Cognitive evoked potential (P<sub>300</sub>): a metric for cerebral concussion

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ABSTRACT - Cognitive impairment has been reported to occur in minor head injury (concussion). The value of the P<sub>300</sub> evoked potential as a measure of cerebral concussion was studied in 20 patients with minor head injury and compared with the data from 20 normal subjects. Significant abnormalities of the P<sub>300</sub> latency and amplitude were noted in these patients in the post-concussion period. The abnormalities improved completely on repeat testing. The correlation of the P<sub>300</sub> to other parameters of head injury is discussed. The P<sub>300</sub> constitutes a simple laboratory test that is a sensitive measure of cerebral dysfunction in concussive head injuries.

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stimuli were presented binaurally through earphones at an intensity of 90 dB. The subjects were seated relaxed and instructed to avoid eye and finger movements. The recording silver chloride electrodes were fixed to the scalp at Cz, with electrode paste and referenced to linked mastoids with FPz as common electrode (10-20 system). Electrode resistance was kept below 2 Kohms. The recording electrodes were led to a Nicolet C3 evoked potential system with filter band pass at 1-30 Hz. An oculogram channel with electrodes above and below the eye was used to monitor eye movement artifacts. Averaging epochs contaminated by artifacts were automatically rejected by the averaging computer. The sampling time was 800 ms and stimulus rate was 0.7/s. The ERPs to both target and non-target stimuli were averaged separately. Each test was repeated once for consistency of results. The latencies of the P2 (P2L) and P3 (P3L) components and amplitude of P3 (P3A) were measured.

In the patient group, the ERPs were done on admission (within 4 days of head injury) and repeated at intervals ranging from 30 to 240 days. The intervals differed for patients. The ERP results in patients were compared to those in normal controls. The P2L and P3L were defined as abnormal if the absolute latency was more than mean plus 2.5 standard deviations (P2L > 228.5 ms and P3L > 383.1 ms). The results were correlated with the GCS, LOC, post-traumatic amnesia (PTA), outcome and development of post-concussional symptoms (PCS).

Results
Normal subjects
The P2L, P3L and P3A data in normal subjects and their correlation with age are shown in Table 1 and Fig. 1. There was no significant difference in P2L, P3L and P3A between males and females.
Patients with concussion

These patients’ ages ranged 10-60 years; there were 18 males and 2 females. At admission they reported LOC ranging 0-30 min (mean 12.8) and PTA ranging 0-60 min (mean 18). Examination revealed GCS ranging 13-15 and no focal neurological deficits (Table 2). They all recovered completely and were discharged within 4 days. PCS of giddiness and loss of memory respectively were seen in 2 patients on follow up (Table 2). The initial P2L and P3L (at admission) were abnormal in 20% and 35% of patients respectively. The initial prolongation of PzL was insignificant (P 0.16) and P3L was significant (P 0.002) by Student t test. A significant initial attenuation of P1L was also seen in comparison to normals (P 0.003).

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However, repeat testing revealed improvements (return to normal values) in P1L, P3L and P3A in all patients (Table 2). The P1L, P3L and P3A improved by mean 32.6 ms, 72.5 ms and 6.3 μV respectively. Paired t tests showed these improvements to be significant P = 0.002, P = 0.0005 and P = 0.0003 respectively for P1L, P3L and P3A. The repeat tests were done after intervals ranging from 30-240 days. The improvements in these parameters were not significantly different between those tested after 30 days and those tested much later. These parameters and their correlation to the neurological assessment are shown in Table 2. Abnormalities of P1L, P3L and P3A did not show significant correlation with the GCS, duration of LOC and PTA or development of PCS.

Discussion

The normal values for P1L, P3L, and P3A are similar to those reported by others (16, 17). However a positive linear correlation with age was not seen in our subjects (Fig. 1). The significant abnormalities of P1L, P3L and P3A in patients at admission and return to normalcy on repeat testing indicate that these are sensitive
Studies of the scalp distribution of the auditory N1-P2 complex in normals, suggest the primary auditory cortex of both hemispheres as the neural generators of these components (19, 20). Intracranial depth electrode and extracranial magnetic recordings have provided evidence that the N1-P2 components are generated by amygdala and hippocampus (21, 22). These ERPs are thought to be time-locked synchronous neural electrical activity that arises from these structures in association with the processes of perception and cognition (7). The abnormalities of these ERPs in the present study indicate dysfunction or damage to these structures consequent to cerebral concussion. The improvement in the ERPs denotes the transient nature of the dysfunction/damage. Rather than the absolute values of the ERP latencies and amplitudes, the abnormality parameters in detecting the cerebral dysfunction that occurs in concussive head injuries. Of the three, P1L is the most sensitive parameter on account of its abnormality in a larger number of patients and a greater degree of abnormality. In their study on patients with severe head injury, Olbrich et al 1986 found the P3 to be a sensitive indicator of cognitive impairment that correlated well with findings on neuropsychological tests. In their patients, repeat testing after 5-6 months showed that while cognitive abnormalities returned to normal, prolongation of P1L persisted, suggesting residual cerebral dysfunction (14). In contrast, in the present study all parameters returned to normal limits indicating the short-lived cerebral dysfunction in cerebral concussion. This fact has been reported by Gentilini et al (1985) who found that cognitive impairment did not persist after one month after minor head injury (3). The abnormalities in P1L, P3L and P3A did not correlate well with parameters of head injury such as LOC, PTA, GCS or development of PCS. A similar lack of correlation between cognitive impairment (on neuropsychological testing) and these parameters in minor head injury has been documented (2).

In conclusion, the P3 constitutes a simple laboratory test that is a sensitive and objective measure of cerebral dysfunction in concussive head injuries and we recommend its regular use for this purpose.

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**References**