Haemodynamics of recovery after strenuous exercise in physically trained hypertensive and normotensive subjects

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1. Central and peripheral post-exercise haemodynamics were studied in 18 physically trained male subjects (10 hypertensive and eight normotensive) engaging in sports activities for 3–5 h/week. After a preliminary multistage bicycle ergometric test to evaluate their maximal oxygen consumption and anaerobic threshold, they underwent prolonged exercise at anaerobic threshold in the semi-supine position at 30° grade until exhaustion (mean duration 60.0 ± 16.7 min in the hypertensive subjects and 61.0 ± 5.7 min in the hypertensive subjects, not significant). During the recovery time, intra-arterial blood pressure, echocardiographic cardiac output and indium-gallium strain-gauge plethysmographic peripheral flow were measured, and total, forearm and leg peripheral resistances were calculated respectively from mean blood pressure/cardiac output and mean blood pressure/peripheral resistance.

2. Systolic blood pressure was decreased during the entire recovery period in comparison with the baseline values (~8.4 mmHg, ~43.8 mmHg and ~39.7 mmHg at the 1st, 5th and 10th min in the hypertensive subjects, \( P = 0.001 \), \( P = 0.0001 \) and \( P = 0.0001 \) respectively; ~18.8, ~25.5 and ~24.1 mmHg in the normotensive subjects, not significant, \( P = 0.01 \) and \( P = 0.01 \), respectively) without any significant difference between the two groups, whereas the reduction in diastolic blood pressure was not statistically significant. Peripheral flow increased and peripheral resistance decreased in parallel in the forearm and the leg and showed similar trends in the hypertensive subjects and the normotensive subjects. The increase in cardiac output and left ventricular ejection fraction and the decrease in total resistance were also similar in the two groups.

3. In conclusion, after long-lasting strenuous leg exercise central and peripheral haemodynamics vary to the same degree in hypertensive and normotensive trained subjects. Peripheral haemodynamics (similar in the leg and the arm in both groups) closely reflect systemic haemodynamics. The blood pressure reduction observed in the recovery period is totally due to the fall in systemic vascular resistance, while cardiac output is increased.

INTRODUCTION

The hypotensive effect of physical exercise has been well documented in hypertensive subjects [1–3]. Isotonic exercise, not only regularly repeated (endurance training) but also acutely performed, is followed by a reduction in blood pressure (BP). According to some investigators, both systolic (SBP) and diastolic (DBP) BP decline after exercise [4–6], whereas according to others there is a drop in SBP but not in DBP [7–9]. Conflicting results have been obtained in normotensive subjects, as in the experience of some authors exercise is followed by a decrease in SBP [3–6], whereas according to others there is a short-lived increase in SBP [10] or no change [4, 5, 11]. Much less is known about the post-exercise haemodynamic response of trained hypertensive subjects, primarily due to the low prevalence of arterial hypertension among athletes (approximately 0.2%) [12]. No studies are available on the comparison of exercise haemodynamics between hypertensive and normotensive athletes, despite a mounting interest for young athletes in the last few years. Moreover, some doubts still exist as to the physiological mechanisms of the post-exercise BP drop, which has been attributed to improved or, on the contrary, to impaired function of the cardio-pulmonary baroreflex [5, 7, 13], to a decrease in sympathetic discharge [4, 8], to a reduction in cardiac output [7, 8, 14] and/or to a drop in peripheral resistance (PR) [4, 5, 13].

Another question is whether or not the blood flow has a different pattern in the leg and the arm after leg exercise [12, 15, 16].

This study was performed in order (1) to define whether or not hypertensive and normotensive trained young subjects have the same BP pattern after prolonged isotonic exercise, (2) to determine

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Abbreviations: BP, blood pressure; CO, cardiac output; DBP, diastolic blood pressure; EF, ejection fraction; HR, heart rate; LVEDV, end-diastolic left ventricular volume; LVESV, end-systolic left ventricular volume; MBP, mean blood pressure; PF, peripheral flow; PR, peripheral resistance; SBP, systolic blood pressure; SV, stroke volume; TR, total resistance; \( V_2 \) max., maximal \( O_2 \) consumption.
which haemodynamic components are mostly involved in determining the post-exercise BP fall, and (3) to clarify whether or not the peripheral flow (PF) and PR vary to a different degree in the leg and the arm in the recovery period after leg exercise.

METHODS

Study population

The study population consisted of 10 male hypertensive subjects, aged 26.1 ± 5.9 years (mean ± SD), referred to our department from the local Centres of Sports Medicine because of the detection of a SBP > 140 mmHg and/or a DBP > 90 mmHg. The hypertensive subjects engaged in sports activities for 3–5 h week, and had never taken any anti-hypertensive medication. Their average office BP at entry (mean of at least three measurements in the lying position at 1 week intervals) was 167.9 ± 22.8/91.2 ± 14.1 mmHg.

Eight male normotensive healthy subjects (controls) aged 24.2 ± 5.9 years with similar training habits were also studied. Their office BP at entry was 138.6 ± 13.0/83.3 ± 18.8 mmHg and had always been lower than 140/90 mmHg at previous measurements.

All subjects were normal by physical examination and standard resting and exercise electrocardiogram. The study protocol was approved by the Ethics Committee of our Department, and all subjects gave their informed consent.

Study protocol

In a preliminary phase, all subjects underwent a proof bicycle ergometric test to exhaustion in a 30% grade semi-supine position with a workload increasing by 20 W at 2 min intervals, in order to evaluate maximal oxygen consumption [\( \text{VO}_2 \text{max.} \), defined as the attainment of a plateau of \( \text{O}_2 \) uptake with increasing work rates (levelling-off criterion)] and anaerobic threshold (Wasserman-McIlroy method) [17]. An average workload of 265.0 ± 41.2 W was reached in the hypertensive subjects and of 256.2 ± 41.7 W in the normotensive subjects (not significant).

Three days or more after this preliminary study, prolonged exercise at the anaerobic threshold to exhaustion was performed in the semi-supine position on the same ergometer used during the preliminary test. During the first 9 min of exercise the workload was progressively increased until the target heart rate (HR) was reached. Then, the subjects exercised until they were unable to continue. The average workload at the anaerobic threshold was 165.5 ± 21.4 W in the hypertensive subjects and 155.0 ± 20.5 W in the normotensive subjects (not significant). The hypertensive subjects reached an average HR of 164.5 ± 16.3 beats/min at the end of the exercise and the normotensive subjects an average HR of 159.7 ± 13.0 beats/min (not significant).

After exercise, subjects stayed on the ergometric bed and BP, HR, PF and cardiac output (CO) were measured with the methods described below, and PR and total resistance (TR) were calculated at the 1st, 5th and 10th min of recovery.

BP monitoring

According to the procedure described elsewhere [18], a Medicut Sherwood catheter was placed in the left radial artery and connected to a Statham P23DB transducer for continuous BP recording. BP signals were recorded on a cassette by means of an Oxford Medilog recorder.

Immediately before each echocardiographic evaluation (see below), an event marker was recorded on the tape and both SBP and DBP in the following 20 s periods were measured and averaged. BP was also simultaneously fed to an ultraviolet printer for direct inspection of the tracing. To make sure that no shift in the zero level occurred, transducer calibrations were performed every 10–15 min throughout the recording period.

Mean BP (MBP) was calculated from the whole sphygmographic profile [18].

PF and PR measurement

An automatic strain gauge plethysmographic fluximeter, which provided the complete automation of occlusion/deflation times and pressures (Angiomed Microlab, Padova, Italy), was used for measuring PF [19, 20]. Once the occlusion pressure of 50 mmHg was programmed, it was repeated at each PF determination without any further operator's intervention. Automatic cuff inflation was periodically obtained through a reservoir. Air to the cuff was delivered through a large-calibre tube in 0.3 s. An electronic calibration equal to a 1% increase of the strain gauge electric resistance was automatically provided before occlusion. At each plethysmographic assessment, four automatic measurements of PF were performed and averaged. PR was calculated at each step as the MBP/PF ratio, and expressed in mmHg min dl ml⁻¹ (units of resistance).

Echocardiogram

After a 30 min rest in a slightly left lateral position with a support under the shoulders, all subjects underwent echocardiogram before exercise and at the 1st, 5th and 10th min of recovery with a SPR 8000 (Hitachi EUP 121, Tokyo, Japan). M-mode echocardiograms were performed under two-dimensional guidance always by the same investigator, following the suggestions of the American Society of Echocardiography [21]. Only good quality images were taken into account. Left ventricular images were obtained at end-expiration with a
cardiac cycles were analysed and averaged at each measurement. Left ventricular end-diastolic and end-systolic diameters were taken into consideration. End-diastolic and end-systolic left ventricular volumes (LVEDV and LVESV, ml) were calculated from the equation:

\[
\text{Volume} = 7 \times \text{diameter} + \frac{7}{2.4} \times \text{diameter}
\]

according to Teicholz et al. [22].

Left ventricular stroke volume (SV, ml) was calculated as LVEDV – LVESV, CO (l/min) as the product of SV and HR, ejection fraction (EF, %) as the percentage ratio between SV and LVEDV, and TR (mmHg min\(^{-1}\)) as the ratio between MBP and CO.

Validation of the procedures

The estensimetric method for measurement of segmental PF has been widely validated [23]. In the Angiomed device, a software procedure also eliminates any abnormal measurement on the basis of a correlation coefficient indicating the degree of dispersion of the measured points around the above-described interpolating curve, i.e. the accuracy of adaptation of the regression curve to the measured points [19, 20]. If this coefficient tends to 1, the value is accepted, otherwise it is rejected.

The Oxford direct BP measurement procedure of Bevan et al. [24] does not need any further validation.

Statistical analysis

First phase. In the first phase of this study, the results were analysed separately for normotensive and hypertensive subjects. Data were expressed as means ± SD. Baseline values were compared with the Bartlett test. Analysis of variance for repeated measures was used in order to compare values at the different steps (before exercise and at the 1st, 5th and 10th min of recovery); between-subjects effects (hypertensive subjects versus normotensive subjects) and within-subjects effects (versus time) were taken into consideration. Significant differences (P < 0.05) were identified by the Tukey post-hoc test. Backward stepwise logistic regression (Wald method) was also performed using hypertension as the dependent variable and PF, PR, CO and TR as independent variables.

Second phase. As hypertensive and normotensive subjects did not appear to differ in any haemodynamic trend (see below), in the second phase they were analysed together. First-degree univariate and Wilk’s lambda multivariate profile analysis according to Morrison [25] and Bock [26] were used in order to evaluate the trends in PF, CO, PR and TR during the recovery time.

RESULTS

First phase: hypertensive versus normotensive subjects

Both baseline intra-arterial SBP and DBP were significantly higher in the hypertensive subjects than in the normotensive subjects (Table 1). CO, TR and HR were similar in the two groups, whereas EF was higher in the hypertensive subjects.

The exercise lasted 61.0 ± 5.7 min (range 50–70 min) in the hypertensive subjects and 60.0 ± 10.7 min (range 50–80 min) in the normotensive control subjects (not significant).

In the hypertensive subjects, HR was 74.0 ± 11.9 beats/min at rest, 133.1 ± 20.4 beats/min at the 1st min (P = 0.0001 versus rest), 109.1 ± 17.3 beats/min at the 5th min (P = 0.001) and 101.7 ± 15.8 beats/min at the 10th min (P = 0.008). In the normotensive subjects it was respectively 70.6 ± 12.8 beats/min, 123.4 ± 16.2 beats/min (P = 0.0001), 104.4 ± 12.8 beats/min (P = 0.0001) and 98.0 ± 11.6 beats/min (P = 0.002). Analysis of variance for HR was as follows: F = 18.055, P = 0.0001, without any between-group statistical difference.

During the recovery period, SBP was constantly lower than before exercise in the hypertensive subjects, and at the 5th and 10th min in the normotensive subjects; the reduction in SBP from the baseline values was 8.4 mmHg (P = 0.001) at the 1st min of recovery, 43.8 mmHg (P = 0.0001) at the 5th min and 39.7 mmHg (P = 0.0001) at the 10th min in the hypertensive subjects, and 18.8 mmHg (not significant), 25.5 mmHg (P = 0.01) and 24.1 mmHg (P = 0.01), respectively in the normotensive subjects, without any between-group statistical difference.

DBP did not show any significant post-exercise change in either the hypertensive subjects or the normotensive subjects (Fig. 1).

Baseline PF was lower and PR insignificantly higher in the leg than in the arm in both the hypertensive subjects and normotensive subjects (Table 1), and remained different during the recovery (between-subjects analysis of variance for repeated measures: F = 7.540, P = 0.011 for PF; F = 8.987, P = 0.006 for PR). Nevertheless, their trend versus time was similar in the two limbs (Fig. 2) as no interaction between group and time was present (F = 2.028, P = 0.139 for PF; F = 0.110, P = 0.954 for PR).

Baseline PF, PR, CO and TR were similar in the hypertensive subjects and the normotensive subjects (Table 1). Logistic regression analysis showed that PF, PR, CO and TR, both before exercise and during recovery were not discriminant variables for hypertension (Table 2). Furthermore, between subject analysis of variance for repeated measures (Table 3) did not show any significant difference between the two groups during recovery, the increases in PF and CO and the decreases in PR and TR being similar in the normotensive subjects and the hypertensive subjects (Fig. 3). Significant changes in PF, CO, PR and TR according to analysis of variance for repeated measures were
found during the recovery in both limbs in each group of subjects (Fig. 3), a finding confirmed by multivariate profile analysis (Table 2).

Second phase: all subjects
Taking into account all subjects irrespective of the diagnosis of hypertension, SBP was constantly lower during recovery than before exercise, whereas DBP was not different (Fig. 4). Significant changes compared with pre-exercise values were observed during the recovery period also for PF, CO, PR, TR, HR and EF.

Repeated measures profile analysis confirmed the
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Table 3. Repeated measures analysis. Between-subject and within-subject analysis for repeated measures in 10 hypertensive and eight normotensive trained subjects.

<table>
<thead>
<tr>
<th></th>
<th>Between-subject analysis</th>
<th>Univariate F-test</th>
<th>Wilk's lambda</th>
<th>Multivariate test</th>
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<tr>
<td></td>
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<td>P</td>
<td>F</td>
<td>P</td>
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![Fig. 1. SBP and DBP at baseline and at the 1st, 5th and 10th min of recovery in the 10 hypertensive (■) and eight normotensive (□) physically trained subjects.](image)

![Fig. 2. PF and PR in the forearm (∙) and in the leg (□) in the 10 hypertensive (right) and eight normotensive (left) physically trained subjects.](image)

DISCUSSION

There is still debate as to whether the reduction in BP that occurs after aerobic exercise is due to a drop in cardiac contractility or vascular resistance, or both. Our study clearly shows that a marked reduction in TR and PR occurred after prolonged exercise in both trained hypertensive and normotensive subjects, which accounted for the drop in BP. On the contrary, CO and left ventricular performance were increased during the recovery time. In agreement with the results of Coats et al. [10], in the present report the main determinant of the high CO during the recovery time was the rise in HR, as the increase in SV was minor.
We also wanted to assess whether hypertensive and normotensive trained subjects have a different haemodynamic pattern after exercise, as experience in this field is still very limited. Kaufman et al. [6] found the same BP drop during the recovery period in eight untrained normotensive and eight hypertensive subjects who underwent a Wilcox exercise protocol [3], but they did not study either central or peripheral haemodynamics. In our study, although SBP decreased to a lesser extent and more gradually in the normotensive than in the hypertensive subjects (Fig. 1) during the recovery period, CO, PF, TR and PR showed a similar trend in the two groups. This is attested by the almost absolute superimposition of the plots of PF and CO versus time in both the normotensive subjects and the hypertensive subjects, without any statistical difference as evaluated by the between-subject analysis. The same applies to PR and TR (Fig. 3). Furthermore, not one of the considered central or peripheral haemodynamic parameters (CO, TR, PF, PR) entered in the logistic regression equation where hypertension was the dependent variable. Thus, no difference was found in post-exercise haemodynamics between the normotensive and the hypertensive trained subjects.

The 140 mmHg intra-arterial systolic BP found in the normotensive subjects is surprising. However, it is likely to reflect the psychological reaction to the hospital environment and to the invasive procedure of the BP measurement. The same reaction was exhibited by the hypertensive subjects whose intra-arterial systolic BP before exercise was higher than the indirect BP measured at entry. On the other hand, the intra-arterial systolic BP of the two groups differed to a high level of statistical significance.

A possible explanation for the similar post-exercise haemodynamic pattern found in hypertensive and normotensive subjects is that the chronic increase in the capillary bed, typical of endurance-trained subjects [27, 28], is capable of preventing or counter-balancing the high-resistance haemodynamic pattern which seems to be characteristic of sedentary hypertensive subjects [14, 29]. This could explain the discordance between our data and those of Hofman et al. [13], who found a 9% lower resting CO and a 15% higher resting TR in 41 untrained hypertensive teenagers compared with 41 age-matched normotensive untrained control subjects (attributed to sympathetic over-activity, as documented by the high plasma catecholamine levels), or those of Takeshita and Mark [30], who found an augmented resting PR and a reduced forearm vasodilator capacity (attributed to structural changes in forearm resistance vessels) in young sedentary men with borderline hypertension.

In nine untrained elderly hypertensive subjects aged 60–69 years, Hagberg et al. [7] observed a reduction in CO as well as in SV from the 20th to the 150th min of recovery after 15 min ergometric bicycle exercise at 50 or 70%. \( \dot{V}O_2 \text{ max} \). The discrepancy between our results and those obtained by the above-mentioned authors could be partially explained by the different ages of the two study groups or the different physical performance (greater in our subjects), or both. In fact, in another study, Hagberg et al. [31] demonstrated that older sportsmen had a higher PR and a lower CO than younger ones when compared at the same relative exercise
intensities, and a markedly lower PR than their sedentary peers.

Another possible explanation for the discrepancies between our results and those of Hagberg et al. [7] is the much longer period of exercise in our study: the higher body temperature consequently developed by our subjects during the mid-late phases of the effort could have entailed a higher degree of cutaneous vasodilatation, a greater increase in skin blood flow [32] and consequently a greater drop in PR. The rapid decrease in plasma noradrenaline concentration [33] and the sympathetic withdrawal [34] which follow strenuous exercise could also account for the post-exercise fall in PR found in the present study. Also, the different timing of the haemodynamic measurements in the two studies could explain the observed differences.

Another interesting finding of the present study is that the trend in PF and PR during the recovery after exercise was similar in the arm and the leg in both the normotensive and the hypertensive subjects (Fig. 2). This is likely to be due to the skin vasodilatation for heat dissipation occurring in both limbs.

This topic is still controversial and information is sparse. Although it is universally accepted that during exercise PF increases and PR decreases in the working muscles, some incertitude does exist about the non-working muscles. Christensen and co-workers [35, 36] and Bishop et al. [37], studying untrained subjects performing a bout of leg exercise, described a slight rise in flow in resting arms in the late phase of the effort and during the recovery period. Lowenthal et al. [38] found that forearm flow was unchanged in the recovery period after leg exercise. Bevergård and Shepherd [14] even found a marked fall in forearm flow during leg exercise, which was attributed to vasoconstriction of the resistance vessels in the resting forearm to avoid any diversion of CO to the inactive muscles. Taylor et al. [39], who recently studied haemodynamics of one resting leg and one resting forearm during a short-lasting one-leg exercise, showed skeletal muscle vasodilatation in the forearm but not in the resting calf.

The discrepancies between the different results obtained by the various authors who have studied this problem is probably attributable to the different length and intensity of the exercise and the different protocols adopted. Our subjects performed prolonged strenuous exercise lasting about 1 h, whereas most of the above-mentioned studies made use of short-lasting exercise. The greater elevation in body temperature which accompanies prolonged exercise is likely to entail vasodilatation in both the muscles and skin of the entire body [27] and therefore a greater involvement of the resistance vessels. The results obtained by Bevergård and Shepherd [14], who obtained a block of the arteriolar vasomotor response by local heating of the forearm, are in keeping with this hypothesis. Also, the results of Blair et al. [16], who described a significant increase in forearm flow after intense leg exercise, but no increase in the recovery period after light exercise [16], are in agreement with our data.

Similar data were also obtained by Coats et al. [10], who found in normotensive subjects a marked vasodilatation in the non-exercising forearm after leg exercise, which was considered as an expression of systemic vasodilatation via sympathetic depression. However, it has to be pointed out that strenuous exercise is accompanied by a noticeable increase in circulating vasodilating substances, such as atrial [40] and brain [41] natriuretic factors and opioid peptides [42], which could favour a generalized muscle vasodilatation.

The quite absolute coincidence between leg and forearm PF and PR, and between peripheral and systemic flow (CO) and resistance (TR), that we found in both normotensive and hypertensive subjects confirms the validity of the strain-gauge plethysmographic method and justifies the extrapolation of limb flow and resistance to systemic haemodynamics, at least in the experimental conditions of the present study.

In conclusion, after long-lasting strenuous leg exercise test in trained subjects: (1) post-exercise PF, PR, CO and TR vary to the same degree in hypertensive and normotensive subjects; (2) post-exercise peripheral haemodynamics are similar in the leg and the arm in both groups, and closely reflect systemic haemodynamics; and (3) the reduction in BP observed in the recovery period is due to a reduction in systemic vascular resistance, while cardiac output and contractility are not decreased.

REFERENCES


