Neuroprotective effects of nasopharyngeal perfluorochemical cooling in a rat model of subarachnoid hemorrhage

Yavuz Samanci MD; Gennaro Calendo; Sandy T Baker; Kadir Erkmen MD; Micheal W Weaver; Marla R Wolfson

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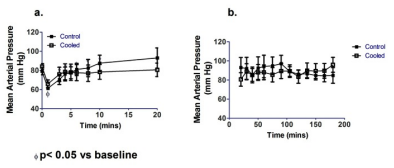
Introduction

Subarachnoid hemorrhage (SAH) results in severe neural damage and hypothermia is known to have neuroprotective effect in ischemic injuries. The aim of this study was to determine whether preferential cerebral hypothermia by nasopharyngeal perfluorochemical (NP-PFC) cooling could be utilized in a rat model of SAH model for neuroprotection.

Methods

SAH was induced in 16 male Sprague -Dawley rats by cisterna magna injection. Brain cooling was performed using NP-PFC method starting from post-SAH 20-minute. Physiological data, CBF values and brain tissues were analyzed.

Figure 1

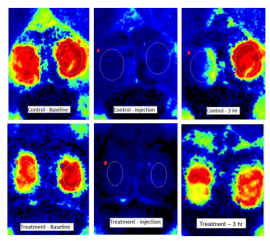


The mean arterial blood pressure responses to injury and treatment

Results

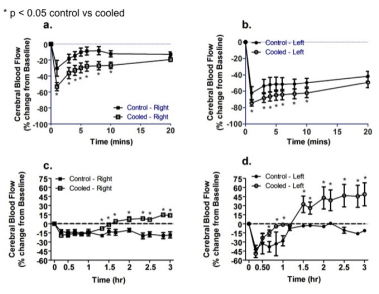
SAH caused an immediate decrease in MAP (17.0 ± 4.90% below baseline). MAP then rapidly approached baseline values (95%) within 4 minutes. SAH induction caused a significant and rapid fall in CBF from baseline (approximately -65%) in both hemispheres, independent of study group. The decrease was significantly greater in the left. By 20 minutes post-injury,CBF in the right hemisphere recovered to within 25% of baseline,while CBF in the left hemisphere remained depressed,independent of group (approximately -60% of baseline). Following initiation of cooling,while there was little change in CBF in the right hemisphere,there was a significant but small increase in CBF in the cooled animals resulting in recovery of CBF in the right hemisphere to baseline values. Right hemisphere CBF remained below baseline in the non-treated animals. In the left hemisphere,cooling facilitated the return of CBF to baseline values within 20 minutes of treatment with further increase in CBF that stabilized by the 2-hour post -injury timepoint. Immunohistochemistry demonstrated significantly greater number of NeuN positive cells in the cortex,significantly fewer IBA-1 positive microglia and GFAP positive astrocytes cells in both cortex and hippocampus in the cooled animals,reflecting preserved neuronal integrity and reduced inflammation.

Figure 2



Representative LASCA images reflecting cerebral blood flow

Figure 3



The CBF profiles over time summarizing CBF responses to injury and

treatment

Conclusions

The data from this study suggest that local hypothermia by NP-PFC cooling is a promising neuroprotective approach to support return of CBF and to suppress inflammatory response in SAH.

Learning Objectives

By the conclusion of this session, participants should be able to:

1) Describe the importance of NP-PFC cooling in neuroprotection in SAH,

2) Identify a possible effective treatment for neural damage in SAH

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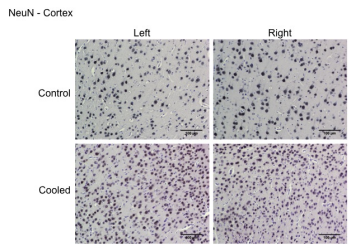
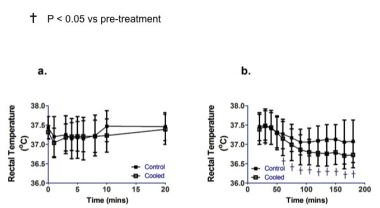
Figure 5



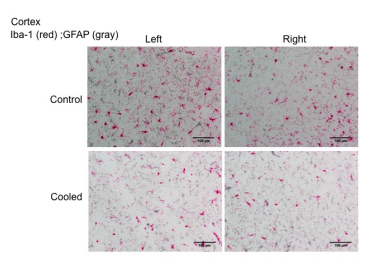
Histomicrographs showing NeuN positive cells in the cortex

Figure 4



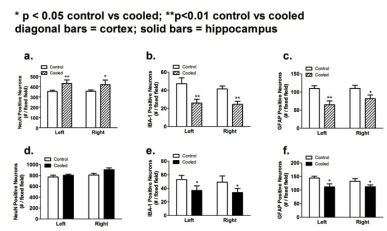
Core temperatures throughout the SAH procedure

Figure 6



Histomicrographs showing Iba-1 positive microglia and GFAP positive astrocytes cells in cortex

Figure 7



Quantitative analyses of the number of NeuN positive, IBA-1 positive, and GFAP positive cells in the cortex and hippocampus