

Whole-body cooling during hyperthermia: physiology versus physics

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Heat exhaustion and illness frequently occur without warning, and are often characterised by a rapid onset. In clinically-significant cases, patient cooling time can impact upon the prognosis. For inanimate objects, the rate of heat loss is a simple function of the thermal gradient that can be established, thus, immersion in ice-cold water is an effective means to rapidly extract heat. However, access to ice or cold water is limited in hot climates in the field, and there are also clinical concerns regarding sudden cold-water immersion in hyperthermic individuals. Furthermore, sympathetically-mediated cutaneous vasoconstriction reduces convective heat transfer from the core. It was therefore hypothesised that a warmer immersion, in which vasoconstriction is less powerful, may facilitate rapid cooling in hyperthermic individuals. To test this hypothesis, eight males participated in three trials, consisting of separate heating and cooling phases. Subjects were heated over 95 min to an oesophageal temperature of 39.5°C, using exercise in the heat (36°C, 50% relative humidity and water-perfusion garment (40°C)). Heating followed a fixed protocol to achieve three target oesophageal temperatures (38.5°C, 39.0°C, 39.5°C), sustained for 30, 20 and 10 min (respectively). Following a 5-min preparation, subjects were cooled using one of three methods: air (20-22°C: control); cold-water immersion (14°C); temperate immersion (26°C). After heating, oesophageal temperatures were 39.4°C (control), 39.3°C (cold immersion) and 39.3°C (temperate immersion). The time taken to reach the target core temperature (37.5°C; Figure) averaged 24.78 min (control), 2.84 min (cold immersion) and 4.56 min (temperate immersion), with each of the between-trial comparisons being statistically significant ($P < 0.05$).

Cooling in water, which has a thermal conductivity > 24 times that of air, will always be more rapid. Nevertheless, it is clear that whole-body cooling in temperate water takes only marginally longer than cold-water cooling. Indeed, one cannot imagine that the time difference of 1.72 min could have any meaningful physiological or clinical implications. In cold water, cutaneous vasoconstriction is maximised, with transcutaneous heat loss being dependent upon conduction. In temperate water, it was assumed that the rapid heat loss was due to a less powerful vasoconstrictor response, with central heat being rapidly transported (convective flow) to the skin surface for dissipation. While the core-to-water thermal gradient was much less during this immersion, a presumably higher skin blood flow supported the rapid convective delivery of heat to the skin, and its subsequent dissipation. Thus, a sustained physiological mechanism (skin blood flow) has countered a less powerful thermal gradient, resulting in clinically insignificant differences in heat extraction.

