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

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1 Leininger BE, Gramling SE, Farrell ED, Kreutzer JS, Peek EA. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *J Neurol Neurosurg Psychiatry* 1990;**53**:293–6.

The paper by Leininger, *et al*[1] reported that the pursuit of litigation had no effect on neuropsychological status after cerebral concussion, and that the cognitive deficits were attributable to cerebral 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 claims for compensation" or "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, and in the United States, been unable to sue their employers, regardless of fault. In nonwork-related motor vehicle accidents, claims for damages may have been made against insurers without any 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 contrary to our own.

In a recent paper Binder and Willis[2] 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 much more 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 tenuous, however. The majority of the minor head injury patients were injured in motor vehicle accidents. Consequently, they may have had orthopaedic 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, or medication use. These variables are also associated with cognitive abilities[3 4] and may have accounted for the differences between the concussed and control subjects. Controlled studies of consecutive acutely injured patients followed prospectively[5] 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 Leininger *et al*.

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1 Leininger BE, Gramling SE, Farrell ED, Kreutzer JS, Peck EA. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *J Neurol Neurosurg Psychiatry* 1990;**53**:293–6.
2 Binder LM, Willis SC. Assessment of motivation after financially compensable minor head trauma. *J Psychological Assessment: A Journal of Consulting and Clinical Psychology* (in press).
3 McAllister TW. Cognitive functioning in the affective disorders. *Comprehensive Psychiatry* 1981;**6**:572–86.
4 McNairy SL, Maruta T, Ivnik RJ, Swanson DW, Ilstrup DW. Prescription medication dependence and neuropsychologic function. *Pain* 1984;**18**:169–77.
5 Dikmen S, McLean A, Temkin N. Neuropsychological and psychosocial consequences of minor head injury. *J Neurol Neurosurg Psychiatry* 1986;**49**:1227–32.

Low plasma iron status and akathisia

Barton *et al*[1] reported a significant inverse correlation between plasma iron levels and akathisia rating. Their findings are important. 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,[2] 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 occur in both conditions),[3] the symptoms of akathisia are prominent throughout the waking hours. Conversely, the symptoms of restless legs are more prominent at night.[4] 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*,[5] 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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1 Barton A, Bowie J, Ebmeier K. Low plasma concentration and akathisia. *J Neurol Neurosurg Psychiatry* 1990;**53**:671–4.
2 Ekbom KA. Restless legs syndrome. *Neurology* 1960;**10**:868–73.
3 Walters AS, Hening WA, Chokroverty S. Frequent occurrence of myoclonus while awake and at rest, body rocking and marching in place in a subpopulation of patients with restless legs syndrome. *Acta Neurol Scand* 1988;**77**:418–21.
4 Walters AS, Hening W. Clinical presentation and neuropharmacology of restless legs syndrome. *Clin Neuropharmacol* 1987;**10**:225–37.
5 Sandyk R. Melatonin stimulating hormone (MSH) in the restless-legs syndrome. *Intern J Neuroscience* 1989;**46**:197–9.

Bowie and Ebmeier reply:

We are pleased to answer Terao's and Yoshimura's comments relating to the differential diagnosis of our akathisic patients, particularly the patients with plasma iron levels about 50 μg/100 ml. Using Walters'[1] summary of the clinical characteristics of restless leg syndrome, the three patients in question: 1) did not suffer parasthesias; 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 wakening 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 Ekbom's seminal paper.[2] In fact, Ekbom found iron deficiency in only 19 of 77 unselected patients. On the other hand, he states that in patients with iron deficiency of less than 60 μg/100 ml, the incidence of restless legs was 24%. This suggests to us that iron deficiency is neither a necessary nor a sufficient cause of restless leg syndrome, or indeed (drug-induced) akathesia.

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1 Walters AS, Hening WA, Chokroverty S. Frequent occurrence of myoclonus while awake and at rest, body rocking and marching in place in a subpopulation of patients with restless legs syndrome. *Acta Neurol Scand* 1988;**77**:418–21.
2 Ekbom KA. Restless legs syndrome. *Neurology* 1960;**10**:868–73.

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[1] is not consistent with greater eyelid separation and meiosis on the side of the headache and the poor correlaton 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 sympathectomised 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 causes unilateral meiosis in normal volunteers[2] thus raising the possibility of a contribution of iris trigeminal fibres towards the development of meiosis during migraine headache through antidromic discharge. In contrast to electrical stimulation of the ophthalmic division of the trigeminal nerve (which was found to be relatively ineffective), mechanical stimulation of the nerve, both with and without stellate ganglionectomy,