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## Advances in Post-Resuscitation Care: the Role of Therapeutic Hypothermia

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**ABSTRACT**

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Mild therapeutic hypothermia (32°C – 34°C) is the only therapy that improved neurological outcome after cardiac arrest in randomized, controlled trials. It protects the brain after ischemia by reduction of brain metabolism, attenuation of reactive oxygen species formation, inhibition of excitatory amino acid release, attenuation of the immune response during reperfusion and inhibition of apoptosis. Its use is recommended by the American Heart Association and the International Liaison Committee on Resuscitation for unconscious adult patients with spontaneous circulation after out-of-hospital ventricular fibrillation cardiac arrest, 12 to 24 hrs following resuscitation. The role of therapeutic hypothermia is uncertain when the initial cardiac rhythm is asystole or pulseless electrical activity, or when the cardiac arrest is primarily due to a noncardiac cause, such as asphyxia or drug overdose. Therefore, in patients with anoxic brain injury after nonventricular fibrillation cardiac arrest, clinicians will need to balance the possible benefit of therapeutic hypothermia with the possible side effects of this therapy.

Mild hypothermia is a safe and effective therapy after cardiac arrest, even in hemodynamically compromised patients and in patients undergoing percutaneous coronary intervention. Because the induction of therapeutic hypothermia has become more feasible with the development of simple intravenous cooling techniques and specialized equipment for improved temperature control in the critical care unit, it is expected that therapeutic hypothermia will become more widely used in the management of anoxic neurological injury whatever the presenting cardiac rhythm. Potential side effects have to be kept in mind and treated accordingly.

**KEY WORDS:** *hypothermia; therapeutic hypothermia; cardiac arrest; cooling; resuscitation*

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