

# Recovery After High-Intensity Intermittent Exercise in Elite Soccer Players Using VEINOPLUS Sport Technology for Blood-Flow Stimulation

François Bieuzen, PhD\*; Hervé Pournot, MS\*‡; Rémy Roulland, PT†; Christophe Hausswirth, PhD\*

\*Research Department, National Institute of Sport, Expertise, and Performance (INSEP), Paris, France; †European Center for Rehabilitation in Sport, Capbreton, France; ‡Laboratory of Human Motricity, Education Sport and Health, University of Nice Sophia Antipolis, Nice, France

**Context:** Electric muscle stimulation has been suggested to enhance recovery after exhaustive exercise by inducing an increase in blood flow to the stimulated area. Previous studies have failed to support this hypothesis. We hypothesized that the lack of effect shown in previous studies could be attributed to the technique or device used.

**Objective:** To investigate the effectiveness of a recovery intervention using an electric blood-flow stimulator on anaerobic performance and muscle damage in professional soccer players after intermittent, exhaustive exercise.

**Design:** Randomized controlled clinical trial.

**Setting:** National Institute of Sport, Expertise, and Performance (INSEP).

**Patients or Other Participants:** Twenty-six healthy professional male soccer players.

**Intervention(s):** The athletes performed an intermittent fatiguing exercise followed by a 1-hour recovery period, either passive or using an electric blood-flow stimulator (VEINOPLUS). Participants were randomly assigned to a group before the experiment started.

**Main Outcome Measures(s):** Performances during a 30-second all-out exercise test, maximal vertical countermovement

jump, and maximal voluntary contraction of the knee extensor muscles were measured at rest, immediately after the exercise, and 1 hour and 24 hours later. Muscle enzymes indicating muscle damage (creatine kinase, lactate dehydrogenase) and hematologic profiles were analyzed before and 1 hour and 24 hours after the intermittent fatigue exercise.

**Results:** The electric-stimulation group had better 30-second all-out performances at 1 hour after exercise ( $P = .03$ ) in comparison with the passive-recovery group. However, no differences were observed in muscle damage markers, maximal vertical countermovement jump, or maximal voluntary contraction between groups ( $P > .05$ ).

**Conclusions:** Compared with passive recovery, electric stimulation using this blood-flow stimulator improved anaerobic performance at 1 hour postintervention. No changes in muscle damage markers or maximal voluntary contraction were detected. These responses may be considered beneficial for athletes engaged in sports with successive rounds interspersed with short, passive recovery periods.

**Key Words:** calf muscle, fatigue, athletes

## Key Points

- After intermittent fatiguing exercise, these elite male soccer players showed better restoration of anaerobic performance with blood-flow stimulation than with passive recovery at 1 hour.
- Neither modality improved clearance of muscle damage markers or maximal voluntary contraction.

Rapid recovery of performance is important for elite athletes engaged in intermittent exercise that involves periods of intense exercise interspersed with short recovery periods (eg, martial arts, ice hockey, field sports). Optimizing training recovery may also be beneficial for performing successive bouts of training or competition over a season without associated fatigue or overtraining effects.

The inability to repeat the same level of performance in short-duration exercise is frequently attributed to peripheral fatigue involving metabolite accumulation and muscle damage<sup>1,2</sup> resulting from mechanical stress, imbalances in muscle cell homeostasis, or local inflammation from exercise.<sup>3</sup> Indeed, the response of different muscle

enzymes (mainly creatine kinase [CK] and lactate dehydrogenase [LDH]) has received researchers' attention because strenuous exercise induces muscle cell structural damage, which results in increased plasma concentrations of muscle enzymes such as CK and LDH.<sup>4</sup> The efflux of CK and LDH proteins from muscle may be attributed to increased permeability of the plasma membrane or intramuscular vasculature (or both).<sup>5</sup> Thus, a reduction in these markers has been proposed as an indicator of recovery after strenuous exercise that induces muscle damage.<sup>6</sup> To optimize recovery, various techniques have been suggested to accelerate the clearance of muscular damage or metabolite accumulations. Usually, these techniques focus on local fatigue. Their main goal is to

treat fatigue by directly applying the recovery method to the working muscles (eg, electromyostimulation, local cryotherapy, or cold-water immersion). This approach showed positive results after muscle damage by reducing local inflammation, especially when cold modalities were used.<sup>7</sup> However, results on peripheral fatigue from metabolite accumulation are inconclusive, probably because the metabolic byproducts are released into the blood. From these findings, a change in the recovery approach from a local treatment to a systemic view was necessary. One possible way to achieve this goal is to improve the peripheral circulation and the venous return by stimulating total blood flow. In athletes, several techniques have been proposed to achieve this result. Of these, active recovery,<sup>8,9</sup> contrast water therapy,<sup>10</sup> compression garments,<sup>11</sup> low-level laser therapy,<sup>12</sup> and low-frequency electromyostimulation<sup>13</sup> have been investigated and compared with passive recovery (PAS).<sup>6,14</sup> The results of these studies provide no definitive consensus on the ability to improve explosive strength and anaerobic capacity performance or clear muscle damage markers after exercise.<sup>15–17</sup> Lattier et al<sup>18</sup> showed no difference in neuromuscular function and maximal test performance after a recovery intervention using blood-flow stimulation from electromyostimulation compared with PAS or active recovery. Based on these observations, several authors concluded that the effects of these techniques are minimal, especially on performance. However, researchers<sup>13,19</sup> have hypothesized that this lack of effect could also be associated with the technique, the device used, or the localization of the electric stimulation (eg, systemic treatment [calf] versus local treatment [quadriceps]), suggesting that the blood flow and, more particularly, the venous return may not be effectively increased. Accordingly, Martin et al<sup>13</sup> recommended optimizing the electric stimulation to better approximate the physiologic contraction of the muscle; a new way of using an electric muscle stimulator on the calf muscles could provide interesting results. This systemic approach is based on results showing that total blood flow can be efficiently stimulated by intensifying the pumping action associated with calf muscle contractions from techniques such as electromyostimulation, cuff inflation, or walking.<sup>20</sup> Indeed, these muscles, which have been termed the “peripheral venous heart,” “calf muscle pump,” and “musculovenous pump,” were responsible for 80% of the venous return<sup>21–23</sup> and considered a second heart. A low-intensity, repetitive mechanical contraction-relaxation muscle cycle may increase local and total blood flow, translocation, and removal of metabolites and reduce intracellular fluid volume.<sup>24</sup> However, using electric muscle stimulation to increase blood flow for exercise recovery has been ineffective despite the emergence of new devices that significantly improved total blood flow and venous return.<sup>25–29</sup> Therefore, we hypothesized that such a device applied to the calf muscles could result in faster restoration in performance and reduce the amount of muscle damage markers after fatiguing exercise.

The purpose of our study was to investigate the effectiveness of muscle stimulation using the VEINOPLUS unit (Ad Rem Technology, Paris, France) on explosive strength and 30-second all-out performance and CK and LDH recovery profiles in professional soccer players after

exhaustive intermittent exercise. We proposed that use of the VEINOPLUS would result in better restoration of anaerobic performance than passive recovery.

## METHODS

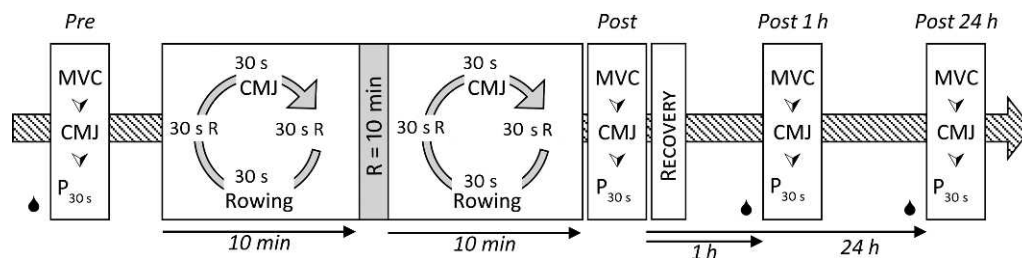
### Participants

Twenty-six healthy professional male soccer players (age =  $25.6 \pm 5.7$  years, height =  $1.77 \pm 0.8$  m, mass =  $75.0 \pm 12.2$  kg,  $\dot{V}O_{2\max} = 60.1 \pm 3.7$  mL·min<sup>-1</sup>·kg<sup>-1</sup>) from 2 teams volunteered to participate in the study and were randomly assigned to 1 of 2 groups: an experimental group using the VEINOPLUS electric blood-flow stimulator (BF<sub>stim</sub>) for recovery or a PAS group. Three participants from the PAS group were unable to complete the fatiguing exercise and were excluded from the study. Inclusion criteria included normal venous capacitance and no venous reflux. None of the participants had a history of heart or circulation disorders. Before the study, each participant was informed about the purpose and risks of the study and signed an informed consent. The experimental protocol was conducted according to the Declaration of Helsinki statement and approved by a local ethics committee, CCP Ile-de-France XI.

### Experimental Design

An overview of the experimental protocol is presented in Figure 1. Participants performed an intermittent fatiguing exercise followed by a 1-hour recovery period. For the next 24 hours, participants had to sleep for at least 9 hours and not play sports or pursue other strenuous activities. Ten minutes before the intermittent fatigue exercise, immediately after, and 1 hour and 24 hours after, the participants performed explosive-strength and 30-second all-out tests. The dependent variables reflecting explosive strength were maximal voluntary contraction (MVC)<sup>30</sup> of the knee extensors and maximal countermovement jump performance (CMJ).<sup>31</sup> Mean power during the 30-second all-out exercise ( $P_{30\text{sec}}$ ) indicated mainly anaerobic capacity.<sup>32</sup> Muscle enzyme analyses indicating muscle damage (CK, LDH) and hematologic profiles were also performed before and 1 hour and 24 hours after the intermittent fatigue exercise. All blood samples were obtained before the explosive-strength and anaerobic-capacity tests to avoid corruption of results. Each participant's perception of quadriceps pain using a visual analogue scale (VAS) was also measured before exercise and on the following day. These measures of muscle enzymes (CK and LDH) and quadriceps soreness are common markers used to assess delayed-onset muscle soreness (DOMS).<sup>7</sup>

**Intermittent Fatigue Exercise.** The intermittent fatigue exercise consisted of 2 sets of 10 minutes, separated by a 10-minute controlled passive rest period. Each set consisted of alternating 30 seconds of CMJ (frequency imposed: 0.7 Hz) and 30 seconds of rowing at power corresponding to 80% of  $P_{30\text{sec}}$  with a recovery period of 30 seconds between exercises. During the 30-second recovery period, participants remained standing. With this protocol, we attempted to generate both systemic metabolic and local muscular fatigue comparable with the demands of a soccer match.<sup>33</sup>



**Figure 1.** Experimental design indicating the exercise performed and data collection for each outcome variable. Electric stimulation for the experimental group was applied during the 20-minute recovery period. Abbreviations: CMJ, vertical countermovement jump; MVC, maximal voluntary contraction;  $P_{30\text{sec}}$ , 30-second all-out rowing exercise; R, Rest. Blood samples are indicated by  $\bullet$ .

**Recovery.** Participants from the  $BF_{\text{stim}}$  and PAS groups remained seated in a chair with minimal movement throughout the 1-hour recovery period. During the first 20 minutes of this recovery period, the  $BF_{\text{stim}}$  group used the electric blood-flow stimulator while seated. The stimulation was applied with 2 skin electrodes ( $8 \times 13$  cm) placed symmetrically on the medial-central part of the calf of both legs (Figure 2). The stimulation consisted of a series of rectangular pulses of low energy ( $<25 \mu\text{C}$ ), low voltage ( $50 V_{\text{peak}}$ ), and low frequency ( $<1.75$  Hz), with a maximum duration of impulse of 240 microseconds. The shape of the current wave was asymmetric and biphasic for each pulse, leading to nearly symmetric contractions of the calf muscles in each leg. The athlete could adjust the current based on tolerance. To limit differences among players, we set a minimal threshold corresponding to a visible but comfortable contraction of the calf muscles. These pulses produced twitches of muscle contractions without pain. The rate of stimulation bursts was preset in the device. The time between bursts changed automatically every 5 minutes. Stimulation frequency was 1 Hz for the first 5 minutes, 1.25 Hz for the subsequent 5 minutes, 1.5 Hz for the next 5 minutes, and 1.75 Hz for the final 5 minutes. The stimulation session lasted 20 minutes and produced 1600 short contractions of each calf muscle.

## Explosive-Strength and 30-Second All-Out Rowing Tests

**Knee Extension Maximal Voluntary Contraction.** Participants reported to the laboratory for familiarization

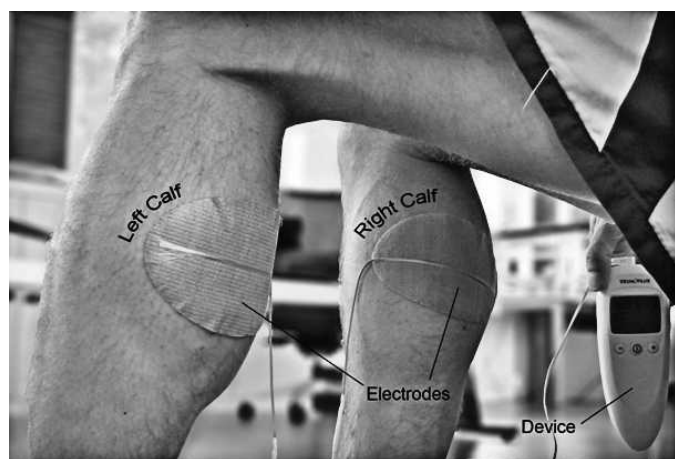
and testing. An isokinetic dynamometer (Biodex System 3; Biodex Medical Systems, Shirley, NY) was used to measure knee extensor torque in the nondominant limb. The device was set up according to the manufacturer's recommendations and anatomical zero was set at a knee angle of  $0^\circ$  (full extension). The MVC was then determined with the knee at a flexion angle of  $70^\circ$ . This angle was marked to ensure consistency during subsequent testing sessions. Before each test began, the moment acting upon the dynamometer power head because of the weight of the lower leg was corrected for gravity. Three maximal-effort knee-extension trial attempts of 3 seconds were performed, separated by 60 seconds of rest. During each test, participants were verbally encouraged to give their maximum effort. The best performance achieved was recorded as the MVC.

**Vertical CMJ.** Jump height was measured using an isoinertial dynamometer (Myotest Pro; Myotest SA, Sion, Switzerland) validated by Jidovstev et al.<sup>34</sup> Participants were asked to keep their hands on their hips to prevent arm movements that could influence vertical-jump performance. They performed 3 maximal CMJs starting from a standing position, with a 1-minute recovery between attempts. Players were asked to jump as high as possible, with the highest jump height used for analysis.

**The 30-Second All-Out Exercise.** The  $P_{30\text{sec}}$  test was performed on an instrumented wind-braked rowing ergometer (Concept2; C2Delivery, Morrisville, VT). After a 5-minute standardized warm-up, participants were asked to row for 30 seconds at maximal effort. Specific attention was given to the use of the lower extremities. Power output values displayed by the ergometer for each stroke were calculated by the C2D system as previously described<sup>35</sup> and recorded using RowPro software (version 1.7; Digital Rowing Inc, Boston, MA). The mean power output ( $P_{30\text{sec}}$ , W) achieved during the test was subsequently calculated from the stroke-by-stroke raw values. We used 80% of this value for the intermittent fatiguing exercise.

## Postexercise Soreness

Perceived soreness, rated using a VAS, was assessed during the performance of a standardized half-squat to ensure a standard reference. Participants ranked their perception of soreness on a scale of 0 (normal) to 10 (extremely sore) before and at 1 hour and 24 hours after the intervention. This method has been used previously as a noninvasive technique to monitor changes in perceived pain after protocols causing muscle damage.<sup>10</sup>



**Figure 2.** Electrode placement for the blood-flow stimulator device.



## Blood Sampling and Processing

**Enzymatic Analyses.** The blood contained in 2 lithium heparin tubes was centrifuged for 10 minutes to obtain plasma. The plasma samples were placed into separated microcentrifuge Eppendorf tubes in multiple aliquots and frozen at  $-80^{\circ}\text{C}$  for subsequent analysis. Plasma CK and LDH concentrations were then determined using an automated clinical chemistry analyzer and reagents (Hitachi 911; Roche Diagnostics Corporation, Indianapolis, IN).

**Hematologic Profile.** With blood from the ethylenediaminetetraacetic acid tube, leukocyte, neutrophil, lymphocyte, monocyte, eosinophil, and erythrocyte counts and hemoglobin and hematocrit concentrations were obtained using an automated cell counter (CELL-DYN Ruby; Abbott Laboratories, Abbott Park, IL).

## Statistical Analysis

Normal distribution of the data was tested using the Kolmogorov-Smirnov test. All variables were expressed as mean  $\pm$  standard deviation. The independent variables of interest were time (before and immediately and 1 hour and 24 hours after exercise) and recovery mode (BF<sub>stim</sub> and PAS). The dependent measure of interest was performance (MVC, CMJ, and P<sub>30sec</sub>), blood variables (CK, LDH, hematologic profile) and perception scale (VAS). A 4 (testing times: before and immediately and 1 hour and 24 hours after exercise)  $\times$  2 (recovery modes) repeated-measures analysis of variance was conducted to compare the effects of recovery interventions on the 3 primary outcome measured variables (MVC, CMJ, and P<sub>30sec</sub>). A 3 (testing times: before and 1 hour and 24 hours after exercise)  $\times$  2 (recovery modes) repeated-measures analysis of variance was calculated to compare the effects of recovery interventions on blood variables (CK, LDH, hematologic profile). A 2 (testing times: before and 24 hours after exercise)  $\times$  2 (recovery modes) repeated-measures analysis of variance was carried out to compare the effects of recovery interventions on the VAS variable. Subsequent post hoc testing was performed using the Tukey test for pairwise comparisons to isolate specific differences when an interaction was observed between time and condition. The data were analyzed using Statistica 7 for Windows (StatSoft, Inc, Tulsa, OK). An  $\alpha$  level of .05 was chosen for all statistical comparisons.

## RESULTS

The P<sub>30sec</sub> values returned to normal more quickly with BF<sub>STIM</sub> than with PAS. In contrast, no difference was detected in MVC, CMJ, blood variables (CK, LDH, and hematologic profile) and VAS score between recovery modalities.

## Performance Analyses

The P<sub>30sec</sub> (expressed as a percentage of the preintervention measurement) data analysis revealed main effects for recovery type ( $F_{1,21} = 10.81$ ,  $P = .003$ ) and for time ( $F_{2,42} = 25.52$ ,  $P < .001$ ). Most importantly, we found a recovery type  $\times$  time interaction ( $F_{2,42} = 3.24$ ,  $P = .04$ ). At 1 hour after intervention, differences were noted between recovery treatments ( $P = .03$ ): the P<sub>30sec</sub> value was higher with BF<sub>stim</sub>

( $101.1 \pm 7.7\%$ ) than with PAS ( $88.8 \pm 9.3\%$ ). No differences were observed at any other time between BF<sub>stim</sub> and PAS ( $P > .05$ ; Figure 3). Examination of the MVC and CMJ data revealed a main effect for time ( $F_{2,42} = 28.13$ ,  $P < .001$ , and  $F_{2,42} = 15.61$ ,  $P < .001$ , respectively); however, there was no recovery effect ( $F_{1,21} = 1.76$ ,  $P = .20$ , and  $F_{1,21} = 0.20$ ,  $P = .67$ , respectively). Similarly, no condition  $\times$  time interaction was seen ( $F_{2,42} = 1.51$ ,  $P = .23$ , and  $F_{2,42} = 0.30$ ,  $P = .74$ , respectively).

## Enzymatic Analyses and Hematologic Profile

Examination of the CK and LDH data revealed a main effect for time ( $F_{2,42} = 15.41$ ,  $P < .001$ , and  $F_{2,42} = 5.19$ ,  $P < .009$ , respectively) but no recovery effect ( $F_{1,21} = .11$ ,  $P = .73$ , and  $F_{1,21} = 2.21$ ,  $P = .15$ , respectively). Again, no condition  $\times$  time interaction was found ( $F_{2,42} = .39$ ,  $P = .67$ , and  $F_{2,42} = 0.21$ ,  $P = .81$ , respectively; Figure 4). The intra-assay and interassay coefficients of variation for all assays were 2.1 to 6.4 and 3.2 to 7.5, respectively.

For both groups, leukocyte, neutrophil, and monocyte counts increased ( $P$  ranged from  $< .001$  to .02; Table) 1 hour postexercise and returned to their baseline values thereafter. No differences were observed for any other hematologic values (ie, lymphocyte, eosinophil, and erythrocyte counts and hemoglobin and hematocrit concentrations) at any point in the protocol.

## Soreness Measurement

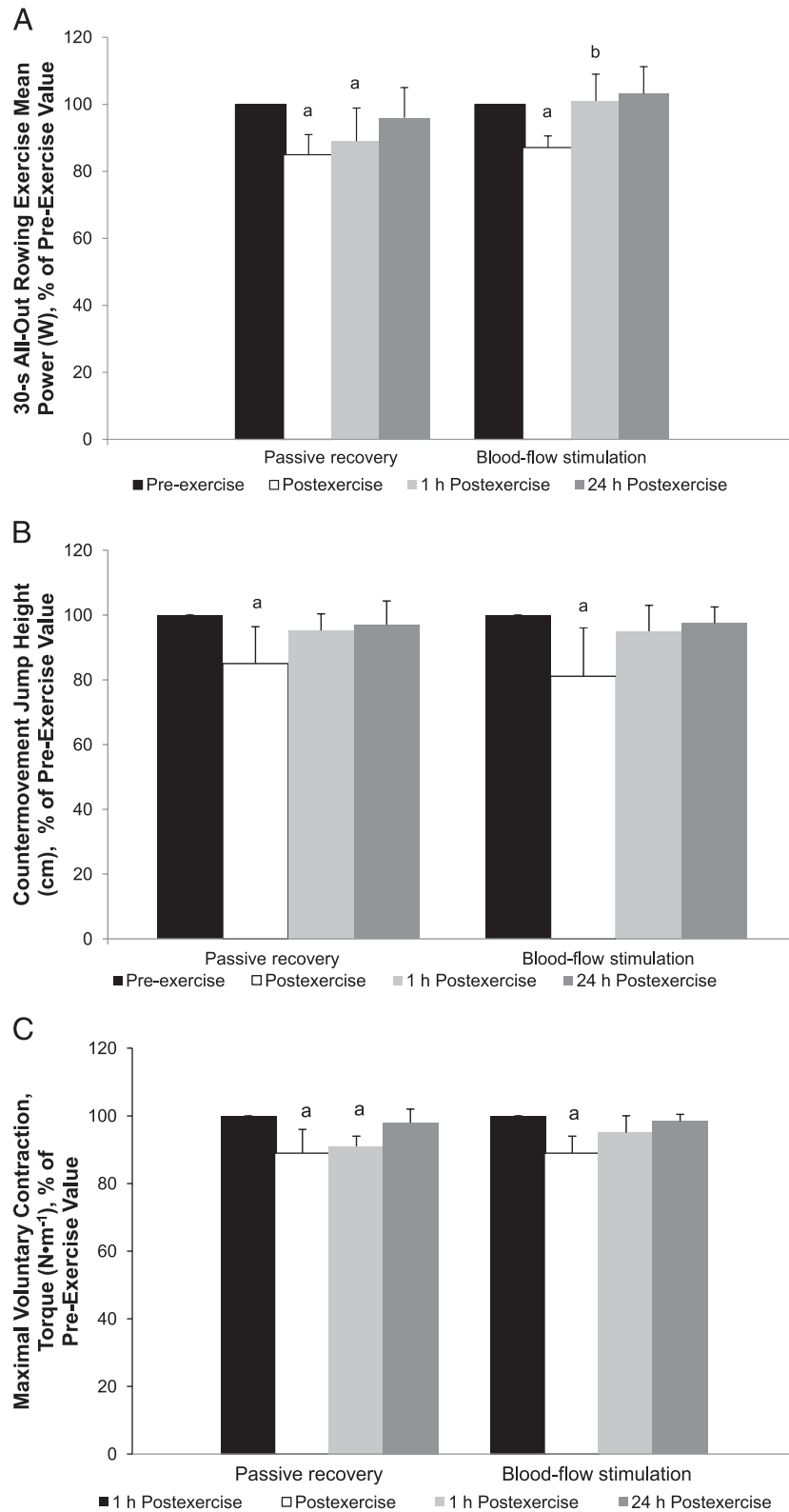
Examination of the VAS score revealed a main effect for time ( $F_{2,42} = 81.28$ ,  $P < .001$ ). However, no recovery effect ( $F_{1,21} = .04$ ,  $P = .84$ ) or condition  $\times$  time interaction was found ( $F_{2,42} = .04$ ,  $P = .84$ ; Figure 4).

## DISCUSSION

Our aim was to determine whether the use of a portable muscle and blood-flow stimulator could improve recovery processes or markers after exhaustive intermittent exercise compared with PAS. By building on the systemic effects of the stimulator, we expected a faster restoration of the explosive-strength properties from increased clearance of the muscle damage markers. This proved to be partly the case. Indeed, the main result indicated faster restoration of the 30-second all-out performance with the BF<sub>stim</sub> recovery intervention in comparison with PAS. However, no difference was noted in values reflecting muscular damage between conditions in the MVC and CMJ data.

## Recovery Modalities and Muscle Damage

**The CK Markers.** Our results show no effect of electromyostimulation on muscle enzyme marker clearance and pain perception. For both groups, CK increased equally 24 hours after exercise, whereas LDH stayed at its initial level. Mixed findings have been reported in the literature, depending on the recovery strategy investigated (eg, wearing compression garments, active recovery, or contrast water immersion) and its localization.<sup>11,14</sup> Duffield et al<sup>11</sup> showed no difference between lower-body compression garments and PAS on CK clearance at 24 hours. In contrast, Gill et al<sup>14</sup> reported faster CK clearance when rugby players used contrast water



**Figure 3.** A, Mean power during 30-second all-out rowing exercise; B, vertical countermovement jump; C, maximal voluntary contraction for the passive and blood-flow stimulation groups (mean  $\pm$  SD). Values were normalized and expressed as a percentage of the pre-exercise values. <sup>a</sup>Different than pre-exercise ( $P < .05$ ). <sup>b</sup>Difference between groups ( $P < .05$ ).

immersion, compression garments, or low-intensity exercise compared with PAS. The main explanations for these heterogeneous results could be the level and time course of the CK enzymes. First, comparing Gill et al<sup>14</sup> and

Duffield et al<sup>11</sup> indicates that the recovery modalities had positive effects during the first 24 hours only when the level of the CK was very high. That was not the case in our study (Gill et al,<sup>14</sup>  $2194.0 \pm 833.7$  U/l; Duffield et al,<sup>11</sup>  $313 \pm$

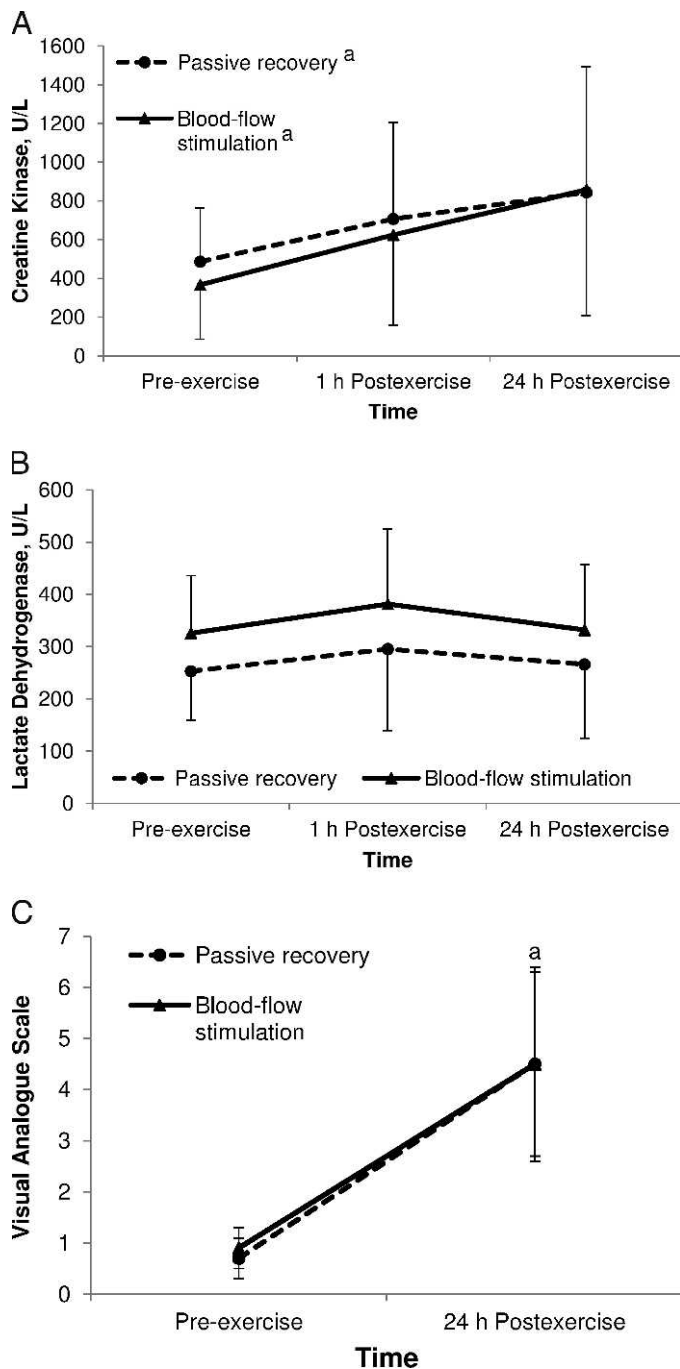


Figure 4. A, Creatine kinase; B, lactate dehydrogenase; and C, perceived soreness (visual analogue scale) before and after the exercise protocol for blood-flow stimulation and passive conditions (mean  $\pm$  SD). Group effect: no difference between the groups for any values: <sup>a</sup>Time effect ( $P < .05$ ).

224 U/L; current study,  $857 \pm 648$  U/L [BF<sub>stim</sub>];  $844 \pm 648$  U/L [PAS]). Second, we focused on short-term recovery ( $\leq 24$  hours), which is a limitation of our study. In the literature,<sup>36</sup> the timing for restoring muscular damage usually exceeded 24 hours. Indeed, for the CK and torque recovery time course, numerous authors have demonstrated that at least 36 hours are needed to notice differences between recovery modalities after an exhaustive eccentric exercise.<sup>37</sup> Finally, most of the researchers who have measured muscle damage used a protocol involving

eccentric exercise,<sup>38</sup> which caused more muscle damage than ours. This kind of exercise may allow better observation of the effect of a particular recovery on CK markers but is further removed from the reality in the field. Moreover, examination of a period exceeding 24 hours may show different results.

**The LDH Markers.** Unlike the CK increases and MVC torque decrease, LDH was not elevated above the baseline in either group. This is consistent with work by Friden et al,<sup>39</sup> who also found elevated serum CK with no change in serum LDH, although the fatiguing protocol was an eccentric exercise of the muscles of the lower leg anterior compartment. Thus, the LDH response may be due to the size of the muscle group affected by the fatiguing protocol. The differences in CK and LDH responses are most likely the result of the structurally different areas in which they are sequestered within the muscle sarcomere and depend on the site of primary mechanical muscle damage.

**Perceived Soreness.** Furthermore, our findings for the muscle enzyme markers were consistent with soreness measurement values for the quadriceps muscles that did not show any improvement despite the recovery modality used. Both results indicate an increase in the markers reflecting DOMS. Two previous groups<sup>13,40</sup> have tested a similar recovery procedure on DOMS after an eccentric exercise protocol. Neither Martin et al<sup>13</sup> using electromyostimulation at 8 Hz on lower limb muscles (pulse width = 400  $\mu$ s, 20–30 mA) nor Weber et al<sup>40</sup> using electromyostimulation at 0.3 Hz (40  $\mu$ A) on upper limb muscles noted a positive effect on DOMS recovery. However, the effectiveness of the tools has been questioned because a frequency that was too high and an intensity that was either too low or too high might not lead to optimal hemodynamic changes. In contrast to these previous studies, Griffin et al<sup>25</sup> measured blood flow and showed that the efficiency of the device in terms of hemodynamic changes cannot be challenged. Despite this, our results demonstrate that the device cannot reduce possible damage to the sarcomere structures or its inflammatory process. Thus, our lack of positive results in reducing damage markers cannot be ascribed to a limited increase in total blood flow. Yet as suggested previously, the muscle contractions involved in the fatiguing exercise of our study were not purely eccentric. From this, we hypothesized that the minimal threshold to observe a positive effect of BF<sub>stim</sub> recovery might not have been reached.

## Recovery Modalities and Performances

Compared with PAS, we observed a positive effect of BF<sub>STIM</sub> recovery modality on the anaerobic performance associated with faster restoration of this component (maximal power output maintained during the 30-second all-out test). In contrast, no effect of recovery treatment was seen on the strength properties (MVC). Both groups reached their baseline CMJ values as soon as 1 hour postintervention, indicating that the fatiguing exercise was not sufficiently exhausting to test a recovery effect. Before discussing these results, we should note that participants were not blinded in our study. We, therefore, cannot, despite the precautions taken in the instructions to limit this negative effect, exclude an ideomotor effect that could influence the results.

**Table. Hematologic Profile Component Measurements Before and 1 Hour and 24 Hours After Fatiguing Exercise, Mean (Range)**

Analyte	Group	Before Exercise	1 h After Exercise	24 h After Exercise
Leukocytes, unit	PAS	5.5 (4.5–7.5)	7.6 (5.2–10.4) <sup>a</sup>	5.2 (3.2–8.2)
	BF <sub>stim</sub>	5.1 (2.8–7.9)	10.0 (4.3–16.5) <sup>a</sup>	5.7 (4.0–8.1)
Neutrophils, unit	PAS	2.67 (1.61–4.17)	4.91 (2.10–7.98) <sup>a</sup>	2.67 (1.48–4.98)
	BF <sub>stim</sub>	2.93 (1.45–4.17)	6.16 (3.64–10.20) <sup>a</sup>	3.05 (1.27–5.08)
Lymphocytes, unit	PAS	2.58 (1.57–7.46)	2.75 (0.90–10.41)	2.78 (1.34–8.17)
	BF <sub>stim</sub>	2.16 (1.63–2.81)	2.57 (2.08–2.98)	1.99 (1.65–2.66)
Monocytes, unit	PAS	0.40 (0.33–0.51)	0.52 (0.34–0.87) <sup>a</sup>	0.42 (0.25–0.63)
	BF <sub>stim</sub>	0.62 (0.32–0.99)	0.81 (0.40–12.10) <sup>a</sup>	0.48 (0.27–0.95)
Eosinophils, unit	PAS	0.23 (0.06–0.60)	0.16 (0.03–0.29)	0.16 (0.04–0.34)
	BF <sub>stim</sub>	0.19 (0.08–0.32)	0.14 (0.06–0.22)	0.18 (0.10–0.30)
Erythrocytes, unit	PAS	4.9 (4.3–5.6)	4.8 (4.4–5.1)	4.8 (4.5–5.3)
	BF <sub>stim</sub>	5.0 (3.9–5.8)	4.8 (3.3–5.5)	4.9 (3.9–5.5)
Hemoglobin, unit	PAS	147 (129–165)	144 (135–153)	145 (135–161)
	BF <sub>stim</sub>	145 (134–157)	141 (107–152)	143 (126–159)
Hematocrit, unit	PAS	43 (38–47)	44 (41–47)	42 (40–47)
	BF <sub>stim</sub>	43 (40–45)	42 (29–45)	42 (38–47)

Abbreviations: BF<sub>stim</sub>, group treated with the blood-flow stimulator; PAS, group treated with passive recovery.

Group effect: no difference between the groups for any values.

<sup>a</sup> Time effect: difference from pre-exercise ( $P < .05$ ).

**The 30-Second All-Out Test.** For many authors, one of the main limits of this short-duration exhaustive exercise (<90 seconds) is related to peripheral fatigue.<sup>1,41</sup> Indeed, high-intensity, short-duration exercise is classically associated with an excess concentration of metabolites, such as inorganic phosphate, hydrogen ions, adenosine diphosphate, free radicals, or carbon dioxide, and occlusion of the circulation.<sup>42,43</sup> Accumulation of metabolites is reported to alter the cross-bridge force production; myofibrillar calcium sensitivity; sarcoplasmic reticulum calcium release, pumping, leakage and binding; and membrane conductance.<sup>44</sup> Neric et al<sup>45</sup> showed that electric muscle stimulation reduced blood lactate after sprint swimming. To our knowledge, this is the only study available in the literature concerning the effect of electric stimulation that shows improvement in lactate clearance but not in anaerobic capacity recovery. Similarly, Yoshida et al<sup>46</sup> demonstrated that enhancing total blood flow and venous return (with active recovery) could improve the removal of inorganic phosphate. In this context, we suggest that greater strength properties and anaerobic performance with BF<sub>stim</sub> at 1 hour postexercise could be mainly attributed to improved removal of metabolites. This was probably due to the efficient and specifically localized stimulation (ie, calf muscles) inducing a systemic action on the blood flow.

**The MVC and CMJ Tests.** The present results indicated similar restoration of the strength properties (MVC and CMJ), no matter which recovery modality was used. Improving blood flow in fatigued or injured muscle has been postulated to enhance recovery by improving microcirculation and regenerative processes and by reducing edema and accumulation of exercise metabolites.<sup>43</sup> In the MVC and CMJ tests, we demonstrated no significant effect of recovery intervention on time during the first hour after the exhaustive task. Mixed findings are reported in the literature depending on the exercise previously performed.<sup>13,18,40,45</sup> In studies using electromyostimulation, the most frequent observations<sup>13,40</sup> showed no further improvement in MVC. These results were also found by researchers<sup>16,31</sup> who attempted to

evaluate the effect of a recovery technique improving blood flow (ie, compression garments) on explosive-strength performance (ie, vertical jump) after a fatiguing exercise. Indeed, they observed no effect of this modality when comparing PAS or contrast water immersion when muscle damage was observed. In these studies, we assume that the fatigue generated was caused more by tissue damage than by metabolite accumulation. In our investigation, the muscle enzyme concentration measured (eg, CK) indicated slight contractile trauma. Even if the muscle trauma was not very marked, previous authors<sup>47</sup> have shown that this level is sufficient to degrade MVC and CMJ performance. From this, we can suggest that the performance decrement in explosive-strength properties could be mainly attributed to tissue damage rather than metabolite accumulation. Based on the previous hypothesis and the CK results, we conclude that the stimulator did not appear to enhance regenerative processes associated with contractile trauma. In the same way, hematologic responses indicated brief increases 1 hour after exercise. This result is classically described in the literature focusing on skeletal muscle damage, but the values we measured do not indicate major tissue damage. Similarly, in the early response to skeletal muscle damage, neutrophils are the most abundant immune cells at the injury site, but within the first 24 hours, if the damage is not too significant, neutrophils start to decline and macrophages increase.<sup>48</sup> Taken together, these results suggest that the specifically localized electric muscle stimulation used in this study may improve the restoration of a part of anaerobic performance. We suggest that this recovery procedure may be attributable to faster clearance of the metabolites and not to decreased tissue damage.

## CONCLUSIONS

The present results indicated enhancement of anaerobic performance restoration (30-second all-out) in elite male soccer players using blood-flow stimulation technology (VEINOPLUS) after intermittent exhaustive exercise compared with forced nonmovement (ie, PAS). In contrast, neither BF<sub>stim</sub> nor PAS improved clearance of muscle



damage markers. The  $BF_{stim}$  responses may be considered beneficial to the specific recovery process of peripheral fatigue but not fully elucidated concerning the enhanced capacity to return to physiologic homeostasis.

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Address correspondence to François Bieuzen, PhD, Research Department, National Institute of Sport, Expertise, and Performance (INSEP), 11 Avenue du Tremblay, 75012 Paris, France. Address e-mail to [francois.bieuzen@insep.fr](mailto:francois.bieuzen@insep.fr).