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Cold-Water Immersion and Lower Limb Muscle Oxygen Consumption as Measured by Near-Infrared Spectroscopy in Trained Endurance Athletes

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Context: Cold-water immersion (CWI) has been reported to reduce tissue metabolism postimmersion, but physiological data are lacking regarding the muscle metabolic response to its application. Near-infrared spectroscopy (NIRS) is a noninvasive optical technique that can inform muscle hemodynamics and tissue metabolism.

Objective: To investigate the effects of CWI at 2 water temperatures (10°C and 15°C) on NIRS-calculated measurements of muscle oxygen consumption (mVO_2).

Design: Crossover study.

Setting: University sports rehabilitation center.

Patients or Other Participants: A total of 11 male National Collegiate Athletic Association Division II long-distance runners (age = 23.4 ± 3.4 years, height = 1.8 ± 0.1 m, mass = 68.8 ± 10.7 kg, mean adipose tissue thickness = 6.7 ± 2.7 mm).

Intervention(s): Cold-water immersion at 10°C and 15°C for 20 minutes.

Main Outcome Measure(s): We calculated mVO_2 preimmersion and postimmersion at water temperatures of 10°C and 15°C. Changes in tissue oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), total hemoglobin (tHb), hemoglobin difference (Hb_{diff}), and tissue saturation index (TSI %) were measured during the 20-minute immersion at both temperatures.

Results: We observed a decrease in mVO_2 after immersion at both 10°C and 15°C ($F_{1,9} = 27.7801$, $P = .001$). During the 20-minute immersion at both temperatures, we noted a main effect of time for O_2Hb ($F_{3,27} = 14.227$, $P = .001$), HHb ($F_{3,27} = 5.749$, $P = .009$), tHb ($F_{3,27} = 24.786$, $P = .001$), and Hb_{diff} ($F_{3,27} = 3.894$, $P = .020$), in which values decreased over the course of immersion. Post hoc pairwise comparisons showed that these changes occurred within the final 5 minutes of immersion for tHb and O_2Hb.

Conclusions: A 20-minute CWI at 10°C and 15°C led to a reduction in mVO_2. This was greater after immersion at 10°C. The reduction in mVO_2 suggests a decrease in muscle metabolic activity (ie, O_2 use after CWI). Calculating mVO_2 via the NIRS-occlusion technique may offer further insight into muscle metabolic responses beyond what is attainable from observing the NIRS primary signals.

Key Words: cryotherapy, optics, distance running, recovery

Key Points

- Cold-water immersion at 10°C and 15°C for 20 minutes decreased muscle oxygen consumption (mVO_2) as measured by near-infrared spectroscopy.
- The reduction in mVO_2 was greater after immersion at 10°C.
- A threshold temperature and immersion duration at which mVO_2 decreases may exist.

Cold-water immersion (CWI) is one of the most commonly applied cooling modalities postexercise.[1,2] It is used to rapidly cool whole limbs and large muscle groups[3] to facilitate reductions in muscle blood flow (mBF) and muscle metabolism. Postexercise cooling is thought to limit the extent of secondary damage via temperature-induced reductions in tissue metabolism and microvascular blood flow to the damaged muscle.[4,5] Indeed, reductions in skin, muscle, and conduit artery blood flow have been shown using a variety of measures, including laser doppler flowmetry, ultrasound, and near-infrared spectroscopy (NIRS).[5–8] A noninvasive optical technique, NIRS is a valid tool for continuously monitoring changes in local muscle hemodynamics and muscle oxygenation. In reviews, researchers have detailed the history and theory of the technique[9] and applications in sport and health.[10] Changes (Δ) in NIRS-derived measures of total hemoglobin (tHb) and tissue saturation index (TSI %) have been used as a proxy for mBF and muscle metabolic activity, respectively,[11,12] providing insight into the physiological effects of CWI.

A postexercise reduction in tHb is commonly observed during CWI,[7,8] and this finding is typically reported as a postimmersion reduction in local muscle blood volume (ie, tHb), explained as a reduction in mBF due to peripheral vasoconstriction. The TSI % response to CWI is less clear, with investigators reporting no change,[7] an increase (suggesting reduced

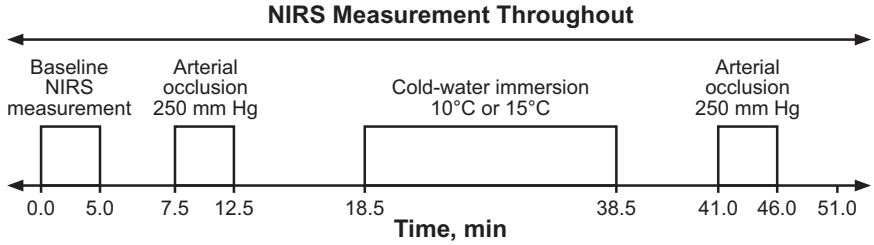

Figure 1. Illustration of the experimental protocol. Abbreviation: NIRS, near-infrared spectroscopy.

metabolic activity),[8] or a decrease (suggesting increased metabolic activity).[13]

Collectively, the authors of these studies have demonstrated the ability of NIRS measurements to monitor local muscle hemodynamics and muscle oxygenation both during and after CWI. However, conflicting findings and interpretations of ΔTSI % and thus muscle metabolic activity exist. To the best of our knowledge, all CWI studies using NIRS have been carried out postexercise, whereby any changes would be largely influenced by increased muscle and skin blood flow.[11] These are confounders of NIRS measurement,[14] which may have influenced previous interpretations. Furthermore, tHb and TSI % signals can provide only indirect and relative changes in local muscle blood volume and muscle oxygen (O_2) saturation, respectively. As such, they cannot be used to accurately quantify changes in either mBF or muscle O_2 consumption (mVO_2).[10,15] To account for these concerns, a physiological intervention in the form of an arterial occlusion (AO), which manipulates blood flow entering and leaving the limb, can be implemented to enable quantification of mVO_2.[16] Furthermore, the application of CWI at rest (as opposed to postexercise) and the use of AO should remove the influence of skin blood flow[9,10] and exercise hyperemia from the NIRS measurement, offering a more robust measurement approach.

To date, no specific guidelines exist regarding the optimal application of CWI to augment recovery from exercise. Durations and temperatures of CWI that have generally shown positive effects on recovery are between 10 and 20 minutes and 10°C and 15°C, respectively,[17] but few researchers have assessed the influence of different CWI protocols (eg, temperatures and durations) on physiological measures of muscle metabolism. Therefore, the purpose of our study was to evaluate the effects of CWI at 2 water temperatures (10°C and 15°C) on NIRS-calculated measurement of mVO_2. Given that colder water temperatures lead to greater reductions in tissue temperature[5,7] and that the premise of CWI is to reduce the metabolic rate of tissue,[18] we hypothesized that the application of CWI at 10°C but not 15°C would reduce mVO_2 postimmersion.

METHODS

Participants

Ten men (age = 23.4 ± 3.4 years, height = 1.8 ± 0.1 m, mass = 68.8 ± 10.7 kg) volunteered for this study. Mean adipose tissue thickness beneath the NIRS device was 6.7 ± 2.7 mm. All participants were long-distance runners competing for the same National Collegiate Athletic Association Division II team. They refrained from consuming caffeine and alcohol for 48 hours before each testing session. All individuals provided written informed consent, and the study was approved by the University of Essex research subcommittee.

Experimental Design

A randomized crossover design was implemented to assess the influence of CWI on mVO_2. Each person was required to participate in 2 testing sessions, separated by at least 24 hours. Participants attended the university sports therapy center at the same time of day, where they were randomly assigned to 1 of 2 recovery interventions, 10°C or 15°C CWI, for 20 minutes. Before and after CWI, AO of the leg undergoing NIRS assessment was performed. An overview of the experimental protocol is shown in Figure 1.

Preparation

The NIRS device was placed on the midportion of the medial gastrocnemius muscle belly. To ensure accuracy of repeat device placement, we drew an outline of the device on the limb using a surgical marker pen. Participants were instructed to maintain the outline during the testing period. Limb skinfold thickness was measured at the site of device attachment using Harpenden skinfold calipers (Baty International). The limb undergoing assessment was randomly assigned.

The CWI Protocol

Participants wore shorts and sat in an upright position in an immersion bath (model S-85-S; Whitehall Manufacturing), which was filled with water to the level of the navel. Water temperature was monitored using a digital aquarium thermometer, maintained within ±0.3°C of 10°C or 15°C, and stirred every 2 minutes using an inbuilt whirlpool machine. Individuals were instructed to keep as still as possible and not activate their lower limb muscles during immersion. After immersion, they exited the recovery bath and quickly towel dried before transitioning to the post-CWI occlusion. This transition period took ≤120 seconds.

Occlusion Protocol

Arterial occlusions were performed for 5 minutes at a pressure of 250 mm Hg[19] before (baseline) and after CWI. A pressure cuff (model SC10D; DE Hokanson, Inc) was attached to the participant's limb just above the knee joint and inflated to the corresponding pressure. The occlusion protocol was identical for both testing sessions, whereby participants lay supine on a therapy plinth next to the CWI bath. The protocol consisted of 1 AO (baseline), followed by a 5-minute recovery before transitioning to CWI. Post-CWI, each person returned to the therapy plinth, and the procedure was repeated.

The NIRS Measurements

A portable NIRS device (model PortaMon; Artinis Medical Systems BV) was fixed with black adhesive tape over

the marked measurement site to prevent signal contamination from external light sources. The device was made waterproof as previously described.[20] The same researcher (R.S.E.) attached each device and applied the adhesive tape for all testing procedures. To account for the external pressure applied to the muscle belly, we used a standardized approach.[21] The PortaMon is a dual-wavelength, continuous-wave system that simultaneously uses the modified Beer-Lambert law and spatially resolved spectroscopy methods. Changes in tissue oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), and tHb were measured using the difference in absorption characteristics of wavelengths at 760 and 850 nm. Values for O_2Hb, HHb, and tHb are reported as a concentration change from baseline (30-second averaging before each test with participants in the supine position) in micromolar units, using a differential path length factor of 4.0. Hemoglobin difference (Hb_{diff}) was calculated as follows: $[Hb_{diff}] = [O_2Hb] - [HHb]$. The TSI % was expressed as a percentage and calculated as follows: $[O_2Hb]/([O_2Hb] + [HHb]) \times 100$. It is independent of the NIR photon path length in muscle tissue and was calculated using the spatially resolved spectroscopy method. Chromophore concentrations were detected using the farthest light-emitting diode (40 mm). Data were collected offline at a sampling rate of 10 Hz.

Muscle Oxygen Consumption

We calculated mVO_2 by evaluating the linear decrease in ($[Hb_{diff}] = [O_2Hb] - [HHb]$), according to Equation 1. The linear decrease in Hb_{diff} was calculated between 30 and 150 seconds as described earlier.[22] Concentration changes of Hb_{diff} were expressed in micromolars per second and converted to milliliters O_2 per minute per 100 g tissue. A value of 1.04 $kg \cdot L^{-1}$ was used for muscle density.[16] The mVO_2 measurement demonstrated good reliability during rest and dynamic exercise, with various estimates of reproducibility shown (coefficient of variation [CV] = about 6% to 20%).[23]

$$mVO_2 = \frac{\left(\left(\left(\frac{\Delta Hb_{diff}}{2} \times 60\right) \div (10 \times 1.04)\right) \times 4\right) \times 22.4}{1000} \quad (1)$$

Statistical Analysis

All results are reported as mean ± SD. Normality and outliers were verified using the Shapiro-Wilk test and by observing the normality plots and residual plots. Preimmersion data of participant 3 were identified as outliers for both temperatures. We calculated imputed estimates using the fully conditional specification method to account for this.[24] Mean values for O_2Hb, HHb, tHb, Hb_{diff}, and TSI % were determined for the 20-minute CWI and for 5-minute segments. Two-way repeated-measures analysis of variance was conducted for all NIRS measures obtained in each 5-minute segment of immersion; within-participant variables were time (5, 10, 15, and 20 minutes) and temperature (10°C and 15°C). When a main effect of time was present, post hoc analysis was performed using a Bonferroni correction factor. Effect sizes were calculated using partial eta squared (η_p^2). Values were interpreted as *small* ($\eta_p^2 = 0.01$), *medium* ($\eta_p^2 = 0.06$), or *large* ($\eta_p^2 = 0.14$) effect sizes. Two-way repeated-measures analysis of variance was computed for mVO_2; within-participant variables were time (pre- and post-CWI) and temperature (10°C and 15°C). Further post hoc analysis was conducted on mVO_2 values for pre- and post-CWI at 10°C and 15°C. We set the level of significance at $P < .05$. All statistical analysis was performed using SPSS (version 25; IBM Corp).

RESULTS

Representative examples of the O_2Hb, HHb, tHb, and Hb_{diff} traces and TSI % during the experimental protocol (10°C) are displayed in Figure 2A and B, respectively. A similar response was seen in the group data.

Pre- and post-AO elicited similar responses. At the onset of AO, a steady and progressive decrease in O_2Hb concomitant with an increase in HHb concentration was present throughout the 5-minute occlusion, which reflects depletion of local O_2 stores in the static compartment of blood. A more rapid decrease in Hb_{diff} concentration occurred at the onset of AO. The Hb_{diff} represents the difference in concentration between O_2Hb and HHb and therefore provides the extent of desaturation taking place during occlusion. The TSI % signal largely followed the same trend as the O_2Hb and Hb_{diff}, with large decreases in saturation during both pre- and post-AO and minimal change during the 20-minute immersion period. The NIRS trace responses were similar for 10°C and 15°C, with no main effects ($P > .05$) across all NIRS measures for temperature or temperature × time interactions. A main effect of time was noted for O_2Hb ($F_{3,27} = 14.227$, $P = .001$, $\eta_p^2 = 0.613$), HHb ($F_{3,27} = 5.749$, $P = .009$, $\eta_p^2 = 0.344$), tHb ($F_{3,27} = 24.786$, $P = .001$, $\eta_p^2 = 0.734$), and Hb_{diff} ($F_{3,27} = 3.894$, $P = .020$, $\eta_p^2 = 0.302$) during the immersion, whereby they decreased over the 20-minute immersion period. No effect of time was evident for TSI % ($F_{3,27} = 2.859$, $P = .056$, $\eta_p^2 = 0.241$).

A nadir or flattening was reached in the O_2Hb, HHb, and Hb_{diff} signals during AO, indicating complete desaturation (Figure 3). The overlapped O_2Hb, HHb, tHb, and Hb_{diff} signals pre- and post-AO for the 10°C immersion trial are shown in Figure 4. The rate of decrease in O_2Hb ([A] in Figure 3) and Hb_{diff} ([D] in Figure 3) concentrations was noticeably slower over the 5-minute occlusion post-AO (the slope value for the line, as calculated using linear regression, confirmed this). Similar end values were achieved. This may reflect a slower depletion of local O_2 stores during the post-AO. After cuff release of both AOs, rapid resaturation occurred and was seen as an increase in O_2Hb ([A] in Figure 3) and Hb_{diff} ([D] in Figure 3) and a concomitant decrease in HHb ([B] in Figure 3), which also displayed an attenuated response during post-AO. Restoration of all signals was demonstrated after 1 to 2 minutes.

Post hoc analysis revealed reductions in the last 5-minute period of immersion (15–20 minutes) versus earlier periods of immersion for O_2Hb (10–15 minutes, $P = .005$; 5–10 minutes, $P = .03$; and 0–5 minutes $P = .007$) and tHb (10–15 minutes, $P = .001$; 5–10 minutes, $P = .002$; and 0–5 minutes, $P = .001$). In addition, we observed reductions in tHb at 10 to 15 minutes (5–10 minutes, $P = .040$; 0–5 minutes, $P = .008$) and 5 to 10 minutes (0–5 minutes, $P = .02$) compared with earlier periods of immersion. The reductions in O_2Hb and tHb represented a change of about 2 to 4 μM (Table).

A main effect of time for mVO_2 pre- and post-CWI for both temperatures ($F_{1,9} = 27.7801$, $P = .001$, $\eta_p^2 = 0.755$) was observed: mVO_2 decreased as a result of CWI (Figure 5). We did not find a main effect of temperature ($F_{1,9} = 1.996$, $P = .19$, $\eta_p^2 = 0.181$) despite the large effect size. We

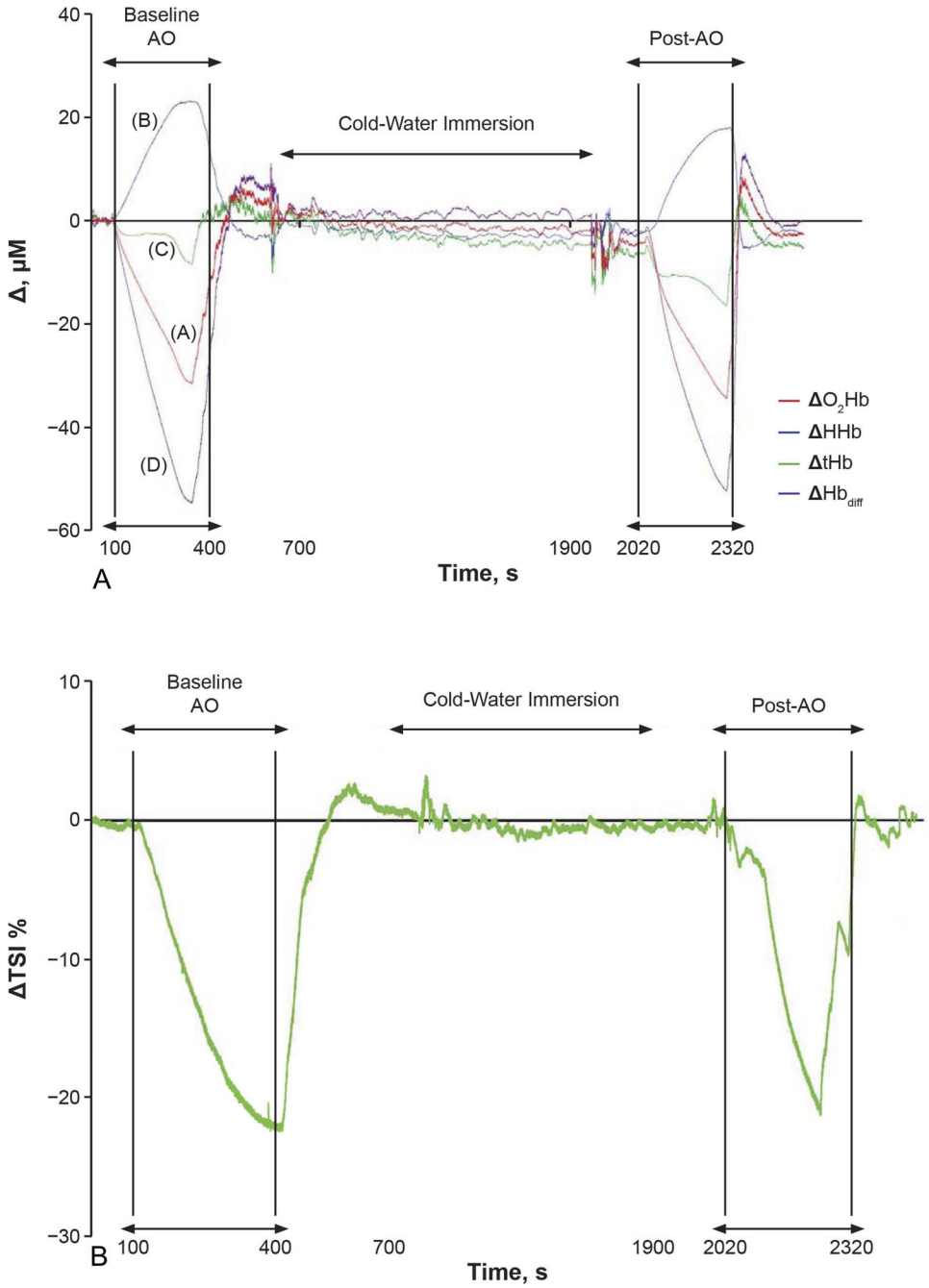

Figure 2. Oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), total hemoglobin (tHb), hemoglobin difference (Hb_{diff}), and tissue saturation index (TSI %) traces from a representative participant during the experimental protocol. Solid black lines indicate the time when arterial occlusions (AOs) took place. In the top panel, A–D denote O_2Hb, HHb, tHb, and Hbdiff traces, respectively. Data for participant 3 were not used for the representative example because the individual's preimmersion data were identified as outliers.

observed a temperature × time interaction ($F_{1,13} = 13.302$, $P = .006$, $\eta_p^2 = 0.592$), with a large effect size.

The main effect for time can be seen in the decrease in mVO_2 from preimmersion to postimmersion ($P = .001$; Figure 5). Figure 5 also shows a decrease in both temperature conditions, and this decrease was bigger postimmersion at 10°C than 15°C. The slope of the line for preimmersion versus postimmersion mVO_2 was clearly steeper at 10°C than at 15°C, as implied by the effect size and difference of the time × temperature interaction effect ($P = .006$). The large main effect for time describes the decline in mVO_2 from preimmersion to postimmersion at both temperatures (ie, the interventions were effective across both temperatures). The magnitude of the interaction effect illustrates the difference that temperature had on the preimmersion to postimmersion decline between conditions. Together, the large main effect for time and large interaction effect showed that immersion at 10°C to 15°C reduced mVO_2 and immersion at 10°C resulted in a larger reduction in mVO_2 than immersion at 15°C.

DISCUSSION

We investigated the effect of 20-minute CWI at 10°C and 15°C on gastrocnemius mVO_2 measured by NIRS. To address

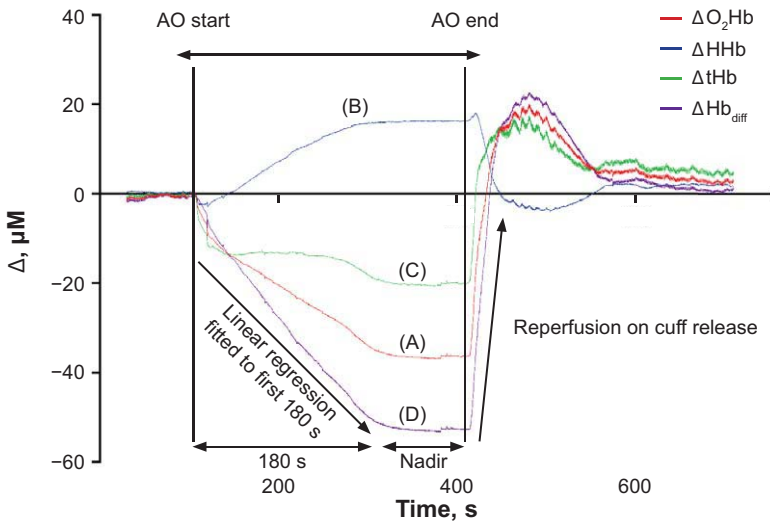

Figure 3. A representative example of oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), total hemoglobin (tHb), and hemoglobin difference (Hb_{diff}) traces during 5-minute arterial occlusions (AOs) and after cuff release (AO end). The experimental approach used to calculate muscle oxygen consumption has been annotated on the Hb_{diff} trace. (A) through (D) denote O_2Hb, HHb, tHb, and Hb_{diff} traces, respectively.

the limitations of previous research, we combined NIRS measures of hemodynamic response with the AO procedure to more robustly quantify mVO_2 post-CWI. The main findings were that CWI decreased mVO_2 after 20-minute immersion at 10°C and 15°C. A greater reduction in mVO_2 was seen at the colder temperature (10°C). These results disagree with our hypothesis that CWI would cause a postimmersion reduction in mVO_2 at 10°C but not 15°C. However, the finding that a colder immersion temperature led to a larger reduction in mVO_2 was in line with our premise.

Muscle Oxygen Consumption

Muscle oxygen consumption was reduced after 20-minute CWI at 10°C and 15°C, indicating the interventions were effective across both temperatures. The reduction was greater

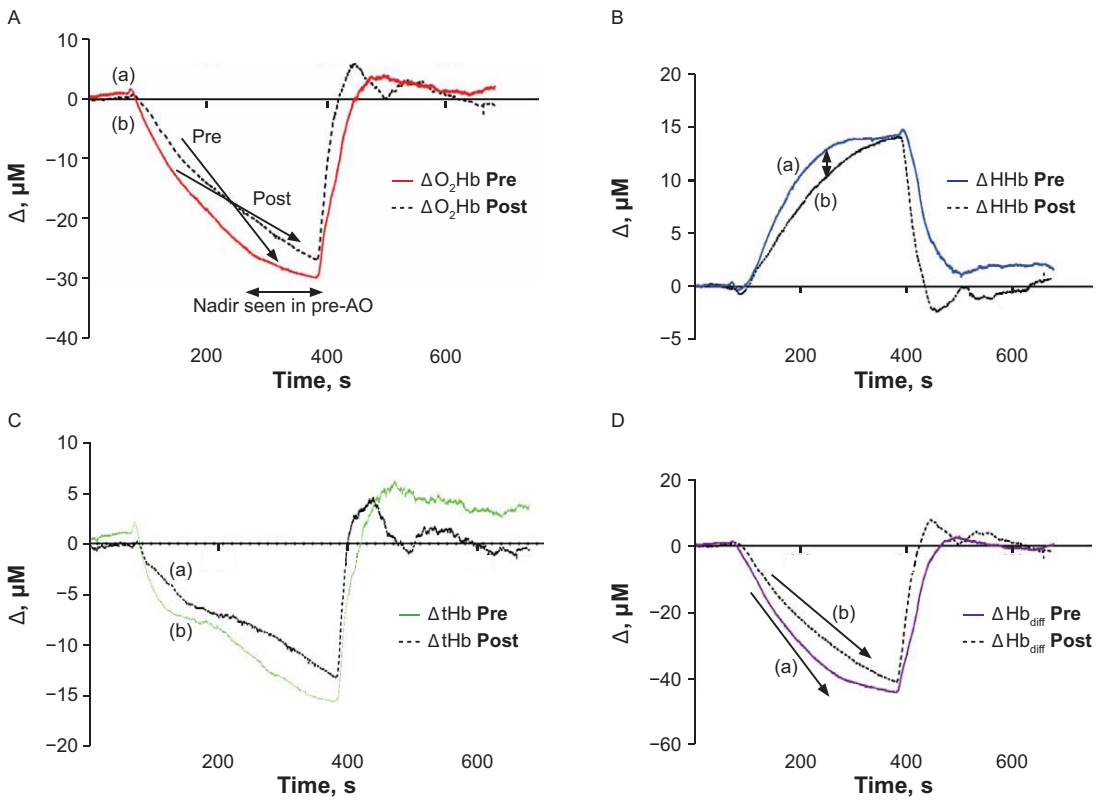

Figure 4. Data for the 10°C immersion group of the oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb), total hemoglobin (tHb), and hemoglobin difference (Hb_{diff}) traces during 5-minute arterial occlusions (AOs) and after cuff release (AO end), with the traces for baseline and post-AO overlapped for visual comparison. Data have been time aligned and normalized for comparison. A, O_2Hb. B, HHb. C, tHb. D, Hb_{diff}. (a) indicates before cold-water immersion; (b), after cold-water immersion.

Table. Results for Near-Infrared Spectroscopy Variables by 5-Minute Period During 20-Minute Cold-Water Immersion, Mean ± SD

Variable	Cold-Water Immersion at 10°C, min				Cold-Water Immersion at 15°C, min			
	0–5	5–10	10–15	15–20	0–5	5–10	10–15	15–20
Oxyhemoglobin, μM	81.2 ± 14.5	80.0 ± 13.3	79.4 ± 12.7	78.6 ± 12.7	80.9 ± 14.2	79.5 ± 13.3	79.1 ± 12.9	78.1 ± 12.8
Deoxyhemoglobin, μM	60.9 ± 15.2	60.1 ± 14.9	59.9 ± 15.0	60.0 ± 14.9	60.7 ± 14.3	60.1 ± 14.2	59.9 ± 14.4	60.1 ± 14.5
Total hemoglobin, μM	142.1 ± 29.4	140.1 ± 27.8	139.3 ± 27.4	138.7 ± 27.4	141.6 ± 28.2	139.6 ± 27.2	139.0 ± 27.0	138.3 ± 27.0
Hemoglobin difference, μM	20.2 ± 4.4	20.0 ± 5.0	19.6 ± 4.7	18.6 ± 4.3	20.1 ± 4.0	19.4 ± 3.9	19.2 ± 4.5	18.0 ± 4.7
Tissue saturation index, %	58.9 ± 3.0	59.9 ± 3.2	59.8 ± 3.5	59.3 ± 3.4	58.8 ± 2.6	59.1 ± 2.9	59.4 ± 3.1	59.0 ± 3.3

at 10°C. This outcome suggests that CWI at 10°C and 15°C reduced O_2 use in the muscle, which was consistent with the mechanism of recovery by which CWI is proposed to prevent inflammation and secondary hypoxic injury.[25,26] To accurately quantify mVO_2, we performed AOs at baseline and post-CWI. These AOs temporarily arrest blood flow, creating a "static" compartment of blood in the muscle in which the linear decrease in O_2Hb or Hb_{diff} directly reflects the rate at which the preexisting O_2 store in the blood compartment is consumed.[16] As such, this method has the advantage of quantifying the O_2 use element of the TSI % measurement while eliminating the influence of further O_2 supply. Researchers have proposed that the reduced tissue metabolism associated with CWI is mediated through reductions in tissue temperature.[5,27] While we did not measure tissue temperature, a reduction likely did occur, as authors using similar CWI protocols observed reductions in tissue temperature during and after immersion.[5,27] A 24% reduction in post-CWI mVO_2 was seen after immersion at 10°C, with a 13% reduction at 15°C, reflecting the influence of the 5°C temperature reduction on mVO_2. Changes in muscle blood volume (discussed later) also occurred with CWI at both 10°C and 15°C but only during the last 5 minutes of immersion. Therefore, an interaction between temperature and duration should be considered when interpreting muscle metabolic responses. Indeed, the greater reduction in mVO_2 at 10°C is an interesting finding and suggests a possible threshold temperature at which reductions in tissue metabolism occur and below which colder temperatures lead to greater reductions in metabolism. In fact, Wakabayashi et al[28] observed the effect of decreasing muscle forearm temperature (via cooling pads) on NIRS-derived mVO_2 and reported higher mVO_2 values at higher tissue temperatures. Yet focusing solely on immersion temperature is potentially misleading, as shown by the large interaction effect, because both time and temperature play roles in reducing tissue metabolism. The benefit at colder immersion temperatures likely relates to efficiency (ie, reduced immersion periods). However, warmer immersions for longer durations could result in the same reduction in metabolism. To the best of our knowledge, we are the first to use the AO technique with NIRS measurements to establish mVO_2 changes in response to different CWI temperatures. Our findings of reduced mVO_2 support earlier reports of reduced muscle metabolic activity.[7,8] Although an accurate quantification of muscle metabolic activity is certainly useful and extends prior results, the threshold or extent of reduced muscle metabolic activity required to aid recovery is still unknown. An optimal reduction threshold may exist, but any quantification is outside the constraints of our study.

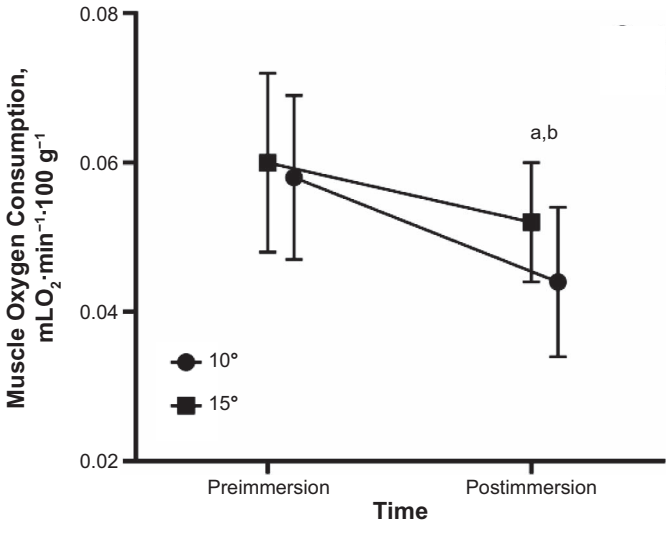

Figure 5. Muscle oxygen consumption measured at each time, mean ± SD (n = 10). [a] *P* = .001. [b] *P* = .006.

Muscle Blood Volume

The NIRS-derived measurements of muscle blood volume (ie, tHb) have shown blood volume reductions during[7,8] and after postexercise CWI,[7,29] with ΔtHb used as an indirect and proxy measure of mBF. Collectively, previous investigators[6,7,30] reported decreases in mBF during and after CWI. We saw an approximately 4-μM reduction in tHb during 20-minute CWI. Specifically, tHb was lower at 15 to 20 minutes during immersion compared with all other measured points, and the reduction in tHb was similar at both temperatures (10°C and 15°C). More substantial decreases in tHb (approximately 20 μM) have been noted over a 20-minute recovery period incorporating three 4-minute CWIs.[31] The different experimental design might be a reason for the disparate findings. Our participants performed CWI in a rested rather than immediate postexercise state, which may more accurately typify the extent of tHb change. Large increases in muscle and skin blood flow are seen postexercise due to exercise hyperemia. As such, a reduction in tHb relative to postexercise values occurs during and after CWI.[7,8] Given that our participants did not exercise before CWI, the change in tHb would be expected to be smaller and provide a greater indication of blood volume changes seen when using CWI at 24 or 48 hours postexercise. During our CWI protocol, peak changes in tHb occurred within the last 5 minutes of immersion (15 to 20 minutes), with minimal changes observed earlier. Furthermore, the 5°C difference in immersion temperature did not influence the magnitude of tHb reduction. Our results, therefore, suggested that a small reduction in muscle blood volume (tHb) can be achieved in either 10°C or 15°C water after at least 15 minutes of immersion. These outcomes may have implications for selecting immersion durations, which currently range from 30 seconds to 30 minutes.[17]

Tissue Saturation Index

Authors of a small number of studies have used the TSI % response during and after CWI, with ΔTSI % considered a proxy for changes in muscle metabolic activity.[7,8,13,29] We identified no changes in TSI % during the 20-minute CWI and marginal reductions in tHb at the end of immersion. No change in TSI %, despite a small reduction in local muscle blood volume (tHb), tentatively suggests decreased O_2 use during this period, possibly reflecting a reduction in muscle metabolic activity. This finding and interpretation have been reported previously.[8] Other researchers[7] detected no change in TSI % values during CWI despite a reduction in tHb, which the authors interpreted as a decrease in O_2 use because ΔtHb is considered a proxy for mBF. However, these and other interpretations should be viewed with caution, as the TSI % measurement is a combination of muscle O_2 supply and use, and thus inferences regarding use cannot be made with any certainty.

Limitations

This study had several limitations. First, the portable NIRS device measures only a small (2–6 cm^3) volume of muscle tissue. The values calculated for mVO_2 are assumed to represent the entire muscle, but given the restricted coverage of the gastrocnemius and its heterogeneity of fiber type and muscle metabolism,[32] the changes likely did not represent the entire muscle. To obtain representative estimations, multisite mVO_2 measurements should be assessed. Second, skin blood flow can influence the NIRS (O_2Hb and tHb) signals. For example, during whole-body heating, elevated skin blood flow influenced the NIRS signal,[14] and it has been well documented that cooling reduced cutaneous blood flow.[5–7] However, the effects of cooling on the NIRS signal have not been fully established. Our measurements of mVO_2 used the Hb_{diff} signal, which is less influenced by skin blood flow and use of AO, which creates a closed loop system, to remove the influence of skin blood flow. Finally, the study sample was exclusively male and comprised well-trained long-distance runners; therefore, extrapolation to female and other sporting populations requires further research.

CONCLUSIONS

We demonstrated that the NIRS-occlusion approach can detect changes in mVO_2 pre- and post-CWI and could provide future investigators with a simple tool to assess muscle metabolic activity. Although our results demonstrated that CWI influenced mVO_2, the significance of a reduction due to CWI is yet to be established. A key finding was that mVO_2 decreased CWI at both temperatures after 20 minutes of immersion. This reduction was most pronounced after the colder immersion temperature. This indicated that colder immersion temperatures elicited a greater decrease in muscle metabolic activity.

The role of mVO_2 in postexercise recovery protocols must be established before the optimal immersion time or temperature for CWI can be determined. Indeed, the application of CWI as a recovery tool is still controversial, with evidence to suggest its regular use can ameliorate training-induced changes after resistance training, with more beneficial findings associated with endurance-based training.[2] The NIRS-occlusion technique may offer further insight into muscle metabolic responses, beyond what is attainable from the NIRS primary signals.

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