Original article

The Effect of Submaximal Exercise on Cutaneous Blood Flow, Thermoregulation and Recovery Hemodynamics Following Endurance Exercise

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Abstract

Aim. There are numerous reports of attenuation of cutaneous blood flow (CBF) during the recovery period after a single bout of exercise, but no one has investigated the CBF response to consecutive short-lasting aerobic exercise (SLAE) sessions following exhaustive endurance exercise (EEE) on daily basis, although this is a commonly used training regime in recreational athletes and could cause a cumulative increase in CBF that may be important for wound healing. This study examined the effects of EEE on forearm skin blood flow (LDF), cutaneous vascular conductance (CVC), and mean arterial pressure (MAP) in response to SLAE sessions performed for 7 days after EEE, as well as the correlation between cutaneous blood flow and indices associated with heart rate (HR) to examine the role of thermoregulation in post-exercise HR regulation.

Methods. In 19 recreational runners, LDF, MAP, HR, heart rate recovery (HRR), and HR variability indices (lnRMSSD, lnHF, and lnLF/ HF) were measured after SLAE in the form of submaximal graded cycling performed on consecutive days after EEE in the form of a half marathon and compared with baseline values before EEE. A significant time effect was observed for all measured parameters throughout the study period.

Results. Postexercise LDF increased 24 hours after EEE compared with baseline (77.814 AU compared with 54.712 AU). Postexercise hypotension was significantly more pronounced immediately after EEE compared with baseline (88.95.3 mmHg compared with 99.33.2 mmHg). However, postexercise CVC showed a progressive increase compared with baseline both immediately and 24 hours after EEE (0.53 0.07 AU/mmHg, 0.66 0.09 AU/mmHg compared with 0.4 0.09 AU/mmHg). A small negative correlation between LDF and HRR was observed throughout the experimental protocol.

Conclusion. These results suggest that EEE strongly influences cutaneous and systemic hemodynamics and cardiac autonomic response during recovery after SLAE. Our most important finding was that EEE improved cutaneous perfusion 24 hours after completion of EEE, which may be important for wound healing. The results of our study are potentially applicable for patients with chronic wounds who should be encouraged to exercise moderately on daily basis and to include endurance exercises occasionally, as this strategy potentiates postexercise cutaneous perfusion.

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Introduction

Recreational athletes often include exercises of varying intensity and duration in their daily training schedule. During several consecutive training sessions and subsequent recovery, a complex interplay of many physiological processes, such as thermoregulation, autonomic nervous system adaptation, and nitric oxide production (NO), establishes a dynamic state that may be reflected in cutaneous blood flow (CBF) as the integration site of all the above processes. The term "training recovery" was introduced by Bishop et al. as recovery between successive exercise sessions (1). It can be assumed that the CBF response in training recovery is the cumulative result of recovery and exercise, especially when exhaustive endurance exercise (EEE), with its long-term effects on cardiovascular function (2), is included in the training process. There are numerous reports of CBF in the recovery phase after a single exercise session (3–7) but no one has investigated the CBF response to successive submaximal, short-lasting aerobic exercise (SLAE) sessions following exhaustive exercise on daily basis, although this is a commonly used training regime in recreational athletes. Knowledge of the CBF response to cumulative exercise could be useful in planning exercise strategies for patients with chronic wounds.

During exercise, cutaneous blood flow increases in hairy skin (5), as it is mainly controlled by sympathetic vasodilator fibers. In addition, vasodilation is provoked in an attempt to achieve thermal equilibrium and thus stable core body temperature to allow heat dissipation (8). However, during the recovery phase after exercise, with a sudden decrease in metabolic demand, loss of muscle pump activity, and significant change in autonomic nervous system activity (ANS), cutaneous blood flow rapidly decreases to near baseline levels (5). Nevertheless, core (9,10) and muscle (9) temperatures remain elevated 60-90 min after exercise cessation. Cutaneous blood vessels in non-glabrous skin are innervated by two branches of sympathetic nerves: noradrenergic vasoconstrictor and cholinergic vasodilator fibers. Consequently, the rapid decrease in cutaneous blood flow after cessation of exercise could be caused by increased vasoconstriction, active vasodilation, or a combination of both. Because nitric oxide mediates 30-45% of active cutaneous vasodilation (11), it could potentially contribute to the modulation of cutaneous blood flow after exercise.

After exercise, heart rate (HR) decreases to pre-exercise levels. The initial, rapid phase of HR recovery caused by parasympathetic reactivation is followed by a secondary, slow decline mediated by continued parasympathetic reactivation and sympathetic withdrawal (2). Several studies have examined the mechanisms underlying these post-exercise autonomic responses and have suggested that exercise-induced thermoregulatory demands play an important role (12). There is evidence for the role of thermoregulation in post-exercise HR recovery (13). A possible link between thermoregulation and HR recovery after exercise could be related to thermoregulatory-induced changes in systemic (mean arterial pressure (MAP)) and/or skin hemodynamics (4).

Recently, it has been shown that HR recovery after a short SLAE in response to EEE differs significantly (2), synchronous with changes in ANS activity. Thus, it can be assumed that cutaneous blood flow is simultaneously affected.

Therefore, the aim of this study was to investigate the effects of EEE on cutaneous blood flow in hairy skin after subsequent SLAEs and the relationship between the changes in HR and cutaneous hemodynamic responses to exercise. Therefore, heart rate, arterial blood pressure, and cutaneous blood flow in the volar forearm were measured simultaneously during recovery from SLAE, repeated sequentially before and immediately, 24 hours, 48 hours, and 7 days after EEE. We hypothesized that EEE provokes an increase in cutaneous perfusion and that skin blood flow correlates with concomitant heart rate changes after exercise.

Methods

Subjects

Nineteen recreational runners (13 men) aged 40.4 ± 15.2 years with a body mass index of 23.0 ± 2.7 were recruited to participate voluntarily in this study, which was in accordance with the Declaration of Helsinki and approved by the National Ethics Committee of the Republic of Slovenia (on 10/ 19/2021/No. 0120-126/2021/10). Written informed consent was obtained from all subjects participating in the study. Physical examination and medical history revealed no autonomic dysfunction, chronic diseases, medication use, or smoking. Their ECG and arterial blood pressure were normal. Individual maximum heart rate (HRmax) was determined according to the formula (14).

Procedure

The study was conducted in an air-conditioned laboratory room between 2 pm and 6 pm. Subjects refrained from physical exertion at least three days before the first exercise test and were asked not to perform any other physical activity during the experimental period. They were not allowed to consume alcohol, caffeine, or tobacco for at least 2 hours before the start of each exercise test and were asked to eat a light meal 1 hour before visiting the laboratory. Each participant visited the laboratory 4 times a week for 5 measurements. On the first visit, EEE was performed in the form of a 21-km run on the predefined outdoor track, on partly sunny days with temperatures between 17˚C and 20˚C and humidity between 55% and 65%.

Experimental sessions. SLAE was repeated at 5 different time points: before, one hour (day 0), 24 hours (day 1), 48 hours (day 2), and 7 days (day 7) after EEE, as shown in Figure 1A. Values recorded in the "before" session are referred to as baseline values. SLAE consisted of a 5-minute rest in a seated position followed by a graded exercise test on the Ergoselect 100 (Ergoline, Germany) cycloergometer, starting at 40 W for 3 minutes and continuing with a load increase of 50 W every 3 minutes until the target heart rate (HRpeak), defined as 85% of the individual HRmax, was reached (Fig. 1B). Immediately thereafter, subjects terminated exercise and remained in a seated position for an additional 15 min to passively recover from SLAE.

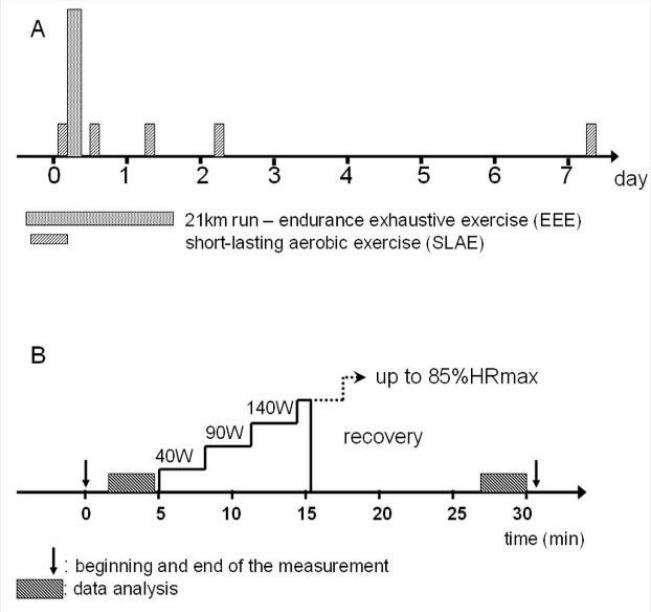

Figure 1. Experimental protocol: schedule of successive exercise bouts (A); short-lasting graded cycling protocol (B)

Measurements

ECG and arterial blood pressure on the middle finger of the right hand were measured continuously (Finometer, Amsterdam, Netherlands). During the measurement, the right arm and hand were placed in an arm rest when sitting on the cycle ergometer that was adjusted to the height of the heart and equipped with hemodynamic sensors. Cutaneous blood flow was measured by laser Doppler (LDF) perfusion monitoring (PeriFlux System 5000, Perimed, Stockholm, Sweden) on the right volar forearm. The principles of LDF monitoring are described elsewhere (15). All signals were recorded simultaneously at 500 Hz using WinDaq data acquisition software (DataQInstruments Inc., Ohio, ZDA). At each SLAE, a time interval of 180 s between the 12th and 15th minutes after cessation of cycling was selected for further analysis. Averaged LDF and MAP were determined using Nevrokard software

(Nevrokard, Slovenia). CVC was calculated as the ratio of LDF to MAP.

Heart rate analysis. RR interval duration was determined beat by beat from ECG using aHRV_file_preparation software (Nevrokard, Slovenia), with artefacts and premature beats corrected. Heart rate (HR) and root mean square of successive differences (RMSSD) as time-domain markers of parasympathetic modulation (Task Force of the European Society of Cardiology) were calculated from RR interval time series using aHRV_analysis_software (Nevrokard), and the natural logarithm of RMSSD (lnRMSSD) was calculated. The following frequency-domain parameters were determined using the autoregressive method (Nevrokard, Slovenia): the power of the high-frequency band (HF; 0.15-0.40 Hz), a marker of parasympathetic modulation, and the LF/HF ratio, which is considered a marker of sympathovagal balance, where LF denotes the power of the low-frequency band (0.04-0.15 Hz) (16). HF and LF/HF were expressed in the natural logarithmic scale (lnHF)

Heart rate recovery in 30 (HRR30) and 60 s (HRR60) was defined as the differences between HRpeak and HR, measured 30 and 60 s after termination of SLAE for each SLAE session.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics, version 27 (IBM, New York, USA). Data were tested for normality and log transformed if they were not normally distributed. We compared the mean differences between the measured parameters over time (before, day 0, day 1, day 2, and day 7) with a one-way repeated-measures analysis ANOVA (rANOVA). The assumption of sphericity was checked with the Mauchly's test; Greenhouse-Geisser or Huynh-Feldt corrections were applied when the sphericity assumption was violated, as published elsewhere (17). When a significant time effect was detected, appropriate contrast tests were used to detect differences between mean values at day 0, day 1, day 2, and day 7 compared with the corresponding baseline (before) values. For post hoc comparisons, the least significant difference test was applied and Bonferroni correction was used to eliminate type I errors in multiple comparisons.

Pearson's product-moment correlation coefficients (r) were used to estimate bivariate correlations between each index of cardiac autonomic activity (HRR30, HRR60, lnRMSSD, lnHF, and lnLF/ HF) and subsequent LDF. SSE performance indices (Pmax, HRstmax, RPE) were combined over the entire experimental period. The following criteria were used to interpret the magnitude of correlations: $r \leq 0.1$ - trivial; $r > 0.1$-0.3 - small; $r > 0.3$-0.5 - moderate; $r > 0.5$-0.7 - large; $r > 0.7$-0.9 - very large (17). Data are presented as mean ± standard deviation, and a confidence level $p < 0.05$ was chosen.

Results

LDF, CVC and MAP

In recovery from subsequent SLAE, there was a significant time effect found in LDF ($F=2.9$; $p=0.026$; $\eta 2=0.08$), CVC ($F=4.5$; $p=0.003$; $\eta 2=0.11$) and MAP ($F=2.8$; $p=0.030$; $\eta 2=0.10$), as displayed on Figures 2B, 2C and 2A, respectively.

LDF was significantly augmented 24 hours after EEE compared to baseline (Fig. 2B), while CVC was enhanced already one hour after EEE cessation and even more 24 hours after EEE compared to baseline conditions (Fig. 2C). MAP was significantly decreased one hour after EEE cessation (Fig. 2A) regarding to baseline.

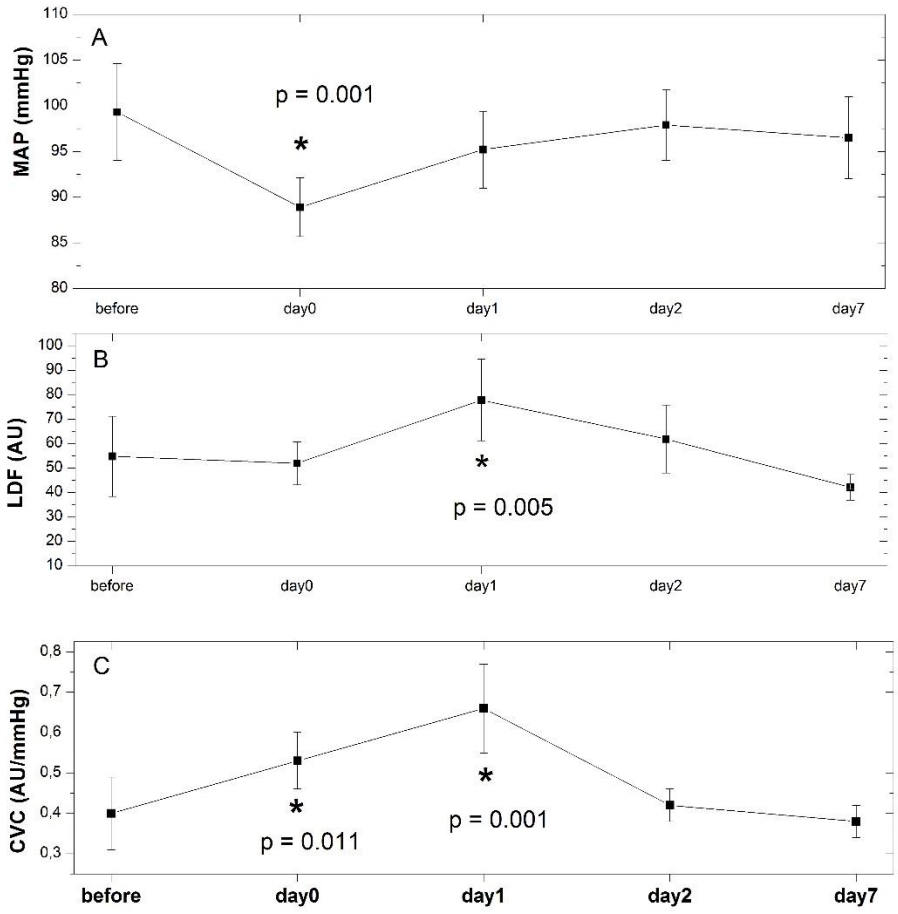

Figure 2: Changes in mean arterial pressure (MAP) (A), laser Doppler flow in the forearm (LDF) (B) and cutaneous vascular conductance (CVC) (C) during experimental protocol (before – before exhausting endurance exercise (EEE); day 0 – 1 hour after EEE; day 1 – 24 hours after EEE; day 2 – 48 hours after EEE, day 7 – 1 week after EEE).

*** - statistically significant compared to baseline (before)**

p – p value according to rANOVA

HR and HR-derived measures

There was a significant time effect found in HR after SLAE ($F=45.6$; $p<0.001$; $\eta 2=0.38$), HRR30 ($F=16.2$; $p<0.001$; $\eta 2=0.20$) and HRR60 ($F=19.6$; $p<0.01$; $\eta 2=0.16$) across all measuring points as displayed in Figure 3. All three parameters exhibited a biphasic change compared to baseline: HR was significantly higher at day 0 and significantly lower at day 1 compared to baseline (Fig. 3), yet HRR30 and HRR60 were significantly lower at day 0 and significantly higher at day 1 compared to baseline (Fig. 3). Additionally, HR after SLAE was decreased also at day 2 compared to baseline (Fig. 3).

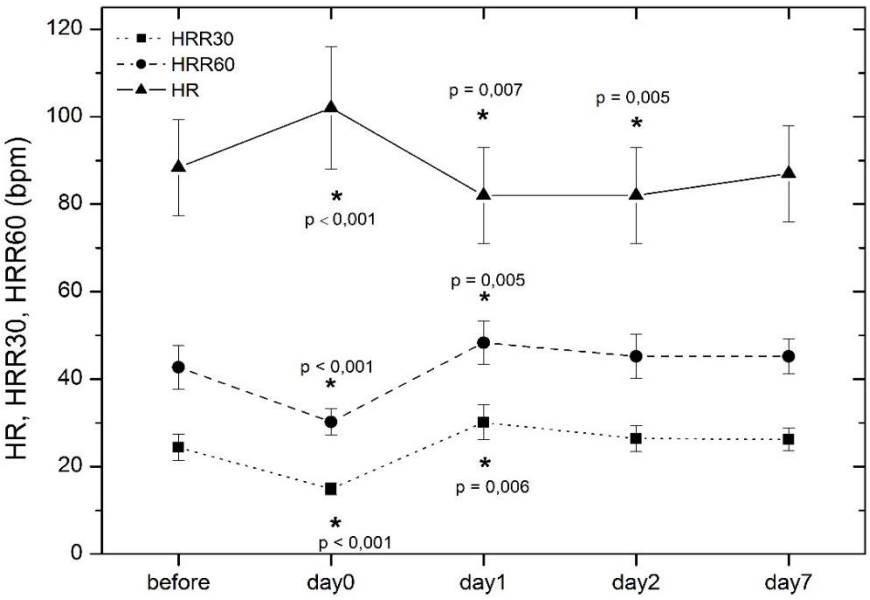

Figure 3: Changes in heart rate (HR) after short-lasting aerobic exercise, heart rate recovery in 30 s (HRR30) and heart rate recovery in 60 s (HRR60) during experimental protocol (before – before exhausting endurance exercise (EEE); day 0 – 1 hour after EEE; day 1 – 24 hours after EEE; day 2 – 48 hours after EEE, day 7 – 1 week after EEE).

*** - statistically significant compared to baseline (before)**

p – p value according to rANOVA

The parameters of heart rate variability, lnRMSSD ($F=10.1$; $p<0.001$; $\eta^2=0.19$), lnHF ($F=7.3$; $p<0.001$; $\eta^2=0.13$) and lnLF/HF ($F=7.6$; $p=0.011$; $\eta^2=0.22$) exhibited significant time effect along measuring protocol (Fig. 4). lnRMSSD behaved biphasically compared to baseline, while lnHF and lnLF/HF differed significantly from baseline only at the day 0 (Fig. 4).

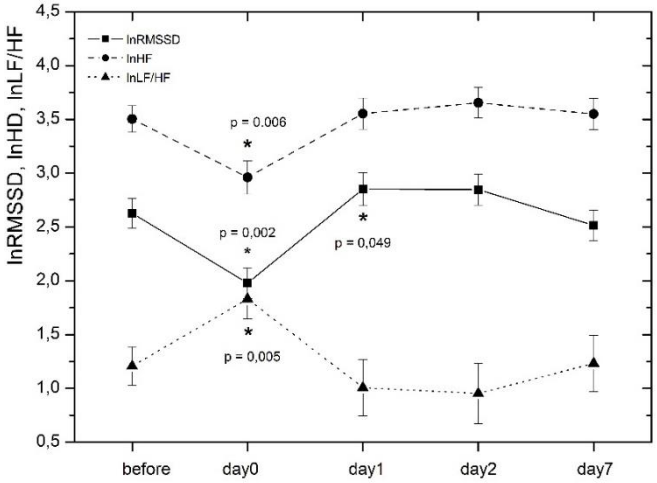

Figure 4: Changes in natural logarithm of root mean square of successive differences (lnRMSSD), the power of high-frequency band (lnHF) and the relationship between the power of low-frequency band to high frequency band (lnLF/HF) during the measuring protocol.

*** - statistically significant compared to baseline (before)**

p – p value according to rANOVA

Bivariate correlations

When all data were pooled there were no clear correlations neither between LDF and HR

(p=0.93), lnRMSSD (p=0.66), lnHF (p=0.17) and lnLF/HF (p=0.34) nor between CVC and HR (p=0.17), lnRMSSD (p=0.78), lnHF (p=0.42), lnLF/HF (p=0.65), HRR30 (p=0.09) and HRR60 (p=0.18), respectively. A small negative correlation was observed between LDF and HRR30 (p=0.036, r= -0.229) as well as between LDF and HRR60 (p=0.032, r= -0.233), as seen in Figures 5A and 5B.

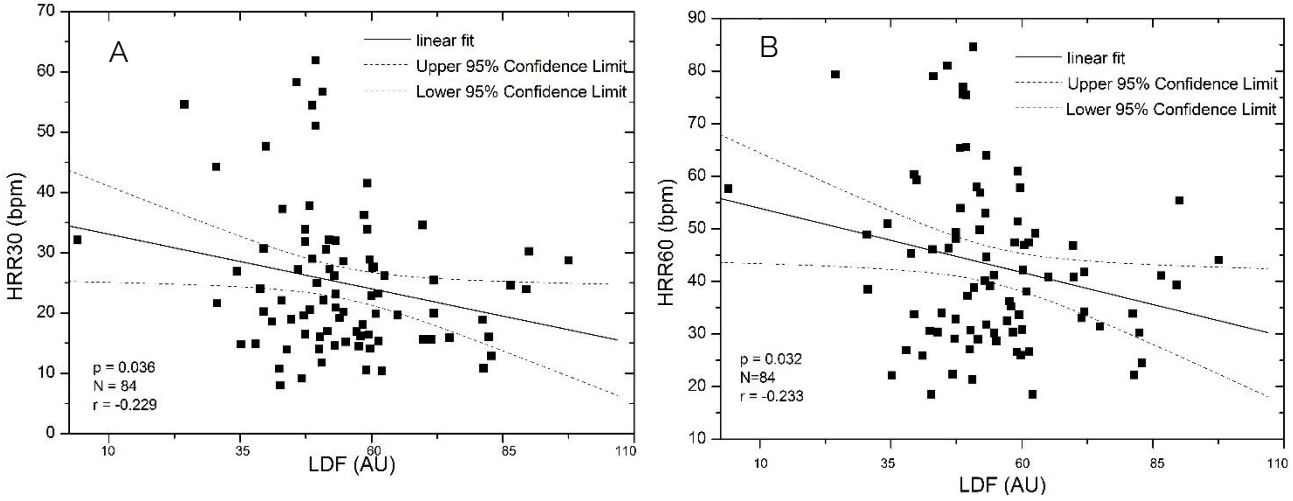

Figure 5: Linear fit between laser-Doppler flow (LDF) in the forearm during recovery from short-lasting aerobic exercise and heart rate recovery in 30 s (HRR30) (A) and heart rate recovery in 60 s (HRR60) (B); 95% confidence limits are indicated by a dashed line. p – p value according to rANOVA; r – Pearson's coefficient; N – number of correlated points

Discussion

In the present study, we investigated the response of cutaneous blood flow in hairy skin in the recovery phase after short lasting aerobic exercise at different time points after exhaustive endurance exercise and compared it with concomitant changes in cardiac autonomic activity in recreational runners. The aim of this study was to contribute to the understanding of the control of cutaneous blood flow in relation to training history. We found that training history has a strong influence on cutaneous (LDF and CVC) and systemic hemodynamics (MAP), as well as on cardiac autonomic (HR, HR derived indices) response during recovery from SLAE. Our most important finding was that exhaustive endurance exercise as part of the training history improved cutaneous perfusion 24 hours after completion of EEE, which may be important for wound healing.

The results of the present study are as follows:

- LDF in the recovery phase at SLAE after exhaustive endurance training was significantly increased 24 hours after EEE compared with baseline, which might be important for wound healing,

- A progressive increase in CVC after training was observed during the first 24 hours after exhaustive endurance training,

- Post-exercise hypotension was significantly increased immediately after exhaustive endurance training, and

- A small bivariate correlation between LDF and HRR30(60) was observed throughout the experimental protocol, indicating the role of thermoregulation in post-exercise regulation HR

To our knowledge, this is the first study to report time-dependent changes in LDF during exercise recovery after exhaustive endurance training. Accordingly, we can only speculate about the mechanisms controlling these changes. During the recovery phase after exercise, there is a rapid decrease in cutaneous perfusion despite a

significant residual heat load (18–21). It has been suggested that both centrally mediated (sympathetic adrenergic and cholinergic innervation) and peripheral endothelium-dependent and independent factors may modulate the control of cutaneous blood flow after exercise (9,22) and that either enhanced vasoconstriction or attenuated active vasodilation may play the role (18). Since nitric oxide mediates 30-45% of active cutaneous vasodilation (18), it seems likely that the bioavailability of NO may additionally influence post-exercise vasodilation. However, the time-dependent contributions of all potentially involved mechanisms during recovery after exhaustive endurance training remain unclear.

According to the literature, postexercise hypotension is caused by at least two mechanisms: increased vasodilation due to thermoregulatory challenges and/or impairment of baroreceptor reflex (23). The predominant mechanism was found to depend on exercise intensity and duration (24). Liu et al. reported that prolonged endurance exercise induces significant postexercise hypotension regardless of exercise intensity (24), and it has been shown that, at least in males, reduced cardiac baroreflex sensitivity triggers postexercise hypotension (25) after a half marathon. In addition, McGinn et al. (26) found decreased baroreflex sensitivity as a major cause of postexercise hypotension after 90 minutes of graded dynamic exercise.

Surprisingly, LDF, CVC, and MAP did not change synchronously during the measurement protocol, although this was generally expected. Based on CVC, which was increased immediately and 24 hours after EEE compared with baseline, one would expect increased postexercise hypotension and skin vasodilation at the same measuring points. However, MAP was significantly decreased only immediately after EEE and LDF was significantly decreased only 24 hours after EEE, suggesting that different mechanisms are involved in the regulation of MAP and LDF. The main cause of the postexercise increase in CVC immediately after EEE appears to be a decreased MAP, whereas its further increase 24 hours after EEE is due to an increased LDF at that measurement time point.

Response immediately after exhaustive endurance exercise

Immediately after EEE, the postexercise decrease of MAP observed in our study compared with baseline (Fig. 2) can be attributed to an impairment of the baroreflex, as suggested by Mourot et al.

After endurance exercise, core body temperature and mean muscle temperature remain elevated for up to 24 hours (20). Therefore, increased cutaneous blood flow during recovery from short lasting aerobic exercise performed immediately after EEE, compared with baseline before endurance exercise, would be expected to eliminate the additional heat load generated during EEE. However, in our study, postexercise LDF was not increased after EEE compared with baseline. The concomitant observed dominance of the cardiac sympathetic nervous system activity over the parasympathetic activity (Fig. 4) suggests that the simultaneously increased adrenergic vasoconstrictor nerve activity affecting the skin may prevent vasodilation.

Another possible explanation would be increased oxidative stress immediately after endurance exercise. Numerous data show that a decreased ability to release NO in the skin microcirculation is associated with increased oxidative stress (19,27). Interestingly, a biphasic response of exhaled NO, a potent surrogate for oxidative stress (28), was reported after endurance exercise (29). More specifically, exhaled NO increased significantly immediately after endurance exercise and decreased 24 hours after endurance exercise compared with the level before endurance exercise. Consequently, we can probably assume that increased oxidative stress immediately after EEE induces endothelial dysfunction with the impairment of NO availability. Immediately after a half marathon, the response of the cardiovascular system to short lasting aerobic exercise has been shown to mimic the response found in cardiovascular dysfunction (2). Thus,

subsequent endothelial dysfunction could be due to cardiovascular dysfunction at that time point.

Dehydration after endurance exercise could lead to decreased MAP and affect cutaneous blood flow. However, concurrent measurements of total body water showed that water volume was preserved after endurance exercise in our study, as reported elsewhere (2).

Response 24 hours after exhaustive endurance exercise

Postexercise MAP was decreased 24 hours after cessation of endurance training compared with baseline, although not to a statistically significant extent (Fig. 2). These results are consistent with the findings of other authors who report that post-exercise hypotension can persist for up to 24 hours (23,30,31).

The main finding of our study was that both LDF and CVC increased significantly at SLAE 24 hours after endurance training compared to baseline (Fig. 2). Since increased skin blood flow accelerates the wound healing process (32), these results support the use of endurance training in wound healing. This finding of our study can be applied in practice by encouraging patients with chronic wounds to include occasional endurance exercise in their regular moderate training schedule to increase skin blood flow and accelerate wound healing. We can only suggest a few possible mechanisms responsible for the improvement in cutaneous blood flow 24 hours after EEE: increased sympathetic cholinergic vasodilation as the main mechanism to eliminate excessive heat, increased NO bioavailability due to suppressed oxidative stress 24 hours after EEE (29), the involvement of adenosine receptors that have been shown to prevent the decrease in skin blood flow after exercise (18), or a complex interplay of many physiological processes leading to cardiovascular supercompensation, which probably includes improved skin blood flow observed by many authors after endurance training (2,33,34). Further studies need to be conducted to substantiate these assumptions.

No significant changes in measured parameters were observed on day 2 and day 7, suggesting that the effect of EEE on cutaneous blood flow diminishes after two days.

Bivariate correlations

We found a small negative correlation between postexercise LDF and HRR30 and HRR60 when data were pooled across the entire measurement protocol (Fig. 5). A correlation of this magnitude requires careful interpretation. However, this correlation suggests that improved cutaneous blood flow delays recovery of HR after SLAEs regardless of endurance exercise. These results are consistent with the conclusions of Pecanha et al. (4), who found that heat stress delays heart rate recovery and highlighted the role of thermoregulation in post-exercise regulation of the HR. They suggested that thermally induced redirection of blood flow to cutaneous vessels, as evidenced by increased LDF, may involve reduced central blood volume and ventricular filling during recovery, resulting in compensatory sympathetic activation and parasympathetic deactivation after exercise (4).

Limitations of our study and perspectives

Our cohort consists of recreational runners, so results are limited to physically active, healthy individuals. Further studies are needed to prove that endurance exercise accelerates wound healing in patients prone to developing chronic wounds, such as those with type 2 diabetes.

Gender and age differences of cutaneous blood flow and postexercise hypotension after endurance exercise described by Stapelton (20) and Mourot et al. (25) were not examined in our study. The analysis of our data should be repeated separately for men and women to identify possible gender differences that could influence our conclusions.

Measurement of core temperature during the study protocol would be helpful to determine the magnitude of heat stress induced by endurance exercise compared with short-lasting exercise.

The proposed mechanisms underlying our results could be better determined if different substances such as bretylium tosylate, an inhibitor of noradrenergic vasoconstriction, and a NO synthase inhibitor (L- NAME) were applied to the skin during the measurement protocol.

Furthermore, spectral analysis based on the fast Fourier or wavelet transform of the periodic oscillations of the LDF after exercise could be used to analyze the periodic oscillations of different frequencies representing the influence of heartbeat, respiration, intrinsic myogenic activity, and neurogenic factors, respectively, on cutaneous blood flow after exercise (3,7). Additional analysis of LDF should be performed according to spectral analysis guidelines.

Conclusion

We measured the response of cutaneous blood flow in the forearm during recovery after short lasting aerobic exercise at different time points after exhaustive endurance exercise and compared it with concomitant changes in cardiac autonomic activity in recreational runners. We aimed to contribute to the understanding of the control of cutaneous blood flow in relation to training history. We found that training history has a strong influence on cutaneous and systemic hemodynamics as well as on cardiac autonomic response during recovery to SLAE. Our most important finding was that exhaustive endurance exercise as a part of the training history improved cutaneous perfusion 24 hours after completion of EEE, which may be important for wound healing. The results of our study are potentially applicable for patients with chronic wounds, who should be encouraged to exercise moderately on daily basis and to occasionally include endurance exercises, as this strategy potentiates postexercise cutaneous perfusion.

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Disclosure

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Competing interests. None to declare.

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