that there is a considerable shortfall in specialist neurological services in the United Kingdom. The question arises as to whether or not the urgent need for a specialist service in the factory 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. The 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

HTLV-I infection: the clinical spectrum widens

A neurological condition causing spastic paraparesis has long been recognised in the West Indies but it has been more recent years that the association between tropical spastic paraparesis (TSP) and human T-cell lymphotropic virus type 1 (HTLV-I) has been confirmed.1,2 Serological tests for HTLV-I 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. Examination revealed weakness of both scapulae with weakness in the deltoids, triceps and biceps, without fasciculations. Distal upper limb muscle wasting was normal. The biceps and supinator jerks were absent and there was an up-going plantar response. 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 raised 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 re-investigation revealed serum antibodies against HTLV-I in a titre of 1 in 6400. In TSP, pain is often a feature of the disease,3 although this pain is frequently due to muscle wasting. In this patient, the biceps and supinator jerks were absent and there was an up-going plantar response. 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 raised and muscle histology revealed neurogenic changes. Myelography and CSF examination were normal. There was a polyclonal increase in the serum immunoglobulins.


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 weeks, 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 same direction, but sometimes reversing, and 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 dizziness and there have been other episodes of dizziness. 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 focal neurological sign of migraine. He derived some benefit from pizotifen which seemed to shorten his aura, but was not helpful much by methysergide.

There were no physical abnormalities on exploration. 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.1 Of occasional patients become drowsy and some symptoms (for example, ataxia, dysarthria and vertigo) have been attributed to disturbances in the vertebro-basilar circulation.1 In one series of patients with transient global amnesia, 42% gave a past history of migraine, a figure considered significantly greater than the prevalence of migraine in the general population.1 It seems likely that some such cases, which are only rarely recurrent, are indeed manifestations of migraine while others are ischaemic in origin.1 Raskin 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,1,1 recurrent transient memory disturbances of the type experienced by this patient, which are reminiscent of deja vu phenomena, do not appear to have been reported in detail, though Saul19 and Sacks13 allude to similar cases.

There is, much, admittedly circumstantial, evidence that the cortical disturbances of classical migraine, due to spreading depression moving across the cortical surface,1,15 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 deja 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 deja vu phenomena, particularly if prolonged.

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