third and nineteenth day from the beginning of the 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 anticryptococcal antigen 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 only a generalised areflexia and a slight dysmetria on the heel-knee test.

Electrophysiological studies (table) were carried out on the sixth 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 block in the remaining nerves was noted. 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.

Until recently the Guillain-Barré syndrome was an entity whose nosological characterisation relied upon a purely descriptive base, with relatively widely accepted diagnostic criteria,[1] Among these, the presence of muscular weakness is the most noticeable, indicating the main affectation 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:[2] rapid onset, distribution widespread and symmetrical, complete or near recovery, high CSF protein content, with few or no cells, and an electrophysiological study compatible with a demyelinative 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;[3] 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.

A case of acute polyneuropathy has been published in which the sensory disorders were also prominent and very similar to those of our patient.[4] 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. These findings, except for the preservation of the motor roots, are identical to those which are described in the Guillain-Barré syndrome.[5]

Our patient had an acute sensory neuropathy after a presumed viral illness and vaccination with a monophasic course and nearly complete recovery—both clinical and electrophysiological—in a short time; 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 haematopoietic system manifested by chronic haemolytic anaemia, leukopenia, 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 a patient with PNH and BCS in whom CVT was angiographically documented. We comment upon the risks of anticoagulants in this unusual situation and the need to remember 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 portocava shunt was performed. Three years later she complained of intensive fronto-occipital pain and clumsiness of the left limbs. Examination showed a mild distal paresis of the left leg with only minimal slowness 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, Bousser *et al*[4] failed to list PNH among the possible causes of CVT. PNH is commonly undiagnosed for a period of months to years, and 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 treatment with heparin is under consideration. The recent German randomised trial demonstrated the benefit of high-dose heparin in patients with CVT.[5] Conversely, therapy with heparin has caused exacerbation of the hemolytic process 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 serotinergic effect.[4] The only described interaction[1 3] is with L-tryptophan which enhances its therapeutic effects, but produces symptoms and signs of intoxication.[5] 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 she 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 and fluoxetine was reduced gradually and there was progressive recovery from the signs and