

Letters to the Editor

Behçet's Disease without Neurological Manifestations

To the Editor

We read with interest the article, "Cognitive event-related potential in Behçet's disease without neurological manifestations" in the Tohoku J. Exp. Med., 2005, **206**, 15-22. The authors suggested that subclinical neurological involvement is undetectable by neurophysiological tests (Ozısık et al. 2005). In previous studies, abnormal BAEP (Brainstem auditory Evoked Potentials), VEP (Visual Evoked Potentials) and MEP (Magnetic Evoked Potentials) values were reported in 31%, 25% and 28% of patients with Behçet's disease without neurological manifestations (Stigsby et al. 1994; Stigsby et al. 2000a, b). Additionally, we reported the patients with Behçet's disease without neurological manifestations have significantly prolonged latencies of P300 and delayed motor response time than controls (Kececi et al. 2001). In our other study (Kececi et al. 2004), ERPs (Event Related Potentials) were recorded from frontal (Fz), central (Cz) and parietal zones (Pz), and we found a significant difference of latencies of P300 between groups. P300 latencies at Fz were not significant whereas at Cz and Pz there was significant difference between patients and controls. The P300 amplitude at Fz was found to be significantly higher in the patient group. Moreover, we showed a lack of habituation of P300 latencies by repetitive auditory stimulus in patients. These findings may reflect that there is an insidious neuronal pathologic process in Behçet's disease. Finally, the results of our two studies related to P300 prolonged latency confirm subclinical involvement of Behçet's disease.

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Response

We have reported that there are no cognitive event-related potential differences in Behçet patients without neurological involvement when compared to control subjects (Ozısık et al. 2005). Kececi et al. (2001, 2004) found a difference when compared to controls but they had recorded from a single electrode in their first study and from 3 mid-zone electrodes in their second study, in contrast to our study. They found an increase in P300 latency in the Cz region and the Cz and Pz regions in the first and second studies, respectively. When compared to our study, the mean age of the patient and control groups were lower and the age range was narrower in their studies (Kececi et al. 2001, 2004). In addition, Gökçay et al. (2004) found no cognitive event-related potential differences between Behçet patients and controls, similar to our study. However, the patient selection favored women (Gökçay et al. 2004) and was different from our study or the studies of Kececi et al. (2001, 2004). Gökçay et al. (2004) also performed electrophysiological recordings from only two regions. These four studies are slightly different in the selection of their patients and control group and the electrode regions from which the event-related potentials were recorded. The limited number of cases may also lead to a conflict between the results. It is well-known that

systemic and/or neurological involvement in Behçet's disease differs from patient to patient and from time to time (Siva et al. 2004). It is therefore possible to resolve the conflict of the results with new studies using a larger number of patients and comprehensively designed electrophysiological tests.

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