is difficult to document until loss of brain bulk can be documented on delayed CT or MRI scans.

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The paper by Leinninger, et al. reported that the pursuit of litigation had no effect on neuropsychological status after cerebral concussion, and that the cognitive deficits were attributable to the head injury and not to litigation. These conclusions, drawn from a clinical series of symptomatic patients, warrant scrutiny.

The authors provide no information as to how they classified their patients into groups "pursuing litigation" and "not pursuing litigation". Merely asking patients if they have filed a lawsuit does not provide the information necessary to classify their claim status. Some of the patients may have been injured in work-related accidents, claims for damages may have been made against insurers without litigation. Furthermore, the patients studied could have filed lawsuits after their neuropsychological evaluations; all of them were seen within 22 months of their injury. These results are not contrary to our own.

In a recent paper Binder and Willis reported a very strong relationship between the pursuit of a claim and performance on a measure specifically designed to assess motivation to remember, the Portland Digit Recognition Test. Our study compared minor head trauma patients to patients with well-documented cerebral dysfunction who were not seeking financial compensation. Our minor head trauma patients were not chronic, seen an average of two years after their trauma, than the patients studied by Leinninger et al, a factor which may have affected the results.

Leinninger et al. equated the cognitive deficits of the concussed patients with cerebral dysfunction. The possible existence of co-morbidities in the minor head injured patients make this relationship tenuous. However, the majority of the minor head injury patients were injured in motor vehicle accidents. Consequently, they may have had orthopedic injuries and been treated with analgesic medications. Some of them may have developed anxiety disorders or depression as a result of their accidents and may have been treated with psychotropic medication. However, the authors provided no information on chronic pain, psychiatric status, or medication use. These variables are also associated with cognitive abilities and may have accounted for the differences between the concussed and control subjects. Controlled studies of consecutive acutely injured patients followed prospectively have shown normalisation of cognition within a few weeks of minor head trauma, using measures no less sensitive than those employed in the study of symptomatic patients by Leinninger et al.  

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Low plasma iron status and akathisia

Barton et al. reported a significant inverse correlation between plasma iron levels and akathisia in a small group of patients. However, three of their akathisic group had low plasma iron levels (about 50 μg/100 ml). Since the association between restless legs syndrome and low plasma iron is generally accepted, their three patients might not have akathisia but the restless legs syndrome.

Although akathisia and restless legs syndrome are clinically similar (floor pacing, marching on the spot, and body rocking in both conditions), the symptoms of akathisia are prominent throughout the waking hours. Conversely, the symptoms of restless legs are more prominent at night.1 It suggests that the circadian rise of possible some hormone could be related to the symptoms of restless legs syndrome, but not to those of akathisia. Sandyk et al. pointed out that one of the possibilities may be melanocyte stimulating hormone (MSH).

Further research is required to differentiate between akathisia and restless legs syndrome, and measuring MSH may be helpful.

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Bowie and Ebenezer reply:

We are pleased to answer Terao's and Yoshimura's comments relating to the differential diagnosis of our akathisic patients, particularly those with mild to moderate insomnia and low plasma iron levels about 50 μg/100 ml. Using Walters' summary of the clinical characteristics of restless leg syndrome, the three patients in question (1) did not suffer pain or paraesthesia; (2) motor restlessness was in evidence during the day, but not during the night; 3) periodic movements in sleep had not been observed, although no systematic observation during sleep had taken place; 4) there was no family history of restless leg syndrome, and finally, 7) symptoms and signs occurred during waking hours and not at night.

We would therefore maintain that these patients had neuroleptic-induced akathisia rather than restless leg syndrome. This, of course, leaves open the possibility of a "common pathway" of both syndromes evidenced by the association with lower iron levels.

Terao and Yoshimura state that "the association between restless leg syndrome and low plasma iron is generally accepted" referring to Ebom's seminal paper.1 In fact, Ebom found iron deficiency in only 19 of 77 unselected patients. On the other hand, he stated that in patients with iron deficiency of less than 60 μg/100 ml, the incidence of restless legs was 24%. This suggests that iron deficiency is neither a necessary nor a sufficient cause of restless leg syndrome, or indeed (drug-induced) akathisia.

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Pupillary disturbances in migraine: what is the relation to autonomic dysfunction?

The proposal that decreased cerebral sympathetic outflow (and an increase in facial blood flow) follows trigeminal nerve activity during migraine1 is not consistent with the greater eyelid separation and meiosis on the side of the headache and the poor correlation between meiosis and ptosis during and between migraine attacks. Although the pupillary reflex to darkness is regarded primarily as a sympathetic reflex, pupillary dilation in darkness occurs in the human sympathetomised eye but is less complete. In the analysis of pupillary light reflexes it is important to remember that a well-stained degree of central sympathetic tone is necessary for the full development of the constrictor action.

Electrical stimulation of the infratrochlear nerve primarily as a sympathetic reflex, pupillary dilation in darkness occurs in the human sympathetomised eye but is less complete. In the analysis of pupillary light reflexes it is important to remember that a well-stained degree of central sympathetic tone is necessary for the full development of the constrictor action.

Electrical stimulation of the intratrochlear nerve primarily as a sympathetic reflex, pupillary dilation in darkness occurs in the human sympathetomised eye but is less complete. In the analysis of pupillary light reflexes it is important to remember that a well-stained degree of central sympathetic tone is necessary for the full development of the constrictor action.

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was found to be highly effective in inducing changes in the intraocular pressure (IOP) and vasodilatation in the uvea in the rabbit; the reaction was best detected in common with an axon reflex mediated by the peripheral branches of the nerve, at the endings of which some active histamine-like substance is liberated, causing pupillary dilatation and increased intraocular vasodilatation.  

Can mechanical activation of iris trigeminal nerve terminals develop naturally and contribute to miosis seen during and between attacks? Acute elevations in the IOP have been shown to discharge impulses in iris nerve fibres (and whole nerve and corneo-scleral fibres) probably due to mechanical distortion of the iris and the chamber angle which suggests the production of painful impulses described in experimental animals.  

An association between migraine and low-tension glaucoma (LTG) has been suggested recently; the differential diagnosis of LTG should include wide diurnal fluctuations in which high pressures are occurring at times when they are not being recorded. Given the central importance of autonomic nervous system effects, the autonomic hypothesis in those with migraine during headache-free intervals allow development of a relatively higher IOP in response to a variety of stimuli and situations, thereby resulting in exaggerated fluctuations in the pressure that possibly contributes to visual field loss and mechanical activation of iris nerve fibres.  

The results of studies of autonomic nervous system dysfunction in migraine have been contradictory. Besides wide normal inter-and intra-individual variations in the reactions of the autonomic nervous system, it may be useful (and not necessarily simplistic) to view autonomic hyperfunction during migraine attacks as an adaptive (secondary stress) response liable to "fatigue" variably in the later stages of severe headache, one function of which may serve to limit the effects of vasodilatation (of intracranial and cranial blood vessels) resulting from antidromic discharge from trigeminal nerve fibres.

**Increased risk of multiple sclerosis among nurses and doctors**

A recent study concluded that the multiple sclerosis (MS) death rates in British nurses and qualified medical practitioners was not greater than expected. However, as part of a population-based prevalence study of MS in North East Scotland, the occupation of all economically-active women over 15 years of age was classified at the time of onset of the disease and compared with the distribution of economically-active males and females in North East Scotland based on the 1961 Census.  

Fifteen female nurses (occupational group 282) had MS (expected 6–2) and four male medical practitioners (occupational group 280) were affected whilst 0–8 were expected (both p < 0.001).

Whilst the actual numbers involved were small, particularly for medical practitioners, an analysis of occupation at the time of onset of MS will nevertheless, produce a less biased assessment than analyses of occupation at the time of death, given the well-recognised downward occupational drift in chronic disabling diseases such as MS and accepted by Dean and Gray.  

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1 Dean D, Gray R. Do nurses or doctors have an increased risk of developing multiple sclerosis? J Neurol Neurosurg Psychiatry 1980;43:899-902.  
Pupillary disturbances in migraine: what is the relation to autonomic dysfunction?

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