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Oral Presentation

Cerebroprotection in Severe Brain Injury

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Abstract

Formula one legend Michael Schumacher hit his head severely while skiing on December 29, 2013. He was operated on for brain decompression and left in a coma fighting for life. In such severe brain injury with cerebral contusion and hemorrhage following head trauma, refractory to medical therapy and with increasing cerebral oedema and intracranial pressure, critically low cerebral perfusion can cause anaerobic cerebral metabolism with secondary severe brain lesions and even death. The prevention of primary brain lesions is only possible by accident prevention measures. Prevention of secondary lesions is a medical challenge. Many medications with suspected cerebroprotective properties show an effect in animal experimental studies but not in human studies. Up to now, the only promising methods in severe cerebral energy crises seem to be hypothermia and/or barbiturate administration. Additionally, clinical and experimental studies showed a close relation between the amount of hypoxic intervals and secondary injury of neural tissue (1), where the application of hyperbaric oxygen (HBO) is the only way to improve oxygen supply to the tissues with higher amounts of oxygen freely dissolved in the plasma. So we examined these methods in experimental studies and clinical application. In experimental studies we used a hypoxic standardized model of brain slices to examine the effect of hypothermia. In clinical cases with critical perfusion pressure below 50 mmHg and severe brain oedema we performed active body cooling with lowering body temperature from 38.2 °C to 35 °C. In 60 patients with reduced cerebral perfusion we administered the barbiturate methohexital and measured lactate concentrations in the blood taken from the appropriate internal jugular vein. EEG analysis was used to reveal electrical brain activity and burst suppression pattern while administrating barbiturates. Ninety-nine patients with midbrain syndrome after severe head injury were randomized. All patients received comparable monitoring and intensive care, while every second patient was additionally subjected to a series of HBO treatment. Neurological follow-up and EEG during and after HBO were registered. Our experimental studies confirmed the cerebroprotective effect of hypothermia in vitro and in vivo in cerebral energy crises, but in contrast a lack of protective effect once hypoxia had occurred under normothermic conditions. In patients with critically low cerebral perfusion, hypothermia is able to improve the clinical outcome. The administration of barbiturates in patients with reduced cerebral perfusion showed a normalization of increased cerebrovenous lactate concentrations with correlation to burst the suppression time. The survival time of brain injured patients under HBO was distinctively longer and the survival rate significantly higher (group B patients). At the end of the study, 74% of the patients in group A. Were dead or apallic as compared to 53% in group B. Complete recovery occurred in only 6% in group A and 33% in group B. In the situation of cerebral energy crises it is a challenge to avoid secondary ischemic brain lesions. Our investigations showed that administration of hypothermia and barbiturates reduces cerebral metabolism and glycolysis with an improved outcome in humans. However, this treatment needs to be administered as soon as possible after reduced cerebral perfusion occurs. As another or additional treatment HBO may be discussed to improve oxygenation of ischemic hypoxic brain tissue in reduced cerebral perfusion. Further investigations and randomized studies should be performed to discuss the best treatment options for patients with a cerebral energy crisis following severe head injury.

Keywords: Brain Injury, Brain Oedema, HBO.

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