Thieme

Effect of External Counterpulsation on Exercise Recovery in Team Sport Athletes

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ABSTRACT

External counterpulsation (ECP), an electrocardiogram-led sequential compression of lower limbs, has been recently proposed for sports recovery, but research is scant. This study examined the effects of an ECP session upon neuromuscular function (vertical jump and torque/velocity characteristics), biochemical responses (creatine kinase, cortisol, testosterone, alpha-amylase and immunoglobulin-A), and muscle soreness (visual analogue scale) following high-intensity exercise. Twenty-one male team sport athletes (age: 21.6 ± 3.4 yrs; height: 182.7 ± 7.3 cm; body mass: 82.7 ± 9.3 kg) recovered from the fatiguing exercise using either ECP or rest. Data collection was conducted at three separate time points: upon arrival (Pre), post-recovery (Post), and 24 h post-recovery (24hPost). Significant main effects for time were observed for increased torque/velocity slope and for decreased isometric extension peak torque ($p < 0.001$). Significant main effects for time were observed for increased creatine kinase, testosterone, alpha-amylase, and muscle soreness (all $p < 0.001$). Significant interaction effects were observed at post-testing following ECP: Cortisol release and the related decline in testosterone/cortisol ratio were attenuated, and immunoglobulin-A was increased following ECP in comparison to the control (all $p < 0.05$). Following high-intensity exercise, ECP has potentially beneficial effects upon biomarkers of recovery, without affecting the neuromuscular function.

Introduction

Athletes are often required to perform repeated high-intensity actions during training and competition [1–3]. This can result in fatigue [4, 5] that leads to degraded athletic performance as well as increases in biochemical markers of stress. Research has demonstrated post-match fatigue-related performance decrements in countermovement jump (CMJ) and sprint performance, while increases in creatine kinase (CK), a biomarker of muscle damage, and cortisol, a stress hormone, have also been documented following fatiguing exercise [6, 7].

Recovery from fatigue is of vital importance in sport. Competing in a state of fatigue can be detrimental in terms of immediate success and can also contribute toward increased injury risk [8–10]. The process of recovery is variable and depends on different factors. Recovery of CMJ, sprint performance, CK, and cortisol to pre-match levels can take up to 96 h across various team sports [6, 9]. However, these timeframes are often not met due to the limited recovery time afforded to athletes owing to demanding training or competition schedules [7, 9]. Thus, means of accelerating the recovery process are of great interest. Massage [11], compression garments [12], cryotherapy [13], cold water immersion [14], and stretching [15] are just a few of the many recovery modalities frequently employed. Although debate exists with respect to what degree such methods truly enhance recovery [16], these approaches are regularly used following training and competition. Given the amount of time and importance conferred to recovery in the ap-

plied setting, research into identifying potential benefits of new modalities is warranted.

External counterpulsation (ECP) is a non-invasive therapy traditionally used to treat patients suffering from angina, heart failure, and other similar conditions [17]. Benefits include, among others, increased exercise capacity [18]. ECP operates using inflatable cuffs that are fastened around the legs and hips. The ECP process is electrocardiogram-led, which ensures that the cuffs inflate and deflate during diastole only, resulting in pressure being relieved prior to ventricular systole. Given that ECP is known to enhance blood flow [19], research has begun to examine the potential for ECP as an effective sports recovery modality. The fact that shear stress associated with ECP has been found to lower pro-inflammatory cytokine levels [20], an effect which could influence the recovery process [21], is one piece of evidence which might support such potential.

Very limited research investigating the use of ECP in sports recovery exists. Catanese [22] examined the effects of ECP on delayed onset muscle soreness (DOMS) and markers of muscle damage and inflammation following long-distance running. ECP was effective at reducing DOMS, inflammation and lactate dehydrogenase. Valenzuela et al. examined the effectiveness of ECP on recovery following high-intensity cycling bouts [23], whereas in a different study they investigated the efficacy of ECP in terms of recovery from exercise-induced muscle damage (EIMD) following plyometric exercise [24]. Results demonstrated neutral effects upon CMJ performance, CK levels, and muscle soreness. However, as acknowledged by the authors, the pressure applied during ECP was only 80 mmHg. Clinical use of ECP involves higher pressures (220–300 mmHg), and as such it is likely that these levels of compression are required to elicit diastolic blood pressure augmentation [18]. The application of higher levels of pressure is a factor we seek to address in our study. Other studies have examined the effects of ECP upon recovery following training and competition in professional rugby league athletes. Results include a neutral effect of ECP upon CMJ performance [25], whereas positive and neutral effects were demonstrated for muscle soreness [25, 26]. Furthermore, testosterone-to-cortisol (T:C) ratio has been shown to increase after recovery with ECP 5 h after a field conditioning session [27] and the day after recovery with ECP one day post-match [26].

Accordingly, this study aimed to examine the effects of ECP in a recovery context upon neuromuscular function, biomarkers of muscle damage, stress and immune function, and subjective perceptions, thereby allowing for analysis of several different components of recovery. Importantly, the pressure applied during ECP was controlled to achieve the therapeutic effect associated with diastolic augmentation, the lack of which has been a limitation of previous work.

Materials and Methods

Subjects and study design

Twenty-one male team sport athletes (age: 21.6 ± 3.4 yrs; height: 182.7 ± 7.3 cm; body mass: 82.7 ± 9.3 kg) volunteered and completed this study. They played at club level in the following sports: rugby, soccer, Gaelic games, basketball and hockey; and trained 3–5 times per week plus one match at the weekend during competitive season. All subjects completed a pre-eligibility questionnaire and provided informed consent. Following completion of a high-intensity exercise (HIE) protocol, subjects received either 20 min of 'ECP' or 20 min of 'Rest' in a randomised, between-subject design. No significant differences were observed in age (20.3 ± 1.0 vs. 23.0 ± 4.5 yrs), height (183.2 ± 5.0 vs. 182.1 ± 9.5 cm) or body mass (82.7 ± 4.7 vs. 82.3 ± 12.9 kg) between the ECP and Rest group, respectively. Given that there is still no consensus on the duration of ECP treatment for sports application, a 20 min protocol was chosen based on previous literature [28] and unpublished data from our laboratory. A single ECP machine (Renew Sport, Singapore) was used to deliver the sessions. Medical screening was conducted for ECP suitability. Eligibility criteria detailed that subjects must be male, involved in a team sport, and aged 18–35, and medical screening consisted of blood pressure and electrocardiogram assessment. The ECP group participants undertook a 20 min familiarisation session of ECP prior to the commencement of the testing period. Testing was conducted at three separate timepoints: upon arrival (Pre), post-recovery (Post), and 24 h post-recovery (24hPost), and consisted of neuromuscular function (NF) testing, biomarker sampling, and assessment of subjective perception of muscle soreness. Biomarker sampling always occurred prior to NF testing. All subjects avoided exercise, and the intake of alcohol, nutritional supplements, caffeine, and anti-inflammatory drugs for at least 36 h pre-treatment, as well as during the 24 h from Pre to 24hPost testing. Ethical approval was granted for this study by the UCD Human Research Ethics Committee – Sciences. The study was conducted according to the ethical standards of the International Journal of Sports Medicine [29].

Neuromuscular function testing

Participants performed two maximal CMJs on a force plate (HUR labs, Tampere, Finland), and a series of knee extensions and flexions on a Cybex dynamometer (Cybex Humac2015®/NORM™, Model 770; Computer Sports Medicine, Stoughton, MA, USA). Standardised warm-up and familiarisation trials of the CMJs and dynamometry preceded data collection.

The CMJ testing required participants to perform a maximal vertical jump without arm swing as is commonly used (e. g., [30]). Jump height, relative peak power and the ratio between flight time and contact time (FT:CT ratio) were analysed as outcome measures.

The participant was then seated in the dynamometer chair with torso, hips, and dominant leg firmly strapped in. The seating position was adjusted appropriately and kept consistent across testing sessions. Alignment of the rotational axis of the dynamometer and the lateral femoral epicondyle was conducted for each participant at each testing period. Subjects were instructed to push 'as hard and as fast as possible' using their dominant leg during testing, and verbal encouragement was provided. Hands were placed across the chest to remove upper body contribution. The testing procedure involved four maximal extensions at 60, 120 and 180 °/s, with 2 min of rest separating each velocity. Additionally, two maximal extensions and two flexions were completed isometrically (0 °/s), with the knee flexed at 60 ° (where 0 ° represents full extension). A 30-second rest period separated each 5-second contraction. The order of the four different movement velocities was randomised for all subjects.

Outcome variables were the T-V slope, calculated from torque results at each of the 4 movement velocities, the isometric hamstring-to-quadriceps ratio (H:Q ratio), and peak isometric torque. To determine the T-V slope, peak torque of each velocity was normalised, with values expressed as a percentage of isometric peak torque at the pre-recovery time point. The slope of the line of these normalised results across the movement velocities was then determined.

Biomarker analysis

Whole-blood CK was assessed from a fingertip capillary sample. A 30 ul sample was taken and immediately analysed using reflectance photometry (Reflotron Plus; Roche Diagnostics GmbH, Mannheim, Germany). Quality control measurements were undertaken using standardised CK strips.

Saliva was collected by unstimulated passive drool. Subjects were instructed not to eat during the 90 min prior to testing and to thoroughly rinse their mouth with water 10 min prior to sampling. Following saliva collection, samples were immediately refrigerated and then frozen at –80 °C within 1 hour. Saliva samples were subsequently analysed for cortisol (C), testosterone (T), immunoglobulin-A (IgA), and salivary alpha-amylase (sAA) by enzyme immunoassay kits (C, T, IgA) and a kinetic enzyme assay kit (sAA) (Salimetrics; Stratech Scientific Ltd., UK) according to manufacturer's instructions. T:C ratio was calculated and results adjusted for secretion rate were also calculated for sAA and IgA, with results expressed in $U \cdot min^{-1}$ and $\mu g \cdot min^{-1}$, respectively. Inter- and intra-assay coefficients of variation (%) for C were 1.41 and 3.80, for T 5.06 and 6.79, for sAA 2.00 and 1.79, and for IgA 4.26 and 4.28, respectively.

HIE protocol

The HIE protocol was completed on an indoor running track, after the completion of a self-directed warm-up. Optojump technology (Optojump Next; Microgate, Bolzano, Italy) was used to assess jump and sprint performance. Subjects began with a maximal CMJ, immediately followed by a '20-metre sprint out' and then a '20-metre sprint back'. On the return sprint, eccentric loading was enforced by a 2.5-metre deceleration zone. Another CMJ was then performed, followed by 30 s of recovery. This constituted one full cycle. The design of the HIE protocol was conducted through review of existing literature and extensive pilot testing. We concluded that 30 s of recovery were sufficient to allow for the completion of repeated cycles, although not so extensive as to allow full recovery. The combination of maximal sprints and CMJs demanded high-intensity, explosive movements from subjects, while also creating large amounts of eccentric loading, all of which would contribute to significant fatigue. CMJ height, sprint time, and rate of perceived exertion were monitored throughout to determine protocol duration. From the average of the first two maximal effort cycles, the criteria for conclusion of the protocol were: a 40 % reduction in CMJ height, a 20 % reduction in sprint time, and a rate of perceived exertion (RPE) of 17 or greater (Borg scale, 6 to 20). When two of these three criteria were met for two consecutive cycles, the protocol came to an end. The number of cycles completed throughout the study ranged from 11–55. The average number of cycles completed was 21.0 ± 13.3 for the Rest group, and 23.4 ± 12.8 for the ECP group. The maximum and minimum number of cycles completed for the Rest and ECP groups respectively were 51 and 12 and 55 and 11. No differences were observed between groups.

ECP and rest conditions

Following the HIE protocol, subjects were given 15 min during which time they were permitted to use the bathroom, to drink water, and to go outside to cool down.

Participants in the ECP condition wore full length trousers and received a 20 min session with individualised cuff sizes. Pressure was increased from 0 to approximately 5 PSI during the first 5 min of treatment, generally remaining between 4 and 5.5 PSI for the rest of the session. The primary aim was to elicit a diastolic-systolic blood pressure peak-to-peak (P-P) ratio of 1.2 or above, with pressure adjusted to achieve this. Pressure modification occurred throughout the session, also accounting for heart rate fluctuation and subject comfort. Average pressure was 4.55 ± 0.52 PSI (235.3 ± 26.9 mmHg), and average P-P ratio was 1.26 ± 0.18 (n = 10: P-P ratio recording unsuccessful for one subject). Across all sessions, pressure never exceeded 6 PSI (310.3 mmHg).

In the Rest condition, the procedure was replicated as detailed above except that cuff inflation did not commence. In both conditions, subjects relaxed for the entire 20 min and were not permitted to use their phone, to read, or to listen to music.

Subjective data

At each testing timepoint, a 200 mm visual analogue scale (VAS) was used to quantify muscle soreness of the thighs [31]. A bodyweight squat was completed, after which the subject placed a mark along the 200 mm scale, with 0 representing no thigh soreness and 200 representing unbearable thigh pain. Following the recovery session, subjects quantified their experience by completing the RPE (Borg Scale, 6 to 20) [32] and Bipolar Comfort (–10 to 10) scales [33]. The collection of subjective data was utilised to capture the participant's experience.

Statistical analysis

Results are expressed as mean (± SD). Variables were checked for normality using a Shapiro–Wilk test and were log-transformed where necessary. NF, biochemical and muscle soreness results were analysed using a 2 (groups: control and intervention) × 3 (time: Pre, Post, and 24hPost) between-within ANOVA. Where a significant interaction F-ratio was found, a Tukey HSD post-hoc analysis was used to identify where differences lay. Effect sizes (ES: Cohen's d) were also calculated, with interpretation thresholds set at 0.00–0.19, 0.20–0.59, 0.60–1.19, 1.20–1.99, and ≥ 2.0, corresponding to trivial, small, moderate, large, and very large effects, respectively [34]. An independent t-test was used to compare RPE and comfort scale at Post between groups. Statistical significance was set at $p < 0.05$. Jamovi software version 0.9.5.14 was used for statistical analysis (www.jamovi.org).

Results

Neuromuscular function testing

▶ **Table 1** shows that no significant time or interaction effects were observed for the CMJ variables, although a main effect for group

was observed for CMJ height ($F = 5.44$, $p < 0.05$). Significant main effects for time were observed for an increase in T-V slope ($F = 9.46$, $p < 0.001$) from Pre to Post and Pre to 24hPost (both $p < 0.05$, $d = 0.85$ and 0.56, respectively), and for a decrease in isometric extension peak torque ($F = 8.67$, $p < 0.001$) from Pre to Post ($p < 0.001$, $d = 0.41$). No main or interaction effect was observed for isometric flexion peak torque and H:Q ratio.

Biomarker analysis

A significant main effect for time ($F = 68.26$, $p < 0.001$) was observed for an increase in CK from Pre to Post, Pre to 24hPost, and Post to 24hPost (all $p < 0.01$, $d = 0.52$, 3.43, and 2.28, respectively) (▶ **Table 2**). Salivary testosterone exhibited a significant main effect for time ($F = 15.02$, $p < 0.001$), with values greater at Post than at Pre and 24hPost (both $p < 0.001$, $d = 1.44$ and 0.62 respectively). sAA demonstrated a significant main effect for time ($F = 10.05$, $p < 0.001$), whereby levels were greater at Post compared to Pre and 24hPost (both $p < 0.01$, $d = 0.29$ and 0.33 respectively). When adjusted for secretion rate, this main effect for time was no longer present, as shown in ▶ **Table 2**. Both raw and secretion rate sAA results showed significant main effects for group ($F = 6.52$, $p < 0.05$ and $F = 5.68$, $p < 0.05$ respectively) (▶ **Table 2**).

Significant interaction effects were observed across three biomarkers. Firstly, for cortisol ($F = 4.07$, $p < 0.05$), ▶ **Fig. 1** shows that levels were greater at Post compared to Pre and 24hPost (both $p < 0.001$, $d = 1.85$ and 1.71 respectively) in the control group, and cortisol in the ECP group was greater at Post compared to 24hPost only ($p < 0.001$, $d = 1.19$). Secondly, in relation to the T:C ratio ($F = 4.54$, $p < 0.05$) and as demonstrated in ▶ **Fig. 2**, values at Post were lower than at Pre and 24hPost (both $p < 0.05$, $d = 0.79$ and 0.31 respectively) in the control group, whereas the ECP group showed a lower result only at Post compared to 24hPost ($p < 0.05$, $d = 3.09$). Finally, ▶ **Fig. 3** shows that for IgA ($F = 4.85$, $p < 0.05$), results in the ECP group at Post were greater than at Pre and 24hPost (both $p < 0.05$, $d = 1.26$ and 0.86 respectively), whereas no change was observed in the control group. When adjusted for secretion rate, no significant interaction effect for IgA remained, as seen in ▶ **Table 2**.

Subjective data

A significant main effect for time ($F = 7.53$, $p < 0.01$) was found for an increase in VAS muscle soreness from Pre to 24hPost and Post to

▶ **Table 1** Summary of neuromuscular function outcome measures (mean ± SD).

	Control			ECP		
Variables	**Pre**	**Post**	**24hPost**	**Pre**	**Post**	**24hPost**
CMJ						
CMJ height (cm) *	34.4 ± 4.1	33.8 ± 5.3	34.6 ± 5.2	40.9 ± 7.8	40.4 ± 7.6	41.3 ± 7.9
CMJ PP/kg (W/kg)	51.3 ± 4.6	50.1 ± 5.1	51.3 ± 6.1	53.6 ± 6.6	52.6 ± 6.2	54.2 ± 6.7
CMJ FT:CT	0.763 ± 0.129	0.738 ± 0.129	0.725 ± 0.143	0.719 ± 0.166	0.741 ± 0.078	0.738 ± 0.082
Dynamometry						
Iso flex PT (Nm)	139.0 ± 19.4	129.1 ± 22.5	139.7 ± 26.7	132.4 ± 19.3	130.1 ± 12.0	132.1 ± 17.5
Iso ext PT (Nm) ^	265.8 ± 56.5	242.4 ± 45.8	255.6 ± 49.9	267.2 ± 44.4	249.7 ± 38.9	257.8 ± 47.1
H:Q ratio	0.539 ± 0.102	0.542 ± 0.100	0.556 ± 0.109	0.500 ± 0.072	0.527 ± 0.059	0.524 ± 0.098
T-V slope ^	–0.219 ± 0.040	–0.166 ± 0.040	–0.186 ± 0.049	–0.197 ± 0.054	–0.167 ± 0.067	–0.175 ± 0.067

CMJ = countermovement jump; PP/kg = peak power per kilogram of body mass; FT:CT = flight time to contact time; Iso flex PT = isometric flexion peak torque; Iso ext PT = isometric extension peak torque; H:Q = hamstring to quadriceps; T-V = torque-velocity; * Significant main effect for group ($p < 0.05$); ^ Significant main effect for time ($p < 0.001$)

▶ **Table 2** Summary of biochemical outcome measures (mean ± SD).

	Control			ECP		
Variables	**Pre**	**Post**	**24hPost**	**Pre**	**Post**	**24hPost**
CK (U/L) *	195.3 ± 145.8	269.1 ± 191.3	529.7 ± 220.2	169.4 ± 113.6	229.3 ± 137.1	697.6 ± 351.9
C (nmol/L)	7.0 ± 6.7	19.4 ± 7.8	6.5 ± 3.8	10.0 ± 6.8	13.4 ± 6.1	6.1 ± 5.8
T (pmol/L) *	696.6 ± 228.5	970.5 ± 456.0	672.8 ± 258.8	685.5 ± 173.7	977.4 ± 543.7	669.2 ± 240.6
T:C ratio	0.17 ± 0.10	0.09 ± 0.14	0.13 ± 0.06	0.10 ± 0.06	0.08 ± 0.03	0.17 ± 0.08
sAA (U/ml) * #	139.5 ± 96.7	155.9 ± 101.5	118.1 ± 69.6	57.8 ± 35.5	87.3 ± 50.2	68.0 ± 72.9
IgA (ug/ml)	229.1 ± 99.4	212.9 ± 88.3	193.0 ± 85.5	179.0 ± 62.9	258.0 ± 119.6	155.4 ± 63.4
sAA S.R. (U·min − 1) #	112.4 ± 90.7	157.3 ± 232.5	184.3 ± 366.7	31.6 ± 20.1	45.0 ± 39.5	50.0 ± 53.2
IgA S.R (ug·min − 1)	183.2 ± 138.7	162.0 ± 119.3	195.7 ± 253.2	91.7 ± 41.4	132.0 ± 125.3	98.9 ± 59.4

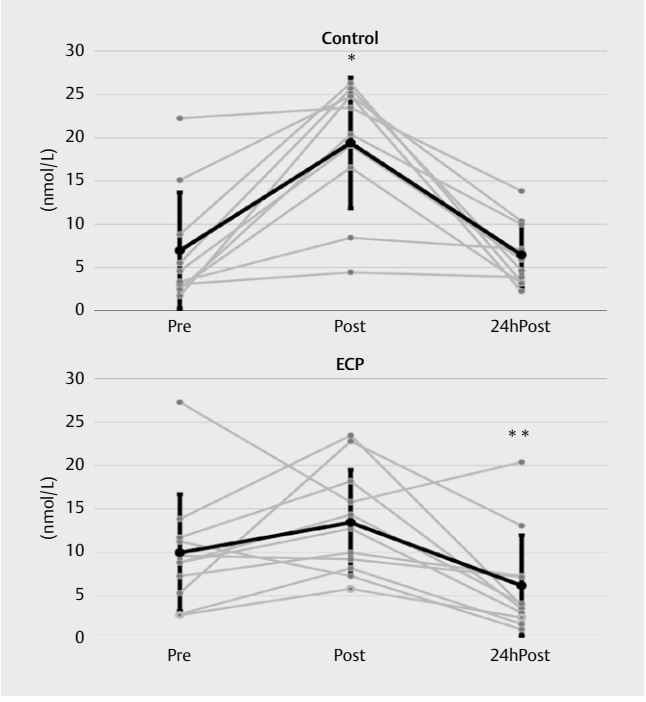
CK = creatine kinase; C = cortisol; T = testosterone; sAA = salivary alpha-amylase; IgA = immunoglobulin-A; S.R = secretion rate; * Significant main effect for time ($p < 0.001$); # Significant main effect for group ($p < 0.05$)

24hPost (both $p < 0.05$, $d = 1.16$ and 0.62 respectively) (▶ **Table 3**). Regarding RPE and comfort scale results, t-tests revealed a significant difference ($p < 0.05$) in RPE between the control and ECP groups, and no difference between groups in comfort rating (▶ **Table 3**).

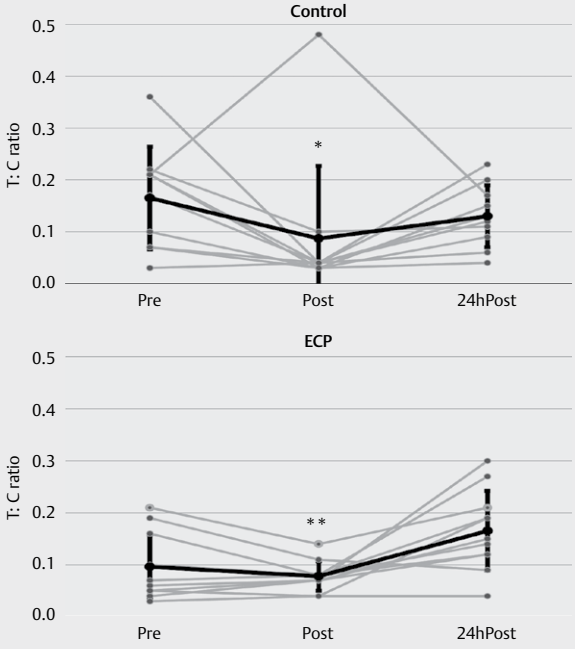
Discussion

This study aimed to build upon the scarce literature on the effect of ECP as a recovery modality following HIE. The effectiveness of the HIE protocol in inducing fatigue was confirmed by an up to threefold increase in CK and VAS muscle soreness at 24hPost, when compared to Pre, with no differences between groups. The main results show a neutral effect of ECP on NF. In contrast, ECP: i) blunted the increase in cortisol at Post; ii) blunted the decline in the T:C ratio at Post; and iii) increased IgA at Post.

ECP demonstrated a neutral effect upon CMJ, which is supported by similar findings [23–25]. Thus we suggest that ECP can be used as a recovery tool without negatively affecting performance 24h later. Congested playing schedules are common, and therefore the need to perform maximally within 24h post-exercise, either for competition or training, is a likely scenario. With regard to the T-V curve, an effect for time was observed for T-V slope. This variable reflects torque-velocity characteristics by representing performance across the different movement velocities. Increased values in this case characterise a greater state of fatigue, as was revealed across both groups at the Post and 24hPost time points. It is notable that this change was manifested through reduced torque-producing capabilities at the slower movement velocities (i. e., 0 and 60 °/s rather than 120 and 180 °/s). Such a finding may also help to explain why no effect of fatigue was found in CMJ performance, because this is a task performed at a high velocity. Al-



▶ **Fig. 1** Salivary Cortisol. Individual values and mean ± SD are presented. * Post significantly different from Pre ($p < 0.001$, $d = 1.74$) and 24hPost ($p < 0.001$, $d = 2.27$). * * 24hPost significantly different from Post ($p < 0.001$, $d = 1.22$).

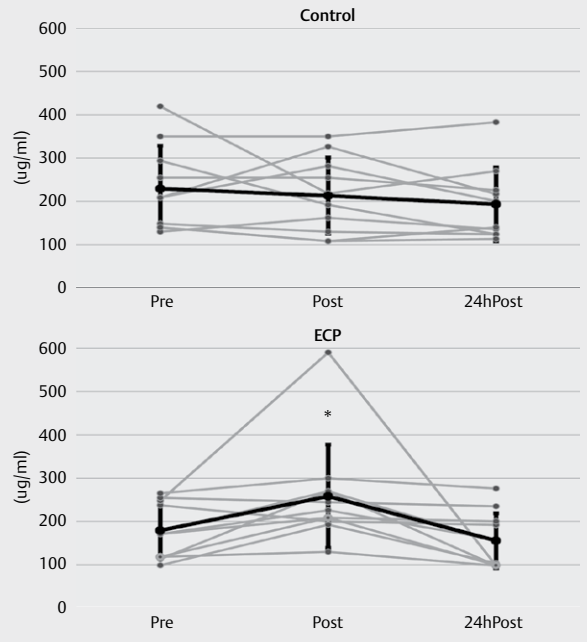


▶ **Fig. 2** Salivary Testosterone to Cortisol ratio. Individual values and mean ± SD are presented. * Post significantly different from Pre ($p = 0.002$, $d = 0.65$) and 24hPost ($p = 0.012$, $d = 0.43$). * * Post significantly different from 24hPost ($p = 0.034$, $d = 1.67$).

▶ **Table 3** Summary of subjective outcome measures (mean ± SD).

	Control			ECP		
Variables	**Pre**	**Post**	**24hPost**	**Pre**	**Post**	**24hPost**
VAS (mm) *	13.3 ± 16.5	28.0 ± 29.4	39.6 ± 33.5	17.9 ± 23.2	18.9 ± 21.0	38 ± 31.6
RPE (6 to 20) ^		6.0 ± 0.0			7.8 ± 2.5	
Comfort (−10 to 10)		6.0 ± 3.5			4.0 ± 3.7	

VAS = visual analogue scale of muscle soreness; RPE = Rate of perceived exertion; RPE and comfort assessed in relation to recovery method;
* Significant main effect for time ($p < 0.01$); ^ Significant difference between groups as assessed by t-test ($p < 0.05$).



▶ **Fig. 3** Salivary Immunoglobulin-A. Individual values and mean ± SD are presented. * Post significantly different from Pre ($p = 0.026$, $d = 0.87$) and 24hPost ($p < 0.001$, $d = 1.12$).

though previous research has demonstrated that fatigue induced at lower velocities can result in torque production at higher velocities being reduced [35], we are unaware of studies reporting how a particular type of fatiguing task affects torque production at a lower but not at a higher velocity; this is an area that warrants further examination.

The acute rise in cortisol levels seen at Post testing in the control group was not observed for subjects who recovered using ECP, suggesting that these subjects experienced a blunting of cortisol release. This finding is supported by previous research which demonstrated lower cortisol levels when ECP was used as part of a recovery protocol [26]. Linked to this change in cortisol levels is the decline observed in the T:C ratio at Post testing for the control group, which was also not found in the ECP group. Existing literature supports the suggestion that the T:C ratio can be positively influenced by ECP [26, 27]. The mechanism, or mechanisms, which may underlie blunted cortisol release following ECP remain unclear. This is especially true given the lack of research with respect to ECP. Recovery methods which involve mechanical compression, such as massage, have been proposed to influence lower cortisol levels through a homeostatic response to stimulation of the parasympathetic branch of the autonomic nervous system [36], although evidence to support this concept is also lacking. Given associations of cortisol with immunosuppressive, catabolic, and protein synthesis-inhibitory effects [37] and physical performance [38], the observed hormonal response to ECP may be associated with several benefits for exercise recovery.

The ECP group also exhibited an acute rise in IgA at Post testing, although no such change was found in the control group. This may represent an improved immune response to HIE with ECP, however must be interpreted with caution as significance was no longer present when concentrations were adjusted for secretion rate. Despite this, the trend between groups from Pre to Post differed in the secretion rate values, as represented in ▶ **Table 2**, whereas a previous study had also demonstrated positive effects of ECP upon markers of immune function [25, 27]. This too represents an area for further research. Overall, the observed effects of ECP upon cortisol, the T:C ratio and IgA may be indicative of a response which reduces catabolism and enhances protein synthesis, which, along with an enhanced immune response, may ultimately contribute to enhanced recovery [37].

The subjective data concerning subject's ratings of their post-HIE recovery indicated a significant difference in RPE, with ECP rated as slightly more exerting than the control condition, a similar finding to [28]. The fact that the mechanical inflation/deflation cycles characterising ECP were perceived as more effortful than rest, but with a low RPE averaging 7.8 (i. e., "very, very light" exertion), and yet not any more uncomfortable than rest, suggests that this would be a readily adopted recovery method in practical settings, with an intensity resembling that of 'active' recovery.

We acknowledge some limitations of the current study. Instead of using a within-subject design, which would have reduced the between-subject variability in response to fatigue, a between/within design was used, with two groups of participants. However, this decision was taken following pilot testing of the HIE protocol, during which evidence of a "repeated bout effect" was observed i. e. participants adapted to a single session of the unaccustomed exercise, with subsequent exercise not resulting in the same level of fatigue as the initial bout [39]. The potential for results to be affected through other factors such as differences in sleep quantity or quality and diurnal variation must also be considered. We did not assess prior sleep patterns, however participants were advised to follow their normal habitual lifestyle. Therefore, although not quantified it is unlikely the ECP group had a significantly different sleep pattern to the control group. Despite attempts to equate testing times exactly between groups, individual subject availability also resulted in imperfect matching where the ECP group began testing 2 h earlier (12 pm) on average than the control group (2 pm). However, no diurnal variation in cortisol would be expected with this time difference [40, 41]. There were no significant differences in baseline cortisol levels between the two groups and there was a 23 hour period, on average, between Post and 24hPost for both groups. Finally, although there did not appear to be any placebo effect, the potential for such an occurrence must be considered, given the challenge associated with blinding in the use of ECP.

To conclude, the use of ECP as a recovery tool following a fatiguing HIE protocol resulted in potentially beneficial effects upon biochemical markers of recovery. This is evidenced by an attenuated rise in cortisol along with an attenuated decline in T:C ratio and increased igA with ECP immediately following exercise compared to control. Thus, with the observation of no negative effects upon performance, in combination with positive biochemical responses which are supported by previous research, we conclude that ECP has the potential to act as an effective sports recovery method following high-intensity fatiguing exercise. Future research should address the use of ECP as a recovery tool in elite athletes.

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Conflict of Interest

Authors declare that they have no conflict of interest.

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