ISCHEMIC RESPONSES IN THE RAT BARREL CORTEX IN VITRO AT DIFFERENT POSTNATAL AGES

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Anoxic depolarization (AD) is a hallmark of ischemic brain damage. In slices of the rat barrel cortex ischemia-like conditions induced by oxygen-glucose deprivation (OGD) evoke AD which manifests as a negative LFP shift and an increase in light transmittanceand resembles spreading depression (SD).AD typically initiated in one or more barrels and further spread across the entire slice with a preferential propagation through L4.

In the present study using simultaneous extracellular local field potential (LFP), optical intrinsic signal (OIS) and whole-cell recordings, we aimed to explorethe OGD-induced AD in slices of the ratbarrel cortex at different postnatal ages (P2-23).We found that OGD-induced ischemic response was not only delayed but also was qualitatively different in the neonatal (P2-6) rats. Ischemic response started with SD-like negative LFP shift associated with transient (~2 min) membrane depolarization of ~20 mV at a single-cell level and transient increase in transparency. Transition from SD to AD was characterized by complete but relatively slowly developing neuronal depolarization ~8 min after SD without any prominent extracellular LFP signal. Delayed AD was also associated with the second wave of transparency increase during OIS imaging. Thus, in contrast to adolescent (>P10) barrel cortex where SD and AD are united, these two processes are dissociated in time in the neonatal rats.

We hypothesize that this developmental differences in the ischemic response involves lower density of voltage-gated channels and synaptic connections, larger extracellular space and lower metabolic demand of immature neurons.

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THE SPECIFICITY OF THE REACTION OF THE CARDIOVASCULAR SYSTEM OF FIRST GRADERS TO PHYSICAL LOAD AT THE BEGINNING AND END OF THE ACADEMIC YEAR

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The study was run on the cardiovascular system in boys 8 years, enrolled in the 1st grade of a public school, in a state of relative rest, after a dosed dynamic load at