

IMMERSION COOLING DURING HYPERTHERMIA: WHY WARMER MAY BE BETTER.

*Joanne N. Caldwell, Anne M.J. van den Heuvel, Pete Kerry, Mitchell J. Clark,
Gregory E. Peoples and Nigel A.S. Taylor*

School of Health Sciences, University of Wollongong, Wollongong, Australia

Contact person: nigel_taylor@uow.edu.au

INTRODUCTION

A significant number of military personnel suffer from exertional heat illness, with some extreme cases resulting in death. These illnesses may occur without warning, they are often characterised by a rapid onset and can occur even in experienced personnel. Various field treatments for hyperthermia have been developed, and one of particular interest is the use cold-water immersion. Not surprisingly, immersion in ice-cold water (0-2°C) has been shown to be an effective means of rapidly reducing body core temperature (Proulx *et al.*, 2003, 2006).

However, some researchers have raised both physiological and ethical concerns regarding this practice, suggesting instead that more conservative and warmer immersion temperatures may be more advisable, and indeed even more practical for field application (Taylor *et al.*, 2008). Such extensive cooling will elicit dramatic reductions in whole-body skin blood flow. This impedes the convective delivery of heat from the deep tissues, and also conductive heat loss to the water by increasing transcutaneous insulation. Furthermore, the sudden immersion in cold water has long been known to precipitate powerful cold-shock responses, and even death (Tipton, 1989). Indeed, it is the rate at which the skin temperature changes that dictates the intensity of this cold shock, so the sudden skin temperature reduction experienced when a hyperthermic individual is suddenly immersed in ice-cold water will evoke very powerful afferent and whole-body sympathetic outflow. Not surprisingly, this practice is not recommended by the current laboratory, except for very serious cases in which a viable peripheral circulation is absent.

Instead, a more conservative water-immersion temperature is advised, since warmer temperatures not only reduce the risk of cold shock, but also satisfy the pragmatic issue of availability within the field. Warmer water temperatures (~26°C) are unlikely to induce a powerful sympathetic response, and sudden cutaneous vasoconstriction should be avoided. As a consequence, a greater skin blood flow will ensure the substantial movement of central body heat to the skin surface, and thereby facilitate rapid heat loss.

We have previously shown that warm-water immersion (26°C) of profoundly hyperthermic individuals enabled core cooling to be achieved just 45 s slower than in water 12°C cooler (Taylor *et al.*, 2008). However, the vascular responses of the skin were not measured in this experiment. Therefore, data were lacking to categorically establish the physiological mechanism that would account for this outcome. Accordingly, the aim of this project was to re-evaluate the effectiveness of two different water-immersion temperatures (14°C and 26°C) on body core cooling from a hyperthermic state, whilst simultaneously quantifying the cutaneous vascular responses. The former temperature was used by Proulx *et al.* (2003, 2006), while the latter is an

approximation of the temperature of water that may be available in the field in hot climatic regions. It was hypothesised that cold-water immersion would elicit more powerful cutaneous vasoconstriction, and this would be less evident during cold-water immersion.

METHODS

Eight males participated in this study. Subjects were healthy, physically active adults, each of whom was screened to eliminate those with a history of cardiovascular, respiratory or thermoregulatory problems. In addition, subjects were screened using an exercise test (PWC170), to ensure a sample that was homogenous for aerobic fitness, and had a predicted mean maximal aerobic power of $58.53 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (SD 9.9). Each subject completed four whole-body, water-immersion trials; two at 14°C , and two at 26°C . Trials A (26°C) and B (14°C) occurred from a thermoneutral state, while trials C (26°C) and D (14°C) were preceded by whole-body heating (exercise in the heat). Thus, the four trials differed in the pre-immersion heating protocol (heating or no heating), and in the form of post-exercise cooling that was provided (14°C or 26°C). Trials were administered in a balanced order across subjects. Immersion was in the horizontal plane (supine), with the subject's head raised sufficiently to enable total immersion without a breathing impediment (Figure 1).



Figure 1: An immersed subject during whole-body cooling.

For trials A and B, immersion followed a 5-min thermoneutral data collection period. For trials C and D, subjects were first heated to achieve an oesophageal temperature of 39.5°C . This was achieved using a combination of exercise (metabolic heat) and exogenous heat (climate chamber (36°C , 50% relative humidity) and water-perfusion garment (40°C)). The exercise protocol followed a four-phase pattern, with three target core temperatures (38.5°C , 39.0°C , 39.5°C) dictating the work rates chosen. The final stage of exercise was of a relatively low intensity (intermittent work and rest), and was designed to clamp the core temperature for 10 min, thereby ensuring a more uniform heat distribution within the body tissues prior to cooling. Within each exercise phase, the work durations were retained (30, 30, 20, 10 min), but the work rate was varied to achieve the target core temperature. That is, the two targets were core temperature and time. If the target core temperature was exceeded, the work ceased. If the target was achieved early, the work rate was reduced to hold the target core temperature until the next stage began. The work rates used in the first trial were then replicated within the subsequent exercise heating

trial. Following heating, subjects were transferred (wheelchair) to the immersion facility adjacent to the chamber, positioned onto a litter (Figure 1) and equipped with skin blood flow apparatus. Immersion commenced 6.34 min (SD 0.92) after heating ceased.

Forearm blood flow was determined using venous-occlusion plethysmography (EC 4 Plethysmograph, D.E. Hokanson, Inc., U.S.A.). Blood flow to the hand was occluded by placing a cuff around the left wrist (inflated to 160 mmHg), while venous return was occluded using a second cuff placed proximal to the left elbow, and inflated to a pressure of 50 mmHg. Forearm arterial flow was approximated from changes in forearm circumference, measured using a mercury-filled strain gauge placed around the forearm. Data were collected continuously (20 Hz) immediately prior to, and during the entire immersion. The wrist cuff was deflated every 10 min. It was assumed that changes in blood flow under these conditions would reflect changes in the cutaneous component only (Johnson and Rowell, 1975). Other physiological measurements included body core and skin temperatures, heart rate and psychophysical responses. Between-trial comparisons were performed using Two-way Analysis of Variance. Sources of significant differences were isolated using Tukey's HSD statistic.

RESULTS AND DISCUSSION

Core temperatures following the thermoneutral and heating phases averaged 37.0°C (± 0.1) and 39.4°C (± 0.1), respectively. Just prior to immersion, these temperatures were 37.0°C (± 0.1) and 38.5°C (± 0.1), respectively. The time taken to reach at core temperature of 37.5°C in trial C was 2.56 min (± 0.64), for trial D this was 1.66 min (± 0.57). Forearm blood flow responses for all trials are shown in Figure 2.

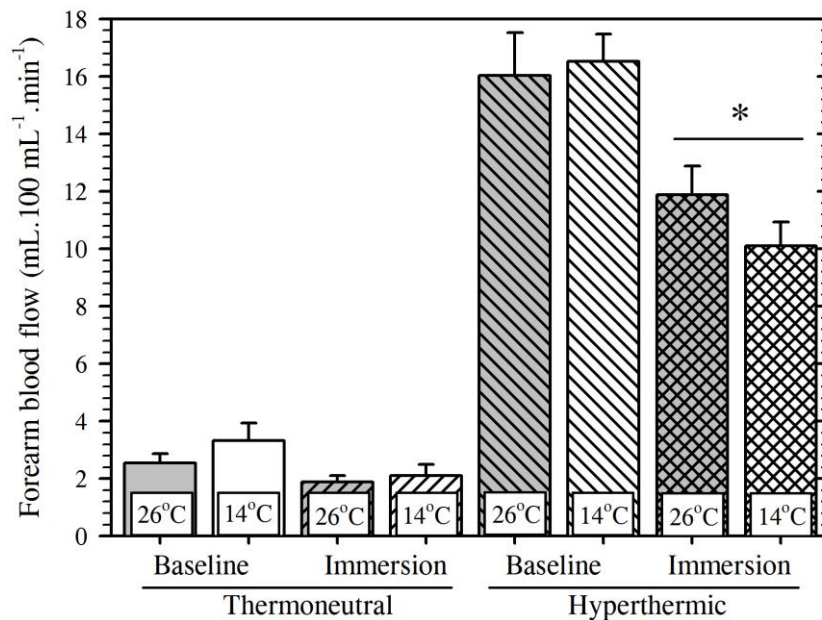


Figure 2: Forearm blood flow immediately prior to, and during the first 5 min of cold-water (14°C) and warm-water immersions (26°C) following prior heating or thermoneutral rest. Data are means with standard errors of the means. * signifies significant difference between treatments.

For each of the water immersions, forearm blood flow decreased significantly relative to the pre-immersion baselines (Figure 2; $P < 0.05$). However, when these immersions followed thermoneutral rest (trials A and B), these blood flow reductions did not differ significantly between the two water temperatures ($P > 0.05$). That is, it appeared that the cutaneous vascular responses were attributable to changes in local tissue temperature, with a slight hydrostatic component, and the 12°C difference in water temperature did not modify the strength of this vasoconstrictor response.

When immersion followed exercise- and heat-induced hyperthermia, the thermal state of the core prior to immersion modulated cutaneous vasoconstriction. During trials C and D, significant vasoconstriction was observed for each immersion temperature, relative to the pre-immersion baselines (Figure 2; $P < 0.05$). However, significantly more powerful vasoconstriction was evident within the cooler water ($P < 0.05$), even though core temperatures did not differ significantly between these trials prior to immersion ($P > 0.05$). This difference must be entirely attributable local thermal influences, either via local thermal effects on vasomotor tone (Pérgola *et al.*, 1993) or through a reflex elevation in sympathetic activation (Kregel *et al.*, 1992).

CONCLUSIONS

In this study, a more powerful reduction forearm blood flow was observed in hyperthermic individuals over the first 5 min of immersion in 14°C water, when compared to water at 26°C. This more powerful vasoconstrictor response will reduce convective heat delivery from the body core to the periphery, as well as suppressing conductive heat loss through the skin. These observations explain why minimal differences in cooling rate have been observed when profoundly hyperthermic individuals were immersed at these water temperatures (Taylor *et al.*, 2008). Therefore, it is recommended that hyperthermic patients with a viable cutaneous circulation be cooled in water at a less stressful temperature (24-26°C), and not in either ice-cold or cold water as others have recommended (Proulx *et al.*, 2003, 2006; Casa *et al.*, 2007).

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