

8. Heckmatt JZ, Kirkham F. Peripheral neuropathy and neuromuscular blockade presenting as prolonged respiratory paralysis following critical illness. *Neuropediatrics* 1993;24:123-125.
9. Segredo V, Caldwell JE, Matthay MA, et al. Persistent paralysis in critically ill patients after long-term administration of vecuronium. *N Eng J Med* 1992;327:524-528.
10. Vanderheyden BA, Reynolds HN, Gerold KB, et al. Prolonged paralysis after long-term vecuronium infusion. *Crit Care Med* 1992;20:304-307.

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.

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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 disorder¹ 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.

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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 advisable.^{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

monitoring that was not detected by SEPs. Disturbingly, the potential importance of this result is completely ignored in the discussion and conclusions.

One other patient had a transient SEP change that resolved spontaneously without EEG alteration. However, the unconvincing illustration shows only a single average below the 50% criterion, which might be explained by the random effects of noise evident in the traces shown.

Because carotid ischemia is usually widespread, both methods are very sensitive, but the situation may be different with more limited disturbances. Proper EEG provides the coverage required to demonstrate extensive or focal cortical ischemia.¹ Although deep subcortical lesions may not cause EEG alterations, this appears to be very rare with good EEG technique.^{1,2} Somatosensory evoked potentials detect subcortical or cortical somatosensory pathway lesions, but are anatomically limited to this specific system only. Therefore, it is physiologically inevitable that motor and other non-sensory neurologic compromise without SEP change will occur exactly as has been found for other surgeries and documented during endarterectomy.^{3,4} It may have even occurred in one of the patients of this study, although apparently unappreciated by the authors. Depending on the undisclosed details of the critical case discussed above, the results of this study could support the contention that the suboptimal EEG techniques used were inadequate or that endarterectomy monitoring should involve both modalities. They do not demonstrate superiority of either, and should not persuade practitioners to rely primarily on SEP monitoring.

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1. Blume WT, Sharbrough FW. Monitoring during carotid endarterectomy and open heart Surgery. In: Niedermeyer E, Lopes Da Silva F (Eds). *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. Baltimore: Williams and Wilkins, 1999: 797-808.
2. Salvian AJ, Taylor DC, Hsiang YN, et alB. Selective shunting with EEG monitoring is safer than routine shunting for carotid endarterectomy. *Cardiovasc Surg* 1997; 5(5): 481-485.

3. Haupt WF, Erasmi-Korber H, Lanfermann H. Intraoperative recording of parietal SEP can miss hemodynamic infarction during carotid endarterectomy: a case study. *Electroencephalogr Clin Neurophysiol* 1994; 92(1): 86-88.
4. Linstedt U, Maier C, Petry A. Intraoperative monitoring with somatosensory evoked potentials in carotid artery surgery--less reliable in patients with preoperative neurologic deficiency? *Acta Anaesthesiol Scand*. 1998; 42(1): 13-16.

RESPONSE

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We agree that 16 channel EEG monitoring provides more widespread coverage of cortical function and it is conceivable that we may, with further experience, encounter a false negative SSEP in the face of a true positive EEG. The opposite was true in the present cohort and this, combined with the fact that we were unable to record EEG in a slightly higher proportion of patients (mainly due to low EEG amplitude after anesthetic induction in patients with previous strokes) led us to conclude that, in our hands, the SSEP was more reliable than EEG.

As we indicated in our discussion, the low intraoperative stroke rate in our series of cases renders our conclusions tentative and we would therefore certainly not attempt to persuade practitioners who are currently employing intraoperative EEG from continuing to do so. We would rather prefer to stimulate additional studies that directly compare monitoring methodologies in the hope that, ultimately, simplification will result. If this occurs, more centres are likely to practise routine intraoperative monitoring for cerebral ischemia with resulting improvement in patient safety.

*David W. Rowed
David A. Houlden*