

EEG ALPHA IS RELATED TO THE COGNITIVE IMPAIRMENT AND „HIDDEN” NEUROPATHOLOGY OF MINOR STROKE

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We aimed this study to investigate the EEG alpha activity at several levels of quantitative EEG analysis, and its relationship to the cognitive functions in the subacute and chronic stages of minor ischemic stroke.

Study included 10 patients with the right middle cerebral artery ischemic stroke and 11 healthy controls. Neurological impairment was measured by National Institute of Health Stroke Scale (NIHSS), whereas the cognitive functions were assessed by Montreal Cognitive Assessment (MoCA) and MoCA memory index (MoCA-MIS). EEG was recorded in resting, awake state with eyes closed using 19 channel EEG system with standard EEG electrode placement. The final 10 minutes artefact-free EEG signals were analyzed in MATLAB R2011a. We have particularly analyzed the EEGs derived from four lateral frontal (F3, F7, F4, F8), and corresponding lateral posterior (P3, P4, T5, T6) electrodes. Quantitative EEG analysis included: the group FFT spectra, the weighted average of alpha frequency (α AVG), the group probability density distributions of all conventional EEG frequency band relative amplitudes (EEG microstructure), the inter-hemispheric and intra-hemispheric coherences, and the topographic distribution of alpha carrier frequency phase potentials (PPs). Statistical analysis was done using the Kruskal-Wallis ANOVA with the post-hoc Mann-Whitney U two-tailed test, and Spearman's correlation.

We have demonstrated the transient cognitive impairment alongside the slower alpha frequency (α AVG) in the subacute stroke patients vs. controls, with no amplitude change, but highly synchronized intra-hemispherically, above the overall ipsi-lesional hemisphere, and inter-hemispherically, above the overall frontal cortex. In addition, the disturbances of EEG alpha activity in subacute stroke patients were expressed as the decrease of alpha PPs over the frontal cortex (indicating a delay of the slower alpha), and the altered "alpha flow", indicating the sustained augmentation of inter-hemispheric interactions.

Although the stroke induced slower alpha was a transient phenomenon, the increased alpha intra- and inter-hemispheric synchronization, the delayed alpha waves, and the newly established inter-hemispheric "alpha flow" within the frontal cortex, remained as a permanent consequence of the minor stroke. This, newly established frontal inter-hemispheric "alpha flow" presented a permanent consequence of the "hidden" stroke neuropathology, despite the cognitive impairment have been returned to the control values. Moreover, all detected permanent changes after minor stroke at EEG level with no cognitive impairment, could be a way for the brain to compensate lesion and restore the lost function.

Our study indicates that slower EEG alpha generation, synchronization and "flow" are related to the cognitive impairment and „hidden" neuropathology of minor stroke, and could present a compensatory post-stroke re-organizational changes, contributing to functional recovery.