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Cold-water immersion and whole-body cryotherapy attenuate muscle soreness during 3 days of match-like tennis protocol

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Abstract

Purpose This study aimed to investigate the effect of whole-body cryotherapy (WBC), cold-water immersion (CWI) and passive recovery (PAS) on tennis recovery.

Methods Thirteen competitive male tennis players completed three consecutive match-like tennis protocols, followed by recovery (WBC, CWI, PAS) in a crossover design. Five tennis drills and serves were performed using a ball machine to standardize the fatiguing protocol. Maximal voluntary contraction (MVC) peak torque, creatine kinase activity (CK), muscle soreness, ball accuracy and velocity together with voluntary activation, low- and high-frequency torque and EMG activity were recorded before each protocol and 24 h following the third protocol.

Results MVC peak torque $(-7.7 \pm 11.3\%; p = 0.001)$ and the high- to low-frequency torque ratio $(-10.0 \pm 25.8\%; p < 0.05)$ decreased on Day 1 but returned to baseline on Day 2, Day 3 and Day 4 (p=0.052, all p > 0.06). The CK activity slightly increased from 161.0 ± 100.2 to 226.0 ± 106.7 UA L⁻¹ on Day 1 (p=0.001) and stayed at this level (p=0.016) across days with no differences between recovery interventions. Muscle soreness increased across days with PAS recovery (p=0.005), while no main effect of time was neither observed with WBC nor CWI (all p > 0.292). The technical performance was maintained across protocols with WBC and PAS, while it increased for CWI on Day 3 vs Day 1 (p=0.017).

Conclusion Our 1.5-h tennis protocol led to mild muscle damage, though neither the neuromuscular function nor the tennis performance was altered due to accumulated workload induced by consecutive tennis protocols. The muscle soreness resulting from tennis protocols was similarly alleviated by both CWI and WBC.

Trial registration IRB No. 2017-A02255-48, 12/05/2017.

Keywords Cold · Recovery · Tennis-induced fatigue · Low and high frequency electrical stimulation

Abbreviations CK Creatine kinase		Creatine kinase	Db100	High frequency doublet peak torque induced by 100-Hz paired stimuli The high to low frequency torque ratio Electromyography Maximal voluntary contraction Passive recovery	
CWI Db10		Cold-water immersion Low frequency doublet peak torque induced by 10-Hz paired stimuli	Db10.Db100 ⁻¹ EMG MVC PAS		
Communicated by Nicolas Place.			RMS	Maximal root mean square (RMS) value of the EMG signal	
	 Mathilde Poignard mathilde.poignard@insep.fr French Institute of Sport (INSEP), Laboratory Sport, Expertise and Performance (EA 7370), 11 Avenue du Tremblay, 75012 Paris, France 		RMSmax	RMS as normalized to the peak-to-peak M-wave amplitude	
1			RPE WBC	Rate of perceived exertion Whole-body cryotherapy	

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Introduction

Players potentially play during the professional tennis season a tournament every week which consists of four or five matches plus the finale, with a limited time frame (<24 h) between matches for recovery (Girard et al. 2014). Therefore, tennis players currently use numerous recovery strategies to minimize their fatigue and improve their recovery between matches and tournaments. Cold-water immersion (CWI) and whole-body cryotherapy (WBC) are particularly popular in tennis despite a lack of specific guidelines for coaches and players (Poignard et al. 2020; Fleming et al. 2018). Indeed, the efficiency of these recovery methods depends particularly on the exercise-induced stress and the beneficial effect of CWI and WBC within the tennis-specific context remains to be demonstrated (Kellmann et al. 2018; Thorpe 2021; Martin and Prioux 2011). Specifically, the application of recovery intervention needs to match with realistic exercise-induced stress. It is thus primarily essential to understand the accumulated tennis-induced fatigue caused by consecutive days of match plays.

Previous studies indicated that tennis matches may induce muscle damage by showing an alteration in voluntary activation accompanied by low-frequency peripheral fatigue (i.e., impair of the excitation-contraction coupling), an increase in creatine kinase activity and a higher score of muscle soreness perceived by the players after 2-4 h of match play (Girard et al. 2008; Vollestad 1997; Ojala and Hakkinen 2013; Gomes et al. 2014; Gescheit et al. 2015; Periard et al. 2014); Gesheit et al. (2015); and Ojala and Hakkinen (2013) suggested that an accumulation of neuromuscular fatigue during successive days of 2-4 h of match play could lead to the decline of physical performances with progressive impairments of lower limb maximal and explosive force and an increase in creatine kinase and muscle soreness up to 48 h after the end of the last match (Ojala and Hakkinen 2013). They also both proposed that decreases in strength, power and velocity could partly explain the alterations in service stroke velocity, service accuracy and movement patterns (i.e., effective playing time, work-to-rest ratio). However, the mechanisms underlying these negative changes in physical and tennis-specific performances remain to be demonstrated, as these studies did not measure central and peripheral fatigue. Moreover, the typical duration is 1.5 h with an effective playing time of 20% for all types of players, though some matches can last more than 5 h (Kovacs 2007). To date, no study has investigated the perceptual, physiological and physical responses induced by shorter tennis matches over consecutive days.

CWI and WBC both aim to decrease tissue and muscle temperatures, and thereby reduce the blood flow and the intramuscular metabolism to limit the inflammation associated with the secondary exercise-induced muscle damage and the resulting muscle soreness (Mawhinney et al. 2017; Bleakley et al. 2012). Previous studies inferred that CWI attenuates secondary exercise-induced muscle damage from a lower increase in creatine kinase activity when compared to a control condition (Ascensao et al. 2011; Pooley et al. 2019). There is still no evidence of such effect after WBC exposure (Bouzigon et al. 2021). CWI is also effective to decrease the perception of muscle soreness. For these reasons, it may attenuate delayed-onset muscular soreness over consecutive days in field conditions (Tavares et al. 2019; Rowsell et al. 2009) and in an ecological tennis setting (Duffield et al. 2014). To date, the beneficial effects of WBC on muscle soreness have been only demonstrated in a protocol detached from contextual performance (Fonda and Sarabon 2013; Hohenauer et al. 2018; Abaidia et al. 2017) or following simulated trial (Hausswirth et al. 2011). CWI and WBC may accelerate the recovery of neuromuscular function by improving physical and perceptual states (Leeder et al. 2012; Abaidia et al. 2017; Hausswirth et al. 2011) and limit decrements in functional performance (Ascensao et al. 2011). In the context of performance, a few studies have demonstrated that CWI can efficiently limit performance decrements, while alleviating muscle soreness after intermittent sprint exercises and/or team sports (Rowsell et al. 2009; Tavares et al. 2019). CWI in combination with compression garments and sleep hygiene recommendations can efficiently improve the playing time and lower-body power-generating capacity after 1 day of repeated on-court tennis training (Duffield et al. 2014). In contrast, the potential benefits of WBC in field conditions remain to be demonstrated (Russell et al. 2017; Grainger et al. 2019). It is noteworthy that CWI could be more efficient than WBC, due to the hydrostatic pressure of water and to the conductive effect conferring a greater ability to reduce tissue temperature (Mawhinney et al. 2017). However, previous studies directly comparing the potential effects of WBC and CWI on muscle soreness and functional recovery in a laboratory setting have reported conflicting results (Abaidia et al. 2017; Wilson et al. 2019). Further investigations are required in the context of specific exercise-induced fatigue (Ihsan et al. 2020).

This study aimed to investigate the effect of three recovery modalities (WBC, CWI and passive recovery [PAS]) on tennis-induced fatigue recovery. We monitored the fatigue over 3 days of match-like tennis protocols by assessing physiological and neuromuscular markers. We hypothesized that (i) the daily use of WBC or CWI could limit accumulated tennis-induced fatigue after 3 days of tennis protocols when compared with the passive condition; and (ii) CWI could attenuate muscle soreness and the loss of lower limb force-generating capacity to a greater extent than WBC.

Methods

Participants

Thirteen well-trained regional male tennis players (mean \pm SD; age = 28 \pm 6 yr; height = 180 \pm 6 cm; body mass = 73 \pm 6 kg; 2 left-handers; 11 right-handers; mean training volume = 7 \pm 3 h.week⁻¹) volunteered to participate in this study. Before the experiment, all participants underwent a medical examination to rule out potential contraindications to intense cold exposure (cardiovascular risk factor, history of respiratory disease, cold hypersensitivity). Players were informed of the procedures and the risks associated with tests and investigations before providing their written consent. All procedures conformed to the standards of the Declaration of Helsinki and the study was approved by the ethics committee of Sud Méditerranée IV (No. 17 10 05) and by the French Health Agency (IRB no 2017-A02255-48). Sample size was based on previous studies demonstrating significant effects of CWI or WBC compared to a control group on muscle soreness and functional and specific performance variables with a similar methodological design (King and Duffield 2009; Schaal et al. 2015).

Experimental overview

Participants first completed two sessions to become familiar with all neuromuscular, physiological and perceptual measures and to perform each of the recovery interventions. They were also accustomed to each step of the match-like tennis protocol by performing one repetition of tennis serve exercise and one repetition of each of the five included tennis drills. Oral and written instructions regarding expected movement patterns, velocity and accuracy of the strokes were also provided for each exercise and drill. Players completed three 4-day experimental conditions in a randomized order separated by at least 7 days (Fig. 1). A random-numbers generator (www.randomization.com) was used to assign the condition order for each player. Participants were asked to refrain from exercise during the 4 days of each testing session. All trainings performed during the 3 days preceding



Fig. 1 Schematic of the experimental design

each session were collected to control for potential residual fatigue from previous strenuous training. Food and hydration diaries were completed 24 h before and during the 4 days of the first experimental condition and that same diet replicated over the three subsequent experimental conditions. Day 1, Day 2 and Day 3 followed similar testing schedules at the same times of day $(\pm 2 h)$ to minimize the effects of circadian rhythms. Participants were asked to not consume stimulant (caffeine, alcohol) and anti-inflammatory medications during the 3 weeks of the protocol. On arrival, players performed pre-measurements consisting of (i) a psychometric questionnaire, (ii) a fingertip sample of capillary blood to assess creatine kinase activity, (iii) a 10-min warm-up on a cycle ergometer, (iv) neuromuscular tests, and (v) a second 10-min on-court warm-up. Participants then performed the match-like tennis protocol on the tennis court. Within 5 min after the end of the last tennis serve exercise, participants performed post-measurements consisting of (i) neuromuscular tests, (ii) rating of the perceived exertion (RPE) of tennis protocols, (iii) creatine kinase activity assessment, and (iv) completing a psychometric questionnaire. Then, they performed their assigned recovery intervention (Fig. 1). On Day 4, participants carried out only pre-measurements, except the second warm-up. Participants were allowed to consume water ad libitum during all sessions. All testing was performed in a temperature-controlled environment (21 $^{\circ}$ C and 55% relative humidity).

Exercise protocol

Match-like tennis protocol

To properly compare CWI and WBC, a standardized tennis protocol was designed to simulate as closely as possible the workload and physiological stress, which could induce muscular damage and neuromuscular fatigue that can be alleviated by these recovery techniques. The duration of the total work completed during the tennis protocol was designed to reflect the effective playing time (% of total play time) of the typical average match duration (90 min) of a professional tennis event on hard court surface, which is approximately 20% (Kovacs 2007). The protocol was performed on an indoor tennis court (GreenSet surface, GreenSet Worldwide S.L., Barcelona, Spain) and the actual effective playing time was consequently 18 min for each match-like tennis protocol, for a total duration of 37.5 min per condition. Because tactical behavior, game style, and score, can impact player's strokes and movement characteristics (speed, distance, pattern) during real or even simulated match play, the tennis-induced fatigue can be highly variable. Consequently, movement patterns and stroke frequency were also



Fig. 2 a Movement and stroke patterns of the five tennis drills. b Schematic view of the ball machine and target zones for tennis drills (left) and the tennis serve exercise (right) (Brechbuhl et al. 2016)

Table 1	Description	of the	tennis	protocol	l
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Drills	Repetitions	Total strokes/ repetition	Work time (s)	Rest time (s)
Warm-up	_	_	300	_
Tennis serve 1	-	_	60	-
Break	-	-	-	90
Star	6	13	30	15
Break	_	_	_	90
Volley smash	6	13	30	30
Break	_	_	_	90
The box	6	13	30	15
Break	_	_	_	90
Tennis serve 2	_	_	60	_
Break	-	-	-	90
The X	6	13	30	15
Break	_	_	_	90
Suicide	6	11	30	15
Break	-	-	-	90
Tennis serve 3	_	_	60	_
Total	-	378	1380	1170

Warm-up consisted of 1 min of forehand with balls thrown to the central area of the court, 2 min of alternating forehand and backhand strokes with balls thrown into the right and left corners of the baseline, 1 min of volley and smashes and ended with 1 min of tennis serves. During the warm-up, players were told to hit balls crosscourt into one of the two target zones. After the warm-up, players performed tennis matches as follows: Tennis serve 1, 90 s of break, The star, 90 s of break, volley smash, 90 s of break, The box, 90 s of break, Tennis serve 2, 90 s of break, The X, 90 s of break, Suicide, 90 s of break, Tennis serve 3. The star, The box, The X and Suicide drills consisted of six repetitions of 30 s of exercise and 15 s of rest. The volley smash drill consisted of six repetitions of 30 s of exercise and 30 s of rest. Between strokes, players were instructed to return to the center mark

standardized by using five different drills (Fig. 2, Table 1) chosen by the investigators based on previous research (Reid et al. 2008) and interviews of senior national coaches. These drills are often prescribed in the training of top professional tennis players because they elicit all the movement patterns, various game situations and physiological responses consistent with normal or maximum match play demands (Reid et al. 2008). To standardize the match-like tennis protocol, all drills were performed using a ball machine (HighTOF, Echouboulains, France), placed just behind the baseline center mark that supported the setting and recording a predefined program (number and frequency of balls, duration of rallies, ball velocity, ball effects, zones of impact) (Table 1). The reliability and accuracy of this device have been previously demonstrated, with the coefficient of variation for the velocity ranging from 1 to 2% (Brechbuhl et al. 2016). For each of the five drills, a predefined program allowed the ball to be fed to specific areas in the order detailed in Fig. 2a. For each stroke, players had to hit the ball cross-court with the "best possible velocity/accuracy ratio" into one of the two target zones (marked on the court with white tape) (Fig. 2b) defined in previous studies to measure tennis-specific performance (Brechbuhl et al. 2016; Smekal et al. 2000). The same target areas were used for all drills and no further tactical instructions were provided in order to standardize the physiological responses between different tennis protocol days and conditions.

To ensure representation of all the eccentric actions potentially inducing muscle damage and neuromuscular fatigue, tennis serve exercises were also included but were not further analyzed (Table 1). In those exercises, players had to achieve three accurate serves in a specific target zone (Fig. 2b) marked within 1 m of the center service line (Gescheit et al. 2015). They were instructed to serve at a steady pace with the same rule as in tennis drills, with additional serves allowed until time ran out (1 min). Righthanded players served from the deuce side and left-handed players from the advantage side.

Under all conditions, a minimum of 100 balls were used every eight tennis protocols to standardize playing conditions. Strong encouragement and live feedback were provided during all tennis protocols. Participants wore a heart rate monitor (Polar Wearlink coded, Polar Electro Oy, Finland) from the on-court warm-up and throughout all tennis protocols, and data were downloaded to specific software (Polarflow, Polar Electro Oy, Finland) to obtain mean heart rate.

Recovery interventions

Recovery interventions were all performed within 30 min after completion of a tennis protocol and under medical supervision at the National Tennis Center (French Tennis Federation, Paris, France). In the CWI condition, players were seated for 11 min immersed up to the suprasternal notch in water held at 11 °C in a pool of the recovery area according to optimal protocols identified by previous reviews (Machado et al. 2016). The whole-body cryotherapy sessions (WBC) were administered in a system (Zimmer Elektromedizin, Germany) comprising two rooms each maintained at a constant temperature (-60 °C, -110 °C)throughout the protocol. Participants were first asked to dry their skin and to wear underwear or shorts, a surgical mask, a headband, gloves, dry socks and shoes. All piercings, glasses and contact lenses were removed. Participants were then allowed to enter and remain for 20 s in the -60 °C chamber, after which they proceeded to the - 110 °C chamber and remained for 3 min, a duration demonstrated to be effective in inducing the beneficial effects on physiological and functional recovery (Hausswirth et al. 2013; Louis et al. 2020). A doctor had visual and auditory contact with participants at all times and encouraged them to breathe and walk slowly around the chamber. For the passive condition (PAS), participants remained seated for 10 min at room temperature (22 $^{\circ}$ C).

Measurements

Neuromuscular function

During all measurements, participants were seated on an isokinetic dynamometer (Con-Trex, CMV AG, Dübendorf, Switzerland), fastened in by means of adjustable straps across the chest and pelvis and having the distal dominant leg fixed to the dynamometer lever arm with the knee and ankle angles set at 90° (0° = full extension). The torque signal from the isokinetic dynamometer and surface electromyography (EMG) activity were synchronized and recorded at 2000 Hz using an analog-to digital converter designed by our laboratory (Custom DT, INSEP, Paris, France). Mechanical data and EMG activity were analyzed using custom-written scripts (Origin 9.1, OriginLab Corporation, USA). Before all testing, participants performed a standardized warm-up with ten 5-s isometric MVCs of the knee extensors at about 50% of MVC peak torque. Neuromuscular testing procedures consisted of two MVCs performed without electrical stimulation, followed by three trials of electrically evoked contractions (Supplementary Fig. 1). Participants were strongly encouraged, and a visual representation of the exerted torque was displayed on a screen placed in front of them.

Maximal voluntary contraction peak torque

Participants performed two 5-s MVCs of the knee extensor muscles separated by 1-min rest periods. Torque signal was corrected for gravity and low-pass filtered at 20 Hz using a third-order Butterworth filter. The highest peak torque value achieved over the two trials was considered as the peak knee extensor torque.

Electrically evoked contractions

Electrically evoked quadriceps contractions were elicited using a high-voltage stimulator (DS7AH, Digitimer, Hertfordshire, UK) that delivered rectangular pulses of 200- μ s duration with maximal voltage of 400 V. The anode (5 × 13 cm; Schwamedico, France) was located on the gluteal fold. At the beginning of the experiment, a motor-point pen (Compex, France) was used to find the optimal site of stimulation in the femoral triangle. Then, the self-adhesive cathode (10 mm diameter, Micromed, France) was placed on the skin and the skin marked with a permanent marker to standardize the stimulation site. The stimulation intensity required to evoke a maximal isometric twitch was determined by gradually increasing the intensity (10 mA at each step) until no further increases of torque (plateau) and M-wave amplitude were achieved (Rampinini et al. 2011). Then, a supramaximal intensity stimulus (120% of maximal intensity) was delivered to secure supramaximal stimulus during electrically evoked contractions. The intensity of stimulation ranged between 60 and 160 mA. Three trials were performed, separated by 30-s rest periods (Tomazin et al. 2012). The average values across trials were determined for further analysis.

The following parameters were determined from mechanical responses elicited by electrically evoked contractions (Supplementary Fig. 1): (i) Db100, high- frequency doublet peak torque induced by 100-Hz paired stimuli (10 ms interval); (ii) Db10, low-frequency doublet peak torque induced by 10 Hz paired stimuli (100 ms interval); and (iii) Db10.Db100⁻¹, the high to low-frequency torque ratio.

Voluntary activation was determined according to the following formula (Merton 1954):

Voluntary activation =
$$\left(1 - \frac{\text{superimposed}MVCDb100}{Db100}\right) \times 100.$$

EMG Surface EMG activity was recorded from the vastus lateralis muscle during voluntary and electrically evoked contractions using a pair of Ag/AgCl self-adhesive electrodes (recording zone area: 520 mm²; inter-electrode distance: 20 mm; Blue sensor N-00-S, Ambu, Copenhagen, Denmark). Participant skin was carefully shaved, abraded and cleaned with alcohol before positioning electrodes with respect to the underlying muscle fiber arrangement, consistent with the Surface EMG for the Non-Invasive Assessment of Muscles (SENIAM) project's recommendations (Hermens et al. 2000); the reference electrode was placed over the patella. Electrode positions were marked with permanent marker to ensure standardized recordings throughout the study. Raw EMG signals were pre-amplified (input impedance: 10 GQ; CMRR: 100 dB; bandwidth: 6-500 Hz; gain: 1000; Mazet Electronique Model, Electronique du Mazet, Mazet Saint-Voy, France) and band-pass filtered (10-400 Hz, zero lag 3rd-order Butterworth filter).

From the EMG response during evoked contractions, the following parameters were determined: (i) the peak-to-peak M-wave amplitude induced by a single stimulation, (ii) the maximal root mean square (RMS) value of the EMG signal calculated over a moving window of 250 ms during the MVC before the superimposed Db100, and (iii) the RMS as normalized to the peak-to-peak M-wave amplitude (RMS_{max}) (Gandevia 2001).

Creatine kinase activity

A lancing device (Accu-Chek[®] Safe-T Pro Plus, Roche, France) allowed the careful sampling of $32-\mu L$ blood from a fingertip capillary of each player. The blood sample was then placed on a measurement strip and analyzed by a Reflotron Plus system according to the manufacturer's recommendations (Roche Diagnostics, Basel, Switzerland).

Perceptual measures

RPE Ratings of perceived exertion (RPE) were collected immediately after tennis protocols and before neuromuscular function tests. Players responded to the question "How did you perceive your exertion during the tennis protocol?" on a ten-point category-ratio scale (Foster et al. 2001).

Muscle soreness

Every day, before all measurements, players were asked to rate perceived muscle soreness on a 0–10 cm visual analog scale (0.1 cm accuracy) by only one investigator to minimize bias. Muscle soreness was thereafter evaluated after each tennis protocol and before neuromuscular function tests. Each participant was blinded to the results of other participants.

Tennis-specific performance

For each ball stroked by the player, ball velocity and ball accuracy were recorded. Ball velocity (km.h⁻¹) was measured and recorded directly with the multi-camera PlaySight® system (PlaySight Interactive, Ltd., Kokhav Ya'ir, Israel), which was approved by the International Tennis Federation as a tennis player analysis technology for all international tournaments. Throughout each tennis protocol, ball accuracy was measured by a dedicated investigator recording each of the balls that landed in the target zone (labeled as "accurate") and any groundstroke errors (outside the tennis court). At the end of the protocol, all videos recorded by the PlaySight system during the protocol were visualized to double check ball accuracy and ball velocity. In addition, technical performance index was calculated for each repetition of the five tennis drills as the product of ball accuracy and ball velocity of the ball, because the combination of these two variables is purposed to better reflect the overall stroke precision in tennis (Brechbuhl et al. 2016). The PlaySight system did not allow accurate recording of ball velocity during the volley-smash drill. Consequently, the means of each

of the other four drills (The star, The box, The X, Suicide) were averaged over days, conditions and participants.

Data and statistical analysis

Data from one participant were discarded for all parameters due to a significant level of fatigue having been induced by previous training before each condition despite the aforementioned recommendations and the specifications of inclusion criteria. Data are presented as mean + standard deviation. Normality of distribution was checked using the Shapiro-Wilk test. MVC peak torque, CK activity, peak-to-peak M-wave amplitude, RPE, Db10 and RMS were found to be normally distributed, while muscle soreness, voluntary activation, RMS_{max}, Db100, Db10.Db100⁻¹, technical performance, ball accuracy and groundstroke errors were not normally distributed. For normal data, the absence of difference between baseline values of the main parameters (MVC peak torque, CK activity, muscle soreness) were evaluated with a one-way (recovery) within-participant, general linear model ANOVA. Non-parametric baseline data were evaluated using a one-way within-participant Friedman test.

Fatigue induced by a tennis protocol (day 1)

When data met normality assumptions, the effect of tennis protocols (PRE–POST tennis) on Day 1 was evaluated with a two-way (time [PRE, POST]×recovery [WBC, CWI, CON]) general linear model ANOVA. For non-parametric data, a Wilcoxon test was performed to evaluate the effect of tennis protocols and a Friedman test was used to assess the effect of recovery interventions on post-exercise measurements.

Daily pre-tennis values

Comparisons between pre-tennis values (baseline, Day 2, Day 3, Day 4) were analyzed using a two-way (time×recovery [WBC, CWI, CON]) within-participant general linear model ANOVA. For non-normal data, a Friedman test assessed the effect of time and, if an effect was observed, was followed by another Friedman test to investigate between-group difference.

Where Mauchly's assumption of sphericity was violated, Greenhouse–Geisser and Huynh–Feldt corrections were used. If a main effect was observed with a Friedman test, post hoc analyses with Bonferroni corrections were performed. Non-parametric tests and general linear model ANOVAs were analyzed using IBM SPSS Statistics version 25 (IBM, Somers, NY). The magnitude of within-group and between-group changes from baseline for dependent variables were quantified using Cohen's d. These results have not been reported to improve the reader's understanding Table 2 Peripheral and central

fatigue indicators

because values ranged between ± 0.1 (small effect) and ± 0.5 (medium effect) and did not provide any new discussion's element.

Results

There were no significant differences between the recovery groups at baseline (p > 0.23) for the following parameters: MVC peak torque, CK activity, muscle soreness, voluntary

Peripheral fatigue indicators PT10 (N.m) CWI 72.50 \pm 15.92 56.86 \pm 17.82 [#] 78.89 \pm 22.74 77.97 \pm 15.45 71.74 \pm 18.4 WBC 78.74 \pm 17.92 57.88 \pm 15.38 [#] 69.44 \pm 20.52 76.87 \pm 16.46 72.48 \pm 21.2 PAS 73.35 \pm 19.51 53.53 \pm 20.21 [#] 71.33 \pm 22.96 75.45 \pm 22.31 73.07 \pm 21.4 PT100 (N.m) CWI 82.1 \pm 9.7 71.7 \pm 12.8 [#] 82.9 \pm 11.7 82.0 \pm 11.3 82.7 \pm 10.4 WBC 85.1 \pm 10.0 72.9 \pm 11.9 [#] 79.8 \pm 9.9 81.6 \pm 9.1 79.4 \pm 16.4 PAS 82.6 \pm 12.9 70.5 \pm 15.7 [#] 77.1 \pm 19.4 81.0 \pm 16.8 84.5 \pm 15.2 Db10/Db100 (a.u) CWI 0.88 \pm 0.12 0.78 \pm 0.12 [#] 0.95 \pm 0.19 0.95 \pm 0.08 0.86 \pm 0.14 WBC 0.92 \pm 0.12 0.79 \pm 0.13 [#] 0.86 \pm 0.19 0.94 \pm 0.12 0.90 \pm 0.1 PAS 0.90 \pm 0.17 0.74 \pm 0.18 [#] 0.91 \pm 0.15 0.92 \pm 0.12 0.86 \pm 0.14 WBC 9.92 \pm 0.12 0.79 \pm 0.13 [#] 0.86 \pm 0.19 0.94 \pm 0.12 0.90 \pm 0.11 PAS 0.90 \pm 0.17 0.74 \pm 0.18 [#] 0.91 \pm 0.15 0.92 \pm 0.12 0.86 \pm 0.14 WBC 0.92 \pm 0.12 0.79 \pm 0.13 [#] 0.86 \pm 0.19 0.94 \pm 0.12 0.90 \pm 0.11 PAS 0.90 \pm 0.17 0.74 \pm 0.18 [#] 0.91 \pm 0.15 0.92 \pm 0.12 0.86 \pm 0.14 CCWI 91.6 \pm 8.3 87.0 \pm 10.6 [#] 91.3 \pm 6.4 92.9 \pm 7.1 93.5 \pm 6.8 WBC 89.9 \pm 10.9 83.4 \pm 14.4 [#] 87.9 \pm 12.6 87.9 \pm 9.9 90.3 \pm 10.4 PAS 91.1 \pm 12.4 84.7 \pm 17.2 [#] 90.6 \pm 12.9 90.1 \pm 14.0 92.2 \pm 8.9 RMS _{max} (a.u) CWI 0.07 \pm 0.03 0.06 \pm 0.02 ^a 0.07 \pm 0.03 0.06 \pm 0.02 0.07 \pm 0.03 0.06 \pm 0.03 0.07 \pm 0.03 0.06 \pm 0.03 0.07 \pm 0.03 0.06 \pm 0.03 0.07 \pm 0.03 0.07 \pm 0.03 0.07 \pm 0.03 0.06 \pm 0.03 0.07 \pm 0.03 0.06 \pm 0.03 0.07 \pm 0.03 0.02 \pm 0.10 [#] 0.22 \pm 0.09 0.24 \pm 0.11 0.25 \pm 0.04 PAS 0.29 \pm 0.14 0.25 \pm 0.12 [#] 0.29 \pm 0.12 0.27 \pm 0.12 0.26 \pm 0.0 PAS 0.29 \pm 0.14 0.25 \pm 0.12 [#] 0.29 \pm 0.12 0.27 \pm 0.12 0.29 \pm 0.09 PAA (mV) CWI 0.61 \pm 1.72 4.43 \pm 1.61 [#] 4.59 \pm 1.76 4.99 \pm 1.76 4.81 \pm 1.66 WBC 4.63 \pm 1.85 4.74 \pm 1.87 [#] 4.34 \pm 2.00 4.49 \pm 1.70 4.67 \pm 2.0 PAS 4.63 \pm 1.66 4.17 \pm 1.72 [#] 4.15 \pm 1.88 4.29 \pm 1.76 4.81 \pm 1.66	Condition	Baseline	Day 1 post-tennis	Day 2	Day 3	Day 4
PT10 (N.m) CWI 72.50 ± 15.92 56.86 ± 17.82 [#] 78.89 ± 22.74 77.97 ± 15.45 71.74 ± 18. WBC 78.74 ± 17.92 57.88 ± 15.38 [#] 69.44 ± 20.52 76.87 ± 16.46 72.48 ± 21.7 PAS 73.35 ± 19.51 53.53 ± 20.21 [#] 71.33 ± 22.96 75.45 ± 22.31 73.07 ± 21.4 PT100 (N.m) CWI 82.1 ± 9.7 71.7 ± 12.8 [#] 82.9 ± 11.7 82.0 ± 11.3 82.7 ± 10. WBC 85.1 ± 10.0 72.9 ± 11.9 [#] 79.8 ± 9.9 81.6 ± 9.1 79.4 ± 16. PAS 82.6 ± 12.9 70.5 ± 15.7 [#] 77.1 ± 19.4 81.0 ± 16.8 84.5 ± 15.7 Db10/Db100 (a.u) CWI 0.88 ± 0.12 0.78 ± 0.12 [#] 0.95 ± 0.19 0.95 ± 0.08 0.86 ± 0.14 WBC 0.92 ± 0.12 0.79 ± 0.13 [#] 0.86 ± 0.19 0.94 ± 0.12 0.90 ± 0.1 PAS 0.90 ± 0.17 0.74 ± 0.18 [#] 0.91 ± 0.15 0.92 ± 0.12 0.86 ± 0.14 CWI 91.6 ± 8.3 87.0 ± 10.6 [#] 91.3 ± 6.4 92.9 ± 7.1 93.5 ± 6.8 WBC 89.9 ± 10.9 83.4 ± 14.4 [#] 87.9 ± 12.6 87.9 ± 9.9 90.3 ± 10.7 PAS 91.1 ± 12.4 84.7 ± 17.2 [#] 90.6 ± 12.9 90.1 ± 14.0 92.2 ± 8.9 RMS _{max} (a.u) CWI 0.07 ± 0.03 0.06 ± 0.02 ^a 0.07 ± 0.03 0.06 ± 0.02 WBC 0.06 ± 0.03 0.05 ± 0.03 ^b 0.06 ± 0.03 0.07 ± 0.03 0.06 ± 0.02 RMS (mV) CWI 0.32 ± 0.09 0.24 ± 0.08 [#] 0.31 ± 0.10 0.30 ± 0.11 0.26 ± 0.0 WBC 0.28 ± 0.16 0.22 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.0 PAS 0.29 ± 0.14 0.25 ± 0.12 [#] 0.22 ± 0.09 0.24 ± 0.11 0.25 ± 0.0 PAS 0.29 ± 0.14 0	Peripheral fa	atigue indicators				·
CWI 72.50±15.92 $56.86\pm17.82^{\#}$ 78.89±22.74 77.97±15.45 71.74±18.4 WBC 78.74±17.92 $57.88\pm15.38^{\#}$ 69.44±20.52 76.87±16.46 72.48±21. PAS 73.35±19.51 $53.53\pm20.21^{\#}$ 71.33±22.96 75.45±22.31 73.07±21.4 PT100 (N.m) CWI 82.1±9.7 71.7±12.8 [#] 82.9±11.7 82.0±11.3 82.7±10.7 WBC 85.1±10.0 72.9±11.9 [#] 79.8±9.9 81.6±9.1 79.4±16. PAS 82.6±12.9 70.5±15.7 [#] 77.1±19.4 81.0±16.8 84.5±15.7 Db10/Db100 (a.u) CWI 0.88±0.12 0.78±0.12 [#] 0.95±0.19 0.95±0.08 0.86±0.14 WBC 0.92±0.12 0.79±0.13 [#] 0.86±0.19 0.94±0.12 0.90±0.1 PAS 0.90±0.17 0.74±0.18 [#] 0.91±0.15 0.92±0.12 0.86±0.14 Central fatigue indicators VA (%) CWI 91.6±8.3 87.0±10.6 [#] 91.3±6.4 92.9±7.1 93.5±6.8 WBC 89.9±10.9 83.4±14.4 [#] 87.9±12.6 87.9±9.9 90.3±10. PAS 91.1±12.4 84.7±17.2 [#] 90.6±12.9 90.1±14.0 92.2±8.9 RMS _{max} (a.u) CWI 0.07±0.03 0.06±0.02 ^a 0.07±0.02 0.07±0.03 0.06±0.02 WBC 0.06±0.03 0.05±0.03 ^b 0.66±0.03 0.06±0.02 0.06±0.0 WBC 0.06±0.03 0.05±0.03 ^b 0.22±0.10 0.30±0.11 0.26±0.0 WBC 0.28±0.16 0.22±0.10 [#] 0.31±0.10 0.30±0.11 0.26±0.0 WBC 0.28±0.16 0.22±0.10 [#] 0.22±0.09 0.24±0.11 0.25±0.0 PAS 0.29±0.14 0.25±0.12 [#] 0.29±0.12 0.27±0.12 0.29±0.0 CWI 4.61±1.72 4.43±1.61 [#] 4.69±1.76 4.99±1.76 4.81±1.6 WBC 4.63±1.85 4.74±1.87 [#] 4.34±2.00 4.49±1.70 4.67±2.0 PAS 4.63±1.86 4.17±1.72 [#] 4.15±1.88 4.29±1.76 4.39±1.6	PT10 (N.n	1)				
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	CWI	72.50 ± 15.92	$56.86 \pm 17.82^{\#}$	78.89 ± 22.74	77.97 ± 15.45	71.74 ± 18.62
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	WBC	78.74 ± 17.92	$57.88 \pm 15.38^{\#}$	69.44 ± 20.52	76.87 ± 16.46	72.48 ± 21.75
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	PAS	73.35 ± 19.51	$53.53 \pm 20.21^{\#}$	71.33 ± 22.96	75.45 ± 22.31	73.07 ± 21.01
CWI 82.1 ± 9.7 $71.7 \pm 12.8^{\#}$ 82.9 ± 11.7 82.0 ± 11.3 82.7 ± 10.7 WBC 85.1 ± 10.0 $72.9 \pm 11.9^{\#}$ 79.8 ± 9.9 81.6 ± 9.1 79.4 ± 16.7 PAS 82.6 ± 12.9 $70.5 \pm 15.7^{\#}$ 77.1 ± 19.4 81.0 ± 16.8 84.5 ± 15.7 Db10/Db100 (a.u)CWI 0.88 ± 0.12 $0.78 \pm 0.12^{\#}$ 0.95 ± 0.19 0.95 ± 0.08 0.86 ± 0.19 WBC 0.92 ± 0.12 $0.79 \pm 0.13^{\#}$ 0.86 ± 0.19 0.94 ± 0.12 0.90 ± 0.17 PAS 0.90 ± 0.17 $0.74 \pm 0.18^{\#}$ 0.91 ± 0.15 0.92 ± 0.12 0.86 ± 0.19 Central fatigue indicatorsVA (%)CWI 91.6 ± 8.3 $87.0 \pm 10.6^{\#}$ 91.3 ± 6.4 92.9 ± 7.1 93.5 ± 6.8 WBC 89.9 ± 10.9 $83.4 \pm 14.4^{\#}$ 87.9 ± 12.6 87.9 ± 9.9 90.3 ± 10.7 PAS 91.1 ± 12.4 $84.7 \pm 17.2^{\#}$ 90.6 ± 12.9 90.1 ± 14.0 92.2 ± 8.9 RMS _{max} (a.u)CWI 0.07 ± 0.03 0.06 ± 0.02^{a} 0.07 ± 0.03 0.06 ± 0.03 WBC 0.06 ± 0.03 0.05 ± 0.03^{b} 0.06 ± 0.03 0.06 ± 0.03 0.07 ± 0.03 WBC 0.32 ± 0.09 $0.24 \pm 0.08^{\#}$ 0.31 ± 0.10 0.30 ± 0.11 0.26 ± 0.00^{a} WBC 0.28 ± 0.16 $0.22 \pm 0.10^{\#}$ 0.22 ± 0.09 0.24 ± 0.11 0.25 ± 0.02^{a} PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.02^{a} PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$	PT100 (N.	m)				
WBC 85.1 ± 10.0 $72.9 \pm 11.9^{\#}$ 79.8 ± 9.9 81.6 ± 9.1 $79.4 \pm 16.$ PAS 82.6 ± 12.9 $70.5 \pm 15.7^{\#}$ 77.1 ± 19.4 81.0 ± 16.8 84.5 ± 15.7 Db10/Db100 (a.u)CWI 0.88 ± 0.12 $0.78 \pm 0.12^{\#}$ 0.95 ± 0.19 0.95 ± 0.08 0.86 ± 0.14 WBC 0.92 ± 0.12 $0.79 \pm 0.13^{\#}$ 0.86 ± 0.19 0.94 ± 0.12 0.90 ± 0.1 PAS 0.90 ± 0.17 $0.74 \pm 0.18^{\#}$ 0.91 ± 0.15 0.92 ± 0.12 0.86 ± 0.14 Central fatigue indicatorsVA (%)CWI 91.6 ± 8.3 $87.0 \pm 10.6^{\#}$ 91.3 ± 6.4 92.9 ± 7.1 93.5 ± 6.8 WBC 89.9 ± 10.9 $83.4 \pm 14.4^{\#}$ 87.9 ± 12.6 87.9 ± 9.9 90.3 ± 10.4 PAS 91.1 ± 12.4 $84.7 \pm 17.2^{\#}$ 90.6 ± 12.9 90.1 ± 14.0 92.2 ± 8.9 RMS _{max} (a.u)CWI 0.07 ± 0.03 0.06 ± 0.02^{a} 0.07 ± 0.03 0.06 ± 0.02 WBC 0.06 ± 0.03 0.05 ± 0.03^{b} 0.06 ± 0.03 0.07 ± 0.03 0.07 ± 0.03 WBC 0.32 ± 0.09 $0.24 \pm 0.08^{\#}$ 0.31 ± 0.10 0.30 ± 0.11 0.26 ± 0.00^{a} WBC 0.28 ± 0.16 $0.22 \pm 0.10^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.02^{a} PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.02^{a} PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.02^{a} PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ <	CWI	82.1 ± 9.7	$71.7 \pm 12.8^{\#}$	82.9 ± 11.7	82.0 ± 11.3	82.7 ± 10.9
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	WBC	85.1 ± 10.0	$72.9 \pm 11.9^{\#}$	79.8 ± 9.9	81.6 ± 9.1	79.4 ± 16.4
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	PAS	82.6 ± 12.9	$70.5 \pm 15.7^{\#}$	77.1 ± 19.4	81.0 ± 16.8	84.5 ± 15.9
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Db10/Db1	00 (a.u)				
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	CWI	0.88 ± 0.12	$0.78 \pm 0.12^{\#}$	0.95 ± 0.19	0.95 ± 0.08	0.86 ± 0.16
PAS 0.90 ± 0.17 $0.74 \pm 0.18^{\#}$ 0.91 ± 0.15 0.92 ± 0.12 0.86 ± 0.14 Central fatigue indicatorsVA (%)CWI 91.6 ± 8.3 $87.0 \pm 10.6^{\#}$ 91.3 ± 6.4 92.9 ± 7.1 93.5 ± 6.8 WBC 89.9 ± 10.9 $83.4 \pm 14.4^{\#}$ 87.9 ± 12.6 87.9 ± 9.9 90.3 ± 10.9 PAS 91.1 ± 12.4 $84.7 \pm 17.2^{\#}$ 90.6 ± 12.9 90.1 ± 14.0 92.2 ± 8.9 RMS _{max} (a.u)CWI 0.07 ± 0.03 0.06 ± 0.02^{a} 0.07 ± 0.02 0.07 ± 0.03 0.06 ± 0.02 WBC 0.06 ± 0.03 0.06 ± 0.03 0.07 ± 0.03 0.06 ± 0.02 WBC 0.32 ± 0.09 $0.24 \pm 0.08^{\#}$ 0.31 ± 0.10 0.30 ± 0.11 0.26 ± 0.00 WBC 0.28 ± 0.16 $0.22 \pm 0.10^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.00 PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.00 PPA (mV)CWI 4.61 ± 1.72 $4.43 \pm 1.61^{\#}$ 4.69 ± 1.76 4.99 ± 1.76 4.81 ± 1.66 WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00 PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.66	WBC	0.92 ± 0.12	$0.79 \pm 0.13^{\#}$	0.86 ± 0.19	0.94 ± 0.12	0.90 ± 0.11
Central fatigue indicators VA (%) CWI 91.6 \pm 8.3 87.0 \pm 10.6 [#] 91.3 \pm 6.4 92.9 \pm 7.1 93.5 \pm 6.8 WBC 89.9 \pm 10.9 83.4 \pm 14.4 [#] 87.9 \pm 12.6 87.9 \pm 9.9 90.3 \pm 10. PAS 91.1 \pm 12.4 84.7 \pm 17.2 [#] 90.6 \pm 12.9 90.1 \pm 14.0 92.2 \pm 8.9 RMS _{max} (a.u) CWI 0.07 \pm 0.03 0.06 \pm 0.02 ^a 0.07 \pm 0.02 0.07 \pm 0.03 0.06 \pm 0.02 WBC 0.06 \pm 0.03 0.05 \pm 0.03 ^b 0.06 \pm 0.03 0.06 \pm 0.02 0.06 \pm 0.02 PAS 0.07 \pm 0.04 0.06 \pm 0.03 0.07 \pm 0.03 0.07 \pm 0.03 0.07 \pm 0.03 0.07 \pm 0.03 RMS (mV) CWI 0.32 \pm 0.09 0.24 \pm 0.08 [#] 0.31 \pm 0.10 0.30 \pm 0.11 0.26 \pm 0.00 PAS 0.29 \pm 0.14 0.25 \pm 0.12 [#] 0.29 \pm 0.12 0.27 \pm 0.12 0.29 \pm 0.00 PAS 0.29 \pm 0.14 0.25 \pm 0.12 [#] 4.69 \pm 1.76 4.99 \pm 1.76 4.81 \pm 1.6 WBC 4.63 \pm 1.85 4.74 \pm 1.87 [#] 4.34 \pm 2.00 4.49 \pm 1.70 4.67 \pm 2.00 PAS 4.63 \pm 1.66 4.17 \pm 1.72 [#] 4.15 \pm 1.88 4.29 \pm 1.76 4.39 \pm 1.60	PAS	0.90 ± 0.17	$0.74 \pm 0.18^{\#}$	0.91 ± 0.15	0.92 ± 0.12	0.86 ± 0.14
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Central fatig	gue indicators				
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	VA (%)					
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	CWI	91.6 ± 8.3	$87.0 \pm 10.6^{\#}$	91.3 ± 6.4	92.9 ± 7.1	93.5 ± 6.8
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	WBC	89.9 ± 10.9	$83.4 \pm 14.4^{\#}$	87.9 ± 12.6	87.9 ± 9.9	90.3 ± 10.4
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	PAS	91.1 ± 12.4	$84.7 \pm 17.2^{\#}$	90.6 ± 12.9	90.1 ± 14.0	92.2 ± 8.9
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	RMS _{max} (a	u)				
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	CWI	0.07 ± 0.03	0.06 ± 0.02^{a}	0.07 ± 0.02	0.07 ± 0.03	0.06 ± 0.02
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	WBC	0.06 ± 0.03	0.05 ± 0.03^{b}	0.06 ± 0.03	0.06 ± 0.02	0.06 ± 0.02
RMS (mV)CWI 0.32 ± 0.09 $0.24 \pm 0.08^{\#}$ 0.31 ± 0.10 0.30 ± 0.11 0.26 ± 0.00 WBC 0.28 ± 0.16 $0.22 \pm 0.10^{\#}$ 0.22 ± 0.09 0.24 ± 0.11 0.25 ± 0.00 PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.00 PPA (mV)CWI4.61 \pm 1.724.43 \pm 1.61^{\#}4.69 \pm 1.764.99 \pm 1.764.81 \pm 1.66WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00 PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.66	PAS	0.07 ± 0.04	0.06 ± 0.03	0.07 ± 0.03	0.07 ± 0.03	0.07 ± 0.02
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	RMS (mV)				
WBC 0.28 ± 0.16 $0.22 \pm 0.10^{\#}$ 0.22 ± 0.09 0.24 ± 0.11 0.25 ± 0.09 PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.09 PPA (mV)CWI4.61 \pm 1.72 $4.43 \pm 1.61^{\#}$ 4.69 ± 1.76 4.99 ± 1.76 4.81 ± 1.66 WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00 PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.60	CWI	0.32 ± 0.09	$0.24 \pm 0.08^{\#}$	0.31 ± 0.10	0.30 ± 0.11	0.26 ± 0.07
PAS 0.29 ± 0.14 $0.25 \pm 0.12^{\#}$ 0.29 ± 0.12 0.27 ± 0.12 0.29 ± 0.09 PPA (mV)CWI 4.61 ± 1.72 $4.43 \pm 1.61^{\#}$ 4.69 ± 1.76 4.99 ± 1.76 4.81 ± 1.66 WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00 PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.66	WBC	0.28 ± 0.16	$0.22 \pm 0.10^{\#}$	0.22 ± 0.09	0.24 ± 0.11	0.25 ± 0.08
PPA (mV) CWI 4.61 ± 1.72 $4.43 \pm 1.61^{\#}$ 4.69 ± 1.76 4.99 ± 1.76 4.81 ± 1.66 WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00 PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.66	PAS	0.29 ± 0.14	$0.25 \pm 0.12^{\#}$	0.29 ± 0.12	0.27 ± 0.12	0.29 ± 0.09
CWI 4.61 ± 1.72 $4.43 \pm 1.61^{\#}$ 4.69 ± 1.76 4.99 ± 1.76 $4.81 \pm 1.67^{\#}$ WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00^{2} PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.60^{2}	PPA (mV)					
WBC 4.63 ± 1.85 $4.74 \pm 1.87^{\#}$ 4.34 ± 2.00 4.49 ± 1.70 4.67 ± 2.00 PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.60	CWI	4.61 ± 1.72	$4.43 \pm 1.61^{\#}$	4.69 ± 1.76	4.99 ± 1.76	4.81 ± 1.64
PAS 4.63 ± 1.66 $4.17 \pm 1.72^{\#}$ 4.15 ± 1.88 4.29 ± 1.76 4.39 ± 1.60	WBC	4.63 ± 1.85	$4.74 \pm 1.87^{\#}$	4.34 ± 2.00	4.49 ± 1.70	4.67 ± 2.09
	PAS	4.63 ± 1.66	$4.17 \pm 1.72^{\#}$	4.15 ± 1.88	4.29 ± 1.76	4.39 ± 1.66

Mean \pm SD. PPA: peak-to-peak M-wave amplitude; Db10: low-frequency doublet peak torque induced by 10-Hz paired stimuli; Db100: high-frequency doublet peak torque induced by 100-Hz paired stimuli; Db100. Db100⁻¹: the high- to low-frequency torque ratio, %; VA: % of the maximal voluntary activation; RMS: the root mean square of the EMG; RMS_{max}: RMS normalized to the PPA.

CWI: cold-water immersion group, WBC: whole-body cryotherapy group, PAS: passive recovery.

#Significantly lower as compared with baseline value. ^aSignificantly higher as compared with baseline for the CWI group. ^bsignificantly lower as compared with baseline for the WBC group. Baseline: pre-tennis values on Day 1; Day 1 post-tennis: post-tennis values on Day 1. Day 2, Day 3, Day 4 values correspond to pre-tennis values on the respective days.

activation, RMS, peak-to-peak M-wave amplitude, RMS_{max} , Db10, Db100 and Db10.Db100⁻¹. No significant differences between recovery groups were observed on Day 1 (p > 0.05) for tennis-specific performance (technical performance, accuracy and groundstroke errors).



Fig. 3 Changes in pre-match values of MVC torque. Mean \pm SD. *CWI* cold-water immersion group, *WBC* whole-body cryotherapy group, *PAS* passive recovery. (level of significance p < 0.05)

Physiological and neuromuscular fatigue induced by a tennis protocol (day 1)

The tennis protocol induced RPE scores of 7 ± 2 , with no main effect of recovery (p = 0.638). For both MVC peak torque and CK activity, a main effect of time (p=0.001) was observed as a result of tennis protocol (Day 1 post-tennis vs baseline); the MVC peak torque decreased by $7.7 \pm 11.3\%$ and CK activity increased from 161.0 ± 100.2 UA L⁻¹ to 226.0 ± 106.7 UA L⁻¹. In the context of recovery interventions context, the same two metrics neither exhibited a main effect of recovery (p = 0.317 and p = 0.701, respectively) nor a time \times recovery interaction (p = 0.307 and p = 0.347, respectively). For voluntary activation, a main effect of time was observed as a result of the tennis protocol; in the CWI, WBC, and PAS groups, voluntary activation significantly decreased by 5.2 ± 6.5 , 7.9 ± 6.6 and $8.0 \pm 9.0\%$, respectively. No between-groups differences were observed at Day 1 post-tennis (p = 0.529) (Table 2). Regarding mechanical response parameters, we found a main effect of time (p = 0.007) for Db10, which decreased by $24.8 \pm 16.2\%$. However, we neither showed a main effect of recovery (p=0.698) nor a time × recovery interaction (p=0.226). Meanwhile, Db100 significantly decreased by $12.6 \pm 12.3\%$ (p = 0.016), $14.4 \pm 9.1\%$ (p = 0.003), and $14.8 \pm 12.5\%$ (p=0.003) in the CWI, WBC, and PAS groups, respectively, with no significant differences observed between recovery groups (p = 0.695). Likewise, Db10.Db100⁻¹ decreased in all groups (CWI p = 0.033, WBC p = 0.026, PAS p = 0.005) with no significant differences between groups (p = 0.234).





Fig. 4 Changes in pre-match values of **a** creatine kinase activity and **b** muscle soreness. Data are expressed as mean \pm SD. *Significant difference compared with baseline for the PAS group. *Significant time effect for CK activity, regardless of the group. #Significant difference for CK activity, regardless of the group.

ference compared with baseline for the PAS group. *CWI* cold-water immersion group, *WBC* whole-body cryotherapy group, *PAS* passive recovery. (Level of significance p < 0.05)

Daily pre-tennis protocol values

MVC peak torque. No significant effect of time (p=0.052) or time × recovery interaction (p=0.329) were obtained from MVC peak torque values. No differences in the pre-tennis values on Day 2, Day 3 and Day 4 were observed in comparison with the baseline (Fig. 3).

CK activity and muscle soreness. A significant effect of time (p = 0.016) was observed on pre-tennis protocol CK activity pre-tennis protocol. Specifically, the values were increased on Day 4 by 194.4 ± 171.2 U.L⁻¹ with no interaction (time × recovery; p = 0.470; Fig. 4a). No main effect of time was observed on muscle soreness for CWI (p = 0.356) and WBC (p = 0.292), though a main effect was identified for PAS (p = 0.005) with peak values of 1.8 ± 1.1 on Day 3 (p = 0.011) and 2.7 ± 2.5 on Day 4 (p = 0.002) (Fig. 4b). No between-recovery group differences in muscle soreness were observed across days.

Peripheral fatigue. We did not find a difference between recovery groups (all p > 0.05) at any time point and no time effect (p > 0.05) in any group for Db10.Db100⁻¹, Db10 or Db100 (Table 2).

Centrale fatigue. Voluntary activation values did not show a main time effect in any group (all p > 0.05; Table 2).

Tennis-specific performance

Ball accuracy. There was an effect of time for the level of ball accuracy in the CWI group (p = 0.040), which significantly increased by $10.6 \pm 7.1\%$ from Day 1 to Day 3 (p = 0.01). In contrast, there was no effect of time for WBC (p = 0.05) or PAS (p = 0.545). We did not observe differences between recovery groups on Day 2 (p = 0.735) or Day 3 (p = 0.500).

Technical performance. An effect of time was observed on technical performance for CWI (p = 0.017), with a performance significantly increased by $10.6 \pm 11.0\%$ on Day 3 vs Day 1 (p = 0.019). In contrast, there was no effect of time for WBC (p = 0.558) or for PAS (p = 0.920). There was no time group difference on either Day 2 (p = 0.920) or Day 3 (p = 0.558) (Fig. 5a).

Groundstroke errors. Throughout the 3 days of tennis protocols, an effect of time on groundstroke errors was observed with CWI (p = 0.002) and with WBC (p = 0.046). For CWI, the number of errors (i.e., balls outside the target zones) significantly decreased by $12.9 \pm 10.6\%$ on Day 3 vs Day 1 (p = 0.001) and by $5.8 \pm 17.6\%$ vs Day 2 (p = 0.001). For WBC, the number of errors significantly decreased by $10.3 \pm 10.4\%$ on Day 3 compared to Day 2 (p = 0.001). No effect of time was obtained on groundstroke errors for PAS (p = 0.472). There



Fig. 5 Changes in tennis-specific performance in terms of **a** technical performance (Technical performance; accuracy×velocity) and **b** groundstroke errors. *Significant difference compared with Day 1 for the CWI group. ^aSignificantly lower as compared with Day 2 for the CWI group; ^bsignificantly lower as compared with Day 2 for the CWI group; ^csignificantly lower as compared with Day 1 for the WBC group; ^dsignificantly higher as compared with Day 2 for the WBC group. *CWI* cold-water immersion group, *WBC* whole-body cryotherapy group, *PAS* passive recovery (level of significance p < 0.05)

were no between-recovery groups differences on either Day 2 (p = 0.472) or Day 3 (p = 0.472) (Fig. 5b).

Discussion

This study investigated the effects of three recovery modalities (WBC, CWI and passive recovery [PAS]) on physiological, perceptual, and functional parameters, including peripheral and central markers of accumulated tennisinduced fatigue and tennis-specific performance, during three consecutive days of tennis protocols. The consecutive days of tennis protocols led to mild muscle damage, but neuromuscular function and tennis-specific performance were not altered throughout the protocol. Daily application of CWI or WBC attenuated muscle soreness with no further impact on any physiological response or performance index.

During tennis protocol, the effective playing time was around 18 min, which for a 1.5-h match is in line with the 20% effective playing time reported in previous studies (Kovacs 2007). This effort elicited a RPE estimated at 7 and a mean intensity of around 142 beats.min⁻¹, which are likewise consistent with previous data (Reid et al. 2008). The work-to-rest ratio ($\approx 1:1,2$) was higher than that during real match play, which normally ranges between 1:3 and 1:5 (Kovacs 2007). However, as this temporal characteristic is more responsible for metabolic fatigue (Mendez-Villanueva et al. 2010; Ferrauti et al. 2001), this was not considered as a major methodological limitation given the assumptions about the mechanisms of action of cold recovery techniques. Tennis protocol on Day 1 were observed to induce a significant decrease in knee extensor MVC peak torque (-7.7%). Although few studies have evaluated physiological and functional responses to less than 2 h of match play, the decline in force observed in the present study is consistent with the range of lower-body force loss described by Girard et al. (2014) and Periard et al. (2014) after 20 min of effective playing time match play.

The observed torque decrement was accompanied by significant decreases in Db10.Db100⁻¹, Db100, Db10 and peak-to-peak M-wave amplitude, suggesting peripheral failure. Similar to findings from previous studies, the decrease in the Db10.Db100⁻¹ ratio suggests that simulated matches altered excitation-contraction coupling (Girard et al. 2008, 2011), defined as low-frequency fatigue (LFF (Vollestad 1997)). Stretch-shortening cycle exercises potentially trigger LFF caused by metabolic by-products factors and/or muscular damage associated with eccentric actions (Martin et al. 2004). Although no parameters assessed in the present study allowed further investigation of the cause and localization of LFF, we could reasonably speculate that excitation-contraction coupling impairment is due to muscle damage rather than metabolic perturbations, as the recovery duration between drills repetitions was sufficient to limit metabolic by-product accumulation (Girard et al. 2011). This hypothesis is supported by the small, but significant increase in CK activity ($\approx 46 \text{ U.L}^{-1}$) that occurred after the tennis protocol (Fig. 4a). Differences in match play duration with respect to studies by Ojala and Häkkinen et al. (2013) and Gomes et al. (2014) could explain the relatively low-amplitude increase in CK activity elicited by one match-like tennis protocol in the present experiment.

In addition to peripheral failure, the observed decline in voluntary activation (from 91 to 85%, Table 2) demonstrated potential impairment of central neural drive following a tennis protocol. In line with previous studies by Girard et al. (2008; 2011), which reported a tennis match-induced reduction in quadriceps muscle activity, our results suggest that tennis protocols induce alterations in neural drive, which probably contributes to the observed torque loss. Our experimental method did not allow further investigation and localization of the neural input impairment. However, Girard et al. (2011) proposed that exercise-induced impairments in central drive occur at the spinal level (i.e., reduced motoneuron excitability, increased presynaptic inhibition), originating from peripheral reflex inhibition via group III and IV afferents that is ultimately attributable to muscle damage. This is consistent with the observed muscle damage putatively elicited by repeated braking actions included in the present protocol (i.e., cutting manoeuvers, postural adjustments, changes in direction and ground receptions), which are recognized to elicit eccentric contractions of the lower limb extensor muscles (Gandevia 2001; Girard et al. 2011). The observed decrease in MVC peak torque concomitant with both peripheral and central activation failure and a rather small but significant elevation of CK activity can confidently be considered to reflect mild induced muscle damage. Effective playing time. Regarding the time course of fatigue markers, an overall increase in CK activity was evident throughout the 3 days of the protocol. However, no differences were found between tennis protocols, and the magnitude of the CK activity increase was rather small (Day $4: +314 \text{ U.L}^{-1}$ vs baseline) and substantially lower than that reported by Ojala and Hakkinen (2013) after three consecutive 2-h matches. The small increase of CK activity is in line with the slight but significant progressive increase in muscle soreness observed in the passive condition over consecutive tennis protocols, and also with the daily recovery of MVC torque, for which values returned to baseline at Pre 2, Pre 3 and Pre 4 (Figs. 3 and 4). Similarly, the acute decreases in peripheral and central drive observed in Day 1 (baseline to Day 1 post-tennis) were recovered on Day 2 and not exacerbated on subsequent days (Table 2) (Periard et al. 2014). This is consistent with the studies by Girard et al. (2014) and Periard et al. (2014) demonstrating that physical capabilities (i.e., sprint abilities and explosive power) were restored to pre-match level within 24 h following a 20 min of effective playing time tennis match. The muscle damage highlighted by increased CK activity and muscle soreness was probably too light to translate into neuromuscular function markers (i.e., voluntary activation, Db10, Db100, Db10.Db100⁻¹) and subsequent functional parameters (MVC peak torque). Consistently, tennis-specific performance as assessed through accuracy, groundstroke errors and technical performance was not affected by the 3 days of tennis protocols (Fig. 5). The daily status of tennis players observed in this study differed from the findings of Ojala and Hakkinen et al. (2013) and Gescheit et al. (2015), who reported decreased physical capacities (MVC force, maximal and explosive force) and concomitant altered tennis-specific performance (i.e., locomotor movement patterns, stroke play characteristics). These discrepancies are also likely attributable to the shorter and fixed duration of the present tennis protocol, where the above-mentioned studies used actual free-paced matches of 2-4 h (Reid and Duffield 2014; Reid et al. 2008). There is still conflicting evidence regarding the manifestation of fatigue on tennis performance, but results from previous studies suggest that fatigue may rather lead to a reduction of movements patterns or in effective playing time (Reid and Duffield 2014; Gescheit et al. 2015). The present standardized and restricted protocol did not allow to investigate these potential tennis-specific fatigue markers. We could also speculate that the three consecutive matchlike tennis protocols induced low-amplitude muscle damage and neuromuscular impairments that did not trigger any substantial alterations in tennis-specific performance (Ojala and Hakkinen 2013; Gescheit et al. 2015). This may be attributed to the lower braking and acceleration movements involved in this protocol than in a real match play associated with a lower stroke frequency $(0,4.s^{-1})$ in the present protocol vs. $0.9.s^{-1}$ in real match play) and/or habituation of the players to the tennis drills (Martin 2018; Girard et al. 2008). These results supported the assumption of Kraemer et al. (2003) that perception of fatigue can persist despite a full recovery of neuromuscular parameters after 24 h following consecutive days of indoor match play.

Regarding recovery modalities, neither CWI nor WBC were effective at attenuating the efflux of CK over the three consecutive days of tennis protocols. Both CWI and WBC have been proposed to limit inflammation, edema and the appearance of blood markers of muscle damage through cold-induced vasoconstriction, likely reducing delayed onset muscle soreness (Ihsan et al. 2016; Mawhinney et al. 2017; Bleakley et al. 2012). These results are not surprising, as the efficiency of cold modalities in limiting exercise-induced muscle damage and associated blood markers is still widely debated (de Freitas et al. 2019; Rowsell et al. 2009; Ihsan et al. 2016). Only a few studies have reported attenuation of CK after CWI exposure within a performance experimental context (Abaidia et al. 2017; Ascensao et al. 2011; Webb et al. 2013; Grainger et al. 2019; Russell et al. 2017), whereas WBC was efficient to reduce CK activity in only one study, after hamstring eccentric contractions, which is less realistic within sports performance (Abaidia et al. 2017). In contrast, WBC and CWI similarly attenuated muscle soreness throughout the consecutive days of the experiment with no differences between the modalities (Fig. 4b). Although the observed analgesic effect of CWI is consistent with previous studies (Tavares et al. 2019; Rowsell et al. 2009; Duffield et al. 2014), the present work is the first to demonstrate a similar result with WBC in practical settings

(Costello et al. 2015). The lack of any significant differences between the cold modalities contrasts with Abaidia et al. (2017) who reported lower muscle soreness with CWI compared with WBC at 24-48 h after high-intensity eccentric protocol. This may originate from the tennis protocols producing less muscle damage and secondary inflammation relative to the high-intensity eccentric protocol used in the above-mentioned study (Abaidia et al. 2017; Pooley et al. 2019). No temporal relationships were observed between the similar benefits of the cryotherapy modalities on the perception on muscle soreness and the time-course of CK activity during the 3 days of tennis protocol. Considering this, it can be postulated that physiological effects induced by CWI and WBC suggested by Mawhinney et al. (2017) is not the primary mechanism implicated in the reduction of muscle soreness in the present study. This latter could only be valuable after exhaustive exercise protocols.

The beneficial effect of these cold modalities could be rather due to a placebo effect given the attenuated perception of muscle soreness in the absence of decreased functional parameters and tennis-specific performances. The role of the placebo effect is well documented as a means for WBC and CWI to enhance recovery through psychological mechanisms (i.e., improved perception of recovery, decreased muscle soreness and fatigue) (Broatch et al. 2014; Wilson et al. 2019). Such alternative mechanisms should be considered given that muscle pain has been shown to reduce force-generating capacity in the absence of muscle damage (Graven-Nielsen et al. 2002). We could speculate that the greater decreases in groundstroke errors observed with CWI and WBC (Fig. 5) could be related to the modalities' positive effects on muscle soreness (Minett and Duffield 2014). Improvement in perceptual outcomes such as muscle soreness or fatigue could be attributed to the athlete's belief in the intervention and the associated expectance effect (Beedie 2007; Wilson et al. 2018). This hypothesis is reinforced by the conflicting results from previous studies comparing the effectiveness of these cold modalities (Abaidia et al. 2017; Wilson et al. 2019; Hohenauer et al. 2020). Cook and Beaven (2013) demonstrated that perception of the effectiveness of CWI was correlated with subsequent performance. This suggests that perceived recovery should be considered when assessing the efficiency of recovery strategies, especially with athletes (Poignard et al. 2020). However, reduced muscle soreness is not necessarily associated with improved technical performance in the literature. Equivocal findings have been reported on the efficiency of CWI recovery techniques at attenuating manifestations of fatigue and limiting performance decrements over consecutive days of exercise (Leeder et al. 2019; Rowsell et al. 2009; Grainger et al. 2019). As functional parameters (MVC peak torque) and central and peripheral neuromuscular fatigue in this study were fully recovered by the day following a match-like

tennis protocol, we unfortunately could not further investigate this question.

Limitations

Pilot studies allowed the design of a standardized matchlike tennis protocol that induces load, muscle damage and neuromuscular fatigue similar to a 1.5-h tennis match. While this standardized protocol was convenient to properly compare the cold modalities, it included some methodological limitations such as a higher work-to-rest ratio compared to a real match play as well as the impossibility to assess the manifestation of fatigue on effective playing time or pacing strategies from tennis players. Participants probably became also accustomed to the repeated standardized exercises, leading to a protective effect over tennis protocols that reduced later fatigue and muscle damage relative to what would be induced in real match conditions. This could explain the observed cross-day trend toward increased accuracy and technical performance and decreased groundstroke errors. Moreover, it is likely that factors such as one's opponent, environmental conditions and mental exertion could influence external and internal load and the manifestations of fatigue. Nevertheless, no methods have been developed to standardize these factors in tennis practice.

Perspective

The present study suggests that consecutive days of tennis exposure (≤ 1.5 h) in well-trained tennis players do not induce cumulative fatigue during tennis tournaments on hard court. The repeated CWI and WBC do not provide the additional benefits expected from cumulative effects over time. Given the moderate-to-high cost and the limited accessibility of these recovery modalities, recovery through cold exposure is not essential and should focus on sleep, rest, nutrition and hydration. Meanwhile, perception of muscle soreness subsequent to successive matchlike tennis protocols was equally attenuated by CWI and WBC recovery techniques. Since CWI and WBC do not negatively affect performance and improve perceptual outcomes of recovery, they can be recommended especially when one considers the unpredictability of the next match duration or when several matches (or trainings) may be scheduled in a day. Perceptual outcomes and player's belief in the recovery should be considered in training and recovery periodization particularly for high-level athletes modality (e.g., monitoring-based practice), given the potential impact of perceived sensation and fatigue on performance (Marcora 2009). Finally, tennis involving both the lower and upper body, it would be interesting to evaluate the generalization of our results to the upper limbs. This would be useful to develop appropriate preparatory trainings and recovery interventions.

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Availability of data and material Not applicable.

Code availability Not applicable.

Declarations

Conflict of interest No potential conflict of interest was reported by the authors.

Ethical approval All procedures conformed to the standards of the Declaration of Helsinki and the study was approved by the ethics committee of Sud Méditerranée IV (no 17 10 05) and the French Health Agency (IRB no 2017-A02255-48).

Consent to participate All participants received both written and verbal information and gave their written informed consent.

Consent for publication All participants gave their consent to publication.

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