MULTIMODALITY SOMATOSENSORY NEURONAL RESPONSES IN HYPOXIC-ISCHEMIC BRAIN INJURY

by
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Abstract

The hypoxic-ischemic (HI) brain injury is a major complication after cardiac arrest (CA), which may lead to long-term neurological deficits. This thesis has been focused on the loss and regain of somatosensory functions during the injury and recovery periods, using electrophysiological approaches. The somatosensory system is of particular interest in this study because that 1) the somatosensory evoked potential (SSEP) is a most commonly used marker for neuro-monitoring and neuro-diagnosis in clinical settings, and 2) the primary sensory neocortex is known to be preferentially affected by HI insults.

The study was conducted in a well established rodent model of asphyxial CA. In the first half of the thesis, various aspects of the SSEP signal were studies with gradient CA injuries. We used an innovative method to separate the early and late SSEPs which improved the detection of two components. The late SSEP has been emphasized and is shown to have important prognostication values. The high-frequency oscillation of the SSEP was presented with advanced time-frequency transformation methods to explore its physiological relation to subcortical inputs.

In order to understand the underlying neuronal activities that give rise to the SSEP, multiunit activities (MUA) and local field potentials (LFP) from the somatosensory cortex were studied in the second half of the thesis. An interesting pattern of triphasic neuronal discharge in response to somatosensory stimulus was discovered, which contained the short- and long-latency responses (SLR and LLR). The LLR was shown to be more vulnerable to HI injury, which disappeared earlier and recovery later than the SLR. Mild hypothermia as one of the most effective therapeutic interventions was applied.

It was found that the hypothermia selectively potentiated the SLR but suppressed the LLR. We hypothesized that LLR reflected the cortical network activity and it was reduced as a strategy of hypothermic protection. Furthermore, the stereotypical neuronal response was parameterized using the generalized linear model, by which we extracted useful information about the structure of the spike train and the coupling between spike and LFP.

At last, to learn about the subcortical activity which drives the cortical response, a preliminary study about thalamocortical interaction during HI injury was presented. The directional information flow and causal coherence were analyzed. The results indicated that thalamus may play a leading role in arousal in an ischemic brain.

In summary, this thesis study looked into the sequential changes of somatosensory neuronal activity in response to HI injury. We hope to understand the vulnerability of the cortical and subcortical structures and their roles in recovery, so that we may able to improve therapeutic interventions, such as better administration of hypothermia.

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