

Effect of Mild Hypothermia on Neurological Outcome in Patients After Out of Hospital Resuscitation.

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Background: Cardiac arrest (CA) followed by no-flow period and global hypoperfusion state after restoration of spontaneous circulation (ROSC) cause cerebral ischemia and leads to severe and potentially irreversible neurologic damage. We describe our initial experience on the neurological outcome of mild therapeutic hypothermia in patients after out of hospital CA.

Methods: Inclusion criteria were a witnessed CA, VT/VF as the presenting rhythm, age 18 to 75 years. Patients with prolonged (>30 minutes) resuscitation or hypotension, temperature less than 35 ° C, poisoning or terminal illness were excluded. The cooling protocol included: induction phase by combined intravenous 4° C fluids with external cooling (CritiCool System, MTRE) and 24 hours maintenance phase supported only by the external system. The temperature goal was 32-34° C. The last phase, controlled re-warming, programmed at rate of 0.5-1°C per hour. To prevent shivering, paralysis was induced by intravenous tubocurarine added to the sedation archived by propofol. The temperature monitoring was made with an external skin and endotracheal probe.

Results: In the past four months 3 patients were eligible to be treated with mild systemic hypothermia. The first patient was 21 year old lady presenting with CA in a public bus. Bystander resuscitation initiated, and after 18 minutes VF was recognized and successfully converted by electrical shock (DC). The second patient was 64 year old man, his son started the chest compression and after 15 minutes, VF was successfully converted to NSR after DC. Both patients were in deep coma on admission, with objective evidence of anoxic brain damage. The mean time to reach the therapeutic temperature was 4-6 hours. In these two cases after the re-warming phase the patients were awake, without clinical neurological impairment at time of discharge. In a third recent patient, therapy failed to alter the initial neurological state.

Conclusion: In appropriate cases of CA, controlled mild therapeutic hypothermia may attenuate neurological damage as result of prolonged anoxic state. Further experience is needed to assess this promising technique.