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Fatigue and melanin in Parkinson's
disease
Fatigue is a major, although often neglected symptom of Parkinsonism. This is influenced
to some extent by circadian factors, including sleep benefit, diurnal changes in levodopa
metabolism, dopamine receptor sensitivity, and monoamine oxidase activity.1

In normal subjects, one of many factors that influences fatigue may be changes in
melatonin rhythmicity.2 The use in Parkinson's disease of the decarboxylase inhibitor
benzamidazol, which reduces 5-hydroxytryptamine and melatonin concentration in the
rodent pineal1 may therefore indirectly affect fatigue mechanisms. We have investigated
Parkinsonian disability, plasma melatonin levels and urinary 6-hydroxy-melatonin sulphate
(αMT6s) as an index of the evening melatonin rise in untreated subjects and fol-
lowing levodopa and benserazide treatment.

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 2-30). 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
benzamidazol 50 mg orally four times daily, the remaining taking levodopa 200 mg every 4 h
and levodopa 1700 mg daily plus benzamidazol 380 mg daily (n = 7).

Samples for plasma melatonin assay were obtained by indwelling venous catheters at
three hourly intervals overnight and on the following day. Four, six hourly consecutive urinary
collections were carried out during the same 24 hour period. Plasma melatonin concentration
in untreated subjects was assayed by radioimmunoassay as de-
cribed by Fraser et al3 and Arendt et al4
respectively.

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

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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 re-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 opthalmoplegia 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.1). 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 10mg/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.1-3 We are aware of only two reports of multiple lesions of this type2,4 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 explored 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 meningism. 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 globulin 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