Cold Water Immersion Recovery after Simulated Collision Sport Exercise

MONIQUE POINTON and ROB DUFFIELD

School of Human Movement Studies, Charles Sturt University, Bathurst, New South Wales, AUSTRALIA

ABSTRACT

POINTON, M. and R. DUFFIELD. Cold Water Immersion Recovery after Simulated Collision Sport Exercise. *Med. Sci. Sports Exerc.*, Vol. 44, No. 2, pp. 206–216, 2012. **Purpose**: This investigation examined the effects of cold water immersion (CWI) recovery after simulated collision sport exercise. **Methods**: Ten male rugby athletes performed three sessions consisting of a 2 × 30-min intermittent-sprint exercise (ISE) protocol with either tackling (T) or no tackling (CONT), followed by a 20-min CWI intervention (TCWI) or passive recovery (TPASS and CONT) in a randomized order. The ISE consisted of a 15-m sprint every minute separated by self-paced bouts of hard running, jogging, and walking for the remainder of the minute. Every sixth rotation, participants performed 5 × 10-m runs, receiving a shoulder-led tackle to the lower body on each effort. Sprint time and distance covered during ISE were recorded, with voluntary (maximal voluntary contraction; MVC) and evoked neuromuscular function (voluntary activation; VA), electromyogram (root mean square (RMS)), ratings of perceived muscle soreness (MS), capillary and venous blood markers for metabolites and muscle damage, respectively measured before and after exercise, immediately after recovery, and 2 and 24 h after recovery. **Results**: Total distance covered during exercise was significantly greater in CONT ($P = 0.01$), without differences between TPASS and TCWI ($P > 0.05$). TCWI resulted in increased MVC, VA, and RMS immediately after recovery ($P < 0.05$). M-wave amplitude and peak twitch were significantly increased after recovery and 2 h after recovery, respectively, in TCWI ($P < 0.05$). Although TCWI had no effect on the elevation in blood markers for muscle damage ($P > 0.05$), lactate was significantly reduced after recovery compared with TPASS ($P = 0.04$). CWI also resulted in reduced MS 2 h after recovery compared with TPASS ($P < 0.05$). **Conclusions**: The introduction of body contact reduces exercise performance, whereas the use of CWI results in a faster recovery of MVC, VA, and RMS and improves muscle contractile properties and perceptions of soreness after collision-based exercise. **Key Words:** RUGBY, BODY CONTACT, PHYSIOLOGICAL LOAD, NEUROMUSCULAR, EXERCISE PERFORMANCE

Team sports are characterized by intermittent bouts of high-intensity activity, separated by short bouts of low-intensity activity (24). Further, many team sports such as rugby league, rugby union, Australian and American football, and soccer also involve regular collisions between opposing players throughout the course of training and/or match play. For example, participation in rugby league and rugby union requires players to be exposed to numerous (n = 20–40) direct physical collisions and tackles (7,17) throughout training and/or competition. The combative nature of such sports, combining intermittent high-intensity activity and repeated blunt force trauma, may result in microdamage to skeletal muscle and postexercise muscle soreness (MS) (11,23). However, to date, few studies have attempted to quantify the effect of body contact on ensuing exercise performance, and those that have report minimal effect of tackling because of insufficient tackling load (33). Regardless, with many team sports training and competing over successive days, often with collision-based exercise, time available for physiological recovery can be limited (32). Because repeated collisions may result in potential residual MS and damage (25), such outcomes can adversely affect subsequent exercise performance (31). Accordingly, interventions such as cold water immersion (CWI) that are aimed at improving postexercise recovery have become increasingly popular in many team sports.

Although the body of literature on the effects of CWI recovery is growing, findings on potential benefits remain equivocal (20,32). In particular, and despite increased popularity, there is a paucity of research examining the effect of CWI after high-intensity physical collision sport (4). To date, the study by Banfi et al. (4) is the only investigation to examine the effects of CWI recovery after rugby (collision-based) exercise. Although no performance results were reported, CWI stabilized venous blood creatine kinase (CK) values, although CWI was combined with an initial active recovery. As such, the effect of CWI alone on performance and physiological recovery from rugby, or more specifically, collision-based exercise, remains unknown. Recently, Rowsell et al.

Address for correspondence: Monique Pointon, Ph.D., School of Human Movement Studies, Charles Sturt University, Panorama Avenue, Bathurst, New South Wales, Australia; E-mail: mpointon@csu.edu.au.
Submitted for publication March 2011.
Accepted for publication June 2011.

0195-9131/12/4402-0206/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/MSS.0b013e31822b0977

(32) reported a reduced perception of leg soreness and general fatigue with CWI recovery during a 4-d soccer tournament, without any improvement in physical performance. Conversely, Ingram et al. (20) recently demonstrated ameliorated recovery of sprint performance and maximal voluntary contraction (MVC) 48 h after recovery with CWI following 80 min of simulated team sport exercise compared with contrast water immersion and passive recovery. Although findings regarding CWI recovery after team sport exercise indicate potential benefits (20,32), there remains a paucity of evidence examining the effects of exercise-induced direct body collisions on performance and the subsequent result CWI has on physiological, performance, and perceptual recovery.

Despite reports of increased damage and physiological load with collision-based exercise (13,15), few studies quantify the subsequent effects on exercise performance (33). Further, with many professional team sports implementing CWI recovery into training regimes, minimal evidence to support such practice after direct body contact sport exists. Accordingly, the primary aim of this investigation was to examine the effects of CWI on recovery of neuromuscular, physiological, and perceptual functions after simulated collision-based team sport exercise. A secondary aim of this investigation was to quantify the effect of direct lower body collisions (tackles) on intermittent-sprint exercise performance. It was hypothesized that implementation of direct body collisions would result in a significant decrement in exercise performance. Further, we hypothesized that CWI would result in an enhanced recovery of neuromuscular, physiological, and perceptual functions.

METHODS

Participants

Ten male, club-level, team sport athletes (rugby league/union) aged 21.0 ± 1.7 yr, 182.9 ± 6.1 cm tall, and weight 87.2 ± 7.7 kg (mean ± SD) were recruited as participants for this study. At the time of testing, participants completed 2–3 training sessions per week and competed in rugby league/union competition at least once per week. All participants were informed of the requirements of the study, and verbal and written consent was gained before the commencement of testing. Human ethics clearance was granted by the institutional ethics committee before the completion of any testing procedures.

Overview

Participants completed an initial session to ensure familiarity with all physiological, neuromuscular, and perceptual procedures, together with an explanation and demonstration of the tackling procedure to be received. This was followed by three testing sessions in a randomized order separated by at least 7 d. The testing sessions were identical apart from the implementation of tackling within the exercise protocol and the use of a postexercise recovery intervention. Each experimental testing session consisted of a high-intensity intermittent-sprint exercise (ISE) protocol (2 × 30-min halves) performed in an enclosed laboratory on a 20-m synthetic running track at 20.3°C ± 1.1°C and 37.0% ± 1.1% relative humidity. Three testing conditions were implemented and consisted of ISE with tackling and CWI recovery (TCWI), tackling with passive recovery (TPASS), and no tackling with passive recovery (CONT). The aim of implementing three conditions was twofold. The investigation aimed to: first, examine the effects of CWI recovery after ISE involving intense body collisions (TPASS vs TCWI); and second, examine the effect of the additional load of tackling on exercise performance (TPASS vs CONT) to confirm an effect of the tackling protocol used. For the tackling conditions (TPASS and TCWI), the ISE protocol included five explicit, intense lower body collisions (tackles) separated by 10 s performed every sixth minute of the protocol (a total of 40 tackles). The total number of tackles performed corresponds to previous time–motion analysis of rugby league and rugby union match play (7,17). During the control condition (CONT), no tackles were implemented; instead, participants completed 5 × 10-m hard runs along the synthetic track to match the distance covered and the intensity of the run before and after the collisions in TPASS and TCWI. The ISE was followed by 20 min of CWI (TCWI) or passive recovery (TPASS and CONT), with each testing session performed at the same time of day to minimize diurnal variation.

Neuromuscular function was measured before and after exercise and again immediately after recovery and 2 and 24 h after recovery. Participants were required to present in a rested state and avoid consumption of food or drink (including caffeine) 3 h before testing and refrain from alcohol consumption 24 h before testing. All food, drink, and physical activity in the 24 h before the first testing session and during the 24-h recovery period after the exercise protocol were recorded. Participants refrained from any strenuous activity during the 24-h recovery period, and food and activity diaries were monitored throughout. Food, drink, and activity before and during the first testing session were replicated for all testing sessions. During the ISE protocol, 500 mL of water was provided to participants to consume *ad libitum* with full consumption monitored during each testing session.

Exercise Protocol

On completion of preexercise neuromuscular tests (detailed subsequently), participants completed a warm-up involving running at increasing speeds over a 15-m running track for a period of 3 min, followed by three maximal 15-m sprints. The ISE protocol consisted of 2 × 30-min halves, interspersed by a 10-min passive recovery. A 15-m maximal sprint (with a subsequent 5-m deceleration zone before impacting with a large mat) was performed every minute, followed by submaximal exercise of varying intensities in a

self-paced, shuttle-run format (hard running, jogging, walking) for the remainder of the minute (12). Only one exercise mode of hard running, jogging, or walking (rotated each minute) was completed each minute before returning to the starting position to complete the ensuing sprint. Every sixth rotation after the maximal sprint, participants received five intense lower body tackles performed by a trained research assistant. The research assistant was 10 m from the start line and, to replicate training drills, was in a kneeling position. Participants were required to hard run the 10-m before making contact with the tackling research assistant. The research assistant leaned back as the participants neared and lunged into the tackles on contact, aiming to provide direct body contact with their shoulder into the participant's midsection of the lower leg, forcing them to the ground. Participants were then required to jog back to the start line and immediately commence the ensuing tackling bout. Maximal 15-m sprint performance during the exercise protocol was assessed with infrared timing gates (Speed-Light, Swift Performance Equipment, Lismore, Australia), and mean sprint time was calculated. As the exercise intensity was set by the participants, distances covered during the exercise protocol were monitored by the investigators, and appropriate encouragement was provided during each testing session. Distance covered for each respective submaximal exercise bout was determined using 1-m markings alongside the 15-m synthetic track. From pilot data collection (n = 10), intraclass correlations (r) and coefficients of variation (CV) for distance covered were r = 0.82–0.96 and CV = 1.5%–3.2%, respectively.

Recovery Interventions

Within 10 min of completing the exercise protocol, the recovery intervention was administered in a designated area of the laboratory (20.3°C ± 1.1°C, 37.0% ± 1.1% relative humidity). Cold water immersion (TCWI) consisted of seated immersion in an ice bath (plunge pool) (9.2°C ± 0.2°C) to a level of the iliac crest for 9 min followed by 1 min seated at room air temperature (38). This procedure was repeated twice for a total duration of 20 min. For the passive recovery (TPASS and CONT), participants remained seated in the laboratory for 20 min.

Instrumentation

Assessment of neuromuscular function. A 5-min low-intensity warm-up at 60 W on a cycle ergometer (Monark 818E, Varberg, Sweden) was performed before the measurement of neuromuscular function before exercise at 2 and 24 h postrecovery time points to ensure similar levels of potentiation as during the postexercise measurement. For the measurement of neuromuscular function, participants were seated on an isokinetic dynamometer (Humac Norm isokinetic dynamometer; Ausmedic, CSMi Medical Solutions, Stoughton, MA) linked to a BNC2100 terminal block connected to a signal acquisition system (PXI1024; National Instruments, Austin, TX). A/D conversion for torque and EMG data was performed at 16-bit resolution and synchronously sampled all data at a rate of 1 kHz. Participants were seated upright with a 90° hip angle and securely fastened by adjustable straps tightly across the chest and pelvis with the distal right leg fixed to the dynamometer lever arm. The axis of rotation of the dynamometer was aligned to the lateral epicondyle of the femur, indicating the anatomical joint axis of the knee. Lever arm length, chair length, and dynamometer height were recorded during familiarization for accurate repositioning during subsequent testing sessions.

Muscle activation was achieved by stimulating the femoral nerve using a felt pad bar cathodal electrode with a tip spacing of 30 mm (Nicolet Biomedical, Madison, WI) positioned at the medioanterior aspect of the upper thigh, directly below the inguinal fold. The anode was a 90 × 50-mm reusable self-adhesive gel pad electrode (Verity Medical, Ltd., Stockbridge, Hampshire, UK) and positioned on the medioposterior aspect of the upper thigh, directly below the gluteal fold, opposite the cathode. The current applied to the femoral nerve was delivered by a Digitimer DS7AH stimulator (Digitimer Ltd., Welwyn Garden City, Hertfordshire, UK) using a single square-wave pulse with a width of 200 μs (400 V with a current of 100–450 mA) that was driven by a custom-designed instrument using LabView software (version 8.0, LabView; National Instruments). Initially, the current was manually applied and gradually increased until a plateau in twitch and M-wave amplitude was achieved in which time the stimulus intensity was increased by a further 25% to ensure supramaximal stimulation. The site most responsive was marked with a permanent pen to ensure identical placement for subsequent testing. The electrode was then securely fastened in position using a Velcro strap with a constant force of 1.5 kgf applied via an algometer (Pain Test FPI Algometer; Wagner Instruments, Greenwich, CT).

For the measurement of voluntary torque, participants performed 5 × 5-s MVC separated by 5-s rest between each contraction with the knee flexed at 65° (0° being full extension). The production of maximal effort was encouraged for the entire 5 s during which time a superimposed electrical twitch was delivered when a reduction in peak force was observed. The trigger for stimulation was manually primed within 1–2 s after initiation of each contraction, and once primed, the stimulus was automatically triggered when customized LabView software detected a decline in peak force (<1%). Within 3 s after each superimposed contraction, a second stimulus was delivered to the rested muscle to determine potentiated twitch properties. The mean MVC was determined as the peak isometric torque produced during the 25 ms preceding the delivery of the electrical stimulus averaged during the five contractions.

Voluntary activation (VA) was calculated using the twitch interpolation technique (1). Peak superimposed torque was determined during the 50- to 150-ms period after the delivery of the stimulus during each MVC. Interpolated twitch torque (ITT) was subsequently determined as peak superimposed

torque minus voluntary peak torque. VA was determined by expressing the ITT as a percentage of the peak potentiated evoked twitch torque (Pt) obtained at rest between contractions using the following equation: VA (%) = [1 − (ITT / Pt)] × 100. Mean VA was determined from the average of all five superimposed contractions.

Potentiated twitch and M-wave properties were determined from the electrical stimulus initiated ~3 s after the superimposed contraction on the rested muscle. Torque–time curves from the potentiated twitch contractions were averaged across all trials with mean data used to determine the following characteristics: 1) peak potentiated twitch torque (Pt), 2) the rate of torque development (RTD), 3) time to peak twitch torque (TPt), 4) the rate of relaxation (RR), 5) half-relaxation time (½ RT), and 6) contraction duration (CD) (8). Torque onset was determined as the point at which torque increased beyond 2 SDs above the mean torque value calculated during a 50-ms period immediately before stimulation (41). Potentiated M-wave data were averaged across the five trials, with the mean used to determine 1) peak-to-peak amplitude, 2) duration, and 3) latency.

Surface EMG data were obtained from the vastus lateralis (VL), vastus medialis (VM), and the antagonist biceps femoris (BF) for the dominant leg. EMG signals were sampled using differential surface electrodes (Bagnoli-16; Delsys, Inc., Boston, MA) positioned on VL, VM, and BF according to Cram and Kasman (10), with a reusable self-adhesive electrode attached to the patella of the opposing limb to ground the signals. Low impedance was obtained by shaving, abrading, and cleaning the skin before positioning of the electrodes at each testing time point. Electrode placement sites were marked with permanent pen to ensure identical placement for subsequent testing sessions. Voluntary EMG data were obtained during the assessment of MVC with the EMG cables taped to prevent movement artifacts in the EMG signal. EMG signals were preamplified and band-pass–filtered, with a bandwidth frequency ranging from 20 to 450 Hz (common mode rejection ratio >90 dB; impedance input = 100 MΩ; gain = 1000).

Voluntary EMG signals were quantified using the root mean square (RMS). RMS amplitude was calculated as the average of the 25 ms preceding the superimposed twitch during MVC. The EMG signal was then averaged between vasti muscles to provide a global indication of total knee extensor (KE) motor unit activity. All data were processed offline with the determination of MVC, VA, potentiated twitch, and M-wave properties, and RMS achieved using MATLAB software (version R2010a; The MathWorks, Inc., Natick, MA). For the MVC, correction for the effect of gravity on the lower leg during the superimposed and potentiated evoked contractions was calculated as the average load applied to the force transducer during the 50-ms period immediately before force onset and subsequently used to offset force data. Once corrected for gravity, force data were then multiplied by lever arm length and expressed in units of torque ($N \cdot m^{-1}$).

Capillary and venous blood measures. On arrival, a 100-μL sample of capillary blood was obtained for the measurement of lactate (La^-), pH, and bicarbonate (HCO_3^-), with further samples obtained at half time, after exercise, and after recovery. A 5-mL sample of venous blood from the antecubital vein was obtained at preexercise and postrecovery intervals (immediately and 2 and 24 h) for the measurement of CK, C-reactive protein (CRP), and aspartate aminotransferase (AST) as markers of muscle damage and cell inflammation. Using an evacuated venipuncture system and serum separator tubes (Monovette, Sarstedt, Numbrecht, Germany), samples were allowed to clot at room temperature before centrifugation for 10 min at 4000 rpm. Serum was then extracted and stored at −20°C until analysis. Before analysis, the serum was allowed to reach room temperature and was mixed gently via inversion. CK, CRP, and AST were analyzed according to manufacturer's instructions provided in the respective assay kits (Dimension Xpand spectrophotometer; Dade Behring, Atlanta, GA). Intra-assay CVs were <5% for all venous blood analyses.

Physiological and perceptual measures. HR was recorded with an HR monitor and wrist watch receiver (F1, Polar Electro-Oy, Kempele, Finland) and measured before exercise, every 5 min during the exercise protocol, and immediately after recovery. RPE was determined using the Borg 6–20 Scale (26) with perceived MS determined using a 10-point Likert scale (MS: 0 = no pain and 10 = very, very sore). RPE was determined before and after exercise and every 5 min during the exercise protocol, whereas MS was determined before and after exercise and throughout the recovery period (immediately after recovery and 2 and 24 h after recovery).

Statistical Analysis

Data recorded from neuromuscular, physiological, and perceptual measures are reported as means ± SD. A repeated-measures ANOVA (condition × time) was used to determine significant difference between conditions and over time from preexercise values for each experimental condition. Pairwise comparisons were used to determine the time points for significance, which was set at $P < 0.05$. To examine the effect of the recovery intervention, comparisons were made between TPASS and TCWI, whereas the inclusion of tackling on exercise performance was determined with comparisons between TPASS and CONT. The Mauchly test of sphericity was performed to test for the homogeneity of variance (28). All data collected were analyzed using SPSS version 16.0 (Statistical Package for the Social Sciences, Chicago, IL).

RESULTS

Distance covered and sprint time. Total distance covered during the exercise protocol was 4178 ± 195, 4212 ± 201, and 4584 ± 385 m for TPASS, TCWI, and CONT, respectively. A significantly greater distance was covered during CONT compared with TPASS and TCWI ($P = 0.01$),

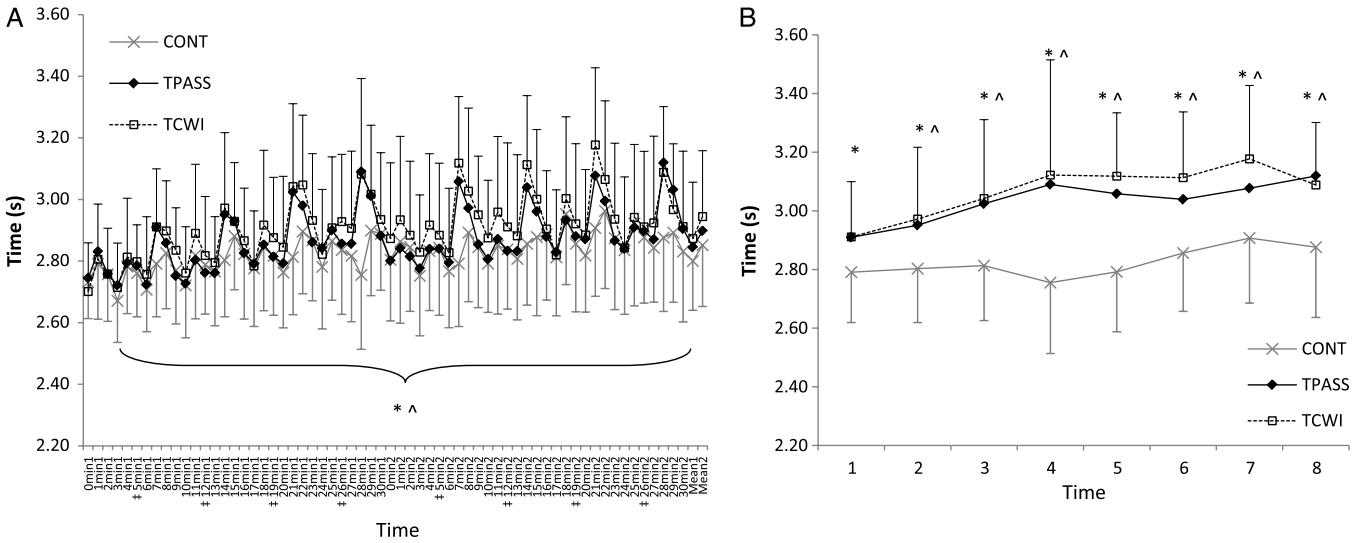

FIGURE 1—Mean ± SD (A) sprint time during the exercise protocol and (B) sprint time after each tackle bout for CONT, TPASS, and TCWI. ∧Significant time effect from preexercise values in all conditions ($P < 0.05$). *Significant difference between TPASS and TCWI versus CONT ($P < 0.05$). ‡Tackling bout.

with no differences evident between TPASS and TCWI ($P > 0.05$). Mean sprint time was significantly increased throughout the duration of the exercise protocol in all conditions ($P = 0.01$; Fig. 1). TPASS and TCWI resulted in a significantly slower mean sprint time compared with CONT ($P = 0.02$ and $P = 0.03$, respectively). Sprint time after the tackle bout was significantly increased throughout the duration of the exercise protocol, with TPASS and TCWI resulting in a significantly slower time compared with CONT ($P = 0.01$; Fig. 1).

Neuromuscular function. The exercise protocol resulted in a significant reduction in MVC and VA in all conditions ($P = 0.01$; Figs. 2A and B, respectively) and remained below preexercise values for the 24-h recovery period. The additional load of tackling resulted in a prolonged reduction in VA with a significant reduction 2 h after recovery in

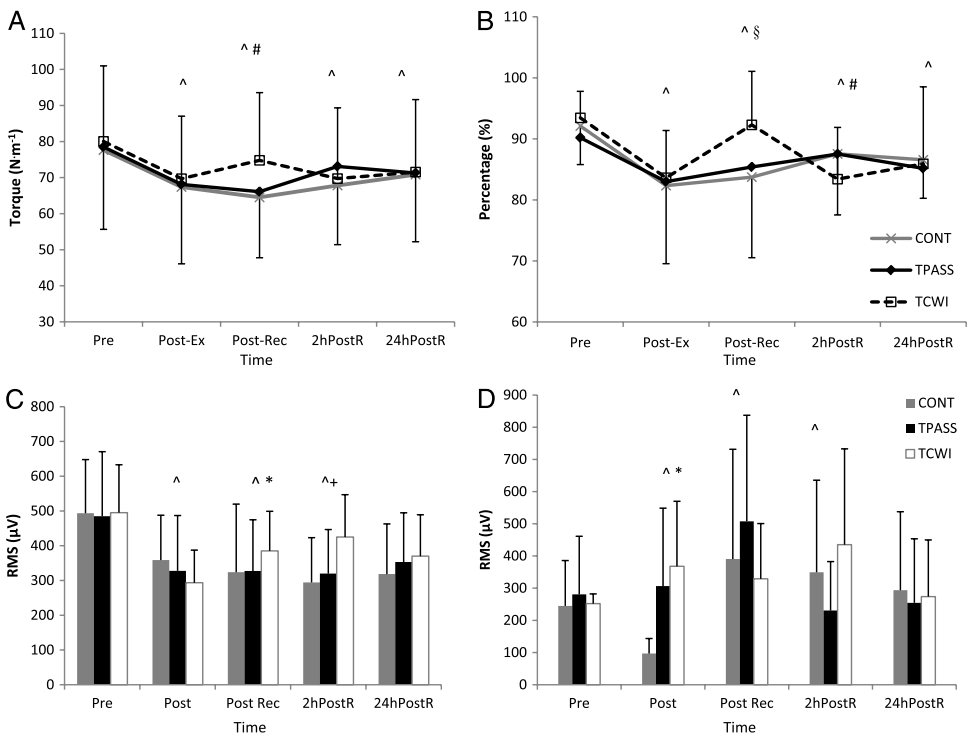

FIGURE 2—(A) Maximal voluntary contraction, (B) VA, (C) RMS EMG of mean ± SD VL and VM, and (D) RMS EMG of hamstrings for CONT, TPASS, and TCWI. *Significant difference between TCWI and CONT. ∧Significant time effect from preexercise values ($P < 0.05$). ∧+Signficant time effect from preexercise values in TPASS and CONT only ($P < 0.05$). #Significant difference between TPASS and CONT versus TCWI ($P < 0.05$). §Signficant difference between TPASS versus TCWI ($P < 0.05$).

TABLE 1. Potentiated twitch properties.

		Before Exercise	After Exercise	After Recovery	2 h after Recovery	24 h after Recovery
Pt	CONT	29.7 ± 4.9	23.5 ± 5.1*	26.8 ± 4.8*	27.6 ± 4.6**	28.6 ± 5.0
	TPASS	29.5 ± 4.8	24.3 ± 4.4*	26.1 ± 4.4*	27.9 ± 4.4**	28.1 ± 4.5
	TCWI	29.7 ± 4.9	24.9 ± 4.7*	24.9 ± 4.9*	28.3 ± 5.8	29.8 ± 5.7
TPt	CONT	86.1 ± 16.8	83.4 ± 15.4	86.8 ± 12.2	93.4 ± 16.6	89.6 ± 9.9
	TPASS	91.8 ± 18.2	94.0 ± 13.9	97.4 ± 27.7	87.8 ± 17.0	92.3 ± 16.8
	TCWI	91.4 ± 15.8	93.7 ± 18.3	90.4 ± 13.5	86.3 ± 10.2	94.0 ± 11.9
½ RT	CONT	49.3 ± 9.4	43.4 ± 5.5	44.7 ± 4.9	49.1 ± 8.6	53.0 ± 13.4
	TPASS	53.0 ± 8.0	48.5 ± 10.9	51.5 ± 10.4	52.1 ± 9.9	57.0 ± 11.7
	TCWI	49.7 ± 7.9	43.0 ± 3.2	58.6 ± 6.8***	51.6 ± 8.2	56.6 ± 6.9
RTD	CONT	352.6 ± 60.8	284.8 ± 54.2*	312.8 ± 55.3*	306.1 ± 80.2	326.0 ± 80.9
	TPASS	343.6 ± 70.2	260.5 ± 39.4*	283.0 ± 70.8*	329.4 ± 70.1	309.5 ± 67.5
	TCWI	338.5 ± 92.2	276.8 ± 78.5*	284.8 ± 83.3*	333.4 ± 80.8	323.2 ± 75.6
RR	CONT	315.5 ± 72.3	277.3 ± 59.5	307.6 ± 62.1	296.0 ± 80.7	287.9 ± 77.7
	TPASS	295.5 ± 64.4	261.8 ± 55.6	266.2 ± 68.3	280.9 ± 62.9	259.9 ± 70.7
	TCWI	295.6 ± 89.5	297.5 ± 59.1	225.8 ± 87.8***	286.2 ± 83.9	269.1 ± 79.0
CD	CONT	135.3 ± 22.2	126.8 ± 17.5	131.5 ± 15.9	142.5 ± 20.9	142.6 ± 16.5
	TPASS	144.8 ± 22.6	142.5 ± 20.2	148.9 ± 30.0	139.7 ± 21.5	148.7 ± 24.9
	TCWI	146.0 ± 27.6	136.7 ± 18.8	156.9 ± 25.3***	137.9 ± 15.0	154.6 ± 11.0
Lat	CONT	36.9 ± 14.7	36.9 ± 14.7	37.0 ± 11.4	34.7 ± 9.7	31.4 ± 12.8
	TPASS	32.8 ± 15.6	22.8 ± 14.2	30.9 ± 12.9	35.7 ± 12.2	31.8 ± 16.5
	TCWI	32.8 ± 16.5	28.6 ± 15.4	30.9 ± 14.0	33.3 ± 10.1	25.5 ± 10.3

Values are mean ± SD.
* Significant time effect from preexercise values in all conditions ($P < 0.05$).
** Signficant time effect from preexercise values in TPASS and CONT only ($P < 0.05$).
*** Significant difference between TPASS and CONT versus TCWI ($P < 0.05$).

TPASS and TCWI ($P = 0.03$ and $P = 0.01$, respectively) compared with CONT. A recovery of CWI resulted in a significantly increased MVC and VA after recovery compared with TPASS ($P = 0.03$ and $P = 0.05$, respectively). No significant differences were evident between the conditions at any other time point ($P > 0.05$).

Mean RMS of VM/VL was significantly reduced after exercise and after recovery in all conditions ($P = 0.01$–0.05; Fig. 2C), with a reduction in RMS also observed 2 h after recovery in TPASS and CONT only ($P = 0.01$). The additional load of tackling resulted in a significantly increased postexercise RMS of the BF in TPASS and TCWI compared with CONT ($P = 0.04$ and $P = 0.01$, respectively; Fig. 2D). A recovery of CWI resulted in a significant increase in RMS of VM/VL compared with TPASS and CONT after recovery ($P = 0.04$).

Significant reductions in postexercise and postrecovery Pt and RTD were observed in all conditions compared with preexercise values ($P = 0.01$; Table 1). At 2 h after recovery, Pt remained decreased compared with preexercise values in TPASS and CONT only ($P = 0.05$). Apart from a reduction in RTD ($P < 0.05$), the exercise protocol did not significantly alter TPt, latency, ½ RT, RR, and CD. However, a recovery of CWI resulted in a significantly slower ½ RT, RR, and CD after recovery compared with TPASS ($P < 0.05$; Table 1).

Amplitude, latency, and duration of the M-wave were not significantly altered by the exercise protocol in any condition ($P > 0.05$; Table 2). The duration and latency of the M-wave in VM was significantly slower after TCWI after recovery compared with TPASS ($P = 0.02$; Table 2). No significant differences were evident between the conditions for duration and amplitude in VL.

Capillary blood, venous blood, and HR. ISE resulted in a significant reduction in pH and HCO_3^- compared with preexercise values, with greater reductions evident in TPASS and TCWI compared with CONT ($P = 0.01$–0.04; Table 3). La^- was significantly elevated after exercise in all conditions ($P = 0.01$), with significantly greater elevations in TCWI and TPASS after recovery compared with preexercise values ($P = 0.02$ and $P = 0.04$, respectively; Table 3). TPASS resulted in a significantly lower La^- after recovery compared with TCWI ($P < 0.04$). There were no significant differences between recovery conditions at any

TABLE 2. Potentiated M-wave properties in VM for CONT, TPASS, and TCWI.

		Before Exercise	After Exercise	After Recovery	2 h after Recovery	24 h after Recovery
Latency	CONT	10.1 ± 2.0	9.9 ± 1.8	10.2 ± 1.9	10.3 ± 1.8	11.0 ± 3.2
	TPASS	10.8 ± 1.7	10.5 ± 2.3	11.5 ± 2.3	11.8 ± 1.8	11.2 ± 1.7
	TCWI	11.0 ± 1.8	10.5 ± 2.5	11.7 ± 2.6*	10.3 ± 2.4	10.4 ± 2.4
Amplitude	CONT	4.1 ± 2.1	3.5 ± 2.3	3.9 ± 2.5	3.9 ± 2.6	4.0 ± 2.5
	TPASS	4.9 ± 3.1	5.0 ± 2.7	5.0 ± 2.4	5.0 ± 2.7	5.3 ± 2.7
	TCWI	5.4 ± 1.9	5.3 ± 1.8	6.4 ± 2.0**	5.9 ± 1.7**	5.9 ± 2.0
Duration	CONT	3.8 ± 0.8	3.9 ± 1.1	4.0 ± 0.9	3.9 ± 1.4	4.0 ± 1.1
	TPASS	4.6 ± 2.0	3.7 ± 1.2	3.6 ± 0.7	4.2 ± 1.1	3.8 ± 0.8
	TCWI	3.7 ± 0.8	3.5 ± 0.6	5.0 ± 1.5*	3.9 ± 0.7	3.9 ± 0.8

Values are mean ± SD.
* Significant difference between TPASS and CONT versus TCWI ($P < 0.05$).
** Significant difference between TCWI versus CONT ($P < 0.05$).

TABLE 3. Capillary and venous blood variables for La^-, pH, HCO_3^-, CK, AST, and CRP for CONT, TPASS, and TCWI.

		Before Exercise	After Exercise	After Recovery	2 h after Recovery	24 h after Recovery
La^-	CONT	1.62 ± 0.48	4.46 ± 2.14*	1.78 ± 0.64**		
	TPASS	1.38 ± 0.28	5.75 ± 2.62*	2.17 ± 0.94***		
	TCWI	1.56 ± 0.17	5.39 ± 2.91*	2.66 ± 1.22***		
pH	CONT	7.34 ± 0.03	7.33 ± 0.03***	7.36 ± 0.02		
	TPASS	7.34 ± 0.02	7.32 ± 0.04***	7.35 ± 0.01		
	TCWI	7.34 ± 0.02	7.32 ± 0.03***	7.34 ± 0.02		
HCO_3^-	CONT	21.6 ± 1.4	19.2 ± 1.2***	21.3 ± 0.8		
	TPASS	21.3 ± 1.4	18.8 ± 2.0***	20.9 ± 0.5		
	TCWI	21.6 ± 1.3	18.6 ± 1.4***	20.6 ± 0.8		
CK	CONT	279 ± 282		389 ± 337*	472 ± 398*	475 ± 374*
	TPASS	281 ± 193		418 ± 256*	457 ± 247*	503 ± 357*
	TCWI	247 ± 143		386 ± 176*	467 ± 209*	567 ± 346*
AST	CONT	32 ± 19		39 ± 18*	39 ± 18*	38 ± 18*
	TPASS	31 ± 13		38 ± 15*	40 ± 14*	39 ± 14*
	TCWI	31 ± 12		38 ± 12*	40 ± 11*	40 ± 12*
CRP	CONT	1.6 ± 0.7		1.8 ± 0.7*	1.6 ± 0.6	1.9 ± 0.8*
	TPASS	1.9 ± 1.3		2.1 ± 1.5*	2.4 ± 2.0*	2.5 ± 2.4*
	TCWI	1.9 ± 1.5		2.0 ± 1.5*	1.9 ± 1.6	3.2 ± 3.0*

Values are mean ± SD.
* Significant time effect from preexercise values in all conditions ($P < 0.05$).
** Significant difference between TPASS and CONT versus CWI ($P < 0.05$).
*** Significant time effect from preexercise values in TPASS and TCWI only ($P < 0.05$).

time point for pH and HCO_3^- ($P > 0.05$). CK, AST, and CRP were all significantly elevated as a result of the exercise protocol in all conditions ($P < 0.05$; Table 3). However, no significant differences were evident between the recovery conditions at any time point ($P > 0.05$). Postexercise HR was significantly increased compared with preexercise values in all conditions ($P = 0.01$; Fig. 3A). No significant differences were evident between the conditions at any time point ($P > 0.05$).

Perceptual measures. Rating of MS was significantly increased at all time points in all conditions compared with preexercise values ($P = 0.01$; Fig. 3C). A recovery of CWI resulted in a significantly lower MS 2 h after recovery compared with CONT ($P = 0.05$) and TPASS ($P = 0.04$). No significant difference was evident between the conditions at any other time point ($P > 0.05$). The additional load of the tackling (TPASS and TCWI) resulted in a significantly increased RPE during the first and second halves of exercise compared with CONT ($P < 0.05$; Fig. 3B). In addition, RPE measured after the tackles significantly increased throughout the duration of exercise, with TPASS and TCWI significantly higher than CONT ($P < 0.05$).

DISCUSSION

This investigation examined the effects of CWI on recovery of muscle function after simulated, collision-based, team sport exercise. Further, the effect of the additional load of tackling on exercise performance was also examined. The exercise protocol sufficiently induced a significant loss in muscle function, as evidenced by reductions in MVC and VA, which did not return to preexercise values during the 24-h recovery period. Further evidence for exercise-induced muscle damage was also observed with significantly elevated blood markers of muscle damage (CK, AST, and CRP), increased perceptions of soreness and reductions in potentiated M-wave amplitude and twitch contractile properties. Despite the significant loss in muscle function, CWI enhanced the immediate recovery of MVC and VA, alongside an ameliorated recovery of RMS, Pt, and MS. Accordingly, a novel finding of this study was that the improvement in acute postexercise MVC with CWI after high-intensity, collision-based exercise is likely attributed to an interaction of enhanced central activation and improved recovery of peripheral contractile function. A further novel finding was the observation of a reduction in intermittent-sprint exercise performance as a direct result of the presence of repeated collision-based body contacts.

To date, Singh et al. (33,34) are the only investigators to specifically examine the effects of direct collisions during simulated team sport activity on ensuing exercise performance. The earlier work of Singh et al. (33) reported no decrement in performance with the addition of tackling during simulated team sport activity; however, using the same contact protocol, Singh et al. (34) recently demonstrated prolonged reductions in 15-m sprint and vertical jump performance 48 h after exercise. Singh et al. (33,34) postulated that the small volume ($n = 20$) and use of simulated body collisions (tackle bags and bump pads) did not elicit significant fatigue to acutely impair performance, but the resulting muscle damage due to contacts produced greater decrements in performance 48 h after exercise. Consequently, in the present study, the higher volume ($n = 40$) and direct physical collisions (lower body-on-body tackles) resulted in a significantly lower total distance covered, alongside reduced mean sprint times in TPASS and TCWI compared with CONT. Accordingly, it seems if sufficient body-on-body contacts occur, an acute impairment of ensuing sprint performance and also submaximal distance covered may result. Moreover, the incorporation of tackling resulted in greater central fatigue compared with the control condition, as observed by a greater reduction in VA. Therefore, the presence of repeated direct and forceful body contact results in acute

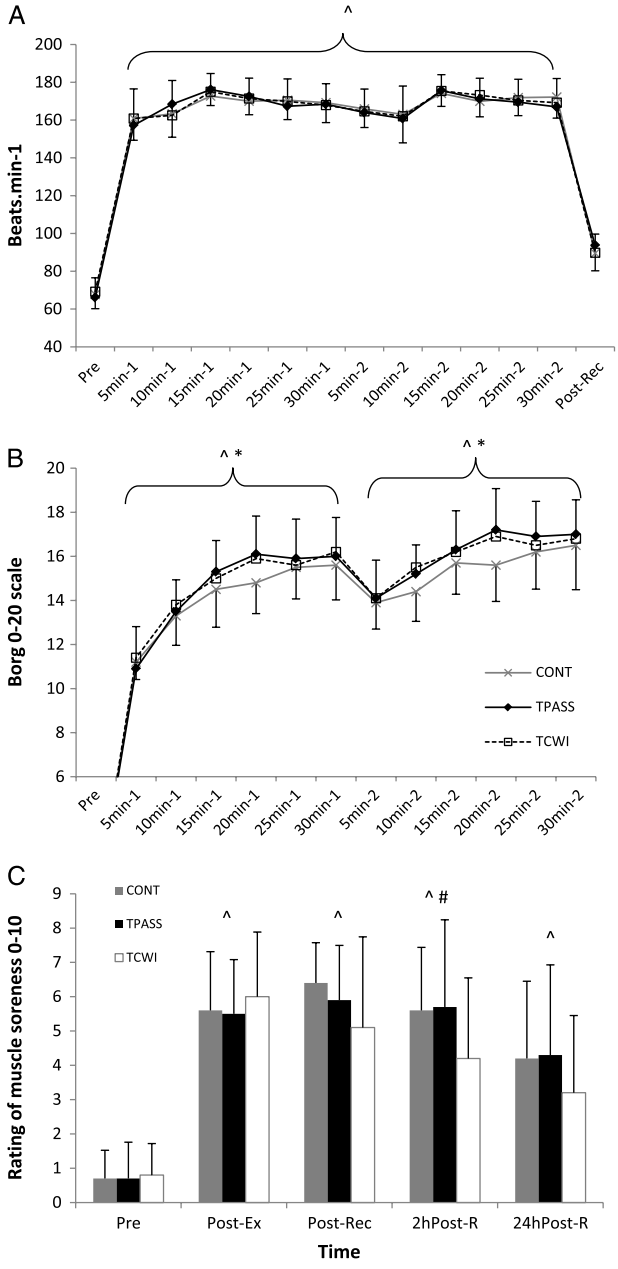

FIGURE 3—Mean ± SD (A) HR during the exercise protocol and after recovery, (B) RPE during the exercise protocol, and (C) rate of perceived MS for CONT, TPASS, and TCWI. ∧Significant time effect from preexercise values in TPASS and TCWI versus CONT ($P < 0.05$). #Significant difference between TPASS and CONT versus TCWI ($P < 0.05$). *Significant difference between TPASS and TCWI versus CONT ($P < 0.05$).

reduction of intermittent-sprint performance and greater central fatigue for team sport athletes.

Despite limited previous research, it is perhaps not unexpected that the presence of simulated tackling results in greater physiological load and reduced performance for team sport exercise. Interestingly, the tackling condition also resulted in an increased coactivation of the antagonist muscle during postexercise MVC, with an increase in hamstring RMS evident compared with CONT. To date, no studies have reported the effect of collision-based exercise on neuromuscular responses. However, previous research has reported a reduction in KE force production due to an increased antagonistic effect during fatiguing isometric contractions (30). The reported increase in central drive to the antagonist muscle was postulated as a protective mechanism against further damage to the agonist (30). This purported protective effect may explain the present findings, with postexercise reductions in MVC and VA and concomitant increases in antagonist RMS observed after intense lower body collisions. Previous research reports that increased volume of collisions during exercise increases the physiological demands of the exercise bout (13,15). Accordingly, the present study complements these findings in that incorporating body contact into any training (or competitive) environment may result in exacerbated performance reductions, possibly via reductions in central recruitment of skeletal musculature. In addition, the present data also highlights the effective use of a body contact intervention in the current study.

Regardless of the effects of body contact on performance, of primary interest, the present study highlights the short-term benefits of CWI after collision-based exercise. These results are in agreement with previous research demonstrating improved recovery of MVC with CWI after intermittent-sprint (3,20) and DOMS-inducing exercise (39). Recently, Ingram et al. (20) demonstrated that after 80 min of simulated team sport exercise, CWI recovery resulted in a smaller decrement in MVC compared with contrast water therapy and a control condition. Further, Vaile et al. (39) reported an improvement in isometric squat strength 48 and 72 h after CWI compared with a passive recovery after DOMS-inducing exercise. Similarly, Bailey et al. (3) reported a lower decrement in MVC of the knee flexors at 24 and 48 h after recovery with CWI. The authors in the aforementioned studies postulated that the mechanisms to explain ameliorated recovery of MVC were due to enhanced recovery of deleterious symptoms associated with exercise-induced muscle damage (EIMD) and fatigue. Although likely, a further explanation is the possibility of centrally mediated mechanisms aiding recovery of voluntary force production (16). To support this notion, recovery of VA and RMS was improved alongside MVC with CWI in the present study. Accordingly, the recovery of MVC after a high-intensity, simulated rugby exercise in the current study is possibly explained by the role of increased centrally mediated skeletal muscle recruitment in improving voluntary force (16). Although the measurement of isometric MVC in the present study was used as a marker of skeletal muscle function, the increase in postrecovery MVC, VA, and RMS with CWI may also provide an explanation for improved exercise performance observed in previous studies (20,38).

Despite the significant reduction in exercise performance and muscle function as a result of the tackling load, CWI recovery ameliorated the decline in MVC and VA, alongside increased RMS of the agonist muscle group and improved perceptions of MS. Although Eston and Peters (14) previously reported a faster return to baseline strength values after

CWI and postulated a reduction in EIMD, to date, the improvement in central activation after CWI has not been investigated, particularly after collision-based team-sport exercise. As such, the results of the present study highlight an increase MVC and a novel finding of an increase in VA and RMS, together with improvements in Pt and M-wave amplitude after CWI. Such findings may suggest that improvements in MVC are a result of enhanced recovery of peripheral contractile ability as well as increased central activation. To date, the precise mechanisms responsible for improved recovery of peripheral contractile ability with CWI are unknown. Although previous research has postulated that beneficial effects of CWI may be partly related to the effect of hydrostatic pressure on decreasing tissue edema (40), recent evidence examining the physiological and neuromuscular effects of CWI compared with temperate water immersion have indicated that physiological alterations are a result of localized cooling rather than hydrostatic pressure *per se* (2,29). As the present study did not include a neutral water immersion control, observed beneficial alterations in skeletal muscle function resulting from colder temperatures or hydrostatic pressure remain to be determined. Further, although the causal relationship between ameliorated recovery of muscle contractile function and physiological parameters influencing central motor drive is unknown, the enhanced recovery of central and peripheral muscle function after CWI in the present study highlights the need for further investigation examining the potential relationship between these variables.

Although CWI resulted in an increase in Pt and M-wave amplitude, ½ RT, RR, CD, M-wave duration, and latency were slower compared with TPASS and CONT. Unfortunately, a limitation of the present study was that the temperature of the muscle during evoked twitches was not measured. Despite this, it is well known that the application of cold significantly alters muscle contractile properties through slowing of nerve conduction velocity (14,35), reduced neuromuscular transmission (lengthened duration of the compound-evoked muscle potential, M-wave), and lengthened contraction and ½ RT of evoked twitches (6). Regardless of such changes in evoked twitch contractile properties, the recovery of MVC after CWI was enhanced immediately after recovery compared with a passive recovery (TPASS). Although CWI resulted in a slower response to the evoked signal, an increased VA and RMS after CWI may highlight the influence of central modulation in enhancing postrecovery MVC, possibly because of other factors including alterations in muscle contractile properties and enhanced perceptions of MS.

Significant postexercise elevations in CK, AST, and CRP were evident in all conditions, suggesting that considerable muscle damage and cell inflammation was present (27). Interestingly, although implementation of tackling in the present study elicited greater reductions in pH and HCO_3^- together with elevated La^- values compared with no tackling, no difference between blood markers of muscle damage was observed between the tackling conditions and CONT, corroborating with recent findings (33,34). Singh et al. (33,34) also failed to observed a difference in CK measures with the inclusion of low-impact tackling (tackle bags and bump pads) compared with noncontact during simulated team sport exercise. As such, elevated CK, AST, and CRP in the present study likely resulted from the eccentric loading associated with high-intensity running (25,37). Indeed, Minett et al. (25) recently reported a strong correlation between very high-intensity running and elevated CK and AST after rugby union match play using a similar subject population as the present study (club-level rugby players). Further, Minett et al. (25) also reported a correlation between the number of contacts and CK elevation, corroborating with previous findings examining rugby league (22) and union match play (36). Thus, it seems that the lower-impact forces associated with simulated tackles (33,34) and forces indicative of training intensities such as the present study may not significantly disrupt muscular contractile integrity to elicit pronounced elevations in blood markers of muscle damage as those observed after actual match play.

Although elevated CK was likely a result of high-intensity running in the present study, postexercise CWI had no significant effect on the appearance of CK compared with a passive recovery, which corroborates previous research using EIMD (3,19,21) and simulated team sport exercise (32). Rowsell et al. (32) recently reported that CWI after each match during a 4-d simulated soccer tournament did not significantly alter the elevation in CK or lactate dehydrogenase. Similarly, despite a postexercise reduction in myoglobin, Bailey et al. (3) reported that CWI had no significant effect on CK response after a 90-min intermittent shuttle running. Furthermore, CWI did not significantly alter the elevation of AST and CRP in the present study. The lack of difference in CRP response is supported by Halson et al. (18) who demonstrated that after a 40-min simulated cycling time trial, CWI recovery did not significantly alter the presence of CRP and additional blood markers of muscle damage and inflammation. The results of the present study support the aforementioned studies and suggest that CWI is not effective in reducing the appearance of indirect blood markers of muscle damage and inflammation in the 24-h recovery period after intense, high-impact collision-based exercise.

Finally, in agreement with previous studies (3,18,32), CWI recovery resulted in a significant reduction in perception of MS evident 2 h after recovery. Rowsell et al. (32) recently demonstrated reductions in perceptions of general fatigue and leg soreness after CWI recovery during a 4-d simulated soccer tournament. Similarly, lower ratings of general fatigue and leg soreness were reported when CWI was implemented after a simulated 20-min cycling time trial (18). Previous investigators suggest that athletes perform better when they believe they have received beneficial treatment (5,9). Therefore, it is possible that the enhanced perception of MS observed in the current study will have arisen

because of a potential placebo effect of CWI. Although the placebo effect is a possible explanation for the observed reduction in perceptions of MS, the acute recovery of MVC, VA, RMS, and Pt was improved after CWI. As such, despite the possibility of a placebo effect enhancing perceptions of MS, CWI implemented in the current study did elicit improvements in acute recovery of skeletal muscle function after intense, collision-based exercise.

CONCLUSIONS

High-intensity, intermittent-sprint exercise with the additional load of intense body contact, simulating collision sports (rugby union/rugby league), resulted in a significant reduction in exercise performance, muscle function, and increased soreness. In comparison to intermittent-sprint exercise alone, the incorporation of lower body tackles (collisions) resulted in an increased physiological load and greater decrement in exercise performance. Despite the reduction in muscle function and exercise performance, CWI improved the acute recovery of MVC compared with a passive recovery. Moreover, CWI resulted in improvements in VA, RMS, perceptions of MS, and potentiated M-wave and twitch responses. As such, acute improvements in MVC after CWI are likely because of an interaction between alterations in the state of peripheral contractile ability and enhanced skeletal muscle recruitment via increased central activation. Accordingly, the CWI-induced improvement in recovery of voluntary force and activation together with enhanced perception of MS would suggest an effective implementation of this recovery strategy in contact-based sports.

The authors thank the subjects for their participation and enthusiasm. In addition, the authors would like to acknowledge Brian Heffernan for his assistance with the blood analysis and Gary McKenzie at SPAN for his help with EMG analysis.

This research undertaking was solely funded by Charles Sturt University and not any of the following organizations: National Institutes of Health, Wellcome Trust, or the Howard Hughes Medical Institute. The authors report no conflicts of interest.

The results of this investigation do not constitute endorsement by the American College of Sports Medicine.

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