Effect of partial brain ischemia on the metabolic and hemodynamic responses to hemorrhage hypotension measured in the brain and small intestine

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Introduction: During hemorrhage blood is redistributed in favor of the vital organs and on the expense of the less vital organs. Bilateral carotid occlusion (BCO) is an animal model of arteriosclerosis, which is considered to be the leading cause of mortality due to reduced blood supply to the brain, in industrialized countries .

Objectives: The purpose of the present study was to investigate how BCO influences the responses of the brain (vital organ) and small intestine (less vital organ) to hemorrhagic hypotension.

Methods: Rats were bled until mean arterial pressure (MAP) of 40mmHg was achived, with or without the induction of BCO 24 hours prior to the bleeding session. MAP level was maintained for 15 minutes, after which the animals were resuscitated with the withdrawn blood. Metabolic and hemodynamic monitoring from both organs were carried out using the Multi-Site Multi-Parametric system, which simultaneously monitors tissue blood flow using laser Doppler flowmeter and mitochondrial NADH redox state using surface fluorometery.

Results: While hemorrhage under normoxic conditions caused a decrease in blood supply (30±7%, $p<0.01$) to the intestine, and mitochondrial dysfunction (132±10% ($p<0.01$), the brain preserved its normal function. However, under partial ischemic conditions hemorrhage caused deterioration in both organs. Blood supply to both brain and intestine rapidly decreased and remained low through the entire hemorrhage period (79.5±8% ($p<0.001$) and 56±10% ($p<0.001$), respectively). In addition, mitochondrial dysfunction was observed in both brain (137±9%, $p<0.01$) and intestine (145±12%, $p<0.01$). Furthermore, the responses of CBF to hypotension, exhibited an event called Ischemic-Depolarization (ID) revealed by a vasoconstriction of small vessels in the cortex.

Conclusions: The Impaired blood supply to the brain decreased cerebral autoregulation abilities and therefore decreased its protection during hemorrhage. The ID demonstrated the severity of the ischemic damage to normal mitochondrial function under combination of partial ischemia and hemorrhagic hypotension. These results emphasis the importance of adequate cerebral perfusion for the maintenance of body homeostasis.