Therapeutic hypothermia in the year 2010: it is about time!

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The use of therapeutic hypothermia (TH) in clinical medicine is no longer a rarity. Since the modern inception of this technique by Fay in the 1940s, TH has been used for a variety of clinical scenarios. (1,2) TH has gained significant popularity as a brain-protection strategy in victims of sudden cardiac death in whom return of spontaneous circulation (ROSC) has been obtained with coma. (3) Nonetheless, many trials and case series have shown the advantageous effects of lowering the body’s core temperature in a variety of other clinical conditions including near-drowning, hypoxemic brain injury, traumatic brain injury, traumatic cardiac arrest, stroke, newborn hypoxic-ischemic encephalopathy, hepatic encephalopathy, bacterial meningitis, congestive heart failure, postoperative neonatal tachycardia, and the acute respiratory distress syndrome. (2,3) However, despite the well documented evidence of its therapeutic value, and the ease of implementation, TH is not used as much as the authors would envision for such a cardiopulmonary and cerebral cornerstone of advanced cardiac life support. (4)

In this issue of Critical Care and Shock, two articles present encouraging data on the use of this therapeutic intervention in the context of cardiovascular conditions in two different countries. Palo and associates, report on their experience in a tertiary care center in the Philippines, utilizing TH within 6 hours of a cardiac arrest with successful return of spontaneous circulation (ROSC) and coma. (5) These investigators found a direct correlation between neurological outcome and the implementation of TH.

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The neurological outcomes presented in the study by Palo and coworkers mirrors those previously reported by others. (2,3) The alleged protective effects of TH in the context of a cardiac arrest, have traditionally been attributed to a reduction of metabolic rate. (6) It is well known that the cerebral metabolism (estimates of oxygen consumption, glucose utilization, and lactate concentration) is temperature-dependent, and hypothermia has been shown to reduce cerebral metabolism by decreasing all of these parameters. At the cellular level, TH protects the cell wall and maintains the lipoprotein membrane integrity. (7) At the tissue level, TH improves oxygen supply to areas of ischemic brain and decreases intracranial pressure. (2)

In another report across the Pacific, in Mexico, González-Chon and coauthors report an interesting case of an individual with refractory cardiogenic shock that underwent TH for 34 hours with significant hemodynamic improvement. (8) As noted by these authors, TH was attempted as a last effort, in order to decrease the myocardial metabolic rate and to allow time for the stunned myocardium to recover. Indeed, in this case there was a dramatic recovery of the left ventricular ejection fraction following the application of TH.

Mild TH is known to decrease the heart rate and increase the systemic vascular resistance. (3) In addition, TH maintains stroke volume and mean arterial blood pressure. In a Scandinavian study in 2007, Hovdenes and associates reported the use of TH for out-of-hospital cardiac arrest victims with ROSC and cardiogenic shock. (9) In their series, TH was justified even in those patients with significant hemodynamic compromise requiring percutaneous coronary...
interventions and intra-aortic balloon counterpulsation. This technique provides neuroprotection against ischemic neuronal injury. In addition to being useful in cardiac arrest situations, TH has proven useful in situations such as cardiogenic shock and is likely to be widely used in this context.

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References