INFLUENCE OF CORTICAL INACTIVATION ON TRAINING ENHANCED VISUAL RESPONSES IN THE RAT'S SUPERIOR COLLICULUS

Kordecka K, Foik A, Wierzbicka A, Waleszczyk W
Nencki Institute of Experimental Biology, Polish Academy of Science, Department of Neurophysiology, Warsaw, Poland University of Warsaw, Faculty of Physics, Warsaw, Poland

The possibility of inducing neuronal plasticity at the cortical level is generally acknowledged. In our previous study we observed that few hours of visual training enhanced responses both at the cortical and subcortical level. The reinforcement of responses at the subcortical level, in the superior colliculus (SC), can occur through descending projection from the visual cortex (VCx). Therefore, in the current study we have attempted to examine how inactivation of the VCx affects tectal responses after visual training.
Experiments were performed on anesthetized rats exposed to flashing white-light-emitting diodes placed 10 cm in front of them. Monocular visual stimulation consisted of series of 300 repetitions of light flashes with 2 s intervals, presented every 15 minutes through 3 hours. Visual evoked potentials (VEPs) were recorded using multichannel linear electrode arrays from the primary VCx and the SC, contralateral to the stimulated eye. In order to block the activity of the cortex, after 3-hour visual stimulations a well above the contralateral VCx was fulfilled with xylocaine solution (2.5%). During cortical inactivation a single series of visual stimulation (300 stimulus repetitions) was presented. The SC VEP amplitudes were offline analyzed and peak-to-peak VEP amplitude was taken as a measure of response magnitude.
We observed that xylocaine used for neuronal inactivation resulted in a strong attenuation of cortical VEP amplitudes. However, cortical inactivation did not cause any significant difference in SC VEP amplitudes. Collicular VEPs were at the high level and significantly differed from control recording at the beginning of training. This indicates a minor impact of the VCx on response enhancement in the SC. As the VEP amplitudes in SC didn't show any decline after cortical inactivation, we conclude that the increase of responses in SC after visual training is most likely a result of the enhancement of the retinal input to the SC.

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