ABSTRACT

Cold water immersion (CWI) and active recovery (ACT) are frequently used as post-exercise 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 ($\dot{Q}$), muscle oxygenation ($\text{SmO}_2$) and blood volume ($\text{tHb}$), muscle temperature ($T_{\text{muscle}}$) 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). $\dot{Q}$ (7.9±2.7 l) and $T_{\text{muscle}}$ (2.2±0.8°C) increased, whereas $\text{SmO}_2$ (−21.5±8.8%) and $\text{tHb}$ (−10.1±7.7 μM) decreased after exercise (p<0.05). During CWI, $\dot{Q}$ (−1.1±0.7 l) and $T_{\text{muscle}}$ (−6.6±5.3°C) decreased, while $\text{tHb}$ (121±77 μM) increased (p<0.05). In the hour after CWI, $\dot{Q}$ and $T_{\text{muscle}}$ remained low, while $\text{tHb}$ also decreased (p<0.05). By contrast, during ACT, $\dot{Q}$ (3.9±2.3 l), $T_{\text{muscle}}$ (2.2±0.5°C), $\text{SmO}_2$ (17.1±5.7%) and $\text{tHb}$ (91±66 μM) all increased (p<0.05). In the hour after ACT, $T_{\text{muscle}}$ and $\text{tHb}$ 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 firstly that hemodynamics and muscle temperature after resistance exercise are dependent on ambient temperature and metabolic demands with skeletal muscle, and secondly, that recovery of strength after resistance exercise is independent of changes in hemodynamics and muscle temperature.

Key words: cryotherapy, muscle oxygenation, blood flow, recovery.
INTRODUCTION

Cryotherapy treatments such as cold water immersion, ice baths and ice massage are frequently used as an aid to recovery from exercise. The use of this method is based partly on the psychological benefits of these treatments, such as lower ratings of muscle fatigue and soreness (51). Some studies have also reported benefits of cold water immersion treatments on muscle function and various aspects of exercise performance [for review see (51)]. Despite its popularity and anecdotal support, the physiological mechanisms underlying the effects of cold water immersion during the post-exercise period are not well established.

Various studies have examined changes in central hemodynamics (e.g., cardiac output, blood pressure, total peripheral resistance) and limb blood flow following immersion in cold water (i.e., ≤15°C) under resting conditions (4, 13, 20, 28, 41, 42). Other studies have investigated the effects of cold water immersion on central and peripheral hemodynamics during recovery from endurance exercise (18, 26, 44, 48). However, the findings from these studies are not necessarily applicable to recovery from resistance exercise, because autonomic regulation of the cardiovascular system differs between recovery from endurance exercise and recovery from resistance exercise (15). Cold water immersion may therefore induce different cardiovascular responses following resistance exercise compared with endurance exercise.

Previous research has used near infrared spectroscopy (NIRS) to measure changes in peripheral hemodynamics, including muscle tissue oxygenation (oxyhemoglobin concentration [O$_2$Hb] or muscle oxygen saturation [SmO$_2$]) and muscle blood volume (total hemoglobin concentration [tHb]) in response to cold water immersion after endurance exercise (18, 44) or to icing after eccentric contractions of the elbow flexors (46). Some of these studies have produced variable findings in relation to changes in SmO$_2$, and only included ‘passive’ measurements of SmO$_2$ and tHb at rest after exercise (18, 46). Measuring changes in SmO$_2$ and tHb in response to maximal muscle contractions arguably provides a
better indication of muscle O$_2$ consumption (rate and level of SmO$_2$) compared with passive measurements (29). Considering the inconsistencies and limitations of previous research (18, 46), more research is needed to gain a better understanding of how cold water immersion affects muscle hemodynamics, particularly after traditional resistance exercise.

A number of studies have evaluated the effects of cold water immersion or ice packs on recovery of strength after eccentric exercise (17, 47), plyometrics (12, 19), resistance exercise (34, 38), intermittent sprint exercise (35) and exhaustive cycling (31, 32). However, many of these studies only measured strength after 24 h of recovery from previous exercise. Less is known about short-term recovery of strength after exercise. Cold exposure can impair muscle function, possibly by altering neuromuscular properties during muscle contractions (7, 30, 52). These effects could have important implications for athletes 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 (SmO$_2$ and tHb), 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 is a valid reflection of current practices in the training of high-performance athletes. We hypothesized that cold water immersion would reduce tissue temperature, cardiac and peripheral hemodynamics, and delay recovery of strength after resistance exercise, whereas active recovery would have the opposite effects.

METHODS
**Subjects**

Ten physically active men (mean ± SD age: 21.4 ± 2 years, 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 months 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 d 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 d apart, in a randomized and counter-balanced manner. Familiarization, baseline testing and the experimental trials were all performed in a temperature-controlled laboratory (mean ± 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 approximately 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 post-exercise 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 illustrated in Figure 1. All trials started at 9 a.m. The participants were asked to eat similar food, and drink 10 ml·kg⁻¹ 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 do 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, pre-exercise 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 in 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 macro- and micro-vascular blood flow in response to cold water immersion at rest (13) 
and following exercise (26)

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 whilst measurements were 
undertaken (accounting for muscle hemodynamic measurements taken during, and in 
response to exercise). The same upright posture (hip-torso angle of ~90°) 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 
minutes 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, CSMI, Stoughton, MA, USA). 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⁻¹. 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⁻¹ 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 torque were recorded from this 10 s contraction. This procedure was repeated before the exercise bout, and 5 min, 20 min 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 Corp., Texas, USA), 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 ~90°, 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 Pty Ltd., Miami, Australia) containing water at 10 ±
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 cardiography electrodes placed on the torso (see cardiac dynamics). Water temperature in the bath was continuously maintained using a circulatory cooling unit (iCool Lite, iCool Australia Pty Ltd., Miami, Australia). For the active recovery therapy, the participants were instructed to exercise on a cycle ergometer (Wattbike®, Wattbike Ltd., Nottingham, UK) for 10 min at a low, self-selected intensity, also adopting a hip angle of ~90°. 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 cardiography (Physioflow®, Manatec Biomedical, Paris, France). This method detects changes in impedance of alternating low magnitude electrical current between electrodes 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 from the brachial artery using an automated sphygmomanometer (Digital blood pressure monitor, UA-767, ADInstruments Ltd., 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 2 beat intervals, and saved on a personal computer for offline analysis. Test-retest coefficients of variation using the Physioflow® 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. Post-exercise coefficients were calculated using data collected during baseline testing and the first experimental trial.
The coefficient of variation post-exercise 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 ($\text{Ln rMSSD}$). 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 0–5 min (5 min), 15–20 min (20 min) and 35–40 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 MSSD (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 2–5 min of each 5 min interval, and from 2–9 min during REC.

**Muscle hemodynamics**

Muscle hemodynamics were assessed using a portable NIRS system (Portamon®, Artinis Medical Systems, 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 mid-line 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 re-positioning 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 on-line at 10 Hz during all trials, using native software (Oxysoft 
V.2.1.6; Artinis Medical Systems, Netherlands). 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 [(O₂Hb / tHb] 
× 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 min, 20 min and 40 min in to 
the recovery period. These 10 s isometric MVCs were used in place of arterial occlusions to
obtain a measure of O\textsubscript{2} consumption because O\textsubscript{2} delivery is substantially occluded due to increased intramuscular pressure of MVCs compressing blood vessels (40). Pre-contraction SmO\textsubscript{2} 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 were 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) and are illustrated in Figure 2.

(i) Minimum ΔSmO\textsubscript{2} amplitude (SmO\textsubscript{2min}), which corresponds to the difference between minimum SmO\textsubscript{2} during the contraction and mean SmO\textsubscript{2} over −4 to −1 s prior to the onset of the contraction. A larger Δ (i.e., more negative) SmO\textsubscript{2min} values indicate greater O\textsubscript{2} consumption relative to O\textsubscript{2} delivery.

(ii) SmO\textsubscript{2} ½ deoxygenation time (SmO\textsubscript{2½ DT}), which corresponds to the period of time between the start of the contraction until SmO\textsubscript{2} reaches 50% of the difference between baseline SmO\textsubscript{2} and the minimum SmO\textsubscript{2} during the contraction. A shorter SmO\textsubscript{2½ DT} (for a similar SmO\textsubscript{2min}) represents a faster O\textsubscript{2} consumption rate.

(iii) SmO\textsubscript{2} maximum amplitude (SmO\textsubscript{2max}) was identified as the amplitude corresponding to the maximum SmO\textsubscript{2} % within 120 s following the end of the contraction.

(iv) SmO\textsubscript{2} ½ reoxygenation time (SmO\textsubscript{2½ RT}), which corresponds to the time required (from the end of the contraction) for SmO\textsubscript{2} to reach 50% of SmO\textsubscript{2max}. A lower value (i.e., faster time) indicates greater O\textsubscript{2} delivery relative to O\textsubscript{2} consumption.

**Temperature measurement**

Intramuscular temperature was measured using a fine-wire implantable probe (T204E, Physitemp Instruments Inc, NJ, USA), while skin temperature was measured with a surface probe (SS-1, Physitemp Instruments Inc, NJ, USA). Temperature was logged at a frequency
of 1 Hz using a portable data logger (SQ2020, Grant instruments, 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 sub-cutaneous 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 for 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 (V.21, IBM, New York, USA). 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 $0.2 = \text{small effect}, 0.5 = \text{moderate effect}, \text{and } \geq 0.8 = \text{large effect}$. 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, SmO$_2$ and tHb. Data for
SmO$_2$ 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 mean ± standard deviation (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 (Figure 3A), stroke volume (Figure 3B) and cardiac output (Figure 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 pre-exercise 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 pre-exercise value, and cardiac output ($p = < 0.0001$ to $0.001$; $d = 2.1$ to 2.4) remained above the pre-exercise 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 pre-exercise rate at 30–60 min after cold water immersion ($p < 0.05$; $d = 0.7$ to 1.4), whereas it remained above the pre-exercise 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 pre-exercise value during cold water immersion ($p = 0.003$; $d = 1.8$), whereas it remained lower than the pre-exercise 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 $0.004$) (Figure 4A).
During cold water immersion, tHb increased above the pre-recovery value during the first 4 min (Figure 4B). It then decreased slightly over the remaining 6 min, but remained higher than the minute 1 value during cold water immersion ($p < 0.05$; $d = 0.6$ to 0.9). tHb increased above pre-recovery value from minute 3 onwards 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 pre-exercise values after either trial (time effect $p = 0.093$) (Figure 4C). tHb was lower than pre-exercise 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 ($p = 0.037$; $d = 1.3$) (Figure 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 pre-exercise temperatures immediately after resistance exercise and before the recovery interventions in both trials ($p < 0.001$) (Figure 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 pre-exercise 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 pre-exercise throughout the active recovery exercise ($p < 0.001$).

After the recovery interventions

Muscle temperature remained below the pre-exercise value for 40 min following cold water immersion ($p < 0.05$; $d = 1.4$ to 4.5), whereas skin temperature remained below the pre-exercise value for the entire period ($p < 0.05$; $d = 0.7$ to 6.0) (Figure 5). Muscle ($d = 2.7$ to 3.8) and skin ($d = 3.3$ to 4.7) temperatures remained above pre-exercise 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 ($p = 0.235$ to 0.618), 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 (Figure 6). The total amount of work completed during the 50 isokinetic contractions was similar at baseline (i.e., ‘pre-exercise’) compared with when the same contractions were performed ~70 min after exercise (Table 3). During the 50 contractions performed ~70 min post-exercise, 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 pre-exercise, SmO$_{2\min}$ $\Delta$ 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 (Figure 7A), and remained lower at 40 min ($d = 1.4$) after active
recovery. Compared with pre-exercise, \( \text{SmO}_2^{\frac{1}{2}} \text{DT} \) during the MVCs did not change significantly at any time after cold water immersion (\( p = 0.155 \) to 0.631). \( \text{SmO}_2^{\frac{1}{2}} \text{DT} \) was longer compared with pre-exercise values at 20 min (\( p = 0.001; d = 1.3 \)) and 40 min (\( p = 0.003; d = 1.2 \)) after active recovery (Figure 7B). Compared with pre-exercise values, \( \text{SmO}_2^{\frac{1}{2}} \text{RT} \) 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 (Figure 7B). \( \text{SmO}_2^{\frac{1}{2}} \text{RT} \) was not significantly different to pre-exercise values at any time (\( p > 0.05 \)) after active recovery. \( \text{SmO}_2^{\text{min}} \) was lower than baseline (pre-exercise) 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 \( \text{SmO}_2^{\frac{1}{2}} \text{DT} \) during the isokinetic contractions, or \( \text{SmO}_2^{\frac{1}{2}} \text{RT} \) 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 (Figure 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 rMMSD 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. SmO2 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 (Figure 4B), indicating an increase in muscle blood volume similar to the cold-induced vasodilation described 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,
immersion, tHb decreased for at least 20 min thereafter (Figure 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. SmO2 amplitude (SmO2_{min}) and deoxygenation time (SmO2_{1/2DT}) derived from NIRS measurements indicate the demand for oxygen in contracting muscle (11, 29). We are confident that occlusion was maintained during the MVCs because (i) full blood flow occlusion occurs at or above 50–60% maximum isometric torque (32), and (ii) torque did not decrease substantially during the MVCs (data not shown). SmO2_{min} during MVCs decreased (relative to pre-exercise values) after both cold water immersion and active recovery (Figure 7A), indicating a reduction in total O2 consumption. This response may reflect temporary muscle fatigue following resistance exercise. SmO2_{1/2DT} during MVCs did not change significantly after cold water immersion, whereas it was longer after active recovery (Figure 7B). This finding indicates that the rate of O2 consumption in muscle during MVCs slowed after active recovery but not after cold water immersion. SmO2 reoxygenation time (SmO2_{1/2RT}) after MVCs tended to be longer \((p = 0.052\) versus pre-exercise values) 40 min after cold water immersion (Figure 7B). This finding suggests that O2 consumption mildly exceeded O2 delivery after MVCs following cold water immersion. This mismatch possibly resulted from the decrease in cardiac output (Figure 3C) and/or muscle perfusion (Figure 4D) that occurred after cold water immersion (14, 25, 27).

Another new and important finding in this study is that cold water immersion prevented any decrease in maximal isometric strength after resistance exercise (Figure 6). By contrast, strength remained below pre-exercise 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) 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, 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 (Figure 4D) and tended to slow the rate of SmO\textsubscript{2} reoxygenation after MVCs (Figure 7B), these effects did not seem to influence muscle function. Active recovery could have interfered with the restoration of sarcoplasmic reticulum function and Ca\textsuperscript{2+} signaling, which have been linked to prolonged muscle fatigue after exercise (16, 50).

**Perspectives and significance**

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 post-exercise recovery or after other modes of exercise. The alterations in muscle temperature, 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 tHb genuinely reflect differences in tissue oxygenation in the muscle, rather than experimental artifact 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 probably would have confounded the effects of the prior recovery interventions. We contend that the muscle function tests are valid because (a) we explicitly set out to examine how differences in muscle temperature after exercise influence recovery of muscle strength and (b) 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.

In summary, this is the first systematic investigation into the effects of two frequently used post-exercise recovery therapies on cardiac dynamics, tissue temperature, muscle function, muscle oxygenation and blood volume. The strength of the study lies in the simultaneous measurement of these physiological variables during and after cold water immersion and active recovery. It is difficult to compare directly the physiological effects of cold water immersion and active recovery. Nevertheless, knowledge of the contrasting effects of these recovery therapies is important to improve our understanding of which modality to use in different circumstances. Future research could compare the effects of different water temperature on these physiological variables and examine in more detail the hyperemic responses in skeletal muscle after cold water immersion.

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REFERENCES


FIGURE LEGENDS

FIGURE 1. Experimental overview.

FIGURE 2. An illustration of near-infrared spectrometry variables during and following an isometric maximum voluntary contraction. Grey section represents the 10 s contraction period. N.B. Axes are not drawn to scale.

FIGURE 3. Heart rate (A), stroke volume (B) and cardiac output (C) before (PRE) and after (POST) resistance exercise, during the recovery interventions (grey section), during the recovery period, and following the dynamic task (PD). Vertical dashed lines represent the beginning of the recovery period (0 min) in the cold water immersion (CWI) and active recovery (ACT) trials. Data are mean ± SD. * p < 0.05 vs pre-exercise.

FIGURE 4. Quadriceps muscle oxygenation (SmO₂) and blood volume (tHb) during (minutes 1-10; panels A and B) and following (minutes 5-40; panels C and D) the cold water immersion (CWI) and active (ACT) recovery interventions. PRE: pre-exercise, post-exercise: POST, pre-recovery therapy: PRE REC. Data are mean ± SD. * p < 0.05 vs pre-exercise.

FIGURE 5. Muscle (A) and skin (B) temperatures before (PRE) and after (POST) the exercise bout, during the recovery interventions (grey section), after the recovery intervention (POST REC), and during the recovery period in the cold water immersion (CWI) and active recovery (ACT) trials. Vertical dashed line represents the beginning of the recovery period (0 min). Data are mean ± SD. * p < 0.05 vs pre-exercise.

FIGURE 6. Peak isometric torque pre-exercise (PRE) and at 5, 20 and 40 min into the recovery period (corresponding to 25, 40 and 65 min after the resistance exercise bout) in the cold water immersion (CWI) and active recovery (ACT) trials. Grey shaded area indicates the period of CWI or ACT. N.B. x-axis is not drawn to scale. Data are mean ± SD. * p < 0.05 vs pre-exercise.

FIGURE 7. Quadriceps muscle oxygenation (SmO₂) kinetics (A) and amplitude (B) during isometric contractions. SmO₂½ DT, half deoxygenation time. SmO₂½ RT, half
reoxygenation time in the cold water immersion (CWI) and active recovery (ACT) trials. N.B. Data to the left of the dotted line are plotted on the left y-axis; data to the right of the dotted line are plotted on the right y-axis. Data are mean ± SD. * $p < 0.05$ vs pre-exercise.
Table 1. Systolic, diastolic, and mean arterial pressure measured before and after exercise, and 1 min after the recovery therapies. *Significantly different vs pre-exercise, p < 0.05. † Significantly different vs post exercise, p <0.05

<table>
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<th>After recovery</th>
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<td>125 ± 8 *</td>
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<td>ACT</td>
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<td>78 ± 6 *†</td>
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<td>ACT</td>
<td>88 ± 4</td>
<td>94 ± 4 *</td>
<td>94 ± 4 *</td>
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Table 2. Peak isokinetic torque (Nm) (mean ± SD) during the fatigue task at baseline, and during the cold water immersion (CWI) and active recovery (ACT) trials. N.B. Baseline performance was measured 7 d prior to the first trial, and was used as a ‘pre-exercise’ comparison for both experimental trials; see Methods. *Significant difference from contractions 1–5 (p < 0.05).

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Table 3. Total work and changes in muscle oxygenation (SmO₂) and blood volume (tHb) during and for 2 min after 50 isokinetic contractions. CWI, cold water immersion; ACT, active recovery (ACT) trials. N.B. Baseline performance was measured 7 d prior to the first trial, and was used as a ‘pre-exercise’ comparison for both experimental trials; see Methods.

* Significantly different from baseline, p < 0.05.

<table>
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<th>Trial</th>
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<th>ACT</th>
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<td>Total work (kJ)</td>
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<td>Contractions 1–25</td>
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<td>Contractions 26–50</td>
<td>2.2 ± 0.6</td>
<td>2.2 ± 0.5</td>
<td>2.2 ± 0.8</td>
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<td>tHb minimum amplitude (µM)</td>
<td>1.1 ± 3.3</td>
<td>−1.3 ± 2.0*</td>
<td>-0.2 ± 3.5</td>
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<td>tHb maximum amplitude (µM)</td>
<td>10.0 ± 5.7</td>
<td>6.9 ± 3.7*</td>
<td>7.0 ± 5.4</td>
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<td>SmO₂min (%)</td>
<td>−14.7 ± 8.4</td>
<td>−17.3 ± 8.0</td>
<td>−14.2 ± 6.5</td>
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<td>SmO₂½DT (s)</td>
<td>11.7 ± 8.3</td>
<td>10.9 ± 3.4</td>
<td>11.8 ± 6.6</td>
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<td>SmO₂½RT (s)</td>
<td>22.5 ± 29.4</td>
<td>19.0 ± 9.6</td>
<td>19.6 ± 8.0</td>
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</tbody>
</table>
Unilateral exercise bout

PRE measures

Transition from recovery room

Recovery intervention

Transition from recovery room

5 min measures

20 min measures

40 min measures

Isokinetic task

Cardiac dynamics

Temperature

NIRS

rMSSD

Trial time (min)
Recovery period (min)
Figure 2
Figure 3

A: Heart rate (b/min⁻¹)

B: Stroke volume (ml)

C: Cardiac output (l/min⁻¹)

ACT

CWI

PRE

POST

Time

*
Figure 4

A. 

B. 

C. 

D. 

- SmO2 (%) 
- tHb (μM) 
- SmO2 change (%) 
- tHb change (μM) 

Legend: 
- ACT 
- CWI 

* statistically significant difference from baseline
Figure 5

Muscle temperature (°C)

Skin temperature (°C)

ACT
CWI

PRE POST POST REC

Time (min)

* * *
Figure 6