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SYSTEMIC AND FOREARM VASCULAR RESISTANCE CHANGES AFTER UPRIGHT BICYCLE EXERCISE IN MAN

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SUMMARY

1. Blood pressure, cardiac function and forearm blood flow following voluntary maximal upright bicycle exercise were studied in thirteen normal volunteers in a cross-over design against a control day.

2. After exercise there was a short-lived (5–10 min) increase in systolic blood pressure, peak aortic blood velocity and aortic acceleration suggesting a persistence of the positive inotropic influence of exercise.

3. Systemic vasodilation, which was seen immediately exercise stopped, lasted at least 60 min. This was associated with a reduction in diastolic blood pressure for the whole hour. After 30 min systolic blood pressure was also reduced. Heart rate and cardiac output were still significantly elevated and systemic vascular resistance still reduced at 60 min post-exercise.

4. A non-exercising limb vascular bed (forearm) showed a marked vasodilation for 1 h after predominately leg exercise indicating the presence of a vasodilatory influence affecting vascular beds other than the exercising muscle groups.

INTRODUCTION

The systemic and regional haemodynamic responses to exercise have been extensively investigated (Rowell, 1974). Much less attention, however, has been directed to events occurring at the conclusion of exercise (Elsner & Carlson, 1962; Bennett, Wilcox & Macdonald, 1984; Somers, Conway, LeWinter & Sleight, 1985). This may be an extremely important period for there are rapid and large changes in the loading conditions of the heart which may precipitate hypotension or arrhythmias. Little is known of the time course of recovery of cardiac output and peripheral resistance after exercise, or the distribution of blood flow to regional vascular beds during this period (Blair, Glover, Roddie, 1961; Zelis, Mason & Braunwald, 1969).

A fall in blood pressure has been reported to follow short periods of exercise (Fitzgerald, 1981; Wilcox, Bennett, Brown & Macdonald, 1982; Somers *et al.* 1985), but these studies have not controlled for the possible increase in pressure which may have occurred in anticipation of exercise. In a preliminary communication we have shown that hypotension does occur after a symptom-limited bicycle ergometer test

in normal volunteers even when compared to a control non-exercising day in which the subject was expecting to exercise (Coats, Conway, Isea, Pannarale, Sleight & Somers, 1987). In the present study we investigate blood pressure and cardiac output following voluntary maximal upright bicycle ergometry, and also investigate regional blood flow to a non-exercising limb vascular bed.

METHODS

Subjects. Thirteen healthy volunteers (seven male, six female) aged 17–47 years agreed to participate in the study. All had normal electrocardiograms and no medical records of raised blood pressure, and were on no medication. All subjects were accustomed to the hospital environment, and were used to cycling, but none was a trained athlete. The procedures were approved by the institutional ethics committee. Subjects were told that they would perform two voluntary maximal bicycle exercise tests on separate days, 1–14 days apart. They were randomly allocated to exercise first, or control first, after the baseline observation period, so that there would be no systematic difference in the level of anticipation between the 2 days. Subjects were instructed to fast for 4 h prior to the test, and the two tests were performed at the same time of the day in a laboratory kept between 20 and 24 °C.

Exercise tests. All subjects completed a voluntary maximal bicycle exercise on a mechanically braked ergometer (Tunturi, Finland) with 5 min incremental stages of 25 W each starting at 50 W (the maximal load achieved ranged from 100 to 250 W). On the control day the subjects sat for approximately 30 min without cycling. Subjects were instructed to rest the right hand on the handle bar but not to grip to ensure that the right arm remained relaxed throughout the test.

Blood pressure. Blood pressure from the brachial artery of the left arm was recorded by an automatic microphonic sphygmomanometer (Copal UA 251, Takeda Medical, Japan), the accuracy of which has been assessed against random zero sphygmomanometry (Conway, Johnston, Coats, Somers & Sleight, 1988). On each of the 2 days, after 15 min rest, blood pressure was recorded every 5 min for 30 min while the subject remained supine in a darkened and quiet environment. The averages of these readings were taken as baseline systolic (SBP) and diastolic (DBP) blood pressures respectively. Mean blood pressure used to calculate vascular resistances was obtained by the formula:

$$\text{Mean blood pressure} = \text{DBP} + \tfrac{1}{3}\ \text{pulse pressure}.$$

After exercise or the control period blood pressure was recorded supine in the same environmental conditions every 2 min for 12 min, and then every 5 min from 15 to 60 min. The blood pressures were averaged for the periods 0–5, 6–10, 12–15, 20–30, 35–45 and 50–60 min.

Heart rate. Heart rate (HR) was derived from an ECG recorded onto audio tape (Racal 4-channel recorder). Heart rate was recorded at 10 and 20 min prior to exercise in the baseline period, and at 5, 10, 15, 30, 45 and 60 min after exercise or the control test.

Cardiac output. Cardiac output was derived using the method of pulsed wave Doppler ultrasonography of the ascending aorta with the pulse volume set immediately above the aortic valve. This methodology has been extensively validated against thermodilution and Fick methods (Gardin, Tobis, Dabestani, Smith, Elkayam, Castleman, White, Allfie & Henry, 1985; Innes, Mills, Noble, Murphy, Pugh, Shore & Guz, 1987). A 2 MHz dedicated pulsed wave ultrasound transducer (Vingmed, Norway) was used to record ascending aortic blood velocities from the suprasternal approach at the same times as heart rate recordings described above. The Doppler ultrasound signals were recorded onto a 4-channel tape-recorder and spectrally analysed on a fast Fourier spectrum analyser (Doptek, Chichester, UK). The intensity weighted mean velocity line was drawn by the analyser for each beat and the velocity–time integral (stroke distance) obtained as the average of five consecutive beats. Stroke volume was calculated as stroke distance × aortic root cross-sectional area, where aortic root diameter was measured by the leading-edge to leading-edge method at a point in the ascending aorta immediately distal to the aortic valve using an echocardiography machine (IREX) using the method recommended by Sahn, DeMaria, Kisslo & Weyman (1978). From stroke volume, heart rate and body surface area, cardiac index (CI) and stroke volume index (SVI) were derived using standard formulae. Systemic vascular resistance (SVR) was derived from the equation:

$$\text{SVR (mmHg min l}^{-1}) = \text{mean BP (mmHg)/cardiac output (l min}^{-1}),$$

assuming that central venous pressure in these healthy subjects was close to zero, and did not significantly change over the course of the experiment.

Aortic velocity parameters. Peak velocity was taken as the highest velocity seen in the ascending aortic velocity spectrum with a cursor placed on the top of the brightest part of the spectral display (representing the modal velocity). Acceleration was taken as the slope of the upstroke line of the intensity weighted mean velocity line from onset to two-thirds peak velocity. We have assessed the reproducibility of these measures and found a standard deviation of percentage differences between repeat recordings of 4·2–5·1 % for peak velocity and 8·5–8·6 % for acceleration compared to 7·7 % for cardiac output estimations. This degree of reproducibility gives the study design a power of 0·9 of detecting a difference of 8 % in cardiac output or acceleration or 6 % for peak velocity (Hills & Armitage, 1979).

TABLE 1. Baseline haemodynamics for the two experimental days

	Control day	Exercise day
SBP (mmHg)	110·2 ± 9·1	113·4 ± 7·4
DBP (mmHg)	62·7 ± 6·9	64·1 ± 7·2
Heart rate (beats min^{-1})	65·3 ± 10·4	68·7 ± 11·0
Cardiac index (l min^{-1} m^{-2})	2·22 ± 0·80	2·48 ± 0·81
SVR (mmHg min l^{-1})	23·3 ± 3·07	20·9 ± 2·64
Peak velocity (cm s^{-1})	105·5 ± 19·4	106·9 ± 16·1
Acceleration (m s^{-2})	28·0 ± 6·20	27·9 ± 5·90
Forearm vascular resistance mmHg min ml^{-1} (100 ml tissue)$^{-1}$	34·2 ± 5·39	34·8 ± 3·76
Pulse wave velocity (ms^{-1})	6·65 ± 0·59	6·96 ± 0·87

Baseline values for all variables for the control and exercise days. Results are expressed as mean ± standard error. There were no significant differences for any variable between the 2 days.

Forearm vascular resistance. Forearm blood flow in ml (100 ml tissue)$^{-1}$ min^{-1} was recorded using mercury-in-Silastic strain-gauge plethysmography (Hokanson, Washington, USA) by standard techniques (Greenfield, Whitney & Mowbray, 1963) at the same times as the haemodynamic variables; forearm vascular resistance (FVR) was derived from simultaneous indirect brachial artery blood pressures from the opposite arm. The reproducibility of this methodology has been reported (Roberts, Tsao & Breckenridge, 1986), and we have found similar results in our laboratory with a within-patient coefficient of variation of 13·5 %.

Pulse wave velocity. A continuous wave Doppler velocity signal from the radial artery of the right arm was recorded on the 4-channel tape-recorder simultaneously with the ascending aortic velocity signal. The time delay between the onset of the aortic velocity signal and the onset of the radial artery velocity signal was measured on the spectral analyser screen. The distance from the aortic Doppler sampling site to the radial artery was estimated as the sum of the pulse depth setting for aortic Doppler recordings and the distance from the sternal notch to the radial artery ultrasound transducer. From the delay and distance the average speed of propagation of the velocity wave in the large and medium sized arteries was derived. This velocity wave propagation velocity is virtually identical to the more commonly measured pressure wave pulse wave velocity (McDonald, 1974) and both depend on vessel diameter, intraluminal pressure and vessel wall stiffness.

Statistical analysis. All comparisons were by analysis of variance, and if significant for any variable, individual time point comparisons were made by Student's paired *t*-test between control and exercise day with a two-tailed level of significance being reported only if at less than the 5 % level. Results are expressed as mean ± standard error, and for the maximum change for each variable 95 % confidence intervals are also reported. Analysis of a two-period cross-over trial by the method of Hills & Armitage (1979) indicated no significant treatment × period effect.

RESULTS

During the baseline period of 30 min there were no significant differences between the exercise and control days for any of the measured variables (Table 1).

Blood pressure

There were significant changes in both SBP and DBP produced by exercise compared to control. SBP was elevated for 5 min after exercise by 11·2±2·5 mmHg ($P < 0·001$) but there was no significant difference from control for the next 25 min. SBP was reduced by 6·2±1·4 mmHg ($P < 0·001$) at 45 min, and by 7·9±1·2 mmHg ($P < 0·001$) at 60 min. DBP was reduced for the whole of the post-exercise hour compared to control (see Fig. 1), with the reduction being greatest at 5 min (12·6±3·0 mmHg, $P < 0·001$) and gradually lessening but remaining significant until 60 min post-exercise (7·2±1·8 mmHg, $P < 0·05$).

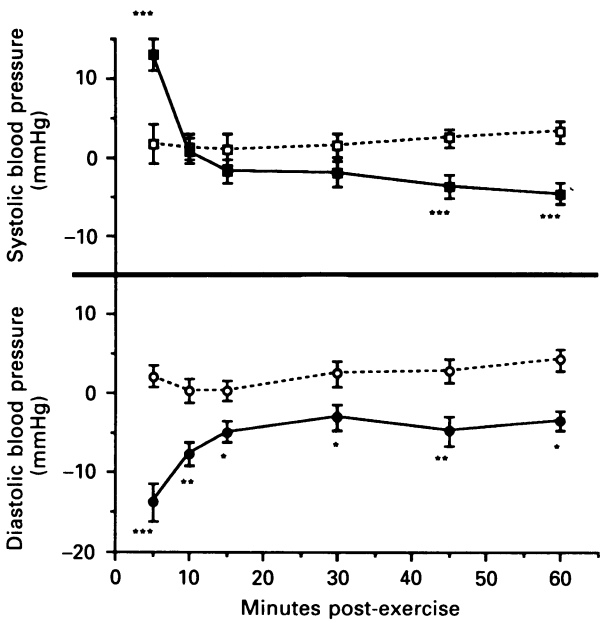

Fig. 1. Change in systolic and diastolic blood pressure (in mmHg) from baseline. Results are means with standard error bars. * = $P < 0·05$, ** = $P < 0·01$, *** = $P < 0·001$ for Student's *t*-test comparison of exercise *versus* control. Open symbols, dotted lines represent the control day and filled symbols, continuous lines represent the exercise day.

Cardiac index, stroke volume index and heart rate

Cardiac index was elevated by exercise for the whole of the post-exercise hour with the increase being greatest at 5 min (1·04±0·20 l min^{-1} m^{-2}, $P < 0·001$) and gradually lessening to an increase of 0·39±0·16 l min^{-1} m^{-2} ($P < 0·05$) at 60 min post-exercise (see Fig. 2). This increase was entirely accounted for by an increased heart rate (+30·9±3·3 beats min^{-1}, $P < 0·001$ at 5 min and +10·5±2·6 beats min^{-1}, $P < 0·001$ at 60 min). There was no change in stroke volume index during the post-exercise hour compared to control (see Fig. 2).

Vascular resistances

Peripheral vasodilatation was seen during the whole of the post-exercise hour, with systemic vascular resistance showing the greatest reduction at 5 min

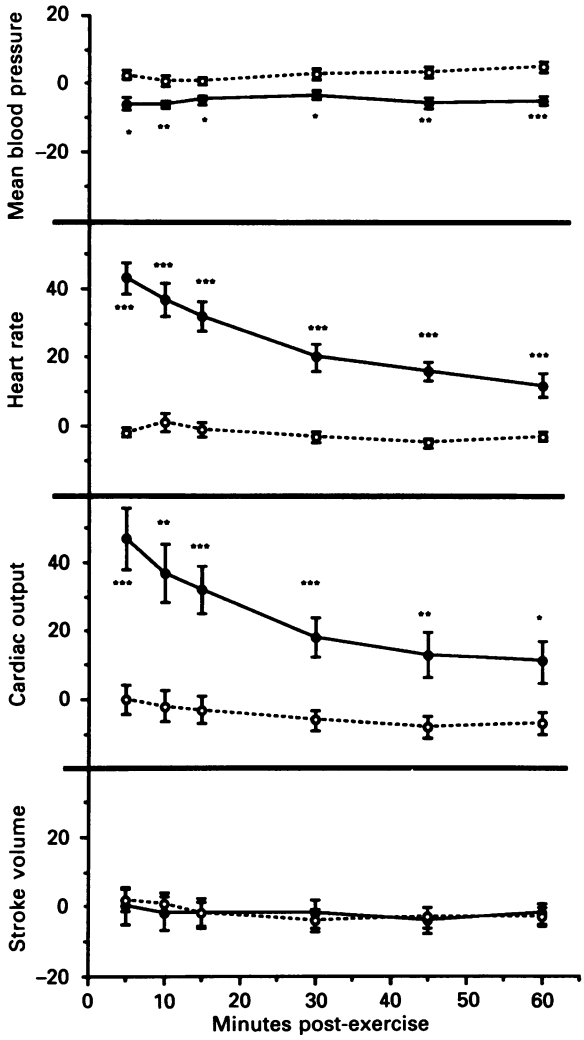

Fig. 2. Percentage changes from baseline for mean blood pressure, heart rate, cardiac output and stroke volume. Results are means with standard error bars. * = $P < 0{\cdot}05$, ** = $P < 0{\cdot}01$, *** = $P < 0{\cdot}001$. Open symbols, dotted lines represent control and filled symbols, continuous lines represent the exercise day.

($-12{\cdot}3 \pm 2{\cdot}2$ mmHg min l^{-1}, $P < 0{\cdot}001$) and the reduction gradually lessening to $-7{\cdot}2 \pm 1{\cdot}8$ mmHg min l^{-1} ($P < 0{\cdot}05$) at 60 min. The vascular bed of a non-exercised limb showed vasodilatation in response to the bicycle exercise for the whole of the post-exercise hour compared to control. Normalized (with reference to the baseline) resistance was reduced by $59 \pm 16{\cdot}2\%$ ($P < 0{\cdot}01$) at 5 min and by $46 \pm 13{\cdot}3\%$ ($P < 0{\cdot}01$) at 60 min post-exercise (see Fig. 3).

Aortic blood velocities

Peak ascending aortic blood velocity and acceleration were both briefly increased during the post-exercise hour compared to control (see Table 2). Acceleration was

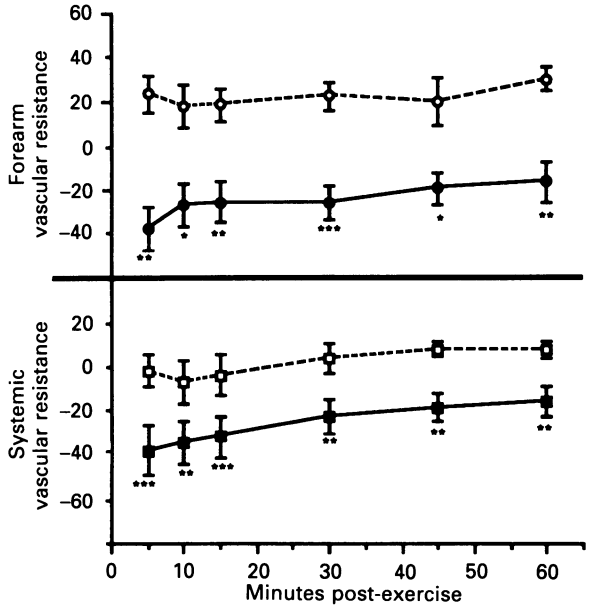

Fig. 3. Percentage changes from baseline for forearm and total systemic peripheral vascular resistances. Results are means with standard error bars. * = $P < 0{\cdot}05$, ** = $P < 0{\cdot}01$, *** = $P < 0{\cdot}001$. Open symbols, dotted lines represent control day and filled symbols, continuous lines represent the exercise day.

TABLE 2. Circulatory changes induced by a period of physical exercise

	Maximum percentage change	Duration (min)
	+10·8 (6·3–15·3)	0–5
SBP	−7·21 (5·03–9·39)	30–60
DBP	−18·5 (12·7–24·3)	0–60
Heart rate	+47·3 (37·3–57·3)	0–60
Cardiac index	+51·5 (33·6–69·4)	0–60
SVR	−32·6 (14·3–50·9)	0–60
Peak velocity	+9·92 (1·83–18·0)	0–5, 45–60
Acceleration	+21·8 (1·6–42·0)	0–10
Forearm vascular resistance	−60·0 (28·1–91·9)	0–60
Pulse wave velocity	−8·27 (3·56–13·0)	30–45

Column 1 shows the maximum percentage difference which occurred in the post-exercise hour compared to the control day (with 95% confidence limits in parentheses), and column 2 indicates the time period over which a significant change is seen.

increased $9{\cdot}2 \pm 2{\cdot}9$ m s^{-2} ($P < 0{\cdot}05$) at 5 min and $5{\cdot}6 \pm 1{\cdot}7$ m s^{-2} ($P < 0{\cdot}01$) at 10 min but not significantly affected for the rest of the hour. Peak velocity showed an increase at 5 min ($+11{\cdot}5 \pm 4{\cdot}3$ cm s^{-1}, $P < 0{\cdot}05$) and at 60 min ($+9{\cdot}8 \pm 2{\cdot}9$ cm s^{-1}, $P < 0{\cdot}05$) but the changes in between did not achieve statistical significance.

Large arterial function

Pulse wave velocity between the aorta and the radial artery was reduced during the second half-hour following the exercise period by $0{\cdot}55 \pm 0{\cdot}16$ m s^{-1} ($P < 0{\cdot}05$) at

45 min and by $0{\cdot}49 \pm 0{\cdot}26$ m s^{-1} (not significant, $P = 0{\cdot}06$) at 60 min (Table 2). Pulse wave velocity for the first half-hour after exercise did not differ from control.

DISCUSSION

Our results confirm and extend initial observations on a hypotensive effect of an acute bout of exercise (Fitzgerald, 1981; Wilcox *et al.* 1982; Bennett *et al.* 1984; Somers *et al.* 1985). The present results are the first, however, to control for the possible hypertensive effects of the anticipation of exercise, and therefore provide a more accurate estimate of the hypotensive effect of exercise.

The first 5 min after exercise are characterized by a state similar to that seen during supine exercise with an increase in cardiac output largely due to an increase in heart rate, a generalized vasodilatation, in particular to the exercising muscles, and an increase in systolic blood pressure (Thadani & Parker, 1978). There is, however, a marked reduction in diastolic blood pressure, which may reflect the sudden withdrawal of the impedance provided by the intermittently contracting musculature pressing on the dilated arterioles of the exercising muscle group. This may be in part artifactual as it is known that in states of high blood flow in the arm the Riva–Rocci method of indirect blood pressure measurement underestimates diastolic blood pressure compared to intra-arterial recordings (Kirkendall, Feinleib, Freis & Mark, 1980). This is only likely to be important for a few minutes after exercise; but the reduction in diastolic blood pressure is maintained for at least 60 min after exercise and, therefore, presumably reflects persisting vasodilatation.

Cardiac output is elevated for the whole of the post-exercise hour, and can be entirely accounted for by a similar increase in heart rate. This is to be expected as all readings were done in the supine posture, in which stroke volume is enhanced at rest and little increase is achieved by exercise (Rowell, 1974; Thadani & Parker, 1978).

The increase in cardiac output probably parallels an increased oxygen consumption due to reversal of the oxygen debt which follows heavy exercise, and which persists for up to 1 h after the end of exercise. This oxygen debt is thought to be a two-phase process, with a rapid phase representing replenishment of high-energy phosphate bonds, and a slower phase representing the time to clear accumulated lactic acid (Asmussen, 1964). The nature of the linking mechanism which couples oxygen consumption to cardiac output in the post-exercise period is not clear.

There is, however, a reduction in systemic vascular resistance for the whole of the post exercise hour. This involves both the exercising and non-exercising limb vascular bed (arm) for at least 60 min after exercise. This is despite strict supervision to ensure the subjects did not grip the handle bars during bicycle exercise. Thus there appears to be radiation of the vasodilatory signal to non-exercising muscle vascular beds which is coincident with the increased oxygen consumption and cardiac output. The linking mechanism may be partly by increased circulating levels of lactic acid, as the time course of the vasodilatation is similar to the second phase of the oxygen debt repayment.

Previous studies have suggested that, during low-level work loads, non-exercising muscle vascular beds are constricted by sympathetic vasoconstrictor efferents

(Bevegard & Shepherd, 1966), but that at higher work loads this is reversed and a vasodilatation is seen (Johnson & Rowell, 1975; Wenger, Roberts, Stolwijk & Nadel, 1975), largely as a result of local thermoregulatory skin vasodilatation. Similarly there are differing results for the post-exercise period with some authors suggesting vasoconstriction to non-exercising muscle vascular beds (Bennett *et al.* 1984), and others showing a tendency to vasodilatation (Blair *et al.* 1961; Zelis *et al.* 1969). The difference almost certainly lies in the intensity of the preceding exercise. We used a maximal exercise test and showed unequivocal vasodilatation in non-exercising vascular beds. We have described a load-dependence of these regional haemodynamic responses following exercise (Coats *et al.* 1988). It would appear that forearm muscle flow is reduced by leg exercise (Zelis *et al.* 1969), but that simultaneously forearm skin flow increases with increasing intensity and duration of exercise (Zelis *et al.* 1969; Johnson & Rowell, 1975). In the post-exercise period there may be an increase in both components (Blair *et al.* 1961), but it is impossible in our experiment to quantify the relative contributions between the muscle and skin vessel beds.

The nature of the vasodilatory signal is not established. Possibilities include a metabolic product released by the exercising muscles (e.g. lactic acid), a resetting of the baroreceptor reflex control of sympathetic tone (Bennett *et al.* 1984; Somers *et al.* 1985), or an effect of increased body heat (Wenger *et al.* 1975). The presence of marked vasodilatation argues against relative hypovolaemia being the cause of the hypotension. There is some evidence that, at least to the exercising limb, sympathetic tone is reduced after exercise (Floras, Seals, Aylward, Sinkey, Thoren & Mark, 1986) but it is not clear if sympathetic tone is reduced to non-exercising vascular beds. There is some evidence to suggest an increase in sympathetic tone to the heart, however, in that heart rate remains elevated for the whole post-exercise hour, associated with an early increase in parameters of cardiac function: aortic blood acceleration and peak velocity. Although both of these parameters are affected by after-load, acceleration has been shown also to reflect changes in the inotropic and functional state of the ventricle (Noble, Trenchard & Guz, 1966; Sabbah, Przybylski, Albert & Stein, 1987), hence their significant increase for 10 min after exercise make it unlikely that sympathetic tone to the heart and hence inotropic state is reduced at this time.

Pulse wave velocity is reduced from 45 to 60 min post-exercise; in part related to reduced blood pressure, but since the hypotension has largely recovered by the time pulse wave velocity is decreasing it is more likely that there is a reduction in arterial wall tension which is only evident when the cardiac activation of exercise has completely disappeared.

This study has implications for the recommendations of exercise outside and within the hospital environment. Exercise training has been recommended as a form of treatment for hypertension (Nelson, Jennings, Esler & Korner, 1986). In hypertensives it is not clear whether a proportion of the hypotensive effect of training is produced by the additive effects of multiple episodes of exercise-induced hypotension. In our study and others (Bennett *et al.* 1984) the period of hypotension lasts more than one hour. It must be stressed, however, that this pertains to subjects maintained in a supine resting state, and that the pressor effects of normal daily activity may mask this hypotension outside the laboratory environment. An

interesting observation from this study is that heart rate and cardiac output are still significantly elevated one hour after exercise. All studies which involve a period of exercise followed by further haemodynamic evaluation (e.g. Thadani & Parker, 1978) are complicated by this changing haemodynamic situation. Studies which compare exercise tolerance or haemodynamics twice on the same day before and after an intervention are potentially biased by this effect.

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