HIGHLIGHTED TOPIC | A Physiological Systems Approach to Human and Mammalian Thermoregulation

Precooling leg muscle improves intermittent sprint exercise performance in hot, humid conditions

Paul C. Castle, Adam L. Macdonald, Andrew Philp, Anthony Webborn, Peter W. Watt, and Neil S. Maxwell

Chelsea School Research Centre, University of Brighton, Eastbourne, United Kingdom

Submitted 11 July 2005; accepted in final form 4 December 2005

Precooling leg muscle improves intermittent sprint exercise performance in hot, humid conditions. J Appl Physiol 100: 1377–1384, 2006. First published December 8, 2005; doi:10.1152/japplphysiol.00822.2005.—We used three techniques of precooling to test the hypothesis that heat strain would be alleviated, muscle temperature (Tmu) would be reduced, and as a result there would be delayed decrements in peak power output (PPO) during exercise in hot, humid conditions. Twelve male team-sport players completed four cycling intermittent sprint protocols (CISP). Each CISP consisted of twenty 2-min periods, each including 10 s of passive rest, 5 s of maximal sprint against a resistance of 7.5% body mass, and 105 s of active recovery. The CISP, preceded by 20 min of no cooling (Control), precooling via an ice vest (Vest), cold water immersion (Water), and ice packs covering the upper legs (Packs), was performed in hot, humid conditions (mean ± SE; 33.7 ± 0.3°C, 51.6 ± 2.2% relative humidity) in a randomized order. The rate of heat strain increase during the CISP was faster in Control than Water and Packs (P < 0.01), but it was similar to Vest. Packs and Water blunted the rise of Tmu until minute 16 and for the duration of the CISP (40 min), respectively (P < 0.01). Reductions in PPO occurred from minute 32 onward in Control, and an increase in PPO by ~4% due to Packs was observed (main effect; P < 0.05). The method of precooling determined the extent to which heat strain was reduced during intermittent sprint cycling, with leg precooling offering the greater ergogenic effect on PPO than either upper body or whole body cooling.

Physiological systems approach; muscle temperature; core temperature; hyperthermia; heat stress

Climatic heat stress has been a common issue at recent Summer Olympic Games (Atlanta 1996, Athens 2004) and remains an important factor for athletes and support staff to consider for the 2008 Beijing Games where it is expected to be, on average, 30°C and 74% relative humidity (www.bbc.co.uk/weather). Such hot, humid conditions reduce short-duration (~20 min) intermittent sprint running performance (32) and sprint times in a 90-min protocol specific to soccer (36, 37). Yet this occurs without alterations in muscle lactate concentrations; glycogen use (32); or plasma ammonia, glucose, or free fatty acid concentration (36). However, increased muscle temperature (Tmu) can quicken the rate of cross-bridge cycling during exercise, such as repeated Wingate sprint bouts (28). This occurs with a subsequently faster rate of fatigue (18) indicated by reduced mean power output (27). Furthermore, heat strain negatively correlated with peak power output (PPO) of individual sprints during the 40-min cycling intermittent sprint protocol (CISP) in hot, humid conditions (11). Therefore, the negative effects of heat stress are apparent in varying durations of intermittent sprint exercise and occur at a local muscular level, through alterations in contractile speed and a whole body “systemic” level of increased heat strain.

Precooling reduces preexercise core temperature, which may allow greater heat storage, improved submaximal exercise performance (8, 26), and thermoregulatory benefits during maximal intermittent sprinting (34) while under heat stress. However, no performance benefits have been reported after precooling for intermittent sprint exercise. This is not too surprising, considering that the methodologies reported only 5 min of precooling (15), which would be unlikely to have cooled the body’s core or deeper tissues (57). Other investigations employed temperate conditions (12, 16), and so performance was unaffected by heat strain (11). Therefore, the potential benefits that precooling may offer to counteract increasing heat strain (systemic effect) (11) and Tmu (local effect) (14) that contribute to reduced exercise performance while under heat stress remains unknown. Different precooling techniques will target either systemic or local effects or both systemic and local effects. Whole body systemic precooling and muscle precooling may be achieved through cold (29.0–24.0°C) water immersion (9), upper body systemic precooling via a modified suit/jacket (56), and muscle cooling directly (37).

Therefore, the aim of this investigation was to examine three techniques of precooling designed to target systemic cooling, local muscle cooling, and both systemic and local muscle cooling before 40 min of intermittent sprint exercise in hot, humid conditions compared with a control condition of no cooling (Control). Systemic cooling was achieved through wearing an ice vest on the upper body (Vest), local muscle cooling was achieved by placing cold packs on the thighs (Packs), and systemic and local muscle cooling was achieved through whole body immersion in water (Water). Therefore, each technique also had differing levels of practical application for field use. It was hypothesized that Vest would reduce heat...
strain, Packs would reduce $T_{\text{mu}}$. Water would reduce both heat strain and $T_{\text{mu}}$, and all techniques would improve intermittent sprint exercise performance.

**METHODS**

**Subjects and Experimental Design**

Twelve male, team games players (mean ± SE: age 22.5 ± 0.6 yr, height 180.3 ± 1.4 cm, body mass 82.5 ± 2.9 kg, sum of four skinfolds 38.0 ± 3.7 cm, peak oxygen consumption 3.9 ± 0.2 l/min) participated in and gave written, informed consent for this study, approved by The University of Brighton Ethics Committee. Subjects were English county standard soccer or rugby players and trained and/or competed in soccer- and rugby-specific activities approximately three times per week during the season before taking part in the investigation. Subjects abstained from alcohol, caffeine, and exercise for 48 h before each test, and they maintained their normal diet; they were asked to replicate this before subsequent visits.

Subjects reported to the laboratory during the British winter season (not heat acclimatized), on six occasions, each 6 days apart, at the same time of day to minimize the influence of circadian variation. (55) The first and second visits were used for preliminary measurements and to familiarize the subjects to the CISP (13). During the remaining four visits, subjects completed the CISP in a randomized order inside an environmental chamber in hot, humid conditions (33.7 ± 0.3°C, 51.6 ± 2.2% relative humidity). Before the subjects entered the environmental chamber, they underwent a 20-min period of either no cooling (Control) or precooling by Vest (Arctic Heat Products), Water, or Pack (3M, United Kingdom, Berkshire, UK).

During the control trial (Control) subjects remained seated. On removal from a −20°C freezer the Vest surface was 10.7 ± 2.5°C, and it weighed 1.4 kg. Subjects wore this for 20 min; the vest was removed for the exercise. In Water, body water immersion (17.8 ± 2.1°C) was to shoulder height. Packs had a surface temperature of −16.0 ± 5.8°C on removal from a −20°C freezer. They were covered with thin cotton cloth and secured to the anterior, lateral, and posterior aspects of the thighs to completely cover the quadriceps and hamstring muscle groups.

**Visit 1**

Sum of skinfolds was determined from four sites using skinfold calipers (Harpenden Instruments, West Sussex, UK) as described by Durnin and Womersley (16). Subjects completed an incremental cycling exercise test on a modified ergometer (Monark 620 Ergomedic, Varberg, Sweden) fitted with power-measuring cranks (SRM; Schroberer Rad Mebtechnik, Julick, Germany). Each individual’s lactate threshold and peak oxygen consumption were measured. Starting workload was set at a calculated power output of 95 W with a target cadence of 80 rpm. Workload was increased by 25 W every 3 min until their lactate threshold was reached, identified using the criteria of Wasserman et al. (51), and thereafter workload was increased 25 W every minute until volitional exhaustion. Whole blood fingertip samples were taken during the last 30 s of each 3-min stage for blood lactate analysis (~25 μl; YSI-2300 Plus, Yellow Springs Instruments, Yellow Springs, OH). Once the lactate threshold had been reached, no further blood samples were taken and the subjects continued the protocol to exhaustion. Oxygen uptake was recorded using open-circuit spirometry with expired air collected for ~45 s during the last minute of each 3-min stage during the lactate threshold test and during every minute thereafter. Heart rate (Polar sports tester, Polar Electro, Kempele, Finland) was taken at the same time points. Expired air was analyzed using a Servomex analyzer (Servomex, Crowborough, UK) for calculating oxygen uptake.

**Visit 2**

Subjects completed a standardized warm-up of cycling at 80 rpm at a calculated power output of 95 W for 5 min, followed by two periods of 30 s of passive rest then 30 s of cycling at 100 rpm at 120 W. Subjects then completed 10 min of the CISP for familiarization. The CISP involved 20 sets of 2-min periods, consisting of 10 s of passive rest, a 5-s maximal sprint from a stationary start against a resistance of 7.5% body mass, followed by 105 s of active recovery. (11) The active recovery intensity of the CISP was equivalent to 35% peak oxygen uptake and calculated from regression analysis of oxygen uptake and power output during the lactate threshold test. When all twenty 2-min blocks were completed, or if subjects felt they could no longer continue, or the safety limit of a high rectal temperature ($T_{\text{re}}$, 9.7°C) was reached, then the subject was removed from the environmental chamber and recovery was monitored.

**Experimental Procedures**

Before the four main visits, 7 of the 12 subjects volunteered to have $T_{\text{mu}}$ measured. On arrival, subjects provided a urine sample for assessment of urine specific gravity as an indicator of hydration status (3) using dip-test strips (Combur10-Test, Roche Diagnostics, Mannheim, Germany). Nude body mass was assessed, and $T_{\text{re}}$ was measured from a depth of 10 cm past the anal sphincter. A physician used diagnostic ultrasound to locate an appropriate site for insertion of an indwelling T-sk (Ellab, Norfolk, UK) into the vastus lateralis of the right leg. Ninety minutes before arrival at the laboratory subjects had placed 5 g of topical anesthetic cream (EMLA, AstraZeneca UK, Luton, UK) on the location marked by the physician. Skin temperature ($T_{sk}$)-measuring thermistors were then attached to the right side of the body on the chest, upper arm, thigh, and calf as described by Ramanathan (45), and a heart rate monitor was fitted to the chest. A resting whole blood fingertip sample was obtained for the determination of blood lactate concentration. Subjects were placed in a supine position, and the anesthetic cream was removed from the leg. The site was sterilized, and a disposable, sterile 18-gauge needle was inserted to 4 cm. The sterile, flexible $T_{\text{mu}}$ thermocouple was fed through the needle to its anatomic site inside the vastus lateralis. The thermocouple was secured to the leg with a sterilized waterproof dressing and adhesive tape. After a 15-min stabilization, resting values were recorded, followed by the 20 min control or precooling. After this, any source of cooling was removed and subjects were given 4 min to enter the environmental chamber to commence the warm-up followed by the CISP.

**Physiological Measures**

No adverse effects were reported by the seven subjects who volunteered to have their $T_{\text{mu}}$ measured, regarding their ability to perform the CISP while the thermistor was in the vastus lateralis. All 12 subjects volunteered for every other measurement. $T_{\text{re}}$ (Libra Medical, Reading, UK), $T_{sk}$ (Squirrel Meter Logger, Grant Instruments, Cambridge, UK), $T_{\text{mu}}$, heart rate, and thermal sensation (49) were recorded every 2 min during the 20-min preexercise control or precooling period. These measures, as well as rating of perceived exertion (RPE) (10), were also recorded at 1 min into each 2-min period during the active recovery part of the CISP replicating Castle et al. (11) and the arm-crank version of the protocol (52). Because $T_{\text{re}}$ can be slow to react to temperature increases during exercise (2), pilot work in our laboratory indicated that 1 min into each 2-min period was sufficient time for $T_{\text{re}}$ to increase during the CISP. Whole blood fingertip samples were obtained, and oxygen uptake was collected for ~45 s during the CISP 1 min into every fourth 2-min period. The calibrated SRM power-measuring cranks recorded power output continuously at a sampling rate of 0.5 Hz. PPO was recorded as the highest recorded power output value for each sprint. Work done during each sprint was calculated from the highest 3-s period of power output.
PRECOOLING AND INTERMITTENT SPRINT EXERCISE

output from each 5-s sprint to omit the inertia required to sprint from a stationary start. No fluid intake was permitted during any condition, in order that postexercise nude body mass could be assessed to indicate nonurine fluid loss. The physiological strain index (PSI) was calculated and then categorized from 0 (no strain) to 10 (very high strain) as described by Moran et al. (34) using the following equation:

\[
PSI = \frac{5(T_{re} - T_{re,0})}{39.5 - T_{re,0}} + \frac{5(HR_t - HR_0)}{180 - HR_0}
\]

where \( T_{re,0} \) and \( HR_0 \) are the initial \( T_{re} \) and heart rate, respectively, and \( T_{re, t} \) and \( HR_t \) are simultaneous measurements taken at any time.

Statistical Analyses

Data were checked for normality and sphericity was adjusted using the Huynh-Feldt method. Paired data from each CISP were compared using two-way ANOVA with repeated measures (condition \times time). Where significance was obtained, Tukey’s honestly significant difference post hoc test was undertaken. Pearson’s product moment correlation coefficient was calculated for identification of relationships between PSI and PPO, \( T_{mu} \), and work done during each sprint. All data were analyzed using a standard statistical package (SPSS version 11.5) and were reported as means \( \pm SE \). Statistical significance was accepted at the level of \( P < 0.05 \).

RESULTS

Precooling and Warm-up

Figure 1 shows \( T_{re} \) remained unchanged for Control during the 20-min period and warm-up. All precooling techniques reduced \( T_{re} \) from rest \((P < 0.01)\) with a cooling rate of 0.006 \( \pm 0.004 \) \(^\circ\)C/min for Vest, 0.001 \( \pm 0.004 \) \(^\circ\)C/min for Packs, and 0.009 \( \pm 0.004 \) \(^\circ\)C/min for Water. Further reductions were observed during the warm-up period so, at the end of the warm-up/start of the CISP, \( T_{re} \) was lower than resting temperatures by 0.3 \( \pm 0.3 \) \(^\circ\)C in Vest \((P < 0.01)\), 0.3 \( \pm 0.3 \) \(^\circ\)C in Water \((P < 0.01)\), and 0.2 \( \pm 0.2 \) \(^\circ\)C Packs \((P < 0.01)\).

\( T_{mu} \) was not reduced during the Control or Vest precooling period, but Water and Packs reduced \( T_{mu} \) \((P < 0.05; \text{Fig. 2})\) at a rate of 0.06 \( \pm 0.002 \) and 0.1 \( \pm 0.03 \) \(^\circ\)C/min \((P < 0.05)\), respectively (Fig. 1). \( T_{mu} \) was lower than resting values at the end of the warm up/start of the CISP by 0.7 \( \pm 0.9 \) \(^\circ\)C due to Water and 1.0 \( \pm 2.5 \) \(^\circ\)C due to Packs, although this was only statistically significant for Packs \((P < 0.05)\).

Water and Packs reduced \( T_{sk} \) from resting values throughout the 20-min period \((P < 0.01)\). This effect remained during the warm-up due to Water \((P < 0.01)\), which also caused a higher heart rate than Control \((\text{main effect, } P < 0.01)\).

Ratings of thermal sensation at rest were 4.0 \( \pm 0 \) units in all conditions, indicating thermal comfort and were unchanged during Control. All precooling techniques reduced thermal sensation values from rest for the 20-min precooling period \((P < 0.01)\). By the end of the warm-up, thermal sensation values increased above resting values for Control and Vest, indicating subjects felt hotter, but remained decreased due to Water at 3.5 \( \pm 1.0 \) units \((P < 0.01)\).

CISP

Performance variables. All subjects completed the CISP in every condition. Packs caused an increase in PPO by \( \sim 4\% \) compared with Control \((\text{main effect, } P < 0.05)\), but no other differences were observed (Table 1). Individual sprint PPO
Reduced from the start of the CISP in Control from sprint 16 (minute 32) onward (Fig. 3; \( P < 0.01 \)). In every condition, \( PPO \) of the final sprint superseded that achieved in the penultimate sprint. Control increased by 21 \( \pm \) 30 W, Water increased by 25 \( \pm \) 25 W, Packs increased by 64 \( \pm \) 18 W, and Vest increased by 93 \( \pm \) 33 W, although this was only significant for Vest (\( P < 0.01 \)).

Work done during each sprint in Control was lower than Packs (main effect; Table 1; \( P < 0.01 \)) but not lower than Vest or Water. Total work done in Control was lower than Vest and Packs (\( P < 0.01 \)) but not different from Water (Table 1). A negative correlation was observed between \( PPO \) and \( \text{PSI} \) and between \( PPO \) and \( \text{Tmu} \) for Control and Packs and between work done during each sprint and \( \text{Tmu} \) in Control, Vest, and Packs (\( P < 0.01 \), Table 2).

**Physiological Variables**

The rate of PSI increase for Control was faster than Water and Packs (main effect; \( P < 0.01 \)) but not faster than Vest. Peak PSI was higher for Control than Water and Packs (Table 3; \( P < 0.01 \)) but not Vest.

Greater \( T_{re} \) was observed in Control (38.2 \( \pm \) 0.3°C) than Vest (38.0 \( \pm \) 0.3°C), Water (37.7 \( \pm \) 0.3°C), and Packs (38.0 \( \pm \) 0.2°C; main effect; \( P < 0.01 \)). \( T_{mu} \) was identical in Control and Vest, both at 39.0 \( \pm \) 0.7°C, which was higher than Water and Packs at 38.2 \( \pm \) 0.7 and 38.4 \( \pm \) 0.9°C, respectively (main effect; \( P < 0.01 \)). Figure 1 shows that Packs maintained lower \( T_{mu} \) until sprint 8 (minute 16) and Water for the duration of the CISP (\( P < 0.01 \)).

Compared with Control, Vest and Packs reduced \( T_{sk} \) until sprint 4 (minute 8; Fig. 2; \( P < 0.01 \)), and Water reduced \( T_{sk} \) for the duration of the CISP (20 sprints, 40 min; \( P < 0.01 \)).

Heart rate in Control (168 \( \pm \) 11 beats/min) was higher than Water (159 \( \pm \) 9 beats/min; main effect; \( P < 0.01 \)), but Vest (171 \( \pm \) 7 beats/min) and Packs (166 \( \pm \) 9 beats/min) were similar. Heart rate was lower than Control until sprint 8 (minute 16) due to water immersion (Fig. 2; \( P < 0.01 \)).

No differences were observed between conditions for the change in nude body mass at \( \sim 0.7 \pm 0.1 \) kg. Table 3 shows

---

**Table 1.** Peak power output, work done each sprint, and total work done during the cycling intermittent sprint protocol in hot, humid conditions after the 20-min control, ice vest, water immersion, and silicate gel ice pack exposure

<table>
<thead>
<tr>
<th>Condition</th>
<th>PPO, W</th>
<th>Work Done Each Sprint, J</th>
<th>Total Work Done, kJ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1,132 ( \pm ) 112</td>
<td>349.1 ( \pm ) 32.5</td>
<td>6.8 ( \pm ) 0.72</td>
</tr>
<tr>
<td>Vest</td>
<td>1,149 ( \pm ) 127</td>
<td>356.4 ( \pm ) 36.7</td>
<td>7.1 ( \pm ) 0.77 ( \dagger )</td>
</tr>
<tr>
<td>Water</td>
<td>1,126 ( \pm ) 118</td>
<td>350.9 ( \pm ) 35.1</td>
<td>6.9 ( \pm ) 0.77</td>
</tr>
<tr>
<td>Packs</td>
<td>1,181 ( \pm ) 149*</td>
<td>366.8 ( \pm ) 42.3*</td>
<td>7.1 ( \pm ) 0.82 ( \dagger )</td>
</tr>
</tbody>
</table>

Values are means \( \pm \) SE. PPO, peak power output; CISP, cycling intermittent sprint protocol; Control, 20-min control; Vest, ice vest; Water, water immersion; Packs, silicate gel ice pack. *Significant difference from Control, \( P < 0.05 \). †Significant difference from Control, \( P < 0.01 \).
there were no differences between values for final thermal sensation, RPE, mean blood lactate concentration, oxygen uptake, and respiratory exchange ratio. No differences were observed for final RPE values ($P = 0.086$), but final thermal sensation values were lower than Control due to Water ($P < 0.01$).

**DISCUSSION**

The aim of this investigation was to examine three techniques of precooling designed to target systemic cooling (Vest), local muscle cooling (Packs), and both systemic and local muscle cooling (Water) for 20 min before the CISP compared with a control condition of no cooling (Control). It was hypothesized that Vest would reduce heat strain, Packs would reduce $T_{mu}$, Water would reduce both heat strain and $T_{mu}$, and all techniques would improve intermittent sprint exercise performance. Accordingly, the first important finding was that the negative effects of heat stress on PPO were evident in Water and Packs, but not in Control. The second important finding was that $T_{mu}$ was negatively correlated to PPO in Control, $r = 0.65*, P < 0.05$. The third and most important finding was that Packs improved PPO by $4\%$ compared with Control.

Reduced performance in Control occurred without differences in oxygen consumption, RER, or blood lactate concentration. This is consistent with other reports in the literature, showing the debilitating effects of heat stress without alterations in muscle metabolism, (30, 31, 35), plasma metabolites (18), fuels (35), or acid-base status (5). Therefore, heat stress appears to add no further metabolic strain, measured by these factors, than already imposed by maximal effort intermittent sprinting.

However, PPO declined in Control with a concomitant $T_{mu}$ of $\sim 40^\circ C$, consistent with observations by Drust et al. (14). Higher $T_{mu}$ speeds the rate of glycogenolysis and phosphate production by as much as $10\%$ (Q10 effect) (7, 28), causing quicker fatigue (5, 14, 46) and reduced mean power output (28). Hence there was a negative correlation between $T_{mu}$ and both PPO and work done during each sprint in Control. However, $T_{mu}$ cannot be the only factor explaining poor performance because $T_{mu}$ also reached $\sim 40^\circ C$ in Vest, but PPO did not decline. The rate of core temperature increase is also important (45, 46), and the PSI, as the integrated response of both $T_{re}$ and heart rate, was negatively correlated to PPO in the present investigation, confirming the link between heat strain and reduced intermittent sprint performance (11).

All precooling techniques reduced $T_{re}$, but only Water and Packs reduced $T_{mu}$. Different water immersion protocols (8, 9, 26) have caused more pronounced reductions in $T_{re}$ than observed in the present investigation. It was, therefore, surprising that Water, Vest, and Packs displayed similar cooling rates and total reductions of $T_{re}$, considering that Water covered the entire body, compared with the regional cooling of Vest and Packs. The sudden change in $T_{sk}$ when entering Water probably caused more severe vasoconstriction than the other conditions alongside increased catecholamine release (24) and blood pressure (22, 23), explaining the elevated heart rate for this condition. Packs was the most effective technique to reduce $T_{mu}$, particularly due to the afterdrop effect (20) when Packs were removed at the start of the warm-up. In contrast to an earlier study by Sleivert et al. (47) the warm-up of the present investigation did not reverse reduced $T_{mu}$ in Packs. However, methodologies were different between studies as Sleivert et al. used 45 min of cooling by a water-perfused cuff ($\sim 4^\circ C$) on a smaller area of the thigh. Therefore, short-term, aggressive cooling on a greater leg area using Packs appears to be more

---

**Table 2. Correlation matrix between peak power output, physiological strain index, muscle temperature, and work done during each sprint**

<table>
<thead>
<tr>
<th>Condition</th>
<th>PPO and PSI</th>
<th>PPO and $T_{mu}$</th>
<th>$T_{mu}$ and work done</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>$-0.95*$</td>
<td>$-0.92*$</td>
<td>$-0.92*$</td>
</tr>
<tr>
<td>Vest</td>
<td>$-0.39$</td>
<td>$-0.30$</td>
<td>$-0.50*$</td>
</tr>
<tr>
<td>Water</td>
<td>0.24</td>
<td>0.56</td>
<td>0.07</td>
</tr>
<tr>
<td>Packs</td>
<td>$-0.75*$</td>
<td>$-0.65*$</td>
<td>$-0.70*$</td>
</tr>
</tbody>
</table>

PSI, physiological strain index; $T_{mu}$, muscle temperature. *Significant correlation, $P < 0.01$. 

---

*Fig. 3. Mean ($\pm$ SE) peak power output CISP after the 20-min Control, Vest, Water, and Packs precooling period in hot, humid conditions. $^a$Significant reduction from sprint 1 in Control, $P < 0.05$. $^b$Significant difference between Control and Vest, $P < 0.01$. $^c$Significant difference between Control and Water, $P < 0.01$. $^d$Significant difference between Control and Packs, $P < 0.01$.
effective in reducing $T_{mu}$, perhaps by inducing greater thermal gradients for conductive cooling.

Peak PSI was reduced by all precooling techniques, indicating the successful manipulation of $T_{re}$ and heart rate that has previously been associated with predominantly submaximal exercise (for review, see Ref. 29). However, the rate of $T_{re}$ increase (displayed by the gradient in Fig. 1) remained unchanged, supporting the theory that precooling creates a sink for the absorption of metabolic and environmental heat (53) without changing the rate of heat gain (52, 53). All techniques also reduced $T_{sk}$, but only Packs and Water reduced $T_{mu}$, which, as an indicator of deeper tissue, suggests that these techniques created a larger heat sink than Vest, explaining the slower rate of PSI increase observed in Packs and Water. It appears that a site/technique-specific, dose-dependent response of precooling exists for the rate of rise in heat strain, which affects performance differently because Vest was ergogenic at the end of the CISP, Water ergolytic at the start and Packs ergogenic by ~4% overall.

The mechanism responsible for the dose dependency of precooling may be explained by alterations in motor unit recruitment, shown in exercise induced hyperthermia (30) and perhaps mediated through a central governor (40). In hot/humid conditions, central activation of exercising muscles can decline at core temperatures of ~38.5°C (48). This is similar to the final $T_{re}$ values in Control, indicating that mild hyperthermia or heat strain can affect the central nervous system without a "critical" $T_{re}$ of 40°C (42) being reached. In response to the anticipated heat gain of exercising under heat stress in Control, afferent feedback from the thermoreceptors may have caused subconscious pacing to reduce motor unit recruitment (30). This has been shown by reduced integrated electromyogram and power output (50), and Oksa et al. (43) suggested that, in response to muscle cooling, alterations in peripheral neural activity occurred to prevent local muscular injury. Therefore, subconsciously, downregulation of motor units to prevent hyperthermia in Control (30) may have been offset by precooling (4). It is likely that this feed-forward-type response would vary, depending on the severity, or "dose" of precooling caused by each technique and monitored through afferent inputs. For example, by slightly reducing $T_{re}$ but not $T_{mu}$, Vest created a small heat sink (53) and allowed greater heat transfer (6), thus addressing the fatigue effect of heat stress at a systemic level (11) but not at a local level (14). Because Vest caused a relatively low dose of afferent feedback from the thermoreceptors, no benefit was observed regarding the PSI, although it is a very practical technique for use in the field.

Although Water and Packs altered PSI and $T_{mu}$ similarly, PPO only increased in Packs. Although elevated $T_{mu}$ reduces intermittent sprint performance in hot, humid conditions (14), cooling muscle by ~8.0°C can reduce PPO by as much as 32% (13) via a Q10 effect on ATP hydrolysis (19). This may partially account for the reduction in PPO of the first two sprints in Water compared with Control, yet, at the same time Packs caused cooler $T_{mu}$ and higher PPO. However, lowering $T_{mu}$ reduces or blocks muscle spindle activity (44) and possibly Golgi tendon organ activity. Thus sensory feedback to the brain may have indicated fewer motor units were active than really the case in Packs. Furthermore, all subjects reported feeling a bearable, but painful burning sensation on the thighs from Packs, indicating a nociceptive sensory feedback due to the extreme cold temperature (21). Paradoxical cooling (21) sensations from the thigh skin thermoreceptors combined with suppressed muscle spindle activity may have been an optimal dose of sensory information. This may have been centrally processed (40) to alter the subconscious pacing strategy (29) and increase the motor unit recruitment and PPO during Packs precooling.

Although suppressed muscle spindle activity probably also occurred in Water, this technique also cooled the majority of the skin, albeit in the absence of painful sensations. It has been hypothesized that reduced cardiorespiratory function reduced time to exhaustion of maximal effort running after whole body cooling (32). However, skin cooling increases mean arterial blood pressure during hyperthermia (54), and little change in cardiorespiratory values was noted in the present investigation. Cooling large areas of the skin does stimulate the central and skin thermosensors (17), and it activates aspects of the insula cortex that are usually activated in response to pain (17). Although there is uncertainty whether thermoreceptive neurons project to the nociceptive system or are separately activated (21), Water may have caused an "overdose" of sensory information, including innocuous, cool nociception (21). Reduced PPO and work done each sprint during Water may be due to impaired exercise capacity (33) as part of a neural protective mechanism (43), although Water did provide the most thermo-regulatory benefit.

The last point of interest is that final sprint PPO improved from the penultimate sprint in every condition. This confirms that peripheral fatigue did not cause the reduced performance in Control, because if that were the case, muscle rigor would be expected (41) and not an improvement in PPO. The more likely explanation is the subconscious pacing strategy already discussed. Although originally derived from self-paced protocols (30, 50), the response has been shown during the Wingate anaerobic test (1), and the PPO profiles of the CISP indicate its presence in the present investigation. Interestingly the greatest final sprint increase was observed in Vest, yet this caused the
PRECOOLING AND INTERMITTENT SPRINT EXERCISE

smallest dose of precooling and all other variables were almost identical to Control. Previously, Arngrimsson et al. (4) observed that an ice vest improved running performance toward the end of the race, although the pace was unchanged at the start. It was suggested that improved thermal responses allowed athletes to realize that they could increase the pace (4). It may be that, because Vest delivered the least sensory information to the brain’s central governor (39), the pacing strategy was too conservative and recalculated toward the end of the CISP to recruit more motor units, explaining the maintenance of PPO compared with Control.

In conclusion, this investigation has highlighted that the negative effects of heat stress manifest themselves through an elevated, local Tミュ and systemic, heat strain level. All three techniques of precooling improved thermoregulation in a dose-dependent response, causing various amounts of sensory feedback. This may alter motor unit recruitment and performance accordingly, such that the experimental hypothesis can only be accepted in part. Thigh precooling with Packs improved inter-

ACKNOWLEDGMENTS

We thank the support of The University of Brighton; the helpful discussions and contributions of Paul McNaught-Davis; and the technical assistance of David Thomas, Anne Atfield, Ian Lloyd, Ron Shepherd, and Alan Allchorn.

REFERENCES

5. Ball D, Burrows C, and Sargeant AJ. Human power output and the effect of acute heat stress during repeated bouts of sprint exercise (Ab-

17. Egan GF, Johnson J, Farell M, McAllen R, Zamarripa F, McKinley MJ, Lancaster J, Denton D, and Fox PT. Cortical,thalamic, and hypothalamic responses to cooling and warming the skin in awake hum-

24. Kozyreva TV, Tkachenko EY, Kozaruk VP, Latysheva TV, and Glinyska MA. Effects of slow and rapid cooling on catecholamine concentra-

28. Linnane DM, Bracken RM, Brooks S, Cox VM, and Ball D. Effects of hyperthermia on the metabolic responses to repeated high-intensity exer-


J Appl Physiol • VOL 100 • APRIL 2006 • www.jap.org