NEUROPHYSIOLOGICAL RESPONSE TO HYPOXIC-ISCHEMIC INJURY
AND MODERATE HYPOTHERMIA

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A thesis submitted to Johns Hopkins University in conformity with
the requirements for the degree of Master of Science in Engineering

Baltimore, Maryland
May, 2010

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Abstract

Poor neurological outcomes are the major cause of morbidity in cardiac arrest (CA) survivors. With a shift in paradigm towards managing CA survivors at an early stage, it is becoming imperative to develop objective electrophysiologic measures for early assessment, because functional evaluation of comatose patients is impractical. This study presents a morphological tool for the quantification of somatosensory evoked potentials (SSEPs), and assesses its prognostic ability in a well-validated rodent model of asphyxial CA. Further, a potential thalamic/brainstem component of the cortical SSEPs is identified and correlated with corresponding functional scores.

Since hypothermia is the most successful therapeutic intervention for CA survivors, this work also explores the effect of temperature modulation on spontaneous and evoked neural activity at clinically relevant temperatures in isoflurane-anesthetized rats with no neural injury. It was observed that hypothermia acts synergistically with isoflurane to tone down basal cortical firing, leading to disinhibition of deep-brain nuclei, and resulting in suppressed EEGs but amplified cortical SSEPs.

To gain a deeper understanding of the neural basis of recovery after hypoxic-ischemia, the final component of this work was a preliminary study done using microelectrodes implanted in the cortex and the ventroposterolateral thalamus. It was observed that the thalamic activity disappeared a few seconds earlier than the cortex after CA and also recovered earlier post-resuscitation. Thalamocortical coupling was also observed during EEG burst-suppression during early recovery. These findings set the stage for a comprehensive study of neural spikes in regions of the brain believed to be important in arousal from ischemic coma.