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 psychogenic 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 "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, and if so, 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 with a catastrophic loss. 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 cannot be applied to our own. In a recent paper Binder and Willius' 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 makes this relationship tentative, 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 anesthetic 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 state, 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 consecutively 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 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. They found a weak but significant correlation. 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 findings may not be an artifact of 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. 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 melatonin stimulating hormone (MSH).

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

TAKESHI TARAOKA, REIJICHI YOSHIMURA
Department of Psychiatry,
University of Occupational and Environmental Health,
School of Medicine,
Kitakyushu 807, Japan


Bowie and Ebenezer 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 paroxysmal, 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 legs syndrome, and finally, 7) symptoms and signs occurred during wakening hours and not at night.

We would therefore maintain that these patients had a hypokinesia 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 Ekbo's seminal paper. In fact, Ekbo 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 that it is the iron deficiency that is neither necessary nor a sufficient cause of restless leg syndrome, or indeed (drug-induced) akathisia.

J BOWIE, K EBENEZER
MRC Brain Metabolism Unit,
Royal Edinburgh Hospital,
Edinburgh, UK

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 migraine is not consistent with 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 sympathetised 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 (and an increase in facial blood flow) follows trigeminal nerve activity during migraine 4 and it is important to remember that this 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.
