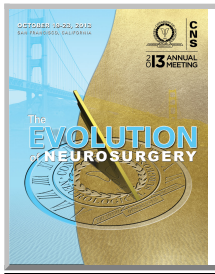

Induced Hypercapnia Enhances CBF and Cerebral Oxygen Saturation Before, During and After Cerebral Vasospasm after Aneurysmal SAH.

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Introduction

In spite of advanced monitoring tools and pharmacological and endovascular treatment options, the occurrence of secondary ischemic deficits and infarctions after aneurysmal subarachnoid hemorrhage (SAH) is still an unsolved problem. This study was conducted to evaluate if the physiological mechanism of pCO2-reactivity is still intact after SAH and if it can be therapeutically used.

Methods

8 patients were included if their clinical state was Hunt/Hess grade 3 or worse, the initial CT-scan showed SAH Fisher grade 3, and if the termination of analgosedation and extubation was not possible at day 4 after SAH and after occlusion of the aneurysm. Starting at a baseline level, pCO2 was decreased to 30 mmHg and then gradually increased to 40, 50, and 60 mmHg by modulation of respirator parameters during mechanical ventilation, respectively. At each level, CBF was measured by an intracerebral thermodilution probe, cerebral tissue oxygenation by near infrared spectroscopy (NIRS), and flow velocities in the cervical internal carotid artery and in the intracranial vessels by transcranial Doppler sonography (TCD).

Results

By continuous monitoring of peripheral oxygen saturation and measurement of arterial blood gases every 5 minutes, a decrease of arterial oxygen partial pressure could be ruled out. ICP decreased during hyperventilation. During hypercapnia, the elevation of ICP was prevented by an increased CSF-outflow. CBF and cerebral oxygen saturation both increased by 20 % during the elevation of arterial pCO2 from baseline (40 mmHg) to 60 mmHg on day 4, 8 and 12 after SAH, respectively. Flow velocities in TCD measurements increased equally in the cervical internal carotid artery (ICA) and the intracranial vessel trunks indicating a global increase of CBF by controlled iatrogenic hypercapnia.

Conclusions

The physiological mechanism of pCO2-regulation of CBF is intact at all stages after aneurysmal SAH. Hypercapnia enhances CBF and cerebral tissue oxygenation and, thus, may be used therapeutically to prevent secondary ischemic events after SAH.

Learning Objectives

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

1) Describe the importance of preventing secondary ischemic events after aneurymsal SAH and the correlation of induced hypercapnia and increased CBF and cerebral oxygen saturation

2) Discuss, in small groups, the role of iatrogenic induced hypercapnia in treatment of vasospasm and prevention of secondary ischemic events after aneurysmal SAH

3) Identify an effective and low-priced treatment of vasospasm and secondary ischemic events after aneurysmal SAH with few negativ side effects

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