

HYPERVENTILATION INDUCED HYPOCAPNIA SHOULD BE AVOIDED IN CASES OF CEREBRAL HYPOPERFUSION

KJ. Szabó¹, B. Rosengarten², L. Csiba¹, L. Oláh¹

¹*Department of Neurology, University of Debrecen, Hungary*

²*Department of Neurology, University of Giessen, Hungary*

dr.katalinszabo@gmail.com

Introduction: Previous studies proved that hypercapnia induced vasodilation does not inhibit the visually evoked flow velocity changes in the posterior cerebral arteries. Our aim was to determine whether vasoconstriction induced by hypocapnia affects the neurovascular coupling.

Methods: By using visual cortex stimulation paradigm, visually evoked flow velocity changes were detected by TCD in both PCAs of young healthy adults. The control measurement was followed by the examination under hyperventilation (HV). Visual-evoked-potentials were also recorded.

Results: Comparing control and HV phases, the breathing frequency significantly increased (16 ± 2 vs. 37 ± 3 /min), resulting in significant decrease of the end-tidal CO₂ (37 ± 3 vs. 25 ± 3 mmHg) and decrease of resting peak systolic flow velocity (58 ± 11 vs. 48 ± 11 cm/s). To allow comparisons between volunteers, relative flow velocity was calculated in relation to baseline. Repeated measures analysis of variance revealed significant difference between the relative flow velocity time courses during hyper- and normoventilation ($p < 0.001$). The maximum changes of visually evoked relative flow velocities were $26\pm 7\%$ and $12\pm 5\%$ during normoventilation and HV ($p < 0.01$), respectively. VEPs did not differ during control and HV phases.

Conclusions: The significantly lower visually evoked flow velocity changes but preserved VEP during HV suggested that hypocapnia induced vasoconstriction significantly inhibited the neuronal activity evoked flow response. Potential vascular effects of HV should not be ignored in patients with advanced stenotic lesions of the brain supplying arteries, because decreased CBF, impaired vasodilation of resistance vessels, and attenuated cortical activity induced flow response, caused by HV, may increase the risk of cerebral ischemia in hemodynamically compromised patients.