Syphils were negative in the blood and the cerebrospinal fluid (CSF). He was HIV negative. CSF protein was 0.25 g/l and there were no cells or oligoclonal bands. Serum creatine phosphokinase was raised at 199 IU/L. There was no evidence of motor sensory conduction velocities, fibrillation and fasciculation potentials and positive sharp waves. Voluntary units were generally small in amplitude and there was gross excess of lower motor neurone lesions. There was no evidence of upper or lower motor neurone weakness.

The patient was HLA Bw 35 antigen positive. The incidence of this HLA antigen was found to be significantly increased in patients with Guamanian type ALS.

There was no pneumococcal, meningococcal or a recent tuberculous meningitis and a road traffic accident in our patient 17 and 13 years before clinical presentation with ALS. Although a history of mechanical trauma or surgical operations was found to be two thirds of idiopathic ALS patients with MND than in matched controls,1 it is unlikely that trauma had played an important role in this patient because of the long latent period between his injuries and the onset of ALS.

ALS in the patient was preceded by a bizarre intermittent illness which at the beginning was thought to be psychiatric in origin. However, the patient did not have a past or family history of psychic disease nor did he have a personality disorder. Furthermore, these symptoms did not respond to adequate psychiatric measures including treatment with anxiolytics, breathing into a paper bag and psychotherapy. The patient's symptoms were therefore almost certainly organic in nature and are probably a hyper-sensitivity reaction to penicillin. In fact, intermittent symptoms lasting weeks or months which might account for those in our patient occasionally result from penicillin hypersensitivity2 and they are known as pseudoa allergic reactions or Hoigne's syndrome. Our patient almost certainly had Hoigne's syndrome which was followed by ALS two to three months later.

The association between MND and ana phylaxis has not been reported before. However, we are aware of another patient who had developed ALS shortly following a severe anaphylactic shock (J Lewis, personal communication). Other neurological disorders are sometimes precipitated by hypersensitivity reactions. We have observed two cases of extrapyramidal disease following insect stings.3 Demyelinating diseases and peripheral neuritis4 have also been reported following anaphylactic reactions. Like trauma, these may damage lower motor neurone ALS or alternatively, accelerate the progression of latent disease. A recent study has demonstrated that non-specific injury outside the CNS, for example, transection of a peripheral nerve, induces strong expression of MHC class 1 and to a lesser extent class 2 antigens in CNS neurons.5 One can postulate a similar mechanism for the role of anaphylaxis in ALS.

Fatigue and melatonin in Parkinson's disease

Fatigue is a major, although often neglected symptom of Parkinsonism. It is influenced to some extent by circadian factors, including sleep benefit, diurnal changes in levodopa metabolism, dopamine receptor sensitivity, and monoamine oxidase activity.6

In normal subjects, one of many factors that influences fatigue may be changes in melatonin rhythm.7 The use in Parkinson's disease of the decarboxylase inhibitor benzerazide, which reduces 5-hydroxytryptamine and melatonin concentration in the rodent pineal8 may therefore indirectly affect fatigue mechanisms. We have investigated the role of urinary 6-hydroxy-dopamine and levodopa in Parkinson's disease.

Eighteen subjects with idiopathic Parkinson's disease were studied, 11 males, seven females, aged 42-81 (mean 64 years) with a mean duration of symptoms of nine years (range 2-20). None had on/off fluctuations. Twelve had a stable response across the day, six progressive fatigue. Six subjects were studied untreated and one week following benzamidase 50 mg orally four times daily, the remaining taking levodopa 100 mg four times daily and levodopa 1700 mg daily plus benzamidase 380 mg daily (n = 7).

Samples for plasma melatonin assay were obtained by indwelling venous catheters at three hourly intervals over a 24 hour period. Four, six hourly consecutive urinary collections were carried out during the same 24 hour period. Plasma melatonin concentration and urinary 6-hydroxy-5-methoxyindole sulphate was assayed by radioimmunooassay as described by Fraser et al.9 and Arendt et al.10 respectively.

Peak plasma melatonin concentrations in six untreated subjects with Parkinson's disease showed wide individual variation

Admitted with blurred vision
sis
CT
and
unremarkable,
conventional doses used in the treatment of Parkinson's disease (p < 0.1). Parkinsonian disability scores (King's College rating scale), presence or absence of diurnal fatigue, peak plasma melatonin and urinary aMT6s concentrations showed no significant correlation.

The pinal gland from three patients (91 and 70 year old females, 78 year old male) with Lewy body associated Parkinson’s disease, treated with levodopa-decarboxylase inhibitor combinations, were examined microscopically. Pinal histopathology was unremarkable, with cells containing brown sérécary granules; there were no intracytoplasmic Lewy bodies.

Plasma melatonin and urinary 6-hydroxy melatonin sulphate concentrations in untreated and levodopa-decarboxylase inhibitor treated subjects with Parkinson’s disease were similar within the normal range as determined in normal healthy young subjects, and it is worth noting that the pinal structure is normal in Parkinsonian patients. Therefore benzerazide inhibits melatonin synthesis in the rodent but not in humans with the conventional doses used in the treatment of Parkinson's disease.

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Cerebral CT lesions in multiple sclerosis mimicking multiple metastases
A 38 year old right handed woman was admitted with blurred vision of the right eye and incoordination of the right limbs. Eighteen months previously she had developed paraesthesia and numbness of the right face and arm, with incomplete recovery and four months previously blurring of vision of the left eye with complete recovery.

Abnormal findings were an ataxic gait, VAR 6/18, bilateral optic disc pallor and ataxia of the right limbs with an extensor right plantar response. Visual evoked responses showed considerably prolonged latencies from each eye. Cranial CT showed a small left frontal enhancing lesion. She was treated with high dose intravenous methylprednisolone with marked improvement.

She was admitted ten months later with a five month history of increasing ataxia, right hemiparesis and expressive speech difficulty. Examination showed expressive dysphasia and dysarthria. There was moderate impairment of cognitive function. Visual acuities were 6/60. There was a bilateral internuclear ophtalmoplegia and right facial weakness. The right hemiparesis was severe and joint position sense was absent in the right foot. Cranial CT showed multiple ring enhancing lesions (fig). The lumbar cerebrospinal fluid (CSF) pressure was 33 cms of water and the CSF protein was elevated at 64 mg/100 ml with equivocal results on immunofluorescence. CSF contained 5 WBC per mm³, mostly lymphocytes. An HIV antibody test was negative.

There was rapid deterioration with drowsiness, dysphagia and weakness of the left arm. Because of doubt about the radiological diagnosis, and the rarity of dysphasia in multiple sclerosis, a CT guided biopsy of the left frontal lobe was carried out. The histology showed demyelination and no evidence of malignancy. She was treated with high dose intravenous methylprednisolone for five days and cyclosporin 10 mg/kg for five months with marked improvement. Six months later speech and limb power were normal and she was able to stand and walk a few steps with assistance. The CT appearances improved.

Enhancing lesions with mass effect have previously been reported in multiple sclerosis.1 2 We are aware of only two reports of multiple lesions of this type3 4 and the florid radiological appearance in our case seems exceptional. The radiological differential diagnoses include abscesses, tumours and vascular lesions. The short term clinical and radiological improvement with corticosteroids is nonspecific and there may continue to be a need to recourse to biopsy in cases of this type.

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Atypical meningitis complicating a penetrating head injury

The bacterial genus bacillus contains predominantly saprophytic organisms with low pathogenicity for humans with the exception of the anthrax bacillus. These organisms may rarely be responsible for serious local and disseminated infections with a significant mortality. To our knowledge there have been only 11 documented cases in which bacillus subtilis has been implicated in such infections. We present a previously unrecorded complication of a penetrating head injury in which a patient survived a self-inflicted crossbow bolt injury but developed meningitis secondary to the introduction of bacillus subtilis spores from the bolt itself.

A 24 year old man with a long history of depressive illness and drug abuse was admitted having been found in his bathroom with a crossbow by his side and a crossbow bolt protruding from his left temporal region. On examination he was conscious and orientated with no focal neurological signs. The right feathers of the bolt were visible in the left temporal region. Skull radiographs showed the metal tip of the bolt lying in the midline (fig 1a).

The bolt was removed by a left temporal craniectomy with a perioperative dose of a broad spectrum antibiotic. The entry point was anterior to the left middle meningeal artery and the tract was fully cleared and irrigated. The immediate post-operative period was uneventful. On the fifth post-operative day the patient developed a pyrexia but with no evidence of meningitis. Lumber puncture showed no evidence of organisms, a polymorph count of 180/ml, a lymphocyte count of 10/ml, a red cell count of 5000/ml, a protein level of 4700 mg/l with a marked globulins increase, and a cerebrospinal fluid (CSF) glucose of 3.8 mmol/l. Computerised tomography (CT) scan showed the bolt tract extending to the frontal horn of the left lateral ventricle.
Fatigue and melatonin in Parkinson's disease.


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