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. Phenytion 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 phenytion 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. Phenytion 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 phenytion 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 diuretics.1 In animal studies it was shown that fluoxetine is a potent inhibitor of hepatic microsomal metabolism,2 this could be responsible for the increase of the phenytion plasma level in these cases. The interaction between the first administration of fluoxetine and the beginning of the phenytion overdose symptoms, also suggests a mechanism of the metabolic alteration in the degradation of the anticonvulsant drug.

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

In non-human primates blockade of the GABAAergic inhibitory strio-pallidial pathways to the lateral segment of the globus pallidus causes chorea, whereas stimulation causes a Parkinsonian syndrome.3 This has led to renewed interest in the potential value of gabaminergic 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 (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 subcutaneous apomorphine, 4 selegiline and 1 bro-mocriptine.

The patients' dyskinesias were assessed over a one week baseline period on optimum anti-Parkinsonian therapy. They were then given 2 Gm/day of GVG 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.

Conducting results with probabide, a gabaminergic agonist, have been reported in the later dopaminergic dyskinesias in Parkinson's disease.7,8 GVG was reported to aggravate Parkinsonism without improving tardive dyskinesias in psychotic patients on sustained neuroleptic therapy.9 GABA mimetic drugs appear 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.

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. This condition commonly results from injury to the brachial plexus at birth.9 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 complete 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 sympathetics may influence iris pigmentation have been proposed.4 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 iris5 or heterochromia iridis,6 where there is increased pigmentation of the iris,7 have been reported following injury to the sympathetic nervous system, but this condition in the acquired state appears to be rare, although it may often be unrecognised.
Acute dystonia due to amitriptyline

Amitriptyline, an anticholinergic drug, is widely used in the treatment of depression, anxiety, and pain. It is known for its sedative, anticholinergic, and antiarrhythmic effects. Dystonia, a movement disorder characterized by sustained muscle contractions leading to abnormal, often grotesque postures, can occur as an adverse effect of amitriptyline therapy.

Case Description:
A 60-year-old man was admitted to the hospital with a sudden onset of severe dystonia involving his limbs, trunk, and face. He had been taking amitriptyline 50 mg daily for several months without any prior history of dystonia. On admission, he was unable to move his eyes, limbs, or trunk, with sustained muscle contractions that prevented normal movement.

Dystonia was confirmed by examination, which revealed sustained muscle contractions primarily involving the trunk, face, and limbs. There was no improvement with the usual anticholinergic treatment. The patient was observed to have severe dystonia with no relief from any of the available treatments, including intravenous prochlorperazine and intramuscular botulinum toxin. These interventions did not alleviate the dystonic symptoms.

Amitriptyline was discontinued, and the patient's symptoms gradually improved over several days. Despite the improvement, dystonia recurred when amitriptyline was reintroduced at a lower dose.

Discussion:
Amitriptyline is a tricyclic antidepressant that functions as an anticholinergic, inhibiting the presynaptic release of acetylcholine. However, its mechanism of action is complex, involving multiple receptor interactions and cholinergic and dopaminergic systems. The occurrence of dystonia in this patient is likely due to its anticholinergic properties, which can lead to suppression of striatal dopamine function, contributing to the development of dystonia.

Amitriptyline-induced dystonia is a well-recognized adverse effect, often characterized by sustained muscle contractions involving the trunk and limbs. Its occurrence is rare but can be severe, necessitating the discontinuation of the drug and the use of alternative treatments. The efficacy of intravenous prochlorperazine and botulinum toxin in this case highlights the need for rapid intervention in cases of suspected amitriptyline-induced dystonia.

This case underscores the importance of recognizing amitriptyline-induced dystonia and its potential for recurrence upon rechallenge. Early discontinuation of the drug and the use of alternative treatments are crucial in managing this adverse effect.

Table: Total Webster scores (10 items, maximum score 50)

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Shoulder pain from glomus tumour.

E W Massey

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