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To the Editor

Re: Exacerbation of Pre-existing Epilepsy by Mild Head Injury

Tai PC, Gross DW. Can J Neurol Sci 2004; 31:394-397.

Drs. Tai and Gross recently reported an exacerbation of pre-existing epilepsy in a series of patients following mild injury to the brain. The authors lay claim to a causal connection by way of cerebral insult rather than the effects of stress.

Unfortunately there was no assessment of seizure frequency in a group of control individuals receiving injuries other than to the brain. The authors suggest that because the increase in seizure frequency was prolonged following the brain injury, it is unlikely that the increase was solely due to stress. However, an adjustment reaction following injury may be prolonged for a period of years, notably in those designated as having post-traumatic stress disorder.[1] Neuronal plasticity changes may take place in the limbic circuitry of chronically stressed individuals regardless of injury or type of injury.[2]

It is possible that, unwittingly, Drs. Tai and Gross may have included two, or even three, injured individuals without brain trauma in their series of five, namely those without a documented blow to the head. The authors assumed there was brain injury solely as a result of deceleration. However, brain injury without head contact in adults is so rare that it is almost never seen in a clinical setting in civilian life.[3]

The authors may be right in supporting a direct relationship between exacerbation of seizure disorder and a minor injury – regardless of whether or not there was trauma to the brain.

Peter M. Rees
Burnaby BC

1. Yehuda R. Post-traumatic stress disorder. N Engl J Med 2002; 346: 108-130.
2. Bremner DJ. Does stress damage the brain? Biol Psychiatry 1999; 45: 797-805.
3. Rees PM. Contemporary issues in mild traumatic brain injury. Arch Phys Med Rehabil 2003; 84:1885-1894.

Response

Exacerbation of Pre-existing Epilepsy by Mild Head Injury

Tai PC, Gross DW. Can J Neurol Sci 2004; 31:394-397.

While we had considered the possible role of stress, none of our patients met DSM-IV-TR criteria for post-traumatic stress disorder[1] and, therefore, it remains our opinion that the most likely explanation for seizure exacerbation was head trauma. As our series was retrospective, some accidents occurred years before presentation. Based on the nature of the accidents, we suspect some degree of head trauma likely was present in all patients. We presented this series because we were struck by the temporal relationship between minor accidents and exacerbation of seizures in epileptic patients. Further study is required to ascertain whether what has previously been considered trivial head injury can provoke seizures in epilepsy patients.

P.C. Tai, D.W. Gross
Edmonton, Alberta

1. 309.81 Posttraumatic Stress Disorder. In: American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric Association 2000:463-468.

To the Editor

Re: Comparison of Monitoring Techniques for Intraoperative Cerebral Ischemia.

DW Rowed, DA Houlden, LM Burkholder, AB Taylor. Can J Neurol Sci 2004;31(3):347-356.

The methods and results of this article do not warrant its conclusion that somatosensory evoked potentials (SEPs) are more reliable than EEG to detect cerebral ischemia. Bilateral median SEPs and four-channel EEG (F3-C3', T3-C3', F4-C4', T4-C4') were monitored in 156 carotid endarterectomies. However, multi-channel recording is fundamental to EEG and 16-channel monitoring is adviseable.[1,2] The EEG was measured from intermittent two-second epochs, but requires a longer time-base for proper analysis.[1] Significant amplitude change was defined as a >50% reduction for SEPs and a >75% reduction of “all activity” for EEG. The reference for the EEG criterion states that major changes "consist of attenuation of all activity by at least 75% and/or a twofold or more increase of ≤ 1 Hz delta activity",[1] but increased delta was ignored and blunted by 1 Hz low frequency filtering. Moderate ischemic EEG changes were also ignored. Finally, the disproportionately high EEG technical failure rate of 5% is contrary to previous experience.[1,2] Fundamentally, SEPs were compared to suboptimal EEG.

No patient with preservation of both modalities at the end of monitoring suffered an intraoperative stroke. Two patients had congruent SEP/EEG deterioration restored after shunting. Two patients suffered intraoperative stroke. One had congruent persistent deterioration of both tests. The other had persistent SEP but “no significant” EEG changes. This single critical case forms the entire basis for the authors' contention that SEP monitoring is superior. Disturbingly, EEG waveforms are not provided and the deficits and imaging results are not described. The reader cannot determine the validity of the EEG interpretation or the lesion's location. If the infarct was deep subcortical, then the EEG may have been unaltered. If it was cortical, then the EEG technique was likely inadequate because a proper EEG should be altered and accepting such an unexpected result requires more proof than that provided.

Furthermore, one patient had significant EEG deterioration reversed after shunting but did not have a significant SEP change. Waveforms are again not provided, but this could have been an example of ischemia detected and reversed by EEG