in the right eye, his left being coloured grey/green.

P BYRNE
C CLOUGH
Brook Hospital, Shooters Hill, London, UK

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 neurona. Glomus tumours are usually benign and rarely cause arm pain distally; rarely they occur proximally.

A 41 year old woman presented with point tenderness in the right infraspinatus muscle 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
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N Turjanski and A J Lees

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