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EARLY RESPONSE OF INFLAMMATORY PROTEINS IN THE STRIATUM AND HIPPOCAMPUS OF NEWBORN PIGLETS FOLLOWING CARDIOPULMONARY BYPASS AND CIRCULATORY ARREST

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Background and aims: Inflammation plays a key role in ischemic brain injury. We examined the levels of select inflammatory proteins in the striatum and hippocampus of newborn piglets in a cardiopulmonary bypass (CPB) and deep hypothermic circulatory arrest (DHCA) ischemia model.

Methods: Piglets were placed on CPB, cooled to 18^{o}C, subjected to 30min of DHCA and 1hr of low-flow (20ml/kg/min), rewarmed to 37°C, separated from CPB, monitored for 2hrs and then striatum and hippocampus were isolated for protein analysis. Protein levels are presented in arbitrary units (mean±SE).

Results:

Striatum: CPB/DHCA increased, as compared with sham operated animals, pro-inflammatory cytokines implicated in ischemic brain injury: interleukin 1 (365±12.7 vs 583±83, $p< 0.047$) and tumor necrosis factor-alpha (318±37 vs 402±18, $p< 0.05$); chemotactic cytokines: growth regulated protein (GRO) (226±16 vs 376±42, $p< 0.02$), GRO-alpha (178±16 vs 297±17, $p< 0.001$) and interleukin-8 (591±47 vs 728±40, $p< 0.05$); chemokines: macrophage inflammatory protein-3 (58±8 vs 88±7, $p< 0.05$) and eotaxin (107±7 vs 164±17, $p< 0.02$). Vascular endothelial growth factor, potentially protective, was decreased (25±5 vs 11±3, $p< 0.05$).

Hippocampus: There were no significant differences in the above proteins after CBP/DHCA. However, potentially protective proteins, interleukin-10 (338±18 vs 405±5, $p< 0.02$) and transforming growth factor beta (389±24 vs 460±16, $p< 0.05$), were increased.

Conclusions: In piglet model of CBP/DHCA, early response of proteins regulating inflammation in the brain is region-dependent: proteins exacerbating ischemic injury are increased in the striatum, whereas those with potential protective role are increased in the hippocampus.

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