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

HAROLD A. WILKINSON
Division of Neurosurgery, Department of Surgery, University of Massachusetts Medical Center, Worcester, MA, USA


The paper by Leininger, et al. reported that the pursuit of litigation had no effect on neuropsychological status after cerebral concussion, and that the cognitive deficits were attributed to the concussion dysfunction. 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 those not. The absence of a control group is a significant limitation. 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, and the Portage, Michigan, area, was unable to sue their insurers, regardless of fault. In nonwork-related motor vehicle 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 comparable 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 Leininger et al., a factor which may have affected the results.

Leininger et al. equated the cognitive deficits of the concussed patients with cerebral dysfunction. The possible existence of comorbidities in the minor head injured patients make this relationship tentative, 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 medications. The authors provided no information on chronic pain, psychiatric status on 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 normalization 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 Leininger et al.

LAURENCE M. BINDER
Psychology Service, VA Medical Center, Portland Division, 3710 Southwest US.

Veterans Hospital Road, Portland, OR 97207, USA


Low plasma iron status and akathisia

Barton et al. reported a significant inverse correlation between plasma iron levels and akathisia in an animal model. However, three of their akathisia 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 while in both conditions), the symptoms of akathisia are prominent throughout the waking hours. Conversely, the symptoms of restless legs are more prominent at night.7 It suggests that the circadian rise of possibly 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.

J MAKI TARAOKI REIJI YOSHIMURA
Department of Psychiatry, University of Occupational and Environmental Health, School of Medicine, Kitakyushu 807, Japan


Bowie and Ebmeier reply:

We are pleased to answer Terao's and Yoshimura's comments relating to the differential diagnosis of our akathisia patients, particularly those who present with 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 from a psychogenic disturbance; 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 some dyskinesia lying still, or sitting quietly, as evidenced by patients' akathisia scores; 5) none of the patients was suffering from sleep disturbance; 6) 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 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 Ebmeier's seminal paper. In fact, Ebmeier 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 we iron deficiency is neither a necessary nor a sufficient cause of restless leg syndrome, or indeed (drug-induced) akathisia.

J BOWIE
Department of Psychiatry, MRC Brain Metabolism Unit, Royal Edinburgh Hospital, Edinburgh, UK


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

The proposal that decreased cervical sympathetic outflow (and an increase in facial blood flow) follows trigeminal nerve activity during migraine is not consistent with greater eyelid separation and meiosis on the right 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 dilatation in darkness occurs in the human sympathetometised eye but is less complete. In the analysis of pupillary light reflexes it is important to remember that a well-defined 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 sympathetometised eye but is less complete. In the analysis of pupillary light reflexes it is important to remember that a well-defined degree of central sympathetic tone is necessary for the full development of the constrictor action.

Electrical stimulation of the infraorbital nerve primarily as a sympathetic reflex, pupillary dilation in darkness occurs in the human sympathetometised eye but is less complete. In the analysis of pupillary light reflexes it is important to remember that a well-defined degree of central sympathetic tone is necessary for the full development of the constrictor action.
was found to be highly effective in inducing changes in the intraocular pressure (IOP) and vasodilatation in the uvea in the rabbit; the reaction seems to be much more common in the presence of 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 intense intraocular vasodilatation.1 Can mechanical activation of iris trigminal nerve terminals develop naturally and contribute to miosis seen during and between attacks? Acute elevation of IOP in the IOP have been shown to discharge impulses in iris nerve fibres (whole nerve and coneo-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.4 An association between migraine and low-tension glaucoma (LTG) has been suggested recently5 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 sympathetic vasomotor dysfunctions and autonomic hyperfunction in those with migraine during headache-free intervals6 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 contribute to both visual field loss and mechanical activation of iris nerve fibres. The results of studies of autonomic nervous system dysfunction in migraine have been contradictory.4 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 intraocular and cranial blood vessels) resulting from antidiromic discharge from trigminal 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 individuals 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.15 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).17 While 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 analysis 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.1 I conclude that, at least in North East Scotland in 1970, there was an excess risk of MS among female nurses and possibly among male doctors.

**References**

1. Dean D, Gray R. Do nurses or doctors have an increased risk of developing multiple sclerosis? J Neurol Neurosurg Psychiatry 1990;53:899-902.

**Dr P Sundhombt**

Psychology Section, Murdoch University, Murdoch, Western Australia


**Dr P Sundhombt**

Psychology Section, Murdoch University, Murdoch, Western Australia


**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 individuals 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.15 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).17

While 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 analysis 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.1 I conclude that, at least in North East Scotland in 1970, there was an excess risk of MS among female nurses and possibly among male doctors.

**References**

1. Dean D, Gray R. Do nurses or doctors have an increased risk of developing multiple sclerosis? J Neurol Neurosurg Psychiatry 1990;53:899-902.

**Dr P Sundhombt**

Psychology Section, Murdoch University, Murdoch, Western Australia