Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function

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Summary

1. Two experiments were carried out in male subjects. In the first experiment heart rate and blood pressures were measured before, during and for 90 min after a 50 min period of intermittent exercise in seven hypertensive subjects. After exercise there was a marked reduction in systolic and diastolic blood pressures; this effect lasted throughout the 90 min observation period and was unaccompanied by tachycardia.

2. In the second experiment heart rate and blood pressures were measured before and during the rest periods in a 50 min session of intermittent exercise in nine hypertensive and nine normotensive subjects. Following the first 10 min bout of exercise, resting blood pressures were significantly reduced in the hypertensive subjects; the reduction in blood pressure progressively increased following successive exercise periods. The normotensive subjects did not show a significant reduction in resting blood pressures until the fifth bout of exercise had been completed.

3. In the second experiment also, the competence of baroreflexes was assessed by measuring cardiovascular responses to lower body subatmospheric pressure (LBSP) 30 min before and 30 and 60 min after exercise. The post-exercise reduction in blood pressure was not due to exercise-induced impairment of baroreflex mechanisms since the reduced blood pressure after exercise was well maintained during lower body subatmospheric pressure. Furthermore, after exercise, exposure to lower body subatmospheric pressure elicited greater increases in heart rate and forearm vascular resistance than were seen before exercise.

Key words: baroreflexes, exercise, hypertension.

Introduction

It is generally agree[d] that during dynamic exercise there is an increase in systolic blood pressure and heart rate, while diastolic blood pressure changes little [1]. The absence of a reflex bradycardia in response to the rise in systolic or mean arterial pressure is attributable partly to a reduction in baroreflex sensitivity [2]. There is less agreement about the changes in blood pressure following exercise; some investigators have observed a slow return of blood pressure to the pre-exercise levels [3], while others have indicated that blood pressure may fall below pre-exercise values [4]. However, these various studies employed widely different types and durations of exercise, and there has been no systematic evaluation of the cardiovascular events following bouts of exercise of moderate intensity and duration. Furthermore, to our knowledge there have been no studies of baroreflex function in the post-exercise period.

Recently [5] we reported that, following intermittent treadmill exercise, a group of hypertensive men showed a significant reduction of systolic and diastolic blood pressures for the 30 min they were monitored after exercise. The present study was carried out to answer three questions: (1) does the post-exercise reduction in blood pressure persist for longer than 30 min?, (2) how much exercise needs to be undertaken to elicit the post-exercise reduction in blood pressure? and (3) is...
the post-exercise reduction in blood pressure accounted for by an exercise-induced incompetence of baroreflexes?

Methods

The study was carried out on weight-matched male hypertensive subjects and normal male volunteers. The patients all had secondary causes of hypertension excluded and were defined as having primary hypertension on the basis of casual clinic measurements showing diastolic blood pressure (phase V) above 95 mmHg while untreated over a period of 3 months. All subjects gave their fully informed, written consent to the study, which had been approved by the Hospital Ethical Committee.

Two experiments were carried out; in the first experiment, seven hypertensive men (mean age 46 years; range 31-62 years) were studied to determine the duration of the post-exercise reduction in blood pressure. Subjects came to the temperature-controlled laboratory at 08.15 hours after an overnight fast, or at 13.00 hours having had a light breakfast only. A cannula (Venflon) was inserted into an antecubital vein and kept patent with a slow infusion of isotonic saline. (The cannula was inserted to provide intravenous access in an emergency; there were no emergencies.) The subjects rested supine for 20 min, during which time brachial arterial blood pressure (by auscultation) and heart rate (from chest ECG electrodes) were measured twice (at 10 and 20 min). The subjects then stood on a treadmill (10% slope) for 5 min and blood pressure and heart rate were measured at the end of this time. The treadmill was then started and the speed adjusted to produce an exercising heart rate between 125 and 135 beats/min (this speed was between 1.8 and 2.2 m.p.h.); the exercise bout lasted 10 min. During the last minute of exercise, arterial blood pressure and heart rate were measured; the subject then sat on a chair on the treadmill for 3 min. The exercise was then repeated so that each subject performed five 10 min bouts of exercise. After the last exercise period, the subject sat in a chair for 90 min and blood pressure and heart rate were measured at 2.5, 5, 7.5, 15, 22.5, 30, 45, 60, 75 and 90 min.

In the second investigation, nine hypertensive men (mean age 44 years, range 29-59 years) and nine normal volunteers (mean age 47 years, range 41-57 years) were studied. The experimental protocol was as stated above, but, in addition, during the last 30 s of each inter-exercise period, resting blood pressures and heart rates were measured. Furthermore, cardiovascular responses to lower body subatmospheric pressure (LBSP) were also measured 30 min before exercise and 30 and 60 min after exercise; this procedure is a means of assessing baroreflex control of blood pressure [6]. Supine subjects were enclosed, to the level of their iliac crests, in an air-tight box from which air could be removed by an industrial vacuum cleaner through a connection that incorporated an adjustable air leak. The sub-atmospheric pressure in the box was monitored with a pressure transducer. Brachial arterial blood pressure was measured by auscultation, heart rate from the ECG, and forearm blood flow by venous occlusion plethysmography using a mercury-in-silastic strain gauge [7]. Forearm vascular resistance was derived by dividing mean arterial pressure \([\text{systolic} + 2(\text{diastolic})/3]\) by forearm blood flow. After a 15 min period of rest, subjects were exposed to 1 min periods of LBSP of 10, 20, 30, 40 and 50 mmHg separated by 1 min rests, during which time variables returned to baseline levels. Responses to LBSP were assessed from a comparison of the values of heart rate, blood pressure and forearm blood flow during the last 15 s of exposure to LBSP, and the values for the same variables measured during the 15 s preceding exposure to LBSP.

Data were analysed using Wilcoxon's signed ranks test.

Results

Experiment 1

Exercise in the hypertensive subjects caused a marked \((P<0.01)\) rise in systolic blood pressure and heart rate, with little change in diastolic blood pressure (Fig. 1). There was a prompt fall in blood pressures after the last bout of exercise, and throughout the 90 min post-exercise period systolic and diastolic blood pressures were significantly \((P<0.01)\) lower than their corresponding pre-exercise values (Fig. 1). Ninety minutes after exercise the mean reduction in sitting blood pressures was 40/15 mmHg (systolic/diastolic) and in standing blood pressures was 25/9 mmHg. At this time the heart rate was not significantly different from that before exercise (Fig. 1).

Experiment 2

Resting blood pressures in the hypertensive subjects showed a significant \((P<0.01)\) reduction after the first 10 min bout of exercise, and this effect was greater following each successive bout of exercise (Table 1). In the normotensive subjects, exercise did not have such a clear-cut effect on blood pressures (Table 1) until 5 min
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after the fifth bout of exercise had been completed (when blood pressure was 101 ± 2/72 ± 2 mmHg, \( P < 0.05 \)), but the hypotension then persisted for the 60 min post-exercise observation period.

The responses to LBSP 30 and 60 min after exercise were not significantly different, so only the results obtained 30 min after exercise are referred to here.

Responses to LBSP before and after exercise in normotensive subjects. Before exercise, exposure of supine, normotensive subjects to LBSP caused no significant change in diastolic blood pressure (Fig. 2); there was a small (\( P < 0.05 \)) reduction in systolic blood pressure with LBSP of 40 and 50 mmHg (Fig. 2), and a small increase in heart rate (\( P < 0.05 \)) during LBSP of 50 mmHg (Fig. 3).

![Arterial blood pressure and heart rate](image)

Fig. 1. Arterial blood pressure and heart rate (mean ± SEM) before, during and after five 10 min periods of treadmill exercise in seven male hypertensive subjects. There was a significant (\( P < 0.01 \)) increase in pressure and heart rate during exercise, but following the last exercise period systolic and diastolic blood pressures fell below (\( P < 0.01 \)) the pre-exercise resting value for the duration of the observation period; there was no persistent tachycardia accompanying the hypotension.

![Systolic and diastolic blood pressures](image)

Fig. 2. Systolic and diastolic blood pressures (mean ± SEM) in a group of supine normotensive subjects (\( n = 9 \)), resting and during LBSP; ○, 30 min before exercise; □, 30 min after exercise. After exercise there was a reduction (\( P < 0.01 \)) in systolic and diastolic blood pressures. During LBSP, systolic blood pressure showed a similar fall before and after exercise, but in the latter situation there was a significant (\( P < 0.02 \)) increase in diastolic blood pressure that was not seen before exercise.

<table>
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<tr>
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<th>Systolic/diastolic blood pressure (mmHg) before exercise and after one, two, three and four bouts of 10 min treadmill exercise in normotensive (( n = 9 )) and hypertensive (( n = 9 )) subjects</th>
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<td>Pre-exercise</td>
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<td>Hypertensive</td>
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* \( P < 0.05 \), **\( P < 0.01 \) for paired comparison (Wilcoxon's test) with pre-exercise control values.
Heart rate and forearm blood flow (mean ± SEM) in a group of supine normotensive subjects ($n = 9$), resting and during LBSP; ○, 30 min before exercise, △, 30 min after exercise. After exercise heart rate was similar to that before, but forearm blood flow was reduced ($P < 0.01$). During LBSP after exercise there was a more marked tachycardia and reduction in forearm blood flow than before exercise.

Forearm blood flow showed a significant ($P < 0.05$) reduction with LBSP of 10 mmHg, and with higher levels of LBSP the effect was more marked ($P < 0.01$; Fig. 3). These changes were due to similarly significant increases in forearm vascular resistance (see Fig. 6).

Following exercise, the supine resting values for systolic and diastolic blood pressures (Fig. 2) and forearm blood flow (Fig. 3) were significantly ($P < 0.01$) lower than before exercise, but there was no difference in heart rates. The reduced forearm blood flow was largely attributable to an increase ($P < 0.01$) in vascular resistance (see Fig. 6). However, in spite of the fact that systemic arterial blood pressure was lower in supine, resting subjects after exercise than it was during LBSP of 50 mmHg before exercise, forearm vascular resistance was significantly ($P < 0.01$) less in the former condition than in the latter (i.e. the reflex vasoconstriction was less although the stimulus was apparently greater). Exposure to LBSP caused an increase in diastolic and a fall in systolic blood pressure (Fig. 2); these changes were significant ($P < 0.02$) with LBSP of 20 mmHg or more. The increase in heart rate was significant ($P < 0.02$) with LBSP of 30 mmHg or more (Fig. 3). Forearm blood flow showed a significant reduction with LBSP of 10 mmHg ($P < 0.05$) or more ($P < 0.01$; Fig. 3) due to similarly significant increases in vascular resistance (see Fig. 6).

Responses to LBSP before and after exercise in hypertensive subjects. Before exercise, exposure of supine, hypertensive subjects to LBSP caused no significant change in diastolic blood pressure (Fig. 4) but systolic blood pressure fell significantly with LBSP of 20 mmHg ($P < 0.05$) or more ($P < 0.01$; Fig. 4). There was a significant ($P < 0.05$) increase in heart rate with LBSP of 50
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Lower body subatmospheric pressure (mmHg)

FIG. 5. Heart rate and forearm blood flow in a group of supine hypertensive subjects \( (n = 9) \), resting and during LBSP; ○, 30 min before exercise; ■, 30 min after exercise. Before and after exercise heart rates were similar, but forearm blood flow was reduced \( (P < 0.01) \) after exercise. During LBSP after exercise there was a more marked tachycardia and reduction in forearm blood flow.

mmHg only (Fig. 5). Forearm blood flow showed a significant \( (P < 0.01) \) reduction with all levels of LBSP, but with the higher levels of LBSP the reduction in blood flow plateaued (Fig. 5), due to a failure of forearm vascular resistance to increase further (Fig. 6).

Following exercise, there was a marked \( (P < 0.01) \) reduction in supine, resting systolic and diastolic blood pressures (Fig. 4) and forearm blood flow (Fig. 5) relative to pre-exercise values, but heart rates were similar (Fig. 5). Although the reduced forearm blood flow was mainly due to a significant \( (P < 0.01) \) elevation in vascular resistance (Fig. 6), the latter was not as high as would have been expected from the relation between forearm blood flow and mean arterial blood pressure during LBSP before exercise (Fig. 6).

Exposure to LBSP after exercise caused a reduction in systolic blood pressure that was significant \( (P < 0.01) \) with LBSP of 20 mmHg or more (Fig. 4) while diastolic blood pressure increased with all levels of LBSP above 20 mmHg \( (P < 0.01); \) Fig. 4). There was a tachycardia with LBSP of 30 mmHg and greater \( (P < 0.01) \). Forearm blood flow was reduced with all levels of LBSP \( (P < 0.01); \) Fig. 5) due to similarly significant increases in forearm vascular resistance (Fig. 6).
Discussion

The present study has shown that following a single 10 min bout of exercise in hypertensive men, blood pressures fell below their pre-exercise value. We have not studied the duration of the reduction in blood pressure after such a short period of exercise. However, the magnitude of the fall increased with repeated exercise bouts. The normotensive subjects were relatively resistant to exercise-induced hypotension, an observation that might be explained by their greater ability to cope with interventions that tend to cause a reduction in blood pressure.

The occurrence of the exercise-induced reduction in blood pressure in the hypertensive subjects after only 10 min of exercise argues against a fall in blood volume or a thermoregulatory vasodilatation being responsible for the hypotension at this stage. Furthermore, we have measured skin temperature throughout intermittent exercise in five hypertensive patients (T. Bennett, R. G. Wilcox & I. A. Macdonald, unpublished work) and there was no indication of marked cutaneous vasodilatation, although a degree of vasodilatation cannot be excluded. After 50 min of exercise it is feasible that there was a reduction in plasma volume [8] associated with vasodilatation in the skeletal muscles of the legs, possibly mediated by circulating metabolites [9]. While such changes may have accounted for the hypotension, this does not explain why the persistent fall in blood pressure did not elicit appropriate reflex increases in forearm vascular resistance or in heart rate. Thus, although the resting forearm vascular resistance post-exercise was higher than it was pre-exercise, it was not as high as would have been expected on the basis of the measured relation between mean systemic arterial pressure and blood flow during LBSP before exercise. Furthermore, 30 min after exercise, when there was a substantial reduction in blood pressure, there was no accompanying tachycardia. One possible explanation of these observations is that baroreflexes were rendered incompe.tent by exercise. However, the response to LBSP showed this was not the case. The reduced mean arterial pressure in hypertensive subjects was better maintained during LBSP following exercise than was their higher mean arterial pressure before exercise. In the normotensive subjects mean arterial pressure was maintained equally effectively during LBSP before and after exercise. However, in both groups of subjects, exposure to LBSP after exercise elicited more marked increases in diastolic blood pressure, forearm vascular resistance and heart rate than before exercise. These observations, together with those above, are consistent with a post-exercise resetting of baroreflexes to the lower pressure level accompanied by an increase in sensitivity of the system. But, it is possible that, following exercise, LBSP caused greater unloading of cardiopulmonary baroreceptors due to a greater fall in central venous pressure (consequent upon increased venous pooling). In line with this, we have observed that the bradycardia following the end of LBSP is greater after, than before, exercise (T. Bennett, R. G. Wilcox & I. A. Macdonald, unpublished work); this may have been due to the return of a larger volume of blood to the central circulation causing a bigger arterial pressure overshoot, and hence a more marked reflex slowing of the heart rate. However, a similar enhancement of the overshoot in arterial pressure following LBSP would be expected if the volume of blood moving back into the central circulation was unchanged, but the peripheral vasculature was more constricted (as we found it to be at each level of LBSP after exercise compared with the corresponding pre-exercise values). An observation that makes it unlikely that there was a greater volume of blood pooled during LBSP after exercise than before exercise is that the absolute fall in systolic blood pressure during LBSP was less after exercise than before exercise in the hypertensive subjects.

A full analysis of the phenomena we have described requires measurements of central venous pressure and cardiac output during LBSP before and after exercise, and more direct assessments of baroreflex sensitivity. Whatever the explanation of the persistent reduction in blood pressure post-exercise, there is no evidence that it was due to impairment of baroreflex function, and there is some evidence that it was accompanied by baroreflex resetting.

The full duration of the post-exercise reduction in blood pressure is not known, since our observations were only made for the 90 min following exercise. Furthermore, our subjects were kept in a temperature-controlled and neutral environment, so we cannot comment on the persistence of the post-exercise reduction in blood pressure under other circumstances.

As a codicil, it is interesting to note that the hypertensive subjects showed a greater proportional increase in forearm vascular resistance with LBSP of 10 mmHg than did the normotensive subjects. This phenomenon has been described recently [10] in patients with borderline hypertension, and has been attributed to an augmentation of the tonic inhibitory influence of cardiopulmonary baroreceptors on the forearm vasculature. In spite of this enhanced responsiveness to low levels of LBSP we observed that exposure to higher levels of LBSP caused a greater
fall in systolic and less of an increase in diastolic blood pressure in hypertensive, compared with normotensive, subjects. These findings corroborate and extend earlier observations on abnormalities in the circulatory responses to the upright posture in patients with benign essential hypertension [11].

References