(range 29–65 pg/ml). Benserazide 200 mg orally did not reduce mean peak plasma melatonin levels or alter urinary aMT6s excretion. Peak levels and time of peak level were similar to values in normal, healthy young laboratory staff. Mean peak plasma melatonin levels and mean urinary aMT6s excretion were slightly lower in levodopa-benserazide treated than in untreated patients with 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 pineal 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. Pineal histopathology was unremarkable, with cells containing brown secretory 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 are within the normal range as determined in normal healthy young subjects, and it is worth noting that the pineal structure is normal in Parkinsonian patients. Therefore benserazide 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. Eight-

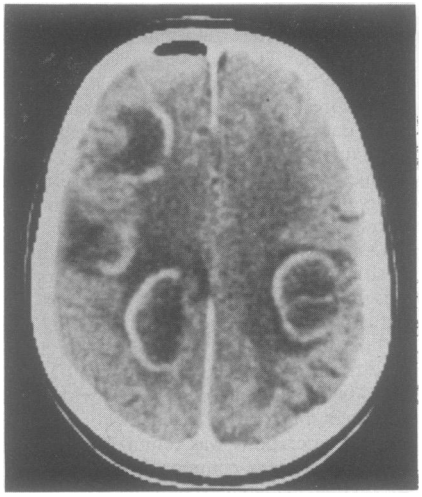

Figure Contrast enhanced CT scan showing multiple ring-like enhancing lesions.

een 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 ophthalmoplegia 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 immunofixation. CSF contained 5 WBC per mm^3, 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.[1-3] We are aware of only two reports of multiple lesions of this type[2,4] 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 a 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 and the flight 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. Lumbar 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

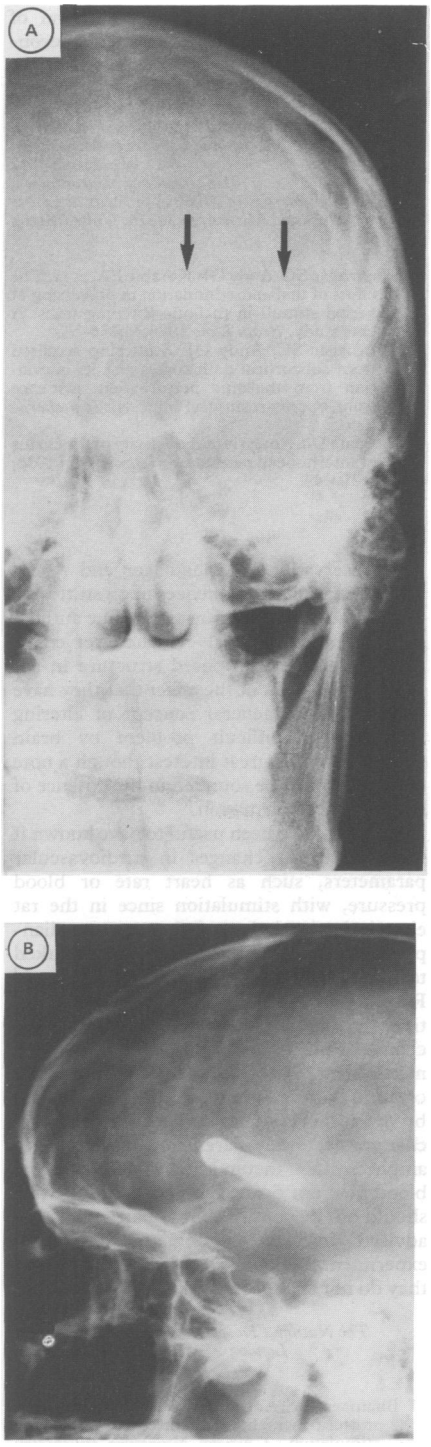

Figure 1 a) Anteroposterior (AP) and b) lateral skull radiographs showing crossbow bolt at presentation.

ventricle (fig 1b), with evidence of intraventricular haemorrhage but no evidence of a cerebral abscess. Despite initial treatment with flucloxacillin and sulphadimidine the pyrexia continued and signs of meningism developed. Penicillin and chloramphenicol were added, after which he showed a progressive recovery over a five day period.

Lumbar punctures performed during this period showed an increasing polymorph count to a maximum of 1400/ml and continued to show a haemolysed supernatent with no visible organism on microscopy. Culture of the initial lumbar puncture CSF grew *bacillus subtilis* after one week but was considered to be of doubtful significance as a primary pathogen. It was, however, subsequently cultured from two further specimens of CSF. It was sensitive to chloramphenicol and penicillin but resistant to sulphadimidine. The organism also possessed a haemolysin which would account for the persistently haemolysed supernatent. Treatment with chloramphenicol was continued for three weeks and penicillin continued for six weeks, at which time lumbar puncture was normal. The patient made a complete recovery with no neurological deficit and was discharged for psychiatric treatment.

The "non-pathogenic" members of the genus bacillus are spore-bearing, aerobic and usually gram positive saprophytic organisms which are ubiquitous in distribution.[1] They have been described as a rare cause of both local and disseminated infections.[2] Some species are used as test organisms for assessing the efficiency of ethylene oxide and ionising radiation sterilisation techniques. In particular *B stearothermophilus* spores which are heat resistant to 121°C, are used to test autoclave function.[1] They are commonly isolated from clinical material and classified as "contaminants". As a result of a complex taxonomic relationship such non-pathogenic isolates are reported as *bacillus subtilis*, and a specific identification is not usually performed.[2] The structure most commonly involved is the eye, although epidemics of food poisoning have been ascribed to it.[3]

The most serious orbital infection, a fulminating panophthalmitis, appears to follow penetrating trauma with direct innoculation of the organism into the vitreous humour.[4] Disseminated infection as a result of primary infection with *bacillus subtilis* is rare, but presents as meningoencephalitis, meningitis, endocarditis and septicaemia in the twelve recorded cases.[2] Nine of these had involvement of the central nervous system which in four cases followed a spinal anaesthetic procedure. It would appear that the organism is introduced during such procedures directly into the subarachnoid space, where the defence mechanisms of the body are minimal. Interestingly, the organism does not appear to cause opportunistic infections in debilitated patients and therefore supports the theory that direct innoculation into a poorly defended area allows a weakly virulent organism to become pathogenic.[2]

In our case the organism appears to have gained entry from the wooden bolt by virtue of the penetrating nature of the injury. Although of low virulence, disseminated infections appear to have a significant mortality of 50% in the 12 cases previously recorded,[2] and our patient was therefore fortunate to have shown a complete recovery.

Although a single dose of a broad spectrum antibiotic was given perioperatively this was obviously not sufficient to prevent infection and in penetrating injuries of this type it is necessary to complete a full course of high dose prophylactic broad spectrum antibiotics to minimise the risk of colonisation. Of further note in this case is the risk of major arterial damage. The crossbow bolt penetrated the brain anterior to the middle cerebral artery and crossed the midline structures. Although not performed in this patient, cerebral arteriography is advisable in any patient before removing a foreign body which has penetrated the skull.

This case illustrates the need to consider *bacillus subtilis* as a primary and potentially serious pathogen following penetrating head injuries and this organism should therefore not be dismissed merely as a laboratory contaminant.

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MATTERS ARISING

Alleviation of acquired stuttering with human centremedian thalamic stimulation

I would like to comment on the article by Bhatnagar and Andy[1] which claimed that thalamic stimulation was effective in the treatment of an acquired stuttering. Their findings could have very substantial theoretical and clinical implications, but they rely on seemingly insubstantial and questionable data.

The authors report on a 61 year old male with a 20 year history of trigeminal pain, beginning with a tic douloureux, who ultimately developed a severe stuttering. The stuttering had a presumed subcortical origin and progressively deteriorated. The patient's self-administered thalamic stimulation, via an implanted electrode, was claimed to have had "a remarkable beneficial effect on the speech dysfluencies." The basis of this claim was a comparison between stutterings counted during preoperative conversation and oral reading tasks, and stutterings counted during a 10 minute postoperative conversation. This information was supplemented by self ratings of "dysfluencies" before and after the implant. It was also claimed that the subject had been "free of his