A Novel Intraoperative Method for Monitoring Hyperacute Changes in Cerebral Ischemia
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INTRODUCTION: We have previously reported cerebral hyperexcitability in rats through excitatory-inhibitory imbalance after 1h of ischemia (Fujioka et al. Stroke 35: e346-, 2004). However, it was unclear whether such an imbalance is induced in the hyperacute stage of ischemia in humans. We addressed the issue in patients undergoing carotid endarterectomy (CEA), wherein cerebral ischemia is induced by temporary carotid occlusion.

Methods: Informed consent was obtained from all patients with indication of CEA (symptomatic patients with >70%stenosis; n = 6, mean age; 72.3 years). Routine shunting was used, and temporary occlusion was applied twice (cf. occlusion (1) and (2) in Figures 1 and 2). Short-term somatosensory evoked potentials (SSEPs) were evoked by paired-pulse stimuli with an inter-stimulus interval of 30 ms, and each peak-to-peak amplitude was measured by using the N20/P25 complex. The excitability index Q was calculated by the amplitude of the second stimulus divided by the first. Near-infrared spectroscopy monitoring was also used to monitor regional cerebral saturation (rSO2).

Results: Compared to each pre- and post-occlusion period, Q values significantly increased during the second occlusion and reversibly decreased when the occlusion was released (Tukey’s post hoc tests; **p < 0.01; Figure 1); however, occlusion did not induce significant changes in amplitudes by traditional single-pulse SSEPs (p = 0.188 by repeated-measures analysis of variance; Figure 2). Asymptomatic cerebral infarct was post-operatively recognized in 1 patient with an increased Q value (Q = 1.1) and a substantial 46.9% reduction of single-pulse SSEP amplitude. Occlusion did not induce significant changes in rSO2.

CONCLUSION: We demonstrated hyperacute ischemia-induced hyperexcitability for the first time in humans. Although single-pulse SSEPs were useful in detecting ischemia, paired-pulse SSEPs proved more reliable in detecting ischemia-induced neuronal imbalance, suggesting that it could be a prognostic monitoring tool for CEA and other ischemia-related cerebrovascular diseases.