

Therapeutic hypothermia

see [Hypothermia in acute ischemic stroke](#).

Targeted temperature management (TTM), or therapeutic hypothermia, is one of the most potent [neuroprotection](#) approaches after ischemic and traumatic brain injuries. TTM has been applied clinically with various methods, but effective achievement and maintenance of the target temperature remain challenging. Furthermore, timing of cooling and target body and brain temperature to optimize effectiveness for neuroprotection and to minimize side effects are yet to be standardized. Focal brain cooling is a potential strategy to minimize adverse effects of systemic TTM.

In a study, Tauchi et al. report on a focal brain cooling device for animals and its effectiveness of focal cooling in several animal models of ischemic cerebral stroke. A focal brain cooling device was constructed using a Peltier's element, a thermoelectric heat pump. The device was validated for its cooling ability, and optimal settings to induce an effective intracranial temperature were determined using male Sprague-Dawley rats. Transient and permanent middle cerebral artery occlusions were experimentally induced, and focal brain cooling was applied using the device varying the timing and duration of cooling. The stroke-induced infarct and edema volumes were evaluated from Nissl-stained cryosections. The focal brain cooling device was able to decrease and subsequently maintained cerebral hypothermia in free-moving rats without altering the core temperature. The device with validated intracranial temperatures produced neuroprotective effects in the acute phase of ischemic neural death, [reperfusion](#) injury, progressing damage to the penumbra, and edema formation. In conclusion, the validated focal cooling device enabled rapid and accurate cerebral TTM in rats. Using this device, they were able to test the neuroprotective effect of focal TTM in several pathological stages of [cerebral ischemia](#), which warrants further studies to develop clinically feasible TTM procedures for patients with cerebral stroke ¹⁾.

[Hypothermia](#) is well recognized to preserve cells and tissue in the face of metabolic challenge. Evidence supports the administration of hypothermia as standard of care for [neuroprotection](#) after cardiac arrest from acute coronary syndromes.

There has been long-standing interest in applying hypothermia to reduce the tissue damage associated with central nervous system trauma; however, benefit cannot be presumed. In addition to suggested neuroprotective effects, hypothermia is well known for its ability to reduce intracranial pressure. However, hypothermia bears risks, including coagulopathy and immunosuppression, and profound hypothermia bears the additional risk of cardiac dysrhythmia and death.

see [Prophylactic Hypothermia for severe traumatic brain injury](#)

In contrast to previous reviews, a systematic review found some evidence to suggest that therapeutic hypothermia may be of benefit in the treatment of traumatic brain injury. The majority of trials were of low quality, with unclear allocation concealment. Low quality trials may overestimate the effectiveness of hypothermia treatment versus standard care. There remains a need for more, high quality, randomised control trials of therapeutic hypothermia after traumatic brain injury ²⁾.

More robust clinical trials have shown a trend toward worse outcomes with hypothermia or were

stopped early because of a low probability of demonstrating superiority ^{3) 4) 5) 6)}.

Studies

see [Therapeutic hypothermia studies](#).

Complications

TBI patients may suffer from hypothermia-induced pulmonary and coagulation side effects, from side effects of vasopressors when re-establishing the hypothermia-induced lowered blood pressure, and from a rebound increase in [intracranial pressure](#) (ICP) during and after rewarming. The difference between body temperature and temperature set by the biological thermostat may cause stress-induced worsening of the circulation and oxygenation in injured areas of the brain. These mechanisms may counteract neuroprotective effects of therapeutic hypothermia. It is a lack scientific support as a first-tier therapy for the use of therapeutic hypothermia in TBI patients for both adults and children, but it may still be an option as a second-tier therapy for refractory intracranial hypertension ⁷⁾.

Children suffered TBI may not be able to benefit from therapeutic hypothermia, it may increase risk of fatality and arrhythmia. There is no evidence that therapeutic hypothermia improves prognosis of children with TBI, and no evidence that therapeutic hypothermia raise pneumonia and coagulation dysfunction. Limited by the quality of the included studies, the results need to be treated with caution. Further large-scale, well-designed RCTs on this topic are still needed ⁸⁾.

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