CORRESPONDENCE

To the Editor

Re: Whiplash and concussion: Similar acute changes in middle-latency SEP's. Can J Neurol Sci. 2006; 33: 379-86.

Dr. Zumsteg and his colleagues[1] have performed a very interesting and provocative study that suggests that both whiplash injury and concussion alter processing of the middle-latency SEP component N60 in the subacute post traumatic period. These changes appeared to normalize between three-six months post injury. The authors speculate that the overlapping clinical symptomatology post whiplash and post concussion may reflect a similar underlying mechanism of rotational mild traumatic brain injury.

The authors do not say whether their comparisons between whiplashed and concussed patients were blinded. They acknowledge that physiological parameters including drowsiness - distinct from pathophysiological changes from a brain injury - can influence the middle-latency SEP components. The abnormal increases in N60 latency were not compared to a control group of patients whose injuries were at a site remote from the head or the neck, for example lower limb injury which by way of pain distraction and other factors such as insomnia and mood change may cause affective and cognitive symptoms indistinguishable from post concussive syndrome.[2,3] The N60 latencies that are said to be generated in areas 1 and 3B of the primary somatosensory cortex, are areas which with painful states may undergo central reorganization from neural activations/deactivations in area 1 and the association areas of the parietal cortex as shown by functional imaging studies.[4] Another confounding variable to potentially influence the SEP, is the demonstrable change to cortical blood flow on SPECT that can be brought about solely by headache[5] as a shared symptom in both concussion and whiplash.

Cited by Zumsteg et al[1] in support of their hypothesis that 'whiplash' without head contact may cause brain injury, were nonhuman primate studies of experimental whiplash including the 1982 experiments of Gennarelli et al[6] that identified structural brain damage but only when the whiplash forces were great enough to cause coma lasting 15 minutes or longer, hardly comparable to the forces that produce human whiplash in everyday life. It is recognized that the human brain may be irreversibly damaged in harnessed pilots crashing a military aircraft in which the rapidly decelerated cranium may never contact a solid object.[7] However, biomechanical calculations of the forces to the head and neck area generated by low-velocity vehicular whiplash[8] are far less than the threshold of about 80-100 G of acceleration/deceleration thought necessary for cerebral injury to occur.[9] Furthermore, in a series of neuropathologic examinations of more than 400 road users fatally injured without receiving head impact, MacLean[10] found no examples of macroscopic or microscopic brain injury.

It will be of interest to repeat the measurements of middle-latency SEP's in people with symptoms arising at injury sites remote from the head and neck and in uninjured people with acute affective/pain disturbances including active headache. On the other side of the coin, will whiplashed 'demolition' derby drivers having no symptoms, also show SEP abnormalities?

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