Intra-Arrest Intra-Nasal Cooling Improves Resuscitation After Prolonged Cardiac Arrest In Pigs

Hao Wang, MD[1]; Denise Barbut, MD, MRCP[3]; Wanchun Tang, MD[1, 2];
Min-Shan Tsai, MD[1]; Shijie Sun, MD[1, 2]; Max Harry Weil, MD, PhD[1, 2]

[1]Weil Institute of Critical Care Medicine, Rancho Mirage, CA
[2]Keck School of Medicine of the University of Southern California, Los Angeles, CA
[3]Benechill Inc, San Diego, CA

Introduction.

Systemic hypothermia initiated after cardiopulmonary resuscitation (CPR) improves survival and neurologic outcomes.[1,2] We previously demonstrated that intra-nasal cooling improved post resuscitation neurologic outcomes.(3) Hypothermia, when induced prior to cardiac arrest, also improves resuscitation.(4) Since hypothermia may be induced with relative ease by the intra-nasal route during CPR in contrast to other available techniques, we investigated this option after prolonged cardiac arrest. We hypothesized that when intra-nasal cooling was initiated during CPR, there was the likelihood that restoration of spontaneous circulation (ROSC) would be increased.

Methods.

In 16 domestic male pigs weighing 42 ± 3 kg, VF was induced electrically and untreated for 15 minutes. Animals were then randomly arranged to either cooling or uncooled control. CPR was administered for 5 minutes prior to attempted defibrillation. Coincident with starting CPR, a device was utilized which provided for cooling with evaporation of perfluorocarbon liquid through the nasal cavity (Figure 1). The cooling was begun simultaneously with precordial compression and discontinued after core temperature of 34ºC was achieved or after four hours. Control animals were identically treated except for intra-nasal cooling. If a biphasic, 150 J electrical shock failed to restore spontaneous circulation, CPR was resumed and continued for 1 minute prior to subsequent defibrillation attempts or for a total of 15 minutes. Brain temperature was measured with a needle temperature sensor (NOVATEMP Myocardial Temperature Probes, NovaMed, NY) through a burr hole in the right parieto-occipital cortex.

Results.

The baseline measurement did not differ between control and cooled animals (Table 1). Seven of eight cooled animals and two of eight in the control group were successfully resuscitated. The differences were highly significant (p=0.01). Resuscitated animals survived for more than 4 hours. At the time of ROSC, core temperature was above baseline in both groups, brain temperature decreased 0.1ºC in the cooled animals and increased 0.3ºC in the control group (Figure 2). Coronary perfusion pressure (CPP), a prediction of the likelihood of ROSC, at the time of the first shock was significantly higher in the cooled group (26±7 mmHg) vs. control (16±4 mmHg) (*P*= 0.01), and therefore predicted successful defibrillation (Table 2). This was also reflected in earlier ROSC (Table 2).

Conclusion.

Intra-nasal cooling initiated during CPR significantly improves the success of resuscitation in a porcine model of prolonged cardiac arrest.

References

1. The Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia improves the neurologic outcome after cardiac arrest. *N Engl J Med*. 2002; 346:549 –556.
2. Bernard SA, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*. 2002; 346:557–563.
3. Jun Guan, et al. Rapid induction of head cooling by the intranasal route during cardiopulmonary resuscitation improves survival and neurological outcomes. *Circulation.* 2007; 116: II-529 – II-530.
4. Boddicker KA, et al. Hypothermia improves defibrillation success and resuscitation outcomes from ventricular fibrillation. *Circulation*. 2005; 111: 3195-3201.