Arterial compliance increases after moderate-intensity cycling

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Arterial compliance increases after moderate-intensity cycling. Am. J. Physiol. 273 (Heart Circ. Physiol. 42): H2186–H2191, 1997.—Exercise training elevates arterial compliance at rest, but the effects of acute exercise in this regard are unknown. This study investigated the effects of a single, 30-min bout of cycling exercise at 65% of maximal oxygen consumption on indexes of arterial compliance. Whole body arterial compliance determined noninvasively from simultaneous measurements of aortic flow and carotid pressure was elevated (66 ± 26%) at 0.5 h postexercise (P < 0.04), followed by a decline to baseline 1 h after exercise. Aortic pulse-wave velocity, which is inversely related to compliance, was reduced (4 ± 2%; P < 0.04) at 0.5 h postexercise. Pulse-wave velocity in the leg decreased by 10 ± 4% at this time (P = 0.01). Mean arterial pressure was unchanged; however, central systolic blood pressure was reduced postexercise (P = 0.03). Cardiac output was elevated after exercise (P = 0.007) via heart rate elevation (P = 0.001), whereas stroke volume was unchanged. Total peripheral resistance was therefore reduced (P = 0.01) and would be expected to contribute to an elevation in arterial compliance. In conclusion, a single bout of cycling exercise increased whole body arterial compliance by mechanisms that may relate to vasodilatation.

arterial stiffness; pulse-wave velocity; aorta; blood pressure

THE IMPORTANCE of a compliant arterial circulation for minimizing cardiac work and providing adequate coronary perfusion has been shown experimentally (1, 16). Furthermore, clinical studies indicate that the physiological consequences of arterial stiffening, as a result of aging or the development of cardiovascular disease, have associations with serious outcomes such as myocardial infarction and death (6, 10). Therapeutic intervention, both pharmacological and nonpharmacological, to raise arterial compliance may thus be of particular benefit to patients with a stiffened proximal circulation underlying either elevated blood pressure or poor coronary perfusion (ischemic heart disease).

Our laboratory (20) and others (9, 27, 32) have previously shown in cross-sectional studies that endurance-trained athletes have greater arterial compliance compared with age-matched sedentary control subjects. Furthermore, Cameron and Dart (5) showed that 4 wk of moderate aerobic-exercise training elevates arterial compliance in previously sedentary individuals. In this latter study, they observed that there were significant changes in arterial compliance independent of mean arterial pressure reduction after only 1 wk of exercise training. Such short-term changes in arterial compliance imply that the mechanism underlying the effect most probably relates to changes in arterial smooth muscle tone, which alters the relative loading of collagen and elastin fibers (1). In particular, altered smooth muscle tone in the vasa vasorum may contribute to the effects of training (31). If such a mechanism contributed to changes in arterial compliance after only 1 wk of training, it is possible that changes in arterial compliance may also be evident acutely after a single bout of exercise.

The present study tested the hypothesis that arterial compliance is elevated in the hours after an acute bout of exercise. We compared, using a randomized, crossover study design, the effects of 30 min of moderate cycling exercise with 30 min of armchair reading on both central and peripheral measures of arterial compliance.

METHODS

Subjects and study design. Twelve male sedentary volunteers gave their written informed consent for participation in the study, which was performed with the approval of The Ethics Committee of The Alfred Hospital (Melbourne, Australia) and carried out in accordance with the Declaration of Helsinki (1989) of the World Medical Association. Participants were healthy, unmedicated nonsmokers with a body mass index < 27 kg/m2, blood pressure < 140/90 mmHg, cholesterol < 5.5 mmol/l, triglycerides < 2 mmol/l, and maximal oxygen consumption (VO2max) > 50 ml·min⁻¹·kg⁻¹ (Table 1). The first laboratory visit for all participants included a medical examination, routine hematological and biochemical blood tests, and assessment of blood pressure and VO2max. This visit occurred at least 1 wk before the first study day, and the participants were advised to avoid exercise for at least 48 h before both study days and to maintain all aspects of their lifestyle constant throughout the study.

The volunteers were aged 24 ± 6 (SD) yr, and each performed 30 min of cycling exercise at 65% of VO2max and 30 min of armchair reading in a randomized order at the same time of day 1 wk apart. Measurement of whole body, central, and peripheral arterial compliances were made both before and after each intervention as described in WBAC and Regional compliance.

Protocol. On each experimental day, the subject arrived at the laboratory at 9 AM after a light breakfast and a 24-h abstinence from caffeinated foods and beverages. A 21-gauge Teflon catheter was inserted under sterile conditions into an antecubital vein, and the subject rested in the supine position for 15 min before and throughout all measurements in a quiet temperature-controlled room maintained at 22°C. Whole body arterial compliance (WBAC) was determined with the area method of Liu et al. (22), and regional compliances of the aorta and leg were determined with pulse-wave velocity (PWV) measurements as described in WBAC and Regional compliance. These measurements were made preintervention and at 0.5, 1, 2, 4, and 24 h postintervention. Heart rate and
Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>24 ± 6*</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>22.9 ± 0.9</td>
</tr>
<tr>
<td>VO_{2max}, ml·kg⁻¹·min⁻¹</td>
<td>47 ± 1</td>
</tr>
<tr>
<td>Total cholesterol, mmol/l</td>
<td>4.1 ± 0.2</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/l</td>
<td>1.10 ± 0.08</td>
</tr>
<tr>
<td>Triglycerides, mmol/l</td>
<td>1.06 ± 0.12</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>118 ± 3</td>
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<tr>
<td>DBP, mmHg</td>
<td>68 ± 2</td>
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</table>

Values are means ± SE except * denotes SD. BMI, body mass index; VO_{2max}, maximal O₂ consumption; HDL, high-density lipoprotein; SBP, brachial systolic blood pressure; DBP, brachial diastolic blood pressure.

Blood pressure measurements were also made at these times with a Dinamap vital signs monitor (1846SX, Critikon, FL). Blood samples (8 ml each) were drawn preintervention and immediately and 2 h postintervention for the determination of blood viscosity and plasma volume changes. Because the indexes of arterial compliance had returned to baseline by 1 h postexercise, the results are reported only for this time period.

VO_{2max}, VO_{2pmax} was assessed during a graded exercise test on an electrically braked cycle ergometer as described previously (14). This test consisted of 1-min periods of bicycle exercise commencing at zero workload and increasing by 20 W/min until any further increase in workload was prevented by fatigue. The criteria for the establishment of VO_{2max} included a plateau in the oxygen consumption with increasing workload, a respiratory exchange ratio > 1.1, and failure to maintain the required work rate despite encouragement. We defined VO_{2pmax}, as the average oxygen consumption during the final 30 s of exercise. Oxygen and carbon dioxide measurements were made with a Medical Graphics 2001 CAD/Net cardiolpulmonary exercise system. Heart rate was derived from the R-R interval measured throughout the exercise test with electrocardiographic recordings from a Hewlett-Packard 43120A defibrillator.

WBAC. WBAC was estimated with the “area method” of Liu et al. (22), which requires measurement of volumetric blood flow and associated driving pressure to derive an estimated compliance over the entire arterial system (22) according to the formula

\[ WBAC = A_d[R(P_s - P_d)] \]  

(1)

where \( A_d \) is the area under the blood pressure diastolic decay curve (from end systole to end diastole), \( R \) is total peripheral resistance, \( P_s \) is the end-systolic blood pressure, and \( P_d \) is the end-diastolic blood pressure.

Continuous ascending aortic flow velocity was measured with a handheld Doppler flow velocimeter (MD1 Multi-Doplex, Huntleigh Technology, Cardiff, UK) placed on the suprasternal notch. This method provides a unique signal related to the velocity of blood in the sample volume, which is an average (approximately root mean square). Because invasive methods use a different measurement technique that derives higher flow values, we have chosen to report our data in arbitrary flow units and arbitrary compliance units that are dimensionally equivalent to liters per minute and milliliters per millimeter of mercury, respectively (5).

Volume flow was calculated as the product of the average systolic flow and aortic root area measured by two-dimensional echocardiography (Hewlett-Packard SONOS 1500, Andover, MA).

Aortic root driving pressure was estimated by applanation tonometry of the proximal right carotid artery with a noninvasive Millar Mikro-Tip pressure transducer (model SPT-301, Miller Instruments). Cameron and Dart (5) previously validated waveforms obtained with this methodology against invasively obtained pressure signals. The pressures obtained by tonometry were calibrated against brachial pressure measurements made simultaneously with a Dinamap vital signs monitor (1846SX, Critikon). Briefly, the brachial mean arterial pressure was assigned the area under the carotid waveform, and the brachial diastolic pressure was assigned the automatically determined end-diastolic point on the carotid waveform. An estimate of central systolic pressure was derived from these calibrations.

Both flow and pressure signals were digitized at 200 Hz with an Industrial Automation AX5411 analog-to-digital conversion board. Data were acquired with purpose-written software using Turbo C++ and analyzed with Pascal Turbo software (Borland International). Further details have been previously published by both Cameron and Dart (5) and Marcus et al. (25).

Regional compliance PWV is inversely related to arterial compliance (AC) according to

\[ AC = \frac{1}{(PWV^2 \cdot \rho)} \]  

(2)

where \( \rho \) is blood viscosity (28). We determined PWV centrally from the carotid to the femoral artery and peripherally from the femoral to the dorsalis pedis artery. Pulse-wave transit time over the trunk and leg was determined by simultaneous applanation tonometry of the carotid and femoral and femoral and dorsalis pedis arteries, respectively. Data were digitized and acquired as described in WBAC. The time of travel between applanation points was calculated from the upstream of the systolic waveform detected as the maximum of the second derivative. Distances from the carotid artery sampling site to the manubrium sternum, manubrium sternum to femoral artery, and femoral artery to dorsalis pedis were measured. The central PWV (aorta to femoral artery) was calculated as manubrium sternum-to-femoral artery distance minus carotid artery-to-manubrium sternum distance divided by the time interval between the carotid and femoral pulses (32). PWV in the leg was calculated as the distance traversed (femoral to dorsalis pedis arteries) divided by time taken.

Blood viscosity. Measurements of blood viscosity were made in 9 of the 12 subjects with a Wells-Brookfield cone-and-plate viscometer (model LVT). Brookfield Engineering Laboratories, Stoughton, MA) with a 1.565° cone spindle. Measurements were made on a 1-ml sample of whole blood maintained in 37°C by an external water bath. Measurements were made in duplicate at two shear rates (230 and 115 s⁻¹), as recommended by the International Committee for Standardization in Hematology (13). The Wells-Brookfield viscometer gives reliable measurements of whole blood viscosity at shear rates > 23 s⁻¹ (29).

Plasma volume. Hematocrit and hemoglobin were used to estimate the percent changes in plasma volume after exercise (7). This method is based on the assumption that the volume of circulating blood cells is constant and that the ratio between the venous hematocrit and whole body hematocrit remains unchanged with dehydration.

Statistics. Results are expressed as means ± SE. Analysis of variance (ANOVA) for repeated measures was used to examine the effects of time, intervention, and order of intervention on measurements of WBAC, PWV, blood pressure, heart rate, cardiac output, stroke volume, total peripheral resistance, and blood viscosity between control and exercise days. A paired t-test was then used to compare baseline values on the two study days and to compare baseline with the 0.5-h time point where indicated. A paired t-test was also
RESULTS

There were no baseline differences between the two study days or any effect of order of intervention on any of the parameters measured. WBAC was elevated by 66 ± 26% (0.9 ± 0.1 to 1.4 ± 0.3 arbitrary compliance units; \( P = 0.04 \)) 30 min postexercise and returned to baseline values by 1 h (Fig. 1). PWV results suggested that there were both central and peripheral contributions to this effect. PWV in the aortofemoral region was reduced 30 min postexercise (from 6.2 ± 0.4 to 5.9 ± 0.3 m/s; \( P = 0.04 \); Fig. 2). Leg PWV, although higher than central PWV, showed similar trends, the reduction at 30 min postexercise being from 8.3 ± 0.3 to 7.5 ± 0.4 m/s (\( P = 0.01 \); Fig. 2), and recovery to baseline values occurred by 1 h. There were no changes in any of the parameters during the control intervention.

Mean arterial and diastolic blood pressures were not different between the two study days, whereas central systolic blood pressure was lower after exercise (\( P < 0.03 \) by ANOVA; Table 2). Cardiac output was elevated after exercise (\( P = 0.007 \)), and this was due to heart rate elevations of 26 ± 4% at 30 min and 17 ± 5% at 1 h postexercise (\( P < 0.001 \) by ANOVA; Table 2). Stroke volume was unchanged. Total peripheral resistance was therefore reduced postexercise (\( P = 0.01 \); Table 2).

Blood viscosity measured at both shear rates was significantly elevated immediately postexercise in all subjects (4.7 ± 0.2 to 5.6 ± 0.2 cP at 115 s\(^{-1} \); \( P < 0.0001 \)) but returned close to baseline levels by 2 h postexercise (5.0 ± 0.1 cP). A reduction in plasma volume of 11 ± 1% contributed to the increased blood viscosity immediately postexercise (\( P = 0.0003 \)) and returned to resting levels by 2 h postexercise. There was no change in either blood viscosity or plasma volume during the control day.

DISCUSSION

An acute bout of cycling exercise increased WBAC via both central and peripheral effects and reduced central systolic blood pressure in the immediate postexercise period independent of changes in mean arterial pressure. Arterial compliance returned to resting levels within 1 h of exercise cessation. Relaxation of vascular smooth muscle, which transfers stress from the less extensive collagen fibers to elastin (1), is likely to account for the acute elevation in compliance. Previous data from our laboratory (5) indicated that moderate exercise training translates this acute elevation in compliance to a sustained elevation. Animal studies have shown that structural vascular adaptations account for elevated compliance when there is more extended training over many months (3, 19, 21, 26). Together, these data implicate arterial compliance in the mechanistic basis for cardiovascular risk reduction both in the immediate postexercise period and chronically with regular exercise training.

The windkessel model-based estimate of WBAC used in this study consists of both central and peripheral components (30). The PWV data suggest that both...
components contributed to the elevation in whole body compliance measured postexercise. The peripheral elevation in compliance would be expected to reduce left ventricular load both directly via physical compliance and via diminished wave reflection (2, 30). The mechanism of the observed increase in both central and peripheral compliance requires consideration of the fundamental interdependence of arterial compliance and mean arterial pressure. Changes in compliance occurring in the presence of mean arterial pressure changes may simply be a consequence of the nonlinearity of the vascular pressure-volume relationship rather than an intrinsic change in vessel wall properties (22).

In the present study, however, the elevation in compliance after exercise occurred without a change in the mean arterial pressure, suggesting a true change in arterial wall properties. This was further substantiated by the lower postexercise central systolic pressure that would be expected to occur secondarily to an elevation in arterial compliance. These findings indicate that an elevation in arterial compliance may underlie the phenomenon of postexercise hypotension, which has been documented as predominantly systolic in origin in normotensive individuals (18).

Heart rate was higher after the exercise compared with the control intervention and may have contributed to the observed changes in compliance. Experimentally, however, Mangoni et al. (23) have shown that in the anesthetized rat acute increases in heart rate are accompanied by reductions in arterial compliance. Similar conclusions were reached in a study of conscious humans using esophageal pacing (Y.-L. Liang, C. D. Gatzka, X.-J. Du, J. D. Cameron, B. A. Kingwell, and A. M. Dart, unpublished observations). The mechanism of this effect most probably relates to a reduction in the time available for arterial recoil during higher heart rates. PWV, however, is unaffected by heart rate at frequencies < 120 beats/min (4). Thus the elevation in heart rate that we measured postexercise cannot account for the increase in arterial compliance or the reduction in both central and peripheral PWVs and, in fact, would have minimized the magnitude of the effect.

Blood viscosity is related to arterial compliance and PWV according to Eq. 2 (28; see METHODS). The inverse nature of the relationship between arterial compliance and blood viscosity, however, indicates that elevation in viscosity would reduce arterial compliance. Thus, as for heart rate, the increase in blood viscosity with exercise could not account for the elevation in arterial compliance and would be expected to minimize these changes.

The mechanisms responsible for the alterations in WBAC in this study are not related to changes in blood pressure, as discussed, nor is the time course of the response consistent with intrinsic changes to the vessel wall structure. The most likely mechanism relates to alterations in the loading of elastin and collagen, which can occur with changes in vascular smooth muscle tone. Vascular smooth muscle relaxation transfers wall stress from the stiffer collagen fibers to the more extensible elastin fibers, thus making the arterial wall more compliant (1). In exercising muscles, factors including local increases in temperature, carbon dioxide, acidity, adenosine, nitric oxide, and magnesium and potassium ions may all contribute to local vasodilation and could account for altered compliance in the leg. Changes in central compliance may also be mediated directly by changes in smooth muscle tone of the aorta, but additional effects, including changes in blood flow of the vasa vasorum of the aortic wall, may also be operative (31). Vasodilation of the vasa vasorum is thought to contribute to increased aortic distensibility, and it has further been demonstrated that the vasodilator capacity of the vasa vasorum is reduced in conditions of reduced compliance, including hypertension (24). Exercise, if acting via this mechanism, may thus improve vasa vasorum blood flow and aortic compliance acutely while also preventing the longer-term medial degeneration and intrinsic stiffening of aortic tissue that is thought to occur as a result of poor perfusion of the vasa vasorum with aging (31).

Some speculation of the mechanisms by which exercise may acutely influence vascular smooth muscle tone of the aorta and vasa vasorum is necessary to complete the hypothesis. Although vasodilatory metabolites may account for peripheral dilation during and immediately after exercise in the exercising limb, other mechanisms are also likely to be operative postexercise, particularly in regions other than the exercising muscle beds. Postexercise sympathoinhibition has been well documented with microneurography (11) in hypertensive patients but does not appear to be a common feature after submaximal exercise in normotensive subjects.

Table 2. Hemodynamics

<table>
<thead>
<tr>
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<th>Control</th>
<th>Exercise</th>
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<tbody>
<tr>
<td></td>
<td>Pre 0.5 h 1.0 h</td>
<td>Pre 0.5 h 1.0 h</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>104 ± 2 104 ± 2 105 ± 3</td>
<td>103 ± 2 100 ± 2 99 ± 2</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>62 ± 3 66 ± 3 62 ± 2</td>
<td>61 ± 2 64 ± 2 61 ± 3</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>78 ± 2 81 ± 2 79 ± 2</td>
<td>78 ± 1 79 ± 2 76 ± 2</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>60 ± 3 59 ± 3 59 ± 2</td>
<td>59 ± 3 74 ± 4 69 ± 3</td>
</tr>
<tr>
<td>CO, afu</td>
<td>12 ± 0.1 12 ± 0.1 11 ± 0.3</td>
<td>13 ± 0.1 15 ± 0.1 15 ± 0.1</td>
</tr>
<tr>
<td>SV, avu</td>
<td>21 ± 1 20 ± 2 19 ± 1</td>
<td>23 ± 2 21 ± 2 22 ± 1</td>
</tr>
<tr>
<td>TPR, aru</td>
<td>17 ± 2 19 ± 2 19 ± 2</td>
<td>17 ± 2 13 ± 13 13 ± 1</td>
</tr>
</tbody>
</table>

Values are means ± SE for measurements taken preexercise (Pre) and 0.5, and 1 h postexercise. SBP, central systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; CO, cardiac output; afu, arbitrary flow units; SV, stroke volume; avu, arbitrary volume units; TPR, total peripheral resistance; aru, arbitrary resistance units. * Significant difference between control and exercise, P < 0.05 by analysis of variance.
adaptations. Effects may be more prolonged via structural vascular muscle beds. With repetition of exercise bouts, the and the vasa vasorum, whereas the peripheral effects be related to vasodilation of the large proximal vessels ing mechanism for the central changes is most likely to central and leg arterial compliances, which was indepen-

trained compared with sedentary Wistar rats (21). More

normally increase progressively with age (17). Further-

by lowering the proportion of polar amino acids that modifies elastin via a reduction in calcium content and collagen and elastin (19). Matsuda et al. (26) have changes in smooth muscle content and the properties of the vascular wall properties would be expected via training. With repeated exercise, intrinsic changes in vessels so that compliance may change independently of changes in arterial diameter.

Vasodilation is likely to represent a short-term mechan-

ism by which compliance is increased with exercise training. With repeated exercise, intrinsic changes in the vascular wall properties would be expected via changes in smooth muscle content and the properties of collagen and elastin (19). Matsuda et al. (26) have shown that swim training Wistar-Kyoto rats for 16 wk modifies elastin via a reduction in calcium content and by lowering the proportion of polar amino acids that normally increase progressively with age (17). Furthermore, a 12-wk running program has been shown to lower the proportion of collagen in the tunica media of trained compared with sedentary Wistar rats (21).

In conclusion, a 30-min bout of aerobic exercise caused an immediate but short-term increase in both central and leg arterial compliances, which was independent of changes in mean arterial pressure. The underlying mechanism for the central changes is most likely to be related to vasodilatation of the large proximal vessels and the vasa vasorum, whereas the peripheral effects are most likely due to vasodilatation of the exercising muscle beds. With repetition of exercise bouts, the effects may be more prolonged via structural vascular adaptations.

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REFERENCES


13. International Committee for Standardization in Hematolo-
gy. Guidelines for measurement of blood viscosity and erythro-


27. Mohiaddin, R. H., S. R. Underwood, H. G. Bogren, D. N. Firmin, R. H. K lipstein, R. S. O. Rees, and D. B. Longmore. Regional aortic compliance studied by magnetic resonance imag-


