third and nineteenth day from the beginning of symptoms, the first one being normal, and the second one showing an increase in protein (0.95 g/l) with 11 cells and negative titres to brucella, syphilis, toxoplasma, herpes simplex, varicella-zoster, and a negative antinuclear determination.

The patient's clinical pattern began to improve two weeks after admission; she was discharged a week later. Follow up a month later revealed some minimal residual areflexia and a slight dysmetria on the heel-knee test.

Electrophysiological studies (table) were carried out on the sixteenth and eighteenth day, which revealed absent sensory potentials. Motor NCS showed less abnormality than that of sensory nerves. There was delay in the distal motor latency in all the nerves tested and there was also reduction in motor amplitude in both median nerves, with a 25-3% drop in peak-to-peak amplitude between wrist and elbow, and a 39-2% drop in amplitude between wrist and axilla in the first right median nerve conduction study. Neither temporal dispersion nor motor conduction between the nerves was found. EMG studies were normal. A third electrophysiological study performed a month after her discharge showed the recovery of the sensory nerve action potentials in the lower and upper extremities, although the amplitude and sensory conduction velocity of the nerves studied were lower than the normal values. The motor NCS, however, were similar to previous values. The EMG study of the right anterior tibialis and right first dorsal interosseous muscles showed compatible changes with minimum denervation.

Our patient with Guillain-Barré syndrome was an entity whose nosological characterisation relied upon a purely descriptive base, with relatively widely accepted diagnostic criteria. Among these, the presence of muscular weakness is the most noticeable, indicating the main affection of the motor roots. However, a purely sensory clinical variant of this illness is also believed to be possible and the following features are necessary for it to be accepted: rapid onset, distribution widespread and symmetrical, complete or near recovery, high CSF protein content, with few or no cells, and an electro-physiological study compatible with a demyelinating process in the peripheral nervous system.

There is still a certain controversy as to whether this clinical variant occurs. In a recent review of 42 patients with acute sensory neuropathy, the authors concluded that this condition is not part of the spectrum of inflammatory demyelinating neuropathies, nevertheless, it is noticeable that only 2 of the 42 patients had complete remission of symptoms and that the course of their disease was very protracted—from six to nine months.

The diagnosis of acute polyneuropathy has been published in which the sensory disorders were also prominent and very similar to those of our patient. The necropsy carried out after the patient's death from pulmonary embolism revealed inflammatory infiltration with segmental demyelination in the peripheral nerves, posterior roots and spinal ganglia, with a marked preservation of the anterior roots. The findings, except for the preservation of the motor roots, are identical to those which are described in the Guillain-Barré syndrome.¹

The patient by an acute sensory neuropathy after a presumed viral illness and vaccination with a monophasic course and nearly complete recovery—both clinical and electrophysiological improvement—CSF protein content was high with a mild pleocytosis. The evidence suggests that this was probably an acute sensory inflammatory demyelinating polyneuropathy.

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Cerebral venous thrombosis in paroxysmal nocturnal haemoglobinuria

Paroxysmal nocturnal haemoglobinuria (PNH) is a rare acquired disease of the haemopoietic system manifested by chronic haemolytic anaemia, leukaopenia, and thrombocytopenia. Patients with PNH have an increased risk of developing systemic venous thrombosis, and obstruction of the hepatic veins, or Budd-Chiari syndrome (BCS) which is a highly fatal complication in about 30% of cases. PNH is also a well-established though extremely uncommon cause of cerebral venous thrombosis (CVT) 1-3. We describe two cases with PNH and BCS in whom CVT was angiographically documented. We comment upon the risks of anti-coagulants in this unusual situation and the need to recognize PNH among the possible causes of CVT.

A 34 year old woman was admitted to hospital because of malaise, low-grade fever, pleural effusion, hepatomegaly and ascites. The diagnosis of PNH was established by the presence of haemosiderinuria, positive Ham acid haemolysis and sucrose lysis tests. Hepatic venogram confirmed the existence of BCS. She received repeated blood transfusions and a year later a portacava shunt was performed. Three years later she complained of intense fronto-occipital pain and clumsiness of the left limbs. Examination showed a mild distal paresis of the leg with only slight sensory impairment. There was minimal slowing of alternating movements in the left arm. There was impairment of position and pain perception with sensory extinction on the left side. The optic discs were slightly blurred. The visual fields were full with a moderately enlarged blind spot on both sides. CT scanning showed a right parietal infarction. Cerebral angiography revealed thrombosis of superior sagittal sinus, straight sinus, lateral sinuses, and internal cerebral veins. The patient made a complete recovery with dexamethasone and coumarin therapy.

In a comprehensive review of 38 cases, Bousset et al. failed to list PNH among the possible causes of CVT. PNH is commonly undiagnosed for a period of months and perhaps its true incidence among patients with CVT is probably underestimated because detailed coagulation studies are not performed in most cases. This may be of the utmost importance when treating patients who have had a CVT and a recent death from high-dose heparin in patients with PNH. Consensus, however, with heparin has caused exacerbation of haemolysis in some patients with PNH, 1,2 and thus might be deleterious in this particularly difficult situation.

PNH should be considered among other possible causes of CVT and ruled out by the appropriate laboratory investigations, especially when treatment with heparin is contemplated.

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Toxic reaction following the combined administration of fluoxetine and phenytoin: two case reports

Fluoxetine is a new antidepressant agent unrelated to the tricyclic antidepressants, whose structure corresponds to a straight chain phenylpropylamide. The drug selectively inhibits reuptake of serotonin but not noradrenaline and has a minimal muscarinic, dopaminergic, histaminergic or serotonergic effect. The only described interaction is with L-tryptophan which enhances its therapeutic effects, but produces symptoms and signs of intoxication. Presently there is no published work on the possible interaction between fluoxetine and anticonvulsant drugs. We describe two patients, who developed symptoms and signs of intoxication with phenytoin a few days after initiating the use of fluoxetine.

An 84 year old woman was treated with phenytoin 300 mg daily, after removal of a chronic subdural haematoma. Two months later the patient developed a depressive syndrome. CT showed no alteration, and the plasma level of phenytoin was 15 µg/ml. Treatment with a dose of 20 mg/day of fluoxetine was given, increasing the dose to 40 mg/day after 10 days. Five days after the beginning of treatment she developed gait ataxia, vertigo, diplopia and alteration of consciousness. Examination also showed dysmetria of the limbs, multidirectional nystagmus, and alteration of judgement with visual hallucinations.

The plasma level of phenytoin was 35 µg/ml. The dose of phenytoin was reduced gradually and there was progressive recovery from the signs and symptoms.

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symptoms, becoming completely normal after 4 weeks. Phenytoin 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 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, tolbutamid and warfarin) and drugs were titrated. 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.

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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. 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 inhibitor of GABAtransaminase, gamma vinyl GABA (GVG) in the treatment of disabling levodopa-induced chorea in 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, 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 3 Gm 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 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 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.

Contradicting results with progabide, a gabamimergic agonist, have been reported in levodopainduced dyskinesias in Parkinson's disease. 3 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.

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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. This condition commonly results from injury to the brachial plexus at birth. 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 of 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 partial 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. 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 acquired Horner's syndrome, is generally thought to be that of failure of pigment development rather than loss of pigment that has already formed. Hypochromia of 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 41 year old woman presented with point tenderness in the right upper chest and lateral supraclavicular 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