Cold water immersion (CWI) and active recovery (ACT) are frequently used as postexercise recovery strategies. However, the physiological effects of CWI and ACT after resistance exercise are not well characterized. We examined the effects of CWI and ACT on cardiac output (Q), muscle oxygenation (SmO₂), blood volume (tHb), muscle temperature (Tmuscle), and isometric strength after resistance exercise. On separate days, 10 men performed resistance exercise, followed by 10 min CWI at 10°C or 10 min ACT (low-intensity cycling). Q (7.9 ± 2.1 l) and Tmuscle (2.2 ± 0.8°C) increased, whereas SmO₂ (−21.5 ± 8.8%) and tHb (−10.1 ± 7.7 μM) decreased after exercise (P < 0.05). During CWI, Q (−1.1 ± 0.7 l) and Tmuscle (−6.6 ± 5.3°C) decreased, while tHb (121 ± 77 μM) increased (P < 0.05). In the hour after CWI, Q and Tmuscle remained low, while tHb also decreased (P < 0.05). By contrast, during ACT, Q (3.9 ± 2.3 l), Tmuscle (2.2 ± 0.5°C), SmO₂ (17.1 ± 5.7%), and tHb (91 ± 66 μM) all increased (P < 0.05). In the hour after ACT, Tmuscle, and tHb remained high (P < 0.05). Peak isometric strength during 10-s maximum voluntary contractions (MVCs) did not change significantly after CWI, whereas it decreased after ACT (−30 to −45 Nm; P < 0.05). Muscle deoxygenation time during MVCs increased after ACT (P < 0.05), but not after CWI. Muscle reoxygenation time after MVCs tended to increase after CWI (P = 0.052). These findings suggest first that hemodynamics and muscle temperature after resistance exercise are dependent on ambient temperature and metabolic demands with skeletal muscle, and second, that recovery of strength after resistance exercise is independent of changes in hemodynamics and muscle temperature.

cryotherapy; muscle oxygenation; blood flow; recovery
letes who use cold water immersion to recover quickly between training sessions or competitive events.

The aim of this study was to examine the effects of cold water immersion on cardiac dynamics (cardiac output, heart rate, cardiac parasympathetic activity), muscle hemodynamics (SmO2 and Hb), thermoregulation (intramuscular and skin temperature), and muscle strength following resistance exercise. We also investigated changes in these variables in response to light-intensity exercise or "active recovery" following resistance exercise, because this method is used frequently as a recovery therapy for athletes (37). These two recovery therapies are very different and are difficult to compare directly. Nevertheless, contrasting physiological effects of these two modalities are a valid reflection of current practices in the training of high-performance athletes. We hypothesized that cold water immersion would reduce tissue temperature and cardiac and peripheral hemodynamics, and delay recovery of strength after resistance exercise, whereas active recovery would have the opposite effect.

METHODS

Subjects

Ten physically active men (mean ± SD; age: 21.4 ± 2.0 yr, height: 1.8 ± 0.1 m, body mass: 83.7 ± 14.8 kg), who were familiar with knee extension exercise and who had been resistance training 2 to 3 times a week for the previous 12 mo, volunteered to participate. Experimental procedures and risks were explained to the participants before they provided their informed consent to take part in the study. The study was approved by the Human Research Ethics Committee of The University of Queensland.

Experimental Design

The participants completed a familiarization trial and baseline testing 7 days before the first of two experimental trials. Each experimental trial involved unilateral knee extensor exercise with the dominant leg, followed by a 10-min period of cold water immersion or active recovery, and a 70-min recovery period. Both experimental trials were performed 7 days apart, in a randomized and counterbalanced manner. Familiarization, baseline testing, and the experimental trials were all performed in a temperature-controlled laboratory (means ± SD: temperature 24.4 ± 0.2°C, humidity 43.5 ± 1.6%).

Familiarization and Baseline Testing

The familiarization session allowed the participants to practice the unilateral leg exercise that they would perform in the experimental trials, and familiarize themselves with the requirements of the physiological measurements. Baseline testing was conducted ~60 min following familiarization and involved recording isokinetic torque during 50 maximal knee extensions (details below). The data recorded for isokinetic torque during this testing were used for comparison with postexercise values after each of the experimental trials. These data were also used to calculate the coefficient of variation of measuring the physiological variables that would be recorded during the experimental trials. We did not measure isokinetic torque before exercise on the day of each experimental trial because the 50 maximal knee extensions would have caused fatigue prior to the unilateral resistance exercise.

Experimental Trials

An overview of the experimental trials is shown in Fig. 1. All trials commenced after 9 AM. The participants were asked to eat similar food and drink 10 ml/kg body mass of water in the 2 h prior to each trial. They were asked to avoid consuming stimulants, alcohol, tobacco, antioxidants, and nutritional supplementation for 24 h preceding all trials and not to undertake any lower body strength exercise for 48 h prior to each trial. Otherwise, they were allowed to exercise between the trials, but physical activity before each trial was not monitored.

Trials commenced with a 15-min rest period, while the apparatus for recording muscle and skin temperature, cardiac dynamics, and muscle hemodynamics was inserted and/or attached. After the apparatus was set up, preexercise data for muscle and skin temperature, cardiac dynamics, heart rate variability, muscle hemodynamics, and isometric strength were collected. The participants then rested for another 5 min before they started the unilateral knee extension exercise (see exercise and performance testing). Over the first 10 min after exercise, heart rate variability was measured again (0–5 min), and the participants moved to the recovery room (5–10 min). At 10 min after exercise, the participants started one of the recovery interventions (cold water immersion or active recovery). Over 5 min after completing the recovery interventions, the participants moved back to the laboratory, where they began the 70-min recovery period. During this recovery period, muscle and skin temperature, cardiac dynamics, muscle hemodynamics, and isometric strength were measured. Resting heart rate variability and resting muscle hemodynamics were measured immediately prior to testing isometric strength at 5, 20, and 40 min during the recovery period. Isokinetic torque was measured at 60 min. We chose this monitoring period based on previous reports of greater macrovascular and microvascular blood flow in response to cold water immersion or active recovery. Over 5 min after completing the recovery interventions, the participants moved back to the laboratory, where they began the 70-min recovery period. During this recovery period, muscle and skin temperature, cardiac dynamics, muscle hemodynamics, and isometric strength were measured. Resting heart rate variability and resting muscle hemodynamics were measured immediately prior to testing isometric strength at 5, 20, and 40 min during the recovery period. Isokinetic torque was measured at 60 min. We chose this monitoring period based on previous reports of greater macrovascular and microvascular blood flow in response to cold water immersion or active recovery.

To minimize the influence of movement, shivering, and posture on cardiac dynamics and muscle hemodynamics, all trials were performed at ambient room temperature of ~24°C. Participants kept their torso and exercising leg as still as possible, while measurements were undertaken (accounting for muscle hemodynamic measurements...
taken during, and in response to exercise). The same upright posture (hip-torso angle of \( \sim 90^\circ \)) was maintained throughout each trial, other than walking to/from the recovery room. During this period, the participants adopted a regular upright walking position. Finally, the participants dried their legs with a towel following cold water immersion and covered their torso with a towel for 5 min following both recovery therapies.

**Unilateral Resistance Exercise, Isometric Strength, and Isokinetic Performance**

All exercise was performed unilaterally with the dominant leg on an isokinetic dynamometer (Cybex 6000; Computer Sports Medicine, Stoughton, MA). Unilateral exercise was implemented to isolate the quadriceps muscles, in particular, the vastus lateralis muscle for temperature and muscle hemodynamic measurements. The dynamometer position was modified to align the lateral condyle of the femur with the fulcrum, and the seat angle was fixed at 90°. The unilateral exercise bout consisted of 10 sets of 20 maximal isokinetic knee extensions in a range from 0 to 90° at a velocity of 90°/s. The participants rested in the seat of the dynamometer for 2 min between sets. Repetition tempo was set at 0.5 Hz by an audio signal, and knee flexion velocity was set at 250°/s to allow passive flexion following each knee extension.

Isometric MVC knee extension strength was measured at a knee flexion joint angle of 70° (full knee extension = 0°). For each contraction, the participants were instructed to extend their knee as forcefully as possible at 70° and continue pushing against the stop point for the required duration. The participants performed two warm-up contractions, each lasting 5 s and separated by 90 s. They then rested for another 90 s before they performed a sustained MVC contraction for 10 s. Maximum and mean isometric torques were recorded from this 10-s contraction. This procedure was repeated before the exercise bout, and 5, 20, and 40 min into the recovery period. The isokinetic performance task was performed during baseline testing (see previous details) and at 60 min during the recovery period. It consisted of 50 sequential maximal isokinetic knee extensions, using the same range, velocity, and tempo as the exercise bout.

All data from the dynamometer were collected at 1,000 Hz using a custom-designed LabVIEW script (LabVIEW, National Instruments, Austin, TX), and stored on a personal computer for offline analysis. Test-retest coefficients of variation were determined from the muscle function testing before both trials. The coefficient of variation for peak torque from the 10-s isometric contraction was 2.5%. The coefficient of variation for total work performed over the isokinetic task was 3.2%.

**Recovery Interventions**

Recovery interventions consisted of cold water immersion or active recovery. For the cold water immersion therapy, the participants adopted a seated position with a hip angle of \( \sim 90^\circ \), with their legs outstretched and fully relaxed. This helped to minimize any confounding effects of muscle contractions on the NIRS signals during the immersion period. The participants were immersed up to the level of the umbilicus continuously for 10 min in an inflatable bath (iBody, iCool Australia, Miami, Australia) containing water at 10.0 ± 0.2°C. The participants only immersed their body to the level of their umbilicus, because immersion up to the neck may have interfered with the impedance cardiology electrodes placed on the torso (see **Cardiac Dynamics**). Water temperature in the bath was continuously maintained using a circulating cooling unit (iCool Lite, iCool Australia). For the active recovery therapy, the participants were instructed to exercise on a cycle ergometer (Wattbike, Wattbike, Nottingham, UK) for 10 min at a low, self-selected intensity, also adopting a hip angle of \( \sim 90^\circ \). The participants cycled a distance of 3.4 ± 0.3 km, at an average power output of 41.1 ± 10.3 W during active recovery.

**Cardiac Dynamics**

Heart rate, stroke volume, and cardiac output were measured continuously by impedance cardiology (Physioslow, Manatec Biomedical, Paris, France). This method detects changes in impedance of alternating low-magnitude electrical current between electrodes placed on the neck (above the supra-clavicular fossa) and the xiphoid process. Calibration was completed at rest before exercise by collecting data over 30 cardiac cycles and inputting data for diastolic and systolic blood pressure measured using an automated sphygmmomanometer (Digital blood pressure monitor, UA-767; ADInstruments, Oxford, UK) over the same period. Blood pressure was recorded again and updated in the calibration 1 min before and 5 min after cold water immersion/active recovery. Data were sampled at two-beat intervals and saved on a personal computer for offline analysis. Test-retest coefficients of variation using the Physioslow at rest were determined between the resting measures collected at the start of each trial. The coefficient of variation at rest was 1.7% for heart rate, 4.1% for stroke volume, and 2.9% for cardiac output. Postexercise coefficients were calculated using data collected during baseline testing and the first experimental trial. The coefficient of variation postexercise was 5.4% for heart rate, 7.0% for stroke volume, and 4.0% for cardiac output.

Heart rate variability was assessed from the time domain by recording the natural logarithm of the square root mean of the sum of the squared differences between adjacent normal sequential R-R intervals (Ln rMSSD). This measurement provided a proxy estimate of parasympathetic nervous activity of the heart (1). R-R intervals were recorded while the participants were seated, using a heart rate monitor (Suunto T6c, Suunto Oy, Vantaa, Finland) at a sampling frequency of 1,000 Hz. Ln rMSSD was calculated for R-R intervals recorded before exercise (PRE), immediately after the resistance exercise (POST), during the recovery interventions (REC), between 0 and 5 min (5 min), 15 and 20 min (20 min), and 35 and 40 min (40 min) during the recovery period. Respiration rate was not controlled during these measurement periods, because heart rate variability indices of parasympathetic activity are similar during controlled or spontaneous breathing (3), and do not influence Ln rMSSD (33). Data files were transferred to a personal computer using Suunto Team Manager Software (Suunto T6c, Suunto Oy, Vantaa, Finland). Offline analysis was conducted from 2 to 5 min during each 5-min interval, and from 2 to 9 min during REC.

**Muscle Hemodynamics**

Muscle hemodynamics were assessed using a portable NIRS system (Portamont, Artinis Medical Systems, Elst, The Netherlands). The NIRS device emits light at 760- and 850-nm wavelengths from three optodes, with an average optode-detector distance of 35 mm. Penetration depth of the light below the skin surface was estimated at 17.5 mm, or half the distance between the optode and the detector (29, 49).

The NIRS probe was placed on the midline of the vastus lateralis muscle, one-third of the linear distance between the superior border of the patella and the inguinal fold. Probe location measurements were recorded and outlined with a marker for repositioning during the second experimental trial. The NIRS probe was covered with a black plastic cloth to protect it from ambient light. It was then placed in a transparent sealed polyethylene bag for waterproofing and firmly secured to the leg to prevent water leaking into the space between the bag and the skin, which could potentially influence scattering of the light (21). Pilot testing revealed no significant impact of the polyethylene bag upon SmO2 and tHb values. The combined adipose tissue and skin thickness of the participants was measured by skinfold calipers (Harpenden skinfold caliper, Baty International, West Sussex, UK). Mean ± SD adipose tissue and skin thickness was 6.5 ± 3.4 mm, calculated as the skinfold thickness divided by two. Therefore, the intramuscular NIRS penetration depth was estimated at 11.5 ± 3.4
mm, calculated as 17.5 mm minus adipose tissue and skin thickness. At this penetration depth, the NIRS signal mainly reflects the metabolic and hemodynamic changes of the muscle (10). The NIRS method is both reliable and valid for the measurement of muscle oxidative metabolism (39). The coefficient of variation for test-retest reliability of resting SmO2 was 2.3% in this investigation.

Data were recorded online at 10 Hz during all trials, using native software (Oxysoft version 2.1.6; Artinis Medical Systems). The software calculates changes in light absorption at the different wavelengths and converts them to relative concentrations of O2Hb and deoxyhemoglobin (HHb) using the modified Lambert law to correct for light scattering within the tissue. tHb was calculated as O2Hb + HHb and SmO2 was calculated as \[ [O_2Hb/Hb] \times 100 \] using the spatially resolved spectroscopy method. Off-line analysis was performed using the same software.

SmO2 and tHb were measured before and after exercise and during the recovery interventions. Changes in SmO2 and tHb were also measured during the 10-s isometric MVCs, which the participants performed before the exercise bout, and 5, 20, and 40 min into the recovery period. These 10-s isometric MVCs were used in place of arterial occlusions to obtain a measure of O2 consumption because O2 delivery is substantially occluded due to increased intramuscular pressure of MVCs compressing blood vessels (40). Precontraction SmO2 and tHb were calculated as the mean from -4 to -1 s prior to the visually estimated onset of the MVCs and isokinetic task. The exact onset of the MVCs and isokinetic task was identified as the time at which tHb decreased below the mean from -4 to -1 s + 2 × SD of the mean from -4 to -1 s. The following muscle hemodynamic variables were calculated (11) (Fig. 2: 1) minimum ΔSmO2 amplitude (SmO2min), which corresponds to the difference between minimum SmO2 during the contraction and mean SmO2 over -4 to -1 s prior to the onset of the contraction. Larger Δ (i.e., more negative) SmO2min values indicate greater O2 consumption relative to O2 delivery; 2) SmO2 ½ deoxygenation time (SmO2½DT), which corresponds to the period of time between the start of the contraction until SmO2 reaches 50% of the difference between baseline SmO2 and the minimum SmO2 during the contraction. A shorter SmO2½DT (for a similar SmO2min) represents a faster O2 consumption rate; 3) SmO2 maximum amplitude (SmO2max Δt) was identified as the amplitude corresponding to the maximum SmO2% within 120 s following the end of the contraction; and 4) SmO2 ½ reoxygenation time (SmO2½RT), which corresponds to the time required (from the end of the contraction) for SmO2 to reach 50% of SmO2max. A lower value (i.e., faster time) indicates greater O2 delivery relative to O2 consumption.

**Temperature Measurement**

Intramuscular temperature was measured using a fine-wire implantable probe (T204E; Physitemp Instruments, Clifton, NJ), while skin temperature was measured with a surface probe (SS-1; Physitemp Instruments). Temperature was logged at a frequency of 1 Hz using a portable data logger (SQ2020; Grant Instruments, Cambridge, UK), and transferred to a portable computer for analysis. An 18-gauge needle was used to insert the implantable probe to a depth of 18 mm beneath the skin surface, 5 cm superior to the NIRS probe. To ensure consistency in the depth of insertion, a piece of medical tape was placed exactly 18 mm from the end of the probe. The probe was then inserted into the muscle until the tape contacted the skin surface. This depth was chosen because it was in the same region as the estimated 17.5 mm subcutaneous working depth of the NIRS probe. Taking into account the thickness of the adipose tissue and skin at this site, the temperature probe was inserted at a mean ± SD intramuscular depth of 11.5 ± 3.4 mm. Once the intramuscular temperature probe was at the required depth, the needle was removed, leaving the probe in place. The skin temperature probe was placed on the quadriceps immediately next to the medial border of the NIRS device. Data were averaged at 1-min intervals before and after the exercise bout, during the recovery interventions, and every 2 min for the recovery period.

**Statistical Analysis**

Statistical analysis was conducted using the Statistical Package for Social Sciences program (version 21; IBM, New York). All data were initially assessed for normality using the Shapiro-Wilk formula. All measures were assessed by one-way ANOVA for each trial. When significant time effects were evident, paired t-tests were used to compare changes over time, and the false discovery rate was used to correct P values for these multiple comparisons. To complement these statistical comparisons, Cohen’s effect sizes (d) were calculated to illustrate the magnitudes of differences in muscle function and physiological variables over time. Effect sizes were assessed as 0.2 = small effect, 0.5 = moderate effect, and ≥0.8 = large effect. Data collected before exercise and prior to the MVCs at 5, 20, and 40 min after cold water immersion/active recovery were pooled to calculate Pearson’s correlations between muscle temperature, skin temperature, cardiac output, heart rate, stroke volume, SmO2, and tHb. Data for SmO2 and tHb during cold water immersion/active recovery were averaged over 10 min to calculate Pearson’s correlations with mean arterial pressure measured 5 min after cold water immersion/active recovery. All data are presented as means ± SD. Significance was set at a level of P < 0.05.

**RESULTS**

**Resistance Exercise Bout**

The total work completed during the unilateral knee extension exercise bout did not differ (P = 0.2) between the active recovery (22.2 ± 4.4 kJ) and cold water immersion (22.8 ± 5.9 kJ) trials.

**Cardiac Dynamics**

After exercise and during recovery interventions. Systolic, diastolic, and mean arterial pressures increased significantly after exercise compared with before exercise (P < 0.05) and did not differ significantly before the two recovery interventions (Table 1). Systolic blood pressure remained higher after cold water immersion than before exercise, whereas systolic,
diastolic, and mean arterial blood pressure remained higher after active recovery than before exercise ($P < 0.05$).

Heart rate (Fig. 3A), stroke volume (Fig. 3B), and cardiac output (Fig. 3C) increased significantly during exercise in both trials ($P < 0.05$). Heart rate ($P = 0.84$), stroke volume ($P = 0.89$), and cardiac output ($P = 0.78$) did not differ before the two recovery interventions. Heart rate remained higher during cold water immersion than before exercise ($P < 0.05$) over the first 2 min and returned to preexercise values after 5 min. Stroke volume decreased gradually during cold water immersion and tended to be lower than before exercise at 7–10 min ($P = 0.1$ to 0.06; $d = -0.3$ to $-0.5$). Consistent with the decrease in stroke volume, cardiac output also decreased during cold water immersion and was lower than before exercise at 8–10 min ($P < 0.05$). During active recovery, heart rate was higher than before exercise and remained elevated ($P = 0.001$; $d = 1.3$ to 4.1). Stroke volume ($P = 0.08$ to 0.03; $d = 0.4$ to 0.6) tended to remain above the preexercise value, and cardiac output ($P = <0.0001$ to 0.001; $d = 2.1$ to 2.4) remained above the preexercise value throughout the recovery therapy.

After the recovery interventions. Heart rate was lower than before exercise at 50–70 min after cold water immersion ($P < 0.05$; $d = 0.7$ to 1.0) but remained higher than before exercise during the entire period following active recovery. Cardiac output also decreased below the preexercise rate at 30–60 min after cold water immersion ($P < 0.05$; $d = 0.7$ to 1.4), whereas it remained above the preexercise value until 20 min after active recovery.

Heart Rate Variability

Ln rMSSD was used as a marker of cardiac parasympathetic activity. Ln rMSSD was lower after the exercise bout in both trials ($P < 0.01$; $d = -2.4$) (data not shown). It rapidly returned toward the preexercise value during cold water immersion ($P = 0.003$; $d = 1.8$), whereas it remained lower than the preexercise value at 5 min after active recovery.

Muscle Hemodynamics

During the recovery interventions. During cold water immersion, SmO$_2$ did not change from minute 1 values ($P > 0.5$), whereas it decreased progressively during active recovery ($P = 0.065$ to 0.004) (Fig. 4A). During cold water immersion, tHb increased above the prerecovery value during the first 4 min (Fig. 4B). It then decreased slightly over the remaining 6 min, but remained higher than the minute 1 value during cold water prerecovery value from minute 3 onward during active recovery ($P < 0.05$; $d = 0.4$ to 1.2). Pooled data for SmO$_2$ and tHb during cold water immersion and active recovery did not correlate with mean arterial pressure measured 5 min after these recovery interventions (SmO$_2$: $r = 0.10$; $P = 0.72$ and tHb: $r = -0.16$; $P = 0.56$).

After the recovery interventions. SmO$_2$ did not change significantly compared with prerecovery values after either trial (time effect: $P = 0.093$) (Fig. 4C). tHb was lower than prerecovery values 5 min ($P = 0.003$; $d = 2.1$) and 20 min ($P = 0.033$; $d = 1.7$) after cold water immersion, whereas it was higher than before exercise 40 min after active recovery ($P = 0.037$; $d = 1.3$) (Fig. 4D). Pooled data for resting SmO$_2$ before exercise and before the MVCs at 5, 20, and 40 min after cold water immersion and active recovery correlated with muscle temperature ($r = 0.59$; $P < 0.01$) and tHb ($r = 0.34$; $P = 0.002$). Pooled data for tHb at the same time points correlated with muscle temperature ($r = 0.62$; $P < 0.001$), skin temperature ($r = 0.59$; $P < 0.001$), and cardiac output ($r = 0.38$; $P < 0.001$).
Skin and Muscle Temperature

After exercise and during the recovery interventions. Muscle and skin temperatures were higher than preexercise temperatures immediately after resistance exercise and before the recovery interventions in both trials ($P < 0.001$) (Fig. 5). There were no differences in muscle temperature ($P = 0.32$) or skin temperature ($P = 0.38$) before the recovery interventions. Muscle ($d = 0.3$ to 2.1) and skin ($d = 0.3$ to 3.3) temperature decreased below preexercise values during cold water immersion ($P < 0.05$), whereas muscle ($d = 4.0$ to 4.8) and skin ($d = 2.1$ to 3.7) temperature remained elevated above preexercise throughout the active recovery exercise ($P < 0.001$).

After the recovery interventions. Muscle temperature remained below the preexercise value for 40 min following cold water immersion ($P < 0.05$; $d = 1.4$ to 4.5), whereas skin temperature remained below the preexercise value for the entire period ($P < 0.05$; $d = 0.7$ to 6.0) (Fig. 5). Muscle ($d = 2.7$ to 3.8) and skin ($d = 3.3$ to 4.7) temperatures remained above preexercise values for the entire period after active recovery ($P < 0.001$). Both muscle ($r = 0.26$; $P = 0.019$) and skin temperature ($r = 0.38$; $P < 0.001$) correlated with cardiac output.

Recovery of Muscle Strength

Peak isometric torque did not change after cold water immersion ($P = 0.24$ to 0.62), whereas it was lower than before exercise at 5, 20, and 40 min ($P < 0.05$; $d = 0.4$ to 0.7) after the active recovery trial (Fig. 6). The total amount of work completed during the 50 isokinetic contractions was similar at baseline (i.e., preexercise) compared with when the same contractions were performed ~70 min after exercise (Table 3). During the 50 contractions performed ~70 min postexercise, isokinetic torque decreased progressively over time (time effect $P < 0.001$) in both the cold water immersion and active recovery trials.

Muscle Hemodynamics in Response to Muscle Contractions

Compared with preexercise, the SmO$_2$ change during the 10 s MVC was smaller ($P < 0.05$) at 5 min ($d = 1$ and 1.3) and 20 min ($d = 0.6$ and 1.4) after both cold water immersion and active recovery, respectively (Fig. 7A) and remained lower at 40 min ($d = 1.4$) after active recovery. Compared with preexercise, SmO$_2$ during the MVCs did not change significantly at any time after cold water immersion ($P = 0.155$ to 0.631). SmO$_2$ was longer compared with preexercise values at 20 min ($P = 0.001$; $d = 1.3$) and 40 min ($P = 0.003$; $d = 1.2$) after active recovery (Fig. 7B). Compared with preexercise values, SmO$_2$ after the MVCs tended to be longer at $20$ min ($P = 0.1; d = 0.4$) and $40$ min ($P = 0.052; d = 0.5$) after cold water immersion (Fig. 7B). SmO$_2$ was not significantly different to preexercise values at any time ($P > 0.05$) after active recovery. SmO$_2$ was lower than baseline (preexercise) values after the isokinetic contractions in the active recovery trial ($P < 0.05$; $d = 3.2$) but not in the cold water immersion trial (Table 3). There were no significant changes in SmO$_2$ during the isokinetic contractions or SmO$_2$ after the isokinetic contractions in either trial (Table 3).

DISCUSSION

The aim of this study was to examine the effects of cold water immersion and active recovery on cardiac dynamics,
muscle hemodynamics, tissue temperature, and strength following resistance exercise. Cold water immersion reduced hemodynamics and tissue temperature and helped to maintain muscle strength after resistance exercise. By contrast, active recovery maintained hemodynamics and tissue temperature and reduced muscle strength after resistance exercise. Collectively, these findings suggest that 1) hemodynamics and muscle temperature after resistance exercise are dependent on ambient temperature and metabolic demands of skeletal muscle, and 2) recovery of strength after resistance exercise is independent of changes in hemodynamics and muscle temperature. Although some of these findings were somewhat predictable, they are nevertheless interesting and valuable, because they represent the first systematic comparison of the physiological effects of these two frequently used recovery therapies.

This is the first study to investigate the effects of cold water immersion on stroke volume and cardiac output during recovery from resistance exercise. Cardiac output and stroke volume decreased rapidly after exercise in response to cold water immersion, whereas they remained moderately elevated in response to active recovery (Fig. 3). The decrease in cardiac output during cold water immersion probably reduced blood flow to peripheral regions of the body to protect and maintain core temperature. By contrast, the maintenance of cardiac output during active recovery most likely conserved oxygen delivery to contracting muscle. These differences in cardiac dynamics between cold water immersion and active recovery probably reflect divergent activity of the parasympathetic and sympathetic nervous systems (1, 43). As a proxy measure of parasympathetic nervous activity, ln rMSSD increased after cold water immersion (data not shown), which provides tentative evidence that cardiac output decreased as a result of greater parasympathetic nervous activity. By contrast, ln rMSSD remained low after active recovery, which suggests that cardiac output remained elevated as a result of lower parasympathetic nervous activity.

We used NIRS to examine changes in muscle oxygenation and blood volume during and after cold water immersion and active recovery (Fig. 5). Muscle oxygenation amplitude (SmO2) decreased rapidly in response to cold water immersion, whereas it remained moderately elevated in response to active recovery (Fig. 7). The decrease in muscle oxygenation during cold water immersion probably reduced oxygen delivery to contracting muscle. These differences in muscle dynamics between cold water immersion and active recovery probably reflect divergent activity of the parasympathetic and sympathetic nervous systems (1, 43). As a proxy measure of parasympathetic nervous activity, ln rMSSD increased after cold water immersion (data not shown), which provides tentative evidence that cardiac output decreased as a result of greater parasympathetic nervous activity. By contrast, ln rMSSD remained low after active recovery, which suggests that cardiac output remained elevated as a result of lower parasympathetic nervous activity.
active recovery. SmO$_2$ did not change significantly during cold water immersion but decreased during active recovery. This difference reflects the greater demand for oxygen in contracting muscle during active recovery. tHb increased during cold water immersion (Fig. 4B), indicating an increase in muscle blood volume similar to the cold-induced vasodilation observed by Gregson et al. (13). This is the first study to combine measurements of central and peripheral hemodynamics after exercise. It is intriguing that muscle blood volume (tHb) increased during cold water immersion, whereas cardiac output decreased. This suggests that changes in central blood flow do not necessarily influence local perfusion within skeletal muscle. In contrast to the increase during cold water immersion, tHb decreased for at least 20 min thereafter (Fig. 4D), which may reflect microvascular adaptation to the cold.

Previous research has used NIRS only to examine resting muscle oxygenation and blood volume after cold water immersion (18, 46). We have extended current knowledge of the effects of cold water immersion by evaluating changes in muscle oxygenation in response to occlusion and hyperemia associated with MVCs. SmO$_2$ amplitude (SmO$_2$min) and deoxygenation time (SmO$_2$2/DT) derived from NIRS measurements indicate the demand for oxygen in contracting muscle (11, 29). SmO$_2$ amplitude (SmO$_2$min) and deoxygenation time (SmO$_2$2/DT) indicated an increase in muscle oxygenation in response to occlusion and hyperemia associated with MVCs. SmO$_2$ amplitude (SmO$_2$min) and deoxygenation time (SmO$_2$2/DT) derived from NIRS measurements indicate the demand for oxygen in contracting muscle (11, 29).

Another new and important finding from this study is that cold water immersion prevented any decrease in maximal isometric strength after resistance exercise (Fig. 6). By contrast, strength remained below preexercise values for at least 40 min after active recovery. Previous research on the short-term effects of cold water immersion on muscle function has produced inconsistent findings. Similar to our findings, Pointon et al. (35) reported that cold water immersion helped to maintain maximal isometric strength immediately after 30 min intermittent sprint exercise. By contrast, other research has demonstrated no effect of cold water immersion on maximal isometric strength at rest (i.e., with no prior exercise) (52) or 1–2 h after exhaustive cycling (31, 32) or resistance exercise (34, 38). This variability may be due to differences in cold water immersion protocols (temperature, duration, or body surface area immersed) and the metabolic and neuromuscular demands of exercise. In the present study, cold water immersion may have helped to maintain strength after resistance exercise through direct and indirect mechanisms involving group III and IV afferents. Cold exposure inhibits the activity of group III and IV afferents in skeletal muscle (24) and reduces the accumulation of lactic acid during muscle contractions (8). Lactic acid stimulates group III and IV afferents in skeletal muscle, resulting in perceptions of fatigue and pain (36). By inhibiting the activity of group III and IV afferents and reducing lactic acid accumulation in muscle after exercise (53), cold water immersion may have minimized the perceptions of fatigue and pain. In turn, this could have allowed the participants to produce greater force during MVCs following cold water immersion. Although cold water immersion reduced resting muscle blood volume (Fig. 4D) and tended to slow the rate of SmO$_2$ reoxygenation after MVCs (Fig. 7B), these effects did not seem to influence muscle function. Active recovery could have interfered with the restoration of sarcoplasmatic reticulum function and Ca$^{2+}$ signaling, which have been linked to prolonged muscle fatigue after exercise (16, 50).

Table 2. Peak isokinetic torque (Nm) during the fatigue task at baseline, and during the cold water immersion and active recovery trials

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<td>Baseline</td>
<td>137±30</td>
<td>152±23</td>
<td>147±20</td>
<td>135±25</td>
<td>123±23$^*$</td>
<td>111±24$^*$</td>
<td>99±24$^*$</td>
<td>86±22$^*$</td>
<td>79±19$^*$</td>
<td>75±17$^*$</td>
</tr>
<tr>
<td>CWI</td>
<td>135±29</td>
<td>156±30</td>
<td>141±31</td>
<td>133±26</td>
<td>115±25$^*$</td>
<td>102±20$^*$</td>
<td>93±18$^*$</td>
<td>86±18$^*$</td>
<td>82±14$^*$</td>
<td>81±18$^*$</td>
</tr>
<tr>
<td>ACT</td>
<td>140±29</td>
<td>150±36</td>
<td>144±39</td>
<td>128±42</td>
<td>116±34$^*$</td>
<td>101±32$^*$</td>
<td>91±32$^*$</td>
<td>82±30$^*$</td>
<td>79±26$^*$</td>
<td>78±26$^*$</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD. Baseline performance was measured 7 days prior to the first trial and was used as a “preexercise” comparison for both experimental trials; see MATERIALS AND METHODS. *Significantly different from contractions 1–5 (P < 0.05).

Table 3. Total work and changes in muscle oxygenation and blood volume during and for 2 min after 50 isokinetic contractions

<table>
<thead>
<tr>
<th>Isokinetic Task Variable</th>
<th>Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Total work, kJ</td>
<td>3.5 ± 0.7</td>
</tr>
<tr>
<td>Contractions 1–25</td>
<td>2.2 ± 0.6</td>
</tr>
<tr>
<td>Contractions 26–50</td>
<td>1.1 ± 3.3</td>
</tr>
<tr>
<td>tHb minimum amplitude, μM</td>
<td>10.0 ± 5.7</td>
</tr>
<tr>
<td>SmO$_2$max, %</td>
<td>14.7 ± 8.4</td>
</tr>
<tr>
<td>SmO$_2$2/DT, s</td>
<td>11.7 ± 9.3</td>
</tr>
<tr>
<td>SmO$_2$2/RT, s</td>
<td>22.5 ± 29.4</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD. CWI, cold water immersion; ACT, active recovery trials; SmO$_2$, muscle oxygenation; tHb, blood volume. Baseline performance was measured 7 days prior to the first trial and was used as a “preexercise” comparison for both experimental trials; see MATERIALS AND METHODS. *Significantly different from baseline, P < 0.05.
Several theoretical and technical issues relating to our measurements of cardiac dynamics, muscle oxygenation, and blood volume are worth considering. First, our findings are specific to the context in which we applied cold water immersion and active recovery. The physiological and functional effects of these recovery treatments will most likely vary depending on the situation. For example, these treatments may induce different effects when they are applied during other phases of postexercise recovery or after other modes of exercise. The alterations in muscle temperature and central and peripheral hemodynamics in response to cold water immersion or active recovery may also influence other aspects of muscle function/exercise performance in different ways.

Second, impedance cardiography and NIRS offer advantages over other more invasive, complicated, or costly methods for assessing central and peripheral hemodynamics. However, we acknowledge that these methods are not without limitations. The main limitation of impedance cardiography is that it is based only on estimates of stroke volume. Anecdotal evidence suggests that impedance cardiography is also affected by shivering (2). The participants in our study probably did shiver to some degree while sitting in cold water, but we can only speculate how much this may have influenced the impedance cardiography data. Impedance cardiography also assumes that central venous pressure remains constant, but it is unlikely that it was constant during cold water immersion or active recovery. Some studies have reported that tissue oxygenation measured using NIRS fluctuates with changes in skin blood flow, which has raised doubts as to whether NIRS signals accurately reflect tissue oxygenation within skeletal muscle (5, 6, 22, 45). The accuracy of the NIRS signal depends on the depth at which the light from the probe penetrates through skin and adipose tissue into skeletal muscle (9). We estimate that our NIRS probe penetrated to 11.5 ± 3.4 mm within skeletal muscle. At this penetration depth, we contend that the changes in SmO2 and Hb genuinely reflect differences in tissue oxygenation in the muscle, rather than experimental artifacts arising from changes in skin temperature and/or blood flow (23).

Third, the lack of any extended warm-up before the MVCs could have influenced muscle function, particularly at 20 and 40 min after exercise. We did not allow the participants to warm up their muscles for any extended period because this would probably have confounded the effects of the prior recovery interventions. We contend that the muscle function tests are valid because 1) we explicitly set out to examine how differences in muscle temperature after exercise influence recovery of muscle strength and 2) we followed the same protocol in both experimental trials. Last, we were careful not to suggest or imply any benefits of cold water immersion over active recovery to the participants in this study. However, it was impossible to blind the participants to the treatments that they received. The participants may also have had some preconceptions about the benefits of cold water immersion as a recovery strategy that could have influenced how they performed in the muscle function tests. Notwithstanding these limitations, we believe our findings advance existing knowledge of the physiological effects of cold water immersion after exercise.


