that there is a considerable shortfall in specialist neurological services in the United Kingdom.⁵

The question arises as to whether or not the urgent/routine classification system is satisfactory. It is surprising that over 50% of new referrals were given a priority classification. This high figure may reflect the known long waiting time for "routine" patients, rather than a truly perceived seriousness of the medical condition. It is worth noting that some patients with serious disease were put on the non-urgent list, thus indicating that in some cases at least the initial priority category was inappropriate. More informative referral letters might assist consultants to classify patients appropriately.

In conclusion, this study highlights particularly: 1) the predominance of the diagnostic role of the neurology outpatient consultation; 2) the small proportion of patients referred with serious disease; 3) the unacceptably long waiting time, and 4) the inappropriate priority classification of some patients.

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We would be pleased to consider for publication short letters describing similar audit of outpatient practice in other countries.

Ed

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HTLV-1 infection: the clinical spectrum widens

A neurological condition causing spastic paraparesis has long been recognised in the West Indies but it is only in recent years that the association between tropical spastic paraparesis (TSP) and human T-cell lymphotropic virus type 1 (HTLV-1) has been

confirmed.¹⁻³ Serological tests for HTLV-1 can now support a diagnosis of TSP in patients with atypical clinical features.

Patient 1 was born in Jamaica and came to the United Kingdom at the age of 38 years. At the age of 61 years she presented with pain in her left shoulder, a three year history of difficulty raising her arms above her head, fatigue and inability to walk long distances.

On examination there was winging of both scapulae with weakness in the deltoids, triceps and biceps, without fasciculations. Distal upper limb musculature was normal. The biceps and supinator jerks were absent and the triceps jerks diminished. There was a mild spastic paraparesis with increased knee jerks, diminished ankle jerks and extensor plantar responses. Sensory testing was normal. The creatine kinase was mildly elevated and muscle histology revealed neurogenic changes. Myelography and CSF examination were normal. There was a polyclonal increase in the serum immunoglobulins.

At first a diagnosis of motor neuron disease was considered but there was no change in her condition during the following four years and reinvestigation revealed serum antibodies against HTLV-1 in a titre of 1 in 6400. In TSP, pain often accompanies or precedes weakness but is characteristically confined to the lower limbs and lumbar spine. This patient is also unusual in the severity of the muscle wasting which in TSP is seldom prominent and usually confined to the intrinsic hand muscles.⁴

Patient 2 was born in Jamaica and came to the United Kingdom aged 42 years. He was first seen aged 63 years following a single generalised convulsion; his right plantar response was extensor but there were no other neurological signs. CT brain scan and EEG were normal. Treponemal serology was positive (VDRL positive 1 in 4; TPHA positive; FTA IgG positive, IgM negative) without a history of previous venereal disease or yaws. Lumbar CSF was acellular with normal protein concentration and the tests for syphilis were all negative; Link's IgG index was 0.78 (upper limit of normal 0.58) suggesting intrathecal immunoglobulin synthesis. He was treated with a course of intramuscular procaine penicillin and regular oral phenytoin.

At the age of 70 years he was seen again for reassessment of his epilepsy. Only on direct questioning did he admit that his legs had become weak. He had difficulty rising from a low chair but otherwise his gait was normal. There was a mild spastic paraparesis with brisk lower limb reflexes and the right plantar response was extensor as before. A CT brain scan was again normal. MRI scan showed several small punctate white matter lesions in both cerebral hemispheres but no abnormality of the spinal cord. Antibodies to HTLV-1 were detected in the serum in a titre of 1 in 8000.

Patient 3 was born in British Guyana and came to the United Kingdom at the age of 34 years. At the age of 59 years she developed bilateral uveitis and was found to have positive treponemal serology (VDRL negative; TPHA positive; FTA IgG positive, IgM negative) without a past history of venereal infection or yaws. CSF contained 32 lymphocytes/mm³ and the protein concentration was raised (0.85 g/l); CSF tests for VDRL, TPHA and FTA were negative. Her vision responded to topical steroids and she took a one month course of oral doxycycline. Six months later she developed weakness and sensory loss in the legs which progressed over

a year. At that time she had a spastic paraparesis with a sensory level at T10 and normal position sense. The upper limbs and cranial nerves were normal. Myelography was normal but was followed by urinary retention requiring catheterisation. The CSF on this occasion contained no cells but the protein concentration remained raised (1.0 g/l). A diagnosis of neurosarcoidosis was considered but the patient showed no response to oral corticosteroid therapy; subsequent bronchoscopy, bronchial biopsy and Kveim test were found to be normal.

At follow up two years later her gait had deteriorated, she had developed mild bilateral nerve deafness and there was muscle wasting in both hands. Antibodies against HTLV-1 were detected in the serum in a titre of 1 in 64000. The blood film was normal but there was a polyclonal increase in the serum immunoglobulins.

The initial detection of HTLV-1 antibodies was by gel particle agglutination assay and confirmed by more specific methods (ELISA, indirect immunoelectrophoresis, IgG antibody capture radio-immunoassay and Western blot techniques).⁵

These patients illustrate many of the recognised features of TSP but despite this, the illness in each of them had been tentatively attributed to another cause: motor neuron disease (patient 1), parasagittal tumour or neurosyphilis (patient 2), neurosarcoidosis or neurosyphilis (patient 3). The initial neurological presentations were pectoral pain and amyotrophy (patient 1), late-onset epilepsy (patient 2) and uveitis (patient 3). Tests for HTLV-1 antibodies clarified the diagnosis on these patients and should be performed in all West Indian patients with spastic paraparesis and with other unexplained neurological syndromes. If effective treatments for HTLV-1 infection become available, early diagnosis will be necessary to identify patients before severe, irreversible neurological damage has occurred. This will require greater awareness of the diverse ways in which TSP may present. The full spectrum of HTLV-1 infection remains to be defined.

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Temporal lobe phenomena during the aura phase of migraine attacks

I report a patient who often experienced temporal lobe phenomena during the aura phase of his migraine attacks.

A 27 year old right handed computer

operator gave an eight year history of attacks of unilateral headache with minimal nausea, lasting for up to two days. These occurred initially at intervals of two months but more recently every three days. At first the attacks were preceded by a typical 25 minute visual aura, in which he experienced a coloured rotating diamond and zig zag lines, usually in the right visual field, but since the age of 25 an increasing proportion of them have been preceded by a momentary feeling that his actual behaviour is unduly familiar, followed by a 20 minute sequence of unpleasant, almost morbid sensations that he felt he had come across before, as if in previous dreams, "in another world". This would be followed by some impairment of memory lasting for three days, and a rather milder, though sometimes more generalised headache with nausea that would last for one day. In addition some attacks were preceded by 15 seconds of quite intense giddiness and there have been rarer episodes of dysphasia. There have never been any lapses of concentration or impairment of consciousness, and he said he was able to carry on speaking and working while the aura was in progress. There is no family history of migraine. He derived some benefit from pizotifen which seemed to shorten his aura, but was not helped much by methysergide.

There were no physical abnormalities on examination. A CT scan was normal and two EEGs showed a generalised excess of slow wave activity.

Visual, sensory and motor symptoms, usually in that order of frequency, are the commonest seen in the aura phase of classical migraine.¹² Occasional patients become dysphasic and some symptoms (for example, ataxia, dysarthria and vertigo) have been attributed to disturbances in the vertebral-basilar circulation.³⁴ In one series of patients with transient global amnesia, 42% gave a past history of migraine, a figure considered significantly greater than the prevalence in the general population.⁵ It seems likely that some such cases, which are only rarely recurrent, are indeed manifestations of migraine while others are ischaemic in origin.⁶⁷ Raskin⁸ describes a young woman who experienced 15 episodes of transient global amnesia, each lasting up to six hours followed by headache, each after drinking a glass of red wine. While olfactory and auditory hallucinations have been described in the aura phase of classical migraine,⁹⁻¹¹ recurrent transient memory disturbances of the type experienced by this patient, which are reminiscent of déjà vu phenomena, do not appear to have been reported in detail, though Saul¹² and Sacks¹³ allude to similar cases.

There is much, admittedly circumstantial, evidence that the cortical disturbances of classical migraine are due to spreading depression moving across the cortical surface.¹⁴¹⁵ Olesen and Jorgensen¹⁶ have speculated that transient global amnesia is due to a wave of spreading depression moving across the hippocampal surface. This patient's déjà vu phenomena, which have a frequency and duration typical of classical migraine, are probably mediated similarly, and it is speculated that this is due to spreading depression in the temporal lobe. Migraine therefore should be considered among the causes of déjà vu phenomenon, particularly if prolonged.

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Subcutaneous apomorphine for Parkinsonian patients with psychiatric side effects on oral treatment

Subcutaneous apomorphine has recently been shown to reduce off periods in patients with Parkinson's disease (PD) who are resistant to conventional management.¹² A previous study indicated that neuropsychiatric symptoms in patients on oral treatment did not recur when apomorphine was used, but the effects of apomorphine in those for whom these complications were the major dose limiting factor has not been investigated.¹ We describe three patients with PD whose optimal control was compromised by severe neuropsychiatric side effects and who responded to treatment with apomorphine.

The first case was a 46 year old woman who had had PD for eight years and was underdosing herself with oral levodopa because of side effects. Bromocriptine had produced psychosis, and levodopa/benserazide (madopar 62.5 mgs) in excess of seven times daily caused hallucinations, paranoid delusions and aggression predominantly in the on periods, which led to two hospital admissions.

Subcutaneous apomorphine infusion was started at 3 mg/hour for 12 hours daily and dramatically reduced her off periods from eight hours daily to one hour. Tremor was abolished and levodopa dosage could be reduced from 350 mg/day to 200 mg/day. She has now been maintained on this treatment for eight months without psychiatric side effects.

The second case was a 67 year old nun who had had PD for 10 years. Eight Madopar 62.5 mg tablets caused severe dyskinesias and visual hallucinations mainly in the on periods. Bromocriptine 2.5 mg four times daily caused visual hallucinations and confusion and was discontinued. She could tolerate seven madopar 62.5 mg tablets and had nine hours off periods daily. Apomorphine was given initially by a "Penject" system and subsequently by subcutaneous infusion at a dose of 40 mg/day. Off periods fell to one hour daily and levodopa could be reduced from 400 mg daily to 300 mg daily. She has now been maintained on apomorphine infusion for 14 months with mild involuntary movements but no psychiatric side effects.

Case 3 was a 74 year old man with severe Parkinsonian tremor and bradykinesia who could not tolerate optimal doses of levodopa, bromocriptine or benhexol. Madopar in excess of 62.5 mg four times daily caused nausea and psychosis, both during on and off phases. Bromocriptine 2.5 mg, three times daily caused visual hallucinations with confusion and the same problems occurred with benhexol 2 mg, twice daily. He tolerated subcutaneous apomorphine infusion at 3 mg/hr for 12 hours daily and his tremor grading (modified King's College scoring system) improved from 3 to 1 (3 = severe, 1 = mild) along with improvement in his bradykinesia. Single blind infusion of normal saline in place of apomorphine resulted in a return of the severe Parkinsonism. His levodopa dosage could be reduced from 200 mg to 150 mg daily and he has been maintained on apomorphine for 14 months without any neuropsychiatric problems except for occasional nocturnal confusion.

Over the past two years we have treated 12 non-demented PD patients with refractory on-off oscillations, using subcutaneous apomorphine. Nine have continued treatment resulting in reduction of their mean daily off periods and levodopa dosage by 85% and 39% respectively. Five of the nine patients had some form of neuropsychiatric complications on oral treatment, before apomorphine was started. Only one patient developed hallucinations for the first time with apomorphine. Thus we have shown that apomorphine will improve the quality of life of PD patients who develop severe neuropsychiatric side effects on optimal doses of oral treatment. Resistant off periods can be reduced without the development of these problems. Lisuride has also been used as parenteral treatment in PD, but neuropsychiatric complications were the major limiting factor in its use.³

It is unclear why apomorphine should be better tolerated than oral therapies. It contains a piperidine moiety which may itself be antipsychotic and it lacks the serotonergic effects of bromocriptine or the ergolides.¹³ Furthermore, its potent effect on both D₁ and D₂ receptors may differentiate it from other dopaminergic agents. For instance, unlike levodopa and amphetamine, small doses of apomorphine in schizophrenics do not exacerbate the psychosis.⁴ Likewise, although it induces stereotyped behaviour in laboratory animals, an emetic dose of apomorphine does not lead to behavioural changes in humans.⁵

To date our experience suggests that PD patients with neuropsychiatric problems with conventional treatment benefit from treatment with apomorphine. Further experience with large number of patients may confirm it