

ing their results into a process, likely with advanced technology to sense the upstroke phase of CPR and to couple it with delivery of electrical energy, opens the door for additional integrative research.

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## Can an ice cream headache save your life?\*

**W**e have all experienced an "ice cream headache" (aka, "brain freeze" or cold-stimulus headache) and would rather avoid another. But could it save a life?

The idea of cooling the brain to protect it from ischemia goes back to the 1950s. Originally, the thinking was that moderate hypothermia (28°C-32°C) was needed. This level of hypothermia was fraught with complications, including arrhythmias, infections, and electrolyte abnormalities. Consequently, the clinical use of hypothermia for resuscitation became dormant, although it became standard for protection during planned ischemia, such as cardiac or neurosurgical procedures.

In the early 1990s, animal studies suggested that small changes in brain temperature could make a significant difference after brain ischemia. Since the

completion of successful clinical trials (1, 2), induction of mild hypothermia (32°C-34°C) for 12-24 hrs in comatose survivors of cardiac arrest has been recommended (3).

The clinical trials used simple techniques (ice packs or convection cooling) and the rate of cooling was relatively slow (1, 2). Subsequently, many researchers have been trying to develop new techniques for cooling, including administration of cold intravenous fluids, surface cooling devices, and intravascular devices, which can be invasive and cumbersome. More importantly, systemic hypothermia is not without risk. Targeted, local/regional cooling could provide benefit while minimizing risks.

Because the brain is the organ most vulnerable to ischemia, selective brain cooling has long been a quest of resuscitation researchers. The simplest approach would be to apply ice packs or an ice cap to the head. This may work in small animals and infants, but not in adults (4). The thickness of the skull and warm arterial blood flow to the brain limit this approach.

Nasopharyngeal cooling (NPC) has been proposed as a noninvasive method for selective brain cooling that could be safe and easy to initiate. Cooling may occur by conduction to adjacent brain structures or by cooling nearby arteries

supplying the brain. Circulating cold fluids in the nasopharynx can establish a brain-systemic temperature gradient (5).

A novel device for NPC is the RhinoChill (Benechill, San Diego, CA), which sprays a perfluorochemical through a set of tubes placed into the nasopharynx. Rapid evaporation of the spray absorbs a large amount of heat. With normal circulation in sheep, this device can cool the brain faster than whole-body surface cooling and establish a brain-systemic temperature gradient (6). There was no damage caused by the perfluorochemical spray and minimal systemic uptake of the perfluorochemical.

In this issue of *Critical Care Medicine*, Yu et al (7) have continued their work on NPC in pigs. In a previous study (8), they compared NPC during cardiopulmonary resuscitation (CPR) with surface cooling started 2 hrs later and with maintenance of normothermia. The NPC group had the lowest brain temperatures as measured by jugular vein temperature, a surrogate for brain temperature, while maintaining a brain to core temperature gradient of approximately 3°C to 5°C. Survival in the hypothermia groups was greater than in the normothermia group. These authors have also shown that NPC improves myocardial function after arrest compared with maintenance of normothermia (9).

### \*See also p. 916.

Key Words: hypothermia; nasopharynx; cardiac arrest  
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In the current study, Yu et al (7) compared NPC with cold saline infusion (CSI) in a swine model of prehospital cardiac arrest with cooling during CPR in both groups. Jugular vein temperature decreased by 1.8°C in the NPC group compared with 0.3°C in the CSI group after 5 mins. Simultaneously, core temperature had decreased by 1.2°C in the CSI group and had not changed in the NPC group.

Perhaps the most interesting and surprising finding of this study (7) is that NPC was associated with a higher coronary perfusion pressure during CPR compared with CSI. This increase in coronary perfusion pressure was associated with improved initial resuscitation rates. The fact that this initial success did not translate into improved long-term outcome reflects the high lethality of this model.

The effect of NPC during CPR, if found to be robust and confirmed by other groups, must be related to an effect of the local cooling near the nasopharynx, because core temperature had not changed and, given low cerebral blood flow during CPR, it seems unlikely that global brain cooling had occurred. The authors suggest that the mechanism of the hemodynamic effects is direct cooling near the hypothalamus, which controls the body's response to cold (10). The hypothalamus integrates afferent information from the peripheral, spinal cord, and brain sensory receptors. The response is mediated mainly via the sympathetic nervous system. Either direct stimulation of the heart or increased systemic levels of vasoconstricting hormones could lead to increased vasomotor tone. The resultant increase in coronary perfusion pressure could readily translate into improved resuscitation rates with decreased need for exogenous epinephrine and countershocks. Others have shown that systemic hypothermia can improve resuscitation and cardiac performance after cardiac arrest, although without increasing coronary perfusion pressure; the mechanism is unclear (11).

An alternative explanation for the findings by Yu et al (7) is that CSI is detrimental. Infusion of large amounts of fluid during CPR may increase forward flow but decrease regional blood flow because of disproportionate increases in central venous and intracranial pressures (12). In one study (13) in swine, rapid cooling with an intravascular catheter led to a better resuscitation rate and myocardial sparing compared with cooling via CSI. In several clinical studies, however, CSI seemed to improve the patients' hemodynamics (14, 15).

The study by Yu et al (7) raises multiple questions that should be explored in future studies. One important question is what is the exact effect of this cooling method on brain temperatures? Use of the jugular vein temperature is questionable. It would be worth conducting an experiment with invasive brain temperature measurements. Some additional minor issues include the optimal duration and temperature goals for NPC.

The most intriguing question is why does NPC have the apparent hemodynamic effects seen here? It will take creative neurophysiologic studies to figure out the answer, including *in vivo* cerebral blood-flow mapping, temperature monitoring, brain histologic damage assessment, sympathetic nerve activity, and hormone release.

A prehospital trial of NPC, recently presented at the Resuscitation Science Symposium of the American Heart Association ([directnews.americanheart.org/extras/sessions2009/slides/13\\_pslides.pdf](http://directnews.americanheart.org/extras/sessions2009/slides/13_pslides.pdf)), demonstrated the safety and feasibility of this approach, decreased time-to-target temperature, and improved outcome when CPR was started within 10 mins.

As a method to improve outcome from cardiac arrest, NPC seems to have great promise, particularly for field use, even if we do not understand all of its mechanisms. Plus, it easily could be combined with a systemic cooling method in the field or in the hospital. In a person who is awake, use of NPC would cause an ice cream headache; however, for the patient who has experienced cardiac arrest, it could save a life.

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