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EFFCTS OF CEREBRAL ISCHEMIA IN STRIATUM AND SOMATOSENSORY CORTEX [1]

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Effects of Cerebral Ischemia in Striatum and Somatosensory Cortex

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Background

Stroke results from an acute occlusion in a cerebral blood vessel, triggering different kinds of lesions and complex sequence of events that results in cell death (Morais et al, 2005). Consequences of a cerebral ischemic are dependent of the seriousness and duration of blood flow reduction. Brain injury and neuronal death necessitate at least 1 to 2 minutes of focal vascular occlusion, so a less serious, but prolonged ischemia can produce damages as a short and serious

ischemia (Morais et al. 2005). The planning and control of motor behaviors is a complex neural process in part dependent of multiple sensory modalities from the body periphery (somatosensation) and external environment (Hummelseim et al. 1988; Riemann and Lephart, 2002. Accordingly, emerging evidence suggests abnormal processing of somatosensory information by the primary somatosensory cortex (S1) contributes to deficits seen in neurological disorders typically classified by motor dysfunction (stroke, parkinson"s disease, dystonia, ataxia etc) (Wolpert et al. 2013; Borich et al. 2015).

The aim of the study was to verify cellular alterations in neurons of the somatosensory cortex after 48 hours of focal ischemia in mice.

Methods

A total of 12 C57/BL6 male mice at 10 weeks weighing 25-30g, were used in this model. Animals were divided into the following experimental groups: Group 1 (control *n*=6), including mice without middle cerebral artery occlusion, and Group 2 (ischemic, *n*=6), including mice induced of distal middle cerebral artery occlusion. After 48 hours occlusion the animals were anesthetized with Ketamine following intracardiac perfusion with saline solution 0,9% and paraformaldehyde solution 4% in phosphate buffer, 0.1M, pH 7.2. The brains were removed and fixed for 48 hours, for histological testing and sectioned coronally at 5 µm. The samples of cerebral tissue were deparaffinized, hydrated, submitted to Leishman and Hematoxylin-Eosin staining. Experiments were performed in accordance with the guidelines by COBEA and were approved in University of Santa Catarina Animal Care Committee (CEUA PP00943).

Results

The model of artery occlusion of distal portion middle cerebral artery produced loss of cellular architecture neuronal with nuclear swelling, nuclear fragmentation and neurons with moderate cytoplasmic and nuclear condensation in the secondary somatosensory cortex, somatosensory primary and caudate putamen (striatum).

Conclusion

In conclusion, neurons of the primary and secondary somatosensory cortex and striatum were susceptible in focal brain ischemia model and can be applied in the experimental conditions of the neuronal injury and brain edema in C57/BL6 mice.