Postexercise Hypotension in Moderately Trained Athletes after Maximal Exercise

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ABSTRACT


Purpose: A similar postexercise hypotension (PEH) has been reported in sedentary and mildly endurance-trained individuals of both sexes after a single dynamic submaximal exercise. In endurance-trained men, the hypotension was associated with a reduction of cardiac output, whereas the peripheral vasodilation was the main mechanism of this fall in other groups. The present study investigated the occurrence and mechanisms of PEH after a short maximal exercise in professional soccer players with greater endurance capacity than previously reported in PEH studies.

Methods: Arterial blood pressure, cardiac output (Q), heart rate (HR), and diffusing lung capacity for carbon monoxide (DLCO) before and 30 and 60 min after short maximal field exercise were studied in 20 professional soccer players.

Results: Diastolic blood pressure (DBP) and systolic blood pressure (SBP), Q, stroke volume, and DLCO decreased, whereas HR increased at both times after exercise. Decreases in DBP were greater in subjects with lesser VO2,,, (r = -0.73, P = 0.0001), whereas SBP was more decreased the higher it was at baseline (r = 0.51, P = 0.023). Total peripheral resistance (TPR) did not change significantly after exercise.

Conclusion: These findings indicate that, in moderately trained athletes, postexercise hypotension is associated primarily with reduced cardiac output because of reduced stroke volume, suggesting venous pooling. In addition, the occurrence of hypotension is more frequent in trained subjects with lower cardiopulmonary fitness level or higher resting SBP.

Key Words: CARDIAC OUTPUT, ARTERIAL BLOOD PRESSURE, MAXIMAL OXYGEN UPTAKE, OXYGEN SATURATION, HUMAN

A single bout of dynamic (6,7) or static (12) exercise can result in a postexercise hypotension (PEH) lasting up to 2 h. PEH has been reported in sedentary and endurance-trained men and women (6,7,19). It is frequently caused by a single bout of submaximal exercise at 60% of maximal oxygen uptake (VO2max) lasting between 40 and 60 min (6,7), although it can occur at exercise intensities ranging from 40 to 70% of VO2max (13). PEH is especially pronounced in hypertensive patients, where it can persist up to 17 h after exercise (15), suggesting that submaximal exercise can be considered as an antihypertensive nonpharmacological treatment (13). The hypotensive effect is predominantly associated with a reduction in TPR that is not counterbalanced with increases in cardiac output (6,7,9).

Senitko et al. (19) found that endurance training did not reduce PEH, either in prevalence or magnitude of response. In endurance-trained women, hypotension was the result of peripheral vasodilatation as had been shown previously in sedentary individuals, whereas in endurance-trained men, it was caused by reduced cardiac output (because of a fall in stroke volume) and unchanged TPR. The aerobic capacity in endurance-trained men in this report, however, was not large (~46 mL·kg⁻¹·min⁻¹) and was equivalent to the aerobic capacity of recreational subjects.

In most studies investigating PEH, submaximal exercise protocols were used, whereas the occurrence and the mechanism of PEH after maximal exercise in moderately endurance-trained athletes are less known. Maximal exercise to exhaustion causes physiological and psychological changes such as metabolic acidosis, hyperventilation, and severe discomfort, not encountered during submaximal exercise. The mechanism of PEH after maximal exercise has been investigated only in sedentary, normotensive individuals (11,17).

The present study investigated the occurrence and mechanisms of PEH in moderately trained athletes after short maximal exercise.

METHODS

Subjects

A total of 20 professional soccer players of the Croatian national champion team, Hajduk Split, were included in the study. Their mean age was 22.0 ± 2.9 (mean ± SD) yr (range 19–31), height 184.4 ± 6.3 cm (range 174–194), and
measurements, which were done 30 and 60 min after the K4 unit and returned to the laboratory for postexercise measurements. The unit was first calibrated to a known concentration of CO, and lung ventilation were determined a with portable K4 breath-by-breath telemetric unit (Cosmed). The unit was first calibrated to a known concentration of O₂ and CO₂, a given volume of air, and to the atmospheric pressure. Respiratory gas concentrations, HR, and the ventilatory volumes were telemetrically relayed to a portable computer. HR and the respiratory data were provided on a report once every 30 s with the values averaged over the last 10 respiratory cycles. Criteria for assessment of VO₂max included a respiratory exchange ratio (RER) ≥1.1 and a plateau (≤150 mL increase) in VO₂, despite an increase in workload. The highest HR attained at maximal oxygen uptake represents maximal HR. Anaerobic threshold was determined from the increase in the ventilatory equivalent for O₂ (VE/VO₂), without a concomitant increase in the ventilatory equivalent for CO₂ (VE/VO₂). The mean relative VO₂max and the maximal HR (HRmax) at VO₂max was 56.3 ± 4.7 mL·kg⁻¹·min⁻¹ and 186.6 ± 6.1 bpm, respectively. The absolute VO₂max was 4633.5 ± 523.7 mL·min⁻¹. Duration of the field test was 11.4 ± 0.9 min, and the running speed at VO₂max was 19.2 ± 1 km·h⁻¹. The anaerobic threshold was at 79.7 ± 6.6% VO₂max.

Cardiac output. Cardiac output (Q) was estimated by the indirect Fick method of carbon dioxide (CO₂) rebreathing to equilibrium, as described previously (3). The equilibrium fraction of CO₂ was given by extrapolation, using the regression line between 8- and 12-s rebreathing intersects at 20 s after the start of the rebreathing maneuver (Cosmed B², Italy). Q was measured in duplicate and the rebreathing bag was thoroughly flushed between two rebreathing periods. Stroke volume (SV) was calculated as Q/HR and TPR as MAP/Q (mm Hg·min⁻¹·L⁻¹). Because a single-breath DLCO maneuver causes decreases in Q, SBP, and DBP (10), in this study Q was measured in the test series before DLCO. Q measurements with CO₂ rebreathing technique before exercise and during PEH have been used by others (2,5,6).

Diffusing lung capacity for carbon monoxide. Diffusing lung capacity for carbon monoxide (CO) (DLCO) was determined by the single-breath method. Concentrations of CO were measured using an infrared analyzer (Quark PFT, Cosmed, Italy). Alveolar volume was measured by single-breath methane dilution and DLCO/VA was calculated.

Statistical Analysis

Data are expressed as mean ± SD. Comparisons between preexercise and postexercise measurements were done with
TABLE 1. Baseline and postexercise hemodynamic data of 20 professional soccer players (mean ± SD).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>Minutes after Exercise</th>
<th>P Values*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>30</td>
<td>60</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>73.0 ± 6.7</td>
<td>70.4 ± 6.8</td>
<td>67.9 ± 6.8</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>129.8 ± 8.8</td>
<td>123.8 ± 10.4</td>
<td>122.7 ± 7.9</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>91.9 ± 6.5</td>
<td>88.9 ± 7.0</td>
<td>86.2 ± 5.8</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>56.9 ± 8.0</td>
<td>53.4 ± 6.8</td>
<td>54.3 ± 10.9</td>
</tr>
<tr>
<td>Heart rate (L.min⁻¹)</td>
<td>59.6 ± 5.9</td>
<td>77.9 ± 8.9</td>
<td>69.9 ± 11.5</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>121.2 ± 25.9</td>
<td>80.9 ± 22.4</td>
<td>95.7 ± 35.3</td>
</tr>
<tr>
<td>Cardiac output (L.min⁻¹)</td>
<td>7.16 ± 1.4</td>
<td>6.2 ± 1.6</td>
<td>6.1 ± 1.6</td>
</tr>
<tr>
<td>Total peripheral resistance (mm Hg.L⁻¹.min⁻¹)</td>
<td>13.2 ± 2.3</td>
<td>15.0 ± 3.4</td>
<td>15.2 ± 4.8</td>
</tr>
<tr>
<td>Diffusing lung capacity (mL.min⁻¹.mm Hg⁻¹)</td>
<td>37.6 ± 8.0</td>
<td>35.0 ± 6.5</td>
<td>34.5 ± 4.9</td>
</tr>
</tbody>
</table>

* P values are for comparisons vs baseline at specific times after exercise (Wilcoxon matched pairs test, following significant result of Friedman ANOVA).

非参数的Friedman和Wilcoxon秩和检验。变量之间的相关性通过Pearson的系数来评价。P < 0.05被认为是显著的。

RESULTS

Baseline vital signs were normal in all subjects, with HR averaging 59.6 ± 5.9 bpm and with SBP and DBP pressure averaging 129.8 ± 8.8 and 73.0 ± 6.7 mm Hg, respectively (Table 1). All 20 subjects successfully completed the study protocol. No subject reported any symptom (e.g., fainting or lightheadedness) after exercise.

Significant lowering of SBP, DBP and MAP were found 30 and 60 min after exercise (Table 1). Interestingly, the decrease in BP was noticed in all of the goalkeepers, but was not present in forward players. The HR increased at 30 min and remained increased at 60 min after exercise, whereas SV, CO, and D_LCO decreased. TPR did not change significantly after exercise. Nonsignificant increases observed, mean that a decrease (but not an increase) in TPR after exercise can be safely excluded (Table 1).

At 60 min postexercise, the lesser the \( VO_2_{max} \), the greater decrease in DBP (\( R = -0.73, P = 0.0001 \), Fig. 1), but not in SBP (\( R = -0.03, P = 0.91 \)) was observed. No correlations were observed at 30 min postexercise. Also, at 60 min postexercise, the greater the SBP, the greater decrease in SBP was observed (\( R = 0.51, P = 0.023 \), Fig. 2), which was not the case for DBP (\( R = 0.24, P = 0.31 \)).

DISCUSSION

In the present study, we investigated the occurrence and cause of postexercise hypotension in moderately trained endurance athletes after short-term maximal exercise. The main findings are (a) there may be less PEH in the subjects with higher \( VO_2_{max} \) values, (b) PEH can be seen after brief but maximal exercise, (c) PEH may occur more frequently in subjects with higher baseline SBP, and (d) in more trained subjects, the mechanisms causing PEH probably differ from the mechanisms in untrained people.

Central and peripheral hemodynamics after maximal exercise. Postexercise hypotension after a single bout of dynamic exercise in sedentary men and women and endurance-trained women is the result of a reduction in TPR (6,7,9). Our results indicate that PEH in endurance-trained soccer players is related to reduced cardiac output caused by stroke volume reduction, because the TPR was unchanged. Similar findings were reported previously by Hagberg et al. (5) in older hypertensive

FIGURE 1—Scatterplot of maximal oxygen uptake (\( VO_2_{max} \)) and decreases in diastolic blood pressure (DBP) at 60 min after maximal exercise in 20 professional soccer players. The best-fit line and linear regression statistics are also presented. Note that the lesser \( VO_2_{max} \), the greater was the decrease in DBP.

FIGURE 2—Scatterplot of baseline systolic blood pressure (SBP) and its decreases at 60 min after maximal exercise in 20 professional soccer players. The best fit line and linear regression statistics are also presented. Note that the greater SBP, the greater was the decrease in SBP.
persons and by Senitko et al. (19) in endurance-trained men after submaximal exercise. Hagberg et al. (5) suggested that reduction in cardiac output was related to reduced myocardial contractility, whereas Senitko et al. (19) explained this effect by reduction in central venous pressure (CVP) and, thus, cardiac preload.

Possible factors affecting stroke volume are preload, afterload, and myocardial contractility. Afterload was likely to be lower after exercise in this study because of lower SBP. A single index of increased efferent sympathetic nervous activity after exercise was persistent tachycardia up to 60 min Raine et al. (17) reported increased ejection fraction from 61% at baseline to 71% after maximal exercise, indicating increased inotropy postexercise. The likely mechanism for reduction of stroke volume after exercise in the present study was a decline in cardiac filling, given that afterload was reduced and that myocardial contractility was probably increased. Cardiac preload is dependent on changes in plasma volume and venous capacitance. Plasma volume change was not measured in this study. It is unlikely that any significant changes in plasma volume occurred in this study, given the short exercise time (9–13 min), cooler environment, and unrestricted pretrial water intake. The main factor reducing cardiac filling in this study is likely the hydrostatic gradient secondary to the upright seated posture as well as pooling of blood in the previously active muscle. Other potential mechanisms underlying the reduced venous return after exercise is the redistribution of cardiac output from less compliant to more compliant vascular beds such as splanchnic organs and skin (18). Halilwill et al. (8) found that whole-limb venous compliance is under negligible active sympathetic control in humans, suggesting the possibility of venous pooling in the leg vasculature in addition to splanchnic and skin beds. Similar vasodilatation in the visceral organs or lower limbs leading to pooling of blood and a decrease in venous return after submaximal exercise was proposed by Brown et al. (2). Renal and splanchnic (16) vascular beds, however, are not involved in postexercise vasodilatation.

Diffusing lung capacity for CO was reduced in this study for about 10% as was previously reported by others after exercise of different duration, intensity, and sport (14). Finding of reduced DLCO in this study appears to support the theory of peripheral pooling and reduced central blood volume after maximal exercise.

It appears that longer bouts of exercise (1 h vs 30 min) produce more PEH (6,7). The effects of exercise intensity and duration on PEH remain unresolved. Probably more sustained PEH occurs after moderate intensity, longer lasting exercise (7), but short, maximal exercise might produce a greater, but less sustained, fall in BP. This study adds to the existing knowledge of the occurrence of PEH by reporting that most of trained soccer players experienced PEH up to 1 h after the maximal field exercise. The average drop in MAP in the supine position was 4–5 mm Hg, but often ranged from 1 to 14 across individuals. The subjects with the greater decrease in SBP had its higher baseline value, which is in accord with findings in hypertensive patients (15). This finding indicates that brief maximal exercise causes a similar fall in BP as longer submaximal protocols, which may prove important in considering hypertension treatment. Future studies should address the sustainability of this effect.

Our subjects had mean VO_{2max} of 56 mL·kg^{-1}·min^{-1}, which places them in the lower range of elite soccer players (10). This was partly because three goalies had relative VO_{2max} of approximately 51 mL·kg^{-1}·min^{-1}. We are lacking data to differentiate between the lack of training (more probably) and limited capacity for higher VO_{2max}. We therefore suggest that the occurrence of PEH in trained subjects is dependent on the cardiopulmonary fitness and is more frequent in the subjects with lower fitness status.

**Study limitation.** The endurance-trained subjects in this study were only moderately trained. Future study therefore needs to evaluate postexercise hypotension in highly trained athletes (e.g., cyclists, rowers, or runners).

**CONCLUSIONS**

This study indicates that in more trained men, the mechanisms causing PEH probably differ from the mechanisms in those who are untrained. We suggest that PEH in trained men is associated with reduced cardiac output, because of reduced cardiac filling. The main factor reducing cardiac filling in this study is likely the hydrostatic gradient secondary to the upright posture as well as the pooling of blood in the previously active muscle. The lower fitness level or higher baseline SBP is associated with higher occurrence of postexercise hypotension.

The authors wish to express their thanks to Jolanda Zloković for her technical assistance. This study was financially supported by the Croatian National Council for Research grant no. 0216006.

**REFERENCES**


