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Cortical function abnormalities in migraine: neurophysiological and neuropsychological evidence from reaction times and event-related potentials to the Stroop Test

Abstract Cortical hyperexcitability was studied in migraine patients using reaction times (RT's) and Event-Related Potentials (ERP) to the Stroop test. We found a slower RTs in patients if compared to controls, supporting the hypothesis of a mild cortical functions impairment even in interictal periods in this group of patients.

Key words Event-related potentials • Habituation physiology • Migraine

Central neuronal hyperexcitability is proposed to be the putative basis for physiologic disturbances in migraine [1–3]. Studies using event-related potentials (ERPs), as well as transcranial magnetic stimulation studies have shown the presence of such hyperexcitability, although results have been conflicting so far [4–6].

The aim of this study was to investigate the involvement of executive functions and to map cortical hyperexcitability using reaction times (RTs) and ERPs to the Stroop test [7], a neuropsychological test involving visuomotor, associative and frontal cortical functions [8].

In our study, 13 right-handed patients with migraine (mean age 42.2 ± 12 years, 6 migraine without aura, 7 migraine with aura), and 13 normal subjects (mean age 40.1 ± 12.5 years) participated in the study. All patients were free from migraine attacks in the 72 h preceding the test.

Stroop test RTs were evaluated in simple, Go–No Go and choice paradigms using a computerised system, and ERPs during mental performance of the Stroop test were obtained from 32 scalp electrodes.

Group grand average Student's t-test analysis of ERP was performed, as well as Student's t-test analysis of RTs, omitted and committed errors at the Stroop test.

Compared to controls, migraine patients had slower ($p < 0.05$) RTs to Stroop Test stimuli in the complex paradigm (Fig. 1), while simple RTs and accuracy by means of committed and omitted errors (Figs. 2 and 3) did not differ between the two groups.

Compared to controls, migraine patients showed a significantly higher amplitude and duration of the positive component at around 400 ms over occipital ($p < 0.01$) and parietal ($p < 0.05$) areas, with no significant differences over the frontal areas (Fig. 4).

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Discussion

Our findings of slower RTs support the hypothesis of a mild impairment of higher cortical functions even in interictal

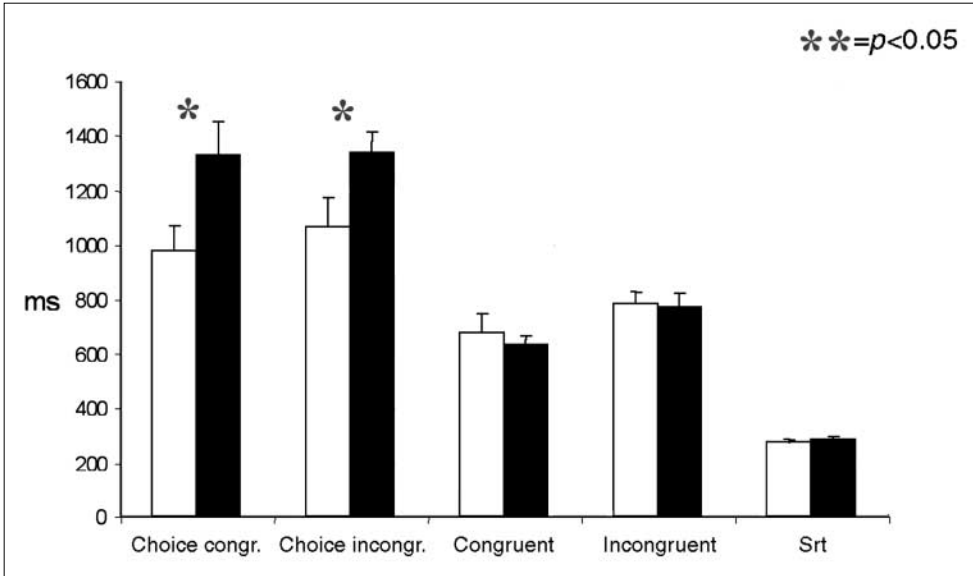


Fig. 1 Reaction times to Stroop test stimuli in migraineurs (black bars) and controls (white bars). Controls showed significantly slower RTs in choice paradigms, both to congruent and incongruent stimuli, compared to healthy subjects

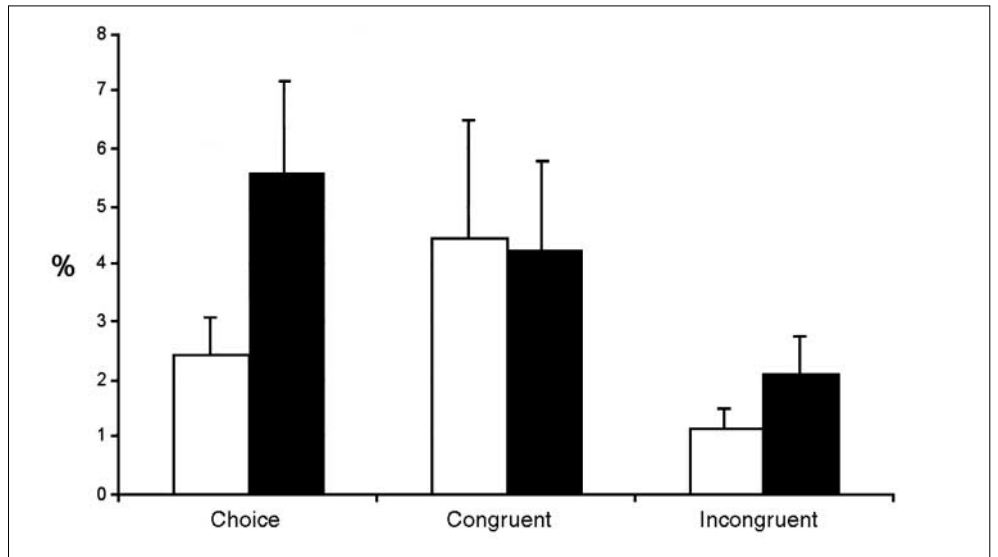


Fig. 2 Committed errors in Stroop test in migraineurs (black bars) and controls (white bars). No significant difference was found between the two groups, although in choice paradigm migraineurs committed more errors than controls

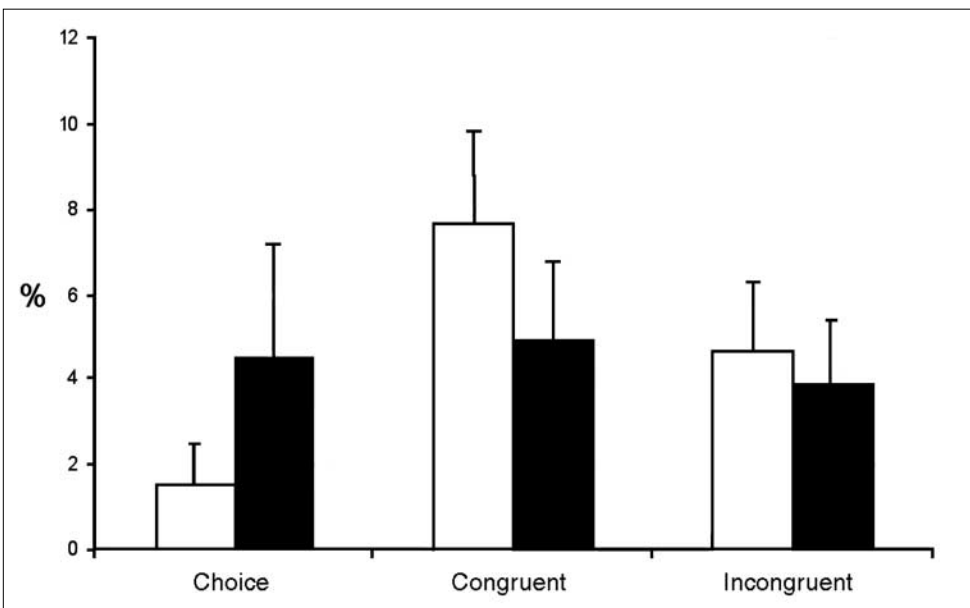


Fig. 3 Omitted errors in Stroop test in migraineurs (black bars) and controls (white bars). No significant difference was found between the two groups, although in choice paradigm migraineurs omitted more responses than controls

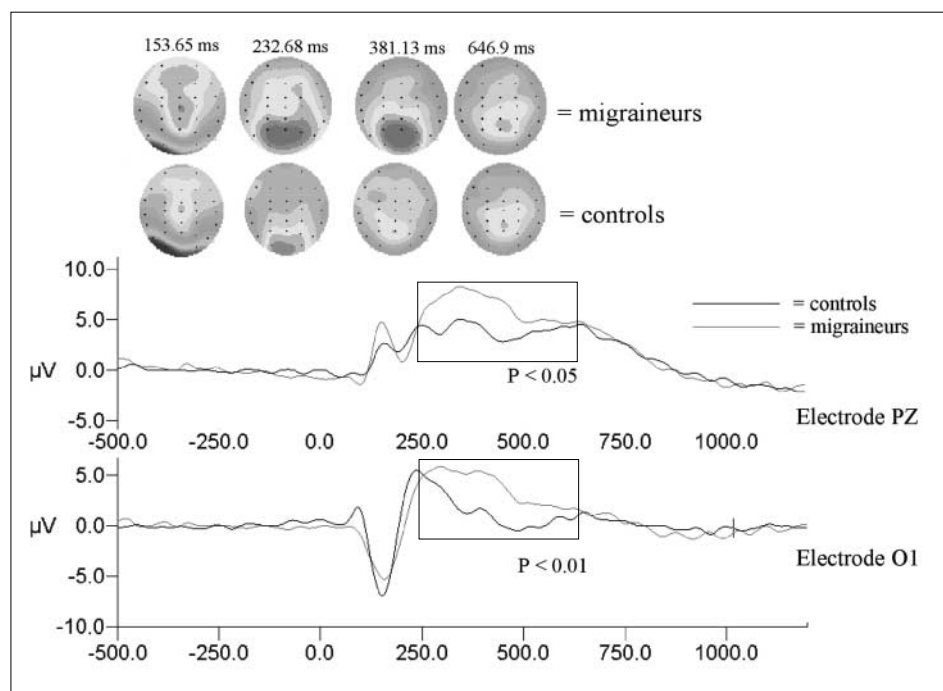


Fig. 4 Group grand average ERPs to Stroop test in migraineurs and controls, recorded in PZ electrode (*upper track*) and O1 electrode (*lower track*). Voltage maps in the upper part of the figure show the persistence of the positive component (*red area*) of the ERP over parieto-occipital areas in migraineurs, even 400 ms after stimulus

periods in migraine patients [9], suggesting the need of high-sensitivity methods to spot out such abnormalities. The higher expression of long-latency ERPs components over occipital and parietal areas supports previous results [10] of lack of habituation and overweight of cortical excitatory vs. inhibitory processes in migraine patients.

Further investigations are needed in order to determine whether the poorer performance at the Stroop test in migraine patients is to be related to aspecific cortical changes such as impairment of inhibitory processes, or to involvement of specific cognitive domains.

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