EXPLORATORY STUDY ON MECHANISM BY WHICH HYPOTHERMIA IMPROVES OUTCOMES OF CPR

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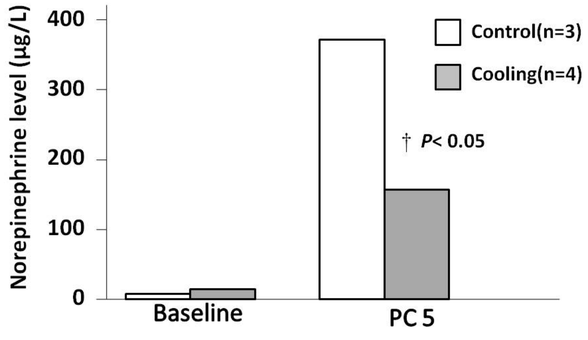
In an earlier report, we demonstrated that when head cooling was initiated during CPR, outcomes were improved. We had also reported in a series of studies the adverse effects on ultimate outcomes after use of adrenergic amines, both α_1 and β adrenergic agonists and improved outcomes with α_1 and β adrenergic blockades. We therefore hypothesized that the benefits of rapid head cooling may at least, in part, relate to reduced endogenous adrenergic stimulation. In a small exploratory study, we measured endogenous plasma concentration of norepinephrine.

Studies were conducted in 7 domestic male pigs weighing 40±5 kg. After 15 minutes of untreated ventricular fibrillation, head cooling was induced in 4 animals with a Rhino Chill device coincident with start of CPR. An additional 3 animals served as identically treated controls without hypothermia. CPR was continued for 5 minutes in both groups prior to defibrillation. Endogenous plasma norepinephrine levels were measured in blood at baseline and 5 minutes after the start of CPR.

Three of 4 head cooling animals were successfully resuscitated, in contrast to none of the control animals. Jugular vein temperature was significantly reduced from 38.0 C to 37.0 C in brain cooled animals, compared to that in control animals (*P*<0.001). Endogenous norepinephrine levels were greatly increased compared to baseline levels in both groups, however, the plasma norepinephrine level was significantly less in brain cooled animals (153 vs. 381 µg/L, *P*<0.05).

Norepinephrine level during CPR: reduction with nasopharygeal cooling



The mechanism by which head cooling during CPR improves outcome of resuscitation may, at least in part, relate to inhibition of the adverse effects of endogenous adrenergic stimulation.