



Hypothermia for Acute Brain Damage: Pathomechanism and Practical Aspects

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The International Brain Hypothermia Symposium 2004 was the second time I have had the honor of opening such a gathering on brain hypothermia treatment. It was a great pleasure to greet the participants in the hope that their valuable contributions would make the Tokyo meeting memorable. Brain hypothermia has long been seen as a promising method that may overcome current limitations on brain resuscitation in patients with severe brain damage. However, although excellent results have been obtained in experimental animal models, for some reason brain hypothermia has not always been successful clinically, and resolving this problem has been a major challenge facing physicians specializing in brain therapies. The ICU management of recent research has uncovered new mechanisms of brain damage not seen in animal models, including brain thermopooling at temperatures above 40 °C in severe brain damage, masking neuronal hypoxia even with normal cerebral blood flow. Stress-related hyperglycemia with brain hypothermia was expected to generate useful results in patients with external injuries, cerebral occlusive stroke, and cardiac arrest. In recent clinical studies of brain hypothermia treatment, many excellent results began being reported on the management of severe brain injury, ischemic stroke, and post-resuscitation after cardiac arrest. However, in clinical brain hypothermia treatment many questions remained about appropriate treatment targets, management technique, prevention of complications, control of brain tissue temperature, management of hypothermia insult, and mechanisms underlying the onset of vegetative states.

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