

Muscular Strength Training Is Associated With Low Arterial Compliance and High Pulse Pressure

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Abstract—Aerobic exercise training increases arterial compliance and reduces systolic blood pressure, but the effects of muscular strength training on arterial mechanical properties are unknown. We compared blood pressure, whole body arterial compliance, aortic impedance, aortic stiffness (measured by β -index and carotid pulse pressure divided by normalized systolic expansion [Ep]), pulse wave velocity, and left ventricular parameters in 19 muscular strength-trained athletes (mean \pm SD age, 26 \pm 4 years) and 19 sedentary controls (26 \pm 5 years). Subjects were healthy, non-steroid-using, nonsmoking males, and athletes had been engaged in a strength-training program with no aerobic component for a minimum of 12 months. There was no difference in maximum oxygen consumption between groups, but handgrip strength (mean \pm SEM, 44 \pm 2 versus 56 \pm 2 kg; P <0.01) and left ventricular mass (168 \pm 8 versus 190 \pm 8 g; P <0.05) were greater in athletes. Arterial stiffness was higher in athletes, as evidenced by lower whole body arterial compliance (0.40 \pm 0.04 versus 0.54 \pm 0.04 arbitrary compliance units; P =0.01), higher aortic characteristic impedance (1.55 \pm 0.13 versus 1.18 \pm 0.08 mm Hg \cdot s \cdot cm⁻¹; P <0.05), β -index (4.6 \pm 0.2 versus 3.8 \pm 0.4; P <0.05), and ln Ep (10.86 \pm 0.06 versus 10.60 \pm 0.08; P <0.01). Femoral–dorsalis pedis pulse wave velocity was also higher in the athletes, but carotid–femoral pulse wave velocity was not different. Furthermore, both carotid (56 \pm 3 versus 44 \pm 2 mm Hg; P <0.001) and brachial (60 \pm 3 versus 50 \pm 2 mm Hg; P <0.01) pulse pressures were higher in the athletes, but mean arterial pressure and resting heart rate did not differ between groups. These data indicate that both the proximal aorta and the leg arteries are stiffer in strength-trained individuals and contribute to a higher cardiac afterload. (*Hypertension*. 1999;33:1385-1391.)

Key Words: mechanical properties, arterial ■ stiffness ■ compliance ■ exercise

Arterial compliance plays a role in determining both arterial systolic and diastolic pressure and therefore, in a clinical context, influences left ventricular size and function, coronary blood flow, and the risk of cerebrovascular accidents.^{1–3} In the past, measures of arterial compliance in humans have been invasive, and therefore surrogate measures such as peripheral pulse pressure have been applied to investigate potential relationships with clinical outcomes. Pulse pressure correlates closely with serious cardiovascular outcomes such as myocardial infarction,⁴ thus highlighting arterial compliance as a potential target for risk reduction therapy.

Arterial compliance decreases with increasing age,^{5–8} in atherosclerosis and coronary artery disease,^{5,9–12} and in hypertensive individuals.^{13–15} Aerobic exercise has well-documented efficacy for cardiovascular risk reduction, and it appears that at least part of its benefit derives from modification of arterial properties. In cross-sectional studies, aerobically trained athletes have a higher arterial compliance than sedentary individuals.^{6,16,17} Furthermore, arterial compliance

is elevated independently of blood pressure reduction in previously sedentary males after a 4-week program of moderate-intensity aerobic exercise training.¹⁸ These data suggest that aerobic exercise structurally modifies the large arteries, a postulate supported by studies of ex vivo aortic properties in rats, after 16 weeks of spontaneous running.^{19,20}

While aerobic exercise is widely recommended as a preventative and therapeutic strategy, resistance-style training is becoming more popular, although it is less well studied with respect to its effects on blood pressure, and no previous study has examined arterial mechanical properties in this context. High-level resistance training is associated with abrupt and large pressor responses²¹ and in the long term leads to a concentric ventricular hypertrophy.^{22–25} We hypothesize that arterial mechanical modification, with a resultant impact on the pulsatile component of arterial pressure, also occurs under these loading conditions. Previous studies have indicated that blood pressure is either reduced or unchanged by a static weight-training program in previously sedentary individuals.^{26–29} The findings of these studies, however, are specific

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to short-term interventions and cannot be extrapolated to provide insight into the effects of high-resistance training performed for many years. Previous cross-sectional studies have failed to find any difference in blood pressure between weight lifters or body builders and untrained controls,^{30–33} a finding attributable in part to the fact that blood pressure was not the primary end point and thus the studies were not ideally designed to address this question. The present study used a cross-sectional design to compare arterial mechanical properties and central and peripheral blood pressures in a group of non-steroid-using muscular strength-trained athletes with a group of well-matched sedentary controls. Measures were made of whole body arterial compliance, aortic impedance, regional aortic stiffness, and pulse wave velocity.

Methods

Subjects and Study Design

Nineteen male muscular strength-trained athletes aged 26 ± 4 (mean \pm SD) years and 19 age-matched sedentary male controls aged 26 ± 5 years participated. All were nonsmoking, normotensive (mean arterial pressure < 105 mm Hg), and normolipidemic (total cholesterol < 5.5 mmol/L, triglycerides < 2 mmol/L), with no history of cardiovascular disease or recent history of upper limb pathology. All athletes were engaged in a regular weight-training program, intended predominantly to build skeletal muscular strength, at a minimum frequency of 3 sessions per week, for a minimum of 12 months, with no aerobic exercise component. Control subjects had not participated in a regular exercise program over the previous 12 months. All subjects gave informed consent for their participation in the study, which was approved by the Ethics Committee of the Alfred Health Care Group and performed in accordance with both the institutional guidelines and the Declaration of Helsinki (1989) of the World Medical Association.

All subjects presented themselves on 2 occasions, at least 24 hours apart but within 7 days of each other. Subjects were requested to refrain from alcohol consumption and intense physical activity for 24 hours and caffeine consumption for 12 hours before their first visit. On the first day of testing, subjects underwent a comprehensive medical examination, including a resting supine 12-lead ECG. The following measurements were then made in sequential order: resting arterial blood pressure, heart rate, arterial compliance, aortic impedance, central and leg pulse wave velocity, regional aortic stiffness, echocardiographic examination, and fitness tests to determine upper limb strength and maximum oxygen consumption. Subjects were then asked to return on a second day for the collection of a routine fasting venous blood sample for lipids, testosterone, follicle-stimulating hormone (FSH), and luteinizing hormone (LH) analysis. At this time they also returned a 24-hour urine sample for assessment of sodium intake.

Resting Hemodynamics

Three brachial arterial blood pressure and 3 heart rate measurements were made at 1-minute intervals with the use of a Dinamap vital signs monitor (1846 SX, Critikon), with subjects remaining in the supine position after 10 minutes of undisturbed rest in a darkened quiet room. The mean of these 3 values was taken to represent resting levels.

Maximum Oxygen Consumption

Maximum oxygen consumption ($\dot{V}O_{2\max}$), and maximum workload (W_{\max}) were assessed during a graded maximum exercise test performed on an electrically braked cycle ergometer (Ergometrics-900, Ergo-line).¹⁶ The criteria for establishment of $\dot{V}O_{2\max}$ included a plateau in oxygen consumption with increasing work rate and a respiratory exchange ratio of > 1.1 . We defined $\dot{V}O_{2\max}$ as the mean oxygen consumption during the final 30 seconds of exercise. Brachial arterial blood pressure measurements were made every

minute with an automated, auscultatory sphygmomanometer incorporated in the cycle ergometer unit.

Handgrip Strength

Maximum handgrip strength was determined in both dominant and nondominant limbs with the use of a Jamar hydraulic hand dynamometer (Sammons Preston); the recommendations of the American Society of Hand Therapists concerning the standardized positioning of subjects during the assessment of handgrip strength were used.³⁴ The dominant limb was tested first in all subjects. Three maximum contractions, each lasting 3 to 5 seconds and 15 seconds apart, were performed in both limbs. The maximum strength score achieved from the 3 trials was taken as being the representative maximum handgrip strength for that particular limb.

Left Ventricular Dimensions, Mass, and Function

Left ventricular end-diastolic posterior wall thickness, interventricular septum wall thickness at end-diastole, left ventricular internal end-diastolic diameter (LVID), and left ventricular internal end-systolic diameter (LVIS) were measured with the use of the American Society of Echocardiography convention from M-mode images of the left ventricle generated in the short-axis view at the level of the mitral chordae by a Hewlett Packard 77020A ultrasound system (Hewlett Packard). Left ventricular mass was calculated,³⁵ and the ratio of average wall thickness (mean of interventricular septal wall thickness and posterior wall thickness) to LVID was used as a measure of left ventricular hypertrophy. Left ventricular systolic function was assessed through the use of fractional shortening (FS), where $FS = 100 \times (LVID - LVIS) / LVID$. Left ventricular diastolic function was assessed from the ratio of peak transmitral blood velocity measurements during early and late diastole with the use of pulsed wave Doppler measurements. The deceleration time of early transmitral flow was used as a measure of left ventricular wall stiffness. Aortic root area was measured at the insertion of the aortic valve leaflets during peak systole to permit calculation of volume flow from velocity flow (see following section).

Arterial Compliance

Whole body arterial compliance was determined by the method of Liu et al³⁶ and validated in our laboratory by Cameron and Dart.^{18,37} Proximal right carotid artery pressure was measured by applanation tonometry with the use of a Millar Mikro-Tip pressure transducer (SPT-301, Millar Instruments). Brachial arterial blood pressure was simultaneously measured with a Dinamap vital signs monitor (1846 SX, Critikon) to permit the calibration of the carotid arterial pressure contour using brachial mean and diastolic blood pressure. This method permits derivation of a carotid systolic blood pressure that has been validated against invasively obtained aortic root pressure measurements.^{18,38} Average aortic systolic flow velocity was measured with a hand-held continuous wave Doppler velocimeter with a 3.5-MHz transducer (Multi-Dopplex MD1, Huntleigh Technology) placed in the suprasternal notch. The pressure waveform was aligned with the flow waveform with the maximum of the second derivative of the systolic upstroke used as a primary match point.³⁹ The average of 10 cardiac cycles is reported. Volume flow was obtained by multiplying velocity flow and aortic root cross-sectional area and is reported in arbitrary flow units (AFU) dimensionally equivalent to $L \cdot \text{min}^{-1}$.^{18,37} Arterial compliance is reported in arbitrary compliance units (ACU); cardiac output derived from velocity flow was also used to calculate total peripheral resistance in arbitrary resistance units (ARU).

Aortic Impedance

From the same 10 individual pressure and flow velocity waveforms as obtained for the compliance determinations, we constructed an ensemble average time series and calculated aortic input impedance, characteristic impedance, reflection factor, and forward and backward components of the pressure waveform.^{40,41} We used only those harmonics in our calculation in which the magnitude of the respective harmonic for both pressure and flow was $> 2.5\%$ of the magnitude of the first harmonic. The highest harmonic thus included

in calculations corresponded to a frequency of 10.5 ± 0.3 Hz. Characteristic impedance was calculated from the arithmetic mean of moduli corresponding to frequencies >2 Hz. The reflection factor was calculated as the ratio between the amplitude of the backward to the amplitude of the forward wave in the time domain.⁴² Calculations were performed with the use of custom written software, MATLAB for Windows version 5.2.1.1420 (The MathWorks, Inc), and Microsoft Excel 97.

Pulse Wave Velocity

Pulse wave velocity, which is inversely related to arterial compliance, was measured centrally between the carotid and femoral arteries and in the leg between the femoral and dorsalis pedis arteries by simultaneous applanation tonometry (SPT-301, Millar Instruments).³⁷

Proximal Aortic Stiffness

The stiffness of the transverse aortic arch was measured from suprasternal M-mode echocardiographic images obtained with the use of a Hewlett Packard model 77020A phased-array sector scanner. Stiffness was quantified with the β -index and E_p .⁵ The β -index is defined as the natural logarithm of the quotient of carotid systolic blood pressure to diastolic blood pressure, all divided by the normalized systolic expansion (S), where $S = (D_s - D_d)/D_d$, D_d is the minimum aortic diameter in diastole, and D_s is the maximum aortic diameter in systole. E_p is defined as carotid pulse pressure divided by the normalized systolic expansion (S). The E_p data were normalized with the use of the natural logarithm. We have previously shown these methods to have high repeatability.^{5,7}

Biochemical Analyses

Blood for analyses was collected into EDTA tubes, placed on ice, and then centrifuged at 3000 rpm within 10 minutes of collection. Plasma was frozen at -20°C . Total, LDL, and HDL cholesterol and triglycerides were determined enzymatically with a Cobas-BIO centrifugal analyzer (Roche Diagnostic Systems).⁴³ FSH, LH, and testosterone were determined by automated immunoassays.

Statistical Analyses

Data from the 38 study subjects were collated and statistically analyzed with the use of SPSS for Windows version 8.0.0. All variables for the athletic and control groups were compared with a 2-tailed unpaired Student *t* test. Statistical significance was deemed to have been achieved when $P < 0.05$. Unless otherwise specified, all group representative results are presented as mean \pm SEM.

Results

Group characteristics are presented in Table 1 and show that the strength-trained athletes and sedentary controls were comparable in age, height, lipid levels, aerobic fitness (assessed by $\text{V}_{\text{O}_2\text{max}}$ and Wmax), FSH, LH, testosterone, and 24-hour urinary sodium output. Body mass and therefore body mass index (BMI) were significantly greater in the athletic group. Confirming the training status of the athletic group was a greater handgrip strength in both the dominant and nondominant forearms.

Cardiac Structure and Function

Absolute left ventricular mass was greater in the athletic group; however, this difference was not significant after normalizing for body surface area (Table 2; $P = 0.14$). Normalizing left ventricular mass by body mass further equalized the 2 groups ($P = 0.82$). In addition, there was no difference between groups in the ratio of wall thickness to lumen or for any echocardiographic measure of systolic or diastolic function, including the aortic root area used to calculate arterial

TABLE 1. Group Characteristics

Variable	Controls (n=19)	Athletes (n=19)
Age, y	26 \pm 1	26 \pm 1
Height, m	1.79 \pm 0.02	1.76 \pm 0.02
Mass, kg	72 \pm 2	84 \pm 3*
Body surface area, m ²	1.90 \pm 0.04	2.00 \pm 0.05
BMI, kg \cdot m ⁻²	22.4 \pm 0.6	26.9 \pm 0.8†
Total cholesterol, mmol \cdot L ⁻¹	4.2 \pm 0.1	4.2 \pm 0.1
HDL cholesterol, mmol \cdot L ⁻¹	1.2 \pm 0.1	1.0 \pm 0.1
LDL cholesterol, mmol \cdot L ⁻¹	2.6 \pm 0.6	2.7 \pm 0.1
Total triglycerides, mmol \cdot L ⁻¹	1.2 \pm 0.1	1.0 \pm 0.1
FSH, U \cdot L ⁻¹	4.2 \pm 0.5	3.8 \pm 0.5
LH, U \cdot L ⁻¹	5.1 \pm 0.5	4.8 \pm 0.4
Testosterone, nmol \cdot L ⁻¹	10.9 \pm 0.7	12.1 \pm 0.9
24-h urinary sodium excretion, mmol	158 \pm 16	183 \pm 13
Dominant handgrip strength, kg	44 \pm 2	56 \pm 2†
Nondominant handgrip strength, kg	42 \pm 2	55 \pm 2†
$\text{V}_{\text{O}_2\text{max}}$, ml \cdot min ⁻¹ \cdot kg ⁻¹	41 \pm 2	44 \pm 2
Wmax , W	227 \pm 13	260 \pm 12

Values are mean \pm SEM.

* $P < 0.05$, † $P < 0.01$, significant difference from controls.

compliance and the diameter of the transverse aorta during diastole or systole (Table 2).

Hemodynamics

Resting heart rate did not differ between controls (62 ± 2 bpm) and athletes (62 ± 3 bpm). Mean resting arterial pressure (controls, 80 ± 1 mm Hg; athletes, 81 ± 2 mm Hg; Figure), cardiac output (controls, 1.20 ± 0.08 AFU; athletes, 1.28 ± 0.10 AFU), and total peripheral resistance (controls, 17.6 ± 1.4 ARU; athletes, 16.7 ± 1.5 ARU) were similar in both groups. The athletes demonstrated a greater brachial systolic blood pressure (controls, 109 ± 2 mm Hg; athletes, 120 ± 3 mm Hg; $P = 0.01$) and lower brachial diastolic pressure (controls, 63 ± 1 mm Hg; athletes, 59 ± 1 mm Hg; $P < 0.05$; Figure). Consequently, the athletic group had a greater resting brachial pulse pressure than controls (controls, 50 ± 2 mm Hg; athletes, 60 ± 3 mm Hg; $P < 0.01$). These differences were similar for both carotid systolic pressure (controls, 110 ± 2 mm Hg; athletes, 118 ± 3 mm Hg; $P < 0.05$) and pulse pressure (controls, 44 ± 2 mm Hg; athletes, 56 ± 3 mm Hg; $P < 0.01$). The difference in brachial systolic pressure was maintained at maximum exercise (controls, 186 ± 5 mm Hg; athletes, 204 ± 5 mm Hg; $P < 0.05$) despite similar maximum heart rates (controls, 183 ± 3 bpm; athletes, 181 ± 3 bpm).

Arterial Mechanical Properties

Whole body arterial compliance was significantly lower and aortic input impedance significantly higher in the athletic group than in the control group (Table 2). The higher values for regional measures of aortic stiffness, including β -index, E_p , and $\ln E_p$ in the athletic group, suggest that stiffening was present in the region of the transverse aortic arch. The similar

TABLE 2. Cardiac Structure and Function and Arterial Mechanical Properties

Variable	Controls (n=19)	Athletes (n=19)
Cardiac structure and function		
Left ventricular mass, g	168±8	190±8*
Left ventricular mass/BSA, g · m ⁻²	87.8±3.3	95.1±3.6
Left ventricular mass/BW, g · kg ⁻¹	2.33±0.09	2.30±0.10
Average wall thickness/LVID	0.19±0.01	0.18±0.01
LVID, cm	5.1±0.08	5.3±0.1
FS, %	36±1	37±1
E/A	1.9±0.1	2.1±0.2
Deceleration time, s	0.17±0.01	0.18±0.01
Aortic root area, cm ²	4.1±0.1	3.9±0.1
Transverse aortic diameter (systole), cm	2.79±0.08	2.74±0.05
Transverse aortic diameter (diastole), cm	2.44±0.07	2.40±0.04
Arterial mechanical properties		
Whole body arterial compliance, ACU	0.54±0.04	0.40±0.04†
Input impedance modulus at 1st harmonic, mm Hg · s · cm ⁻¹	1.64±0.10	2.23±0.24*
Characteristic impedance, mm Hg · s · cm ⁻¹	1.18±0.08	1.55±0.13*
Reflection factor	0.32±0.02	0.37±0.02
Frequency, 1st minimum of impedance modulus, Hz	3.4±0.2	3.8±0.2
Carotid-femoral PWV, m · s ⁻¹	6.6±0.2	6.5±0.2
Femoral-dorsalis pedis PWV, m · s ⁻¹	8.1±0.3	9.2±0.3†
β-Index	3.8±0.4	4.6±0.2*
Ep, kN · m ⁻²	43±4	54±3*
Ln Ep	10.60±0.08	10.86±0.06†

Values are mean±SEM. BSA indicates body surface area; BW, body weight; E/A, ratio of early to late transmitral diastolic blood flow; and PWV, pulse wave velocity.

**P*<0.05, †*P*<0.01, significant difference from controls.

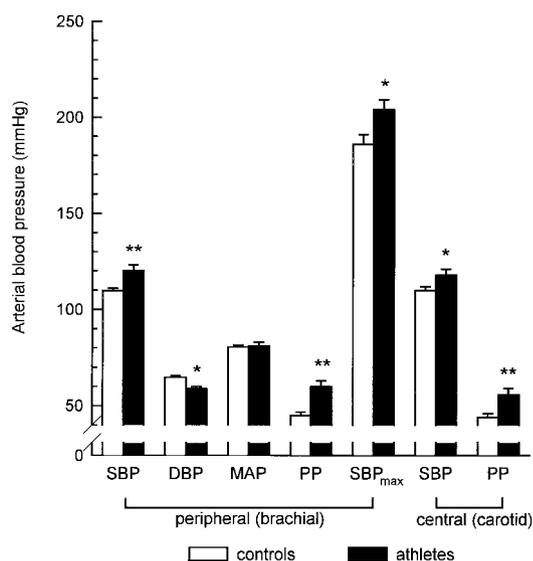
reflection factors and higher characteristic impedance in the athletic group suggest that differences in aortic stiffness had a structural origin. Directly measured carotid-femoral pulse wave velocity, which omits the most proximal part of the

aorta, was not different between groups. In the periphery, athletes exhibited a higher leg pulse wave velocity than controls.

Discussion

This is the first study to systematically characterize central and peripheral blood pressure and mechanical properties in moderately but exclusively power-trained athletes. Our data indicate that whole body arterial compliance is lower in strength-trained men than in age-matched sedentary controls. Furthermore, this difference in arterial compliance cannot be attributed to differences in mean arterial pressure but rather is most likely due to intrinsic differences in the proximal aorta of strength-trained and sedentary men. This can be justified by examining the differences in aortic stiffness indices and characteristic aortic impedance observed between the 2 study groups.

Previous studies have shown a decrease in arterial compliance with advancing age,⁵⁻⁸ in atherosclerosis and coronary heart disease,^{5,10-12} and in hypertension¹³⁻¹⁵ and an increase in endurance-trained athletes.^{6,16-18} The difference observed between the 2 groups cannot be explained by the presence of any of these factors, because age, lipid profiles, resting mean arterial pressures, and aerobic fitness (quantified by $\dot{V}O_2\text{max}$) were comparable in the 2 groups. Undeclared steroid use is another possible confounding factor; however, the reductions in HDL cholesterol,^{44,45} FSH, LH, and testosterone^{46,47} usually observed



Brachial and carotid arterial blood pressure in controls and athletes. All pressures are resting except maximum systolic blood pressure (SBP_{max}), which corresponds to maximum exercise. SBP indicates systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; and PP, pulse pressure. **P*<0.05, ***P*<0.01, significant difference from control group.

with abuse of androgenic-anabolic steroids were not present in our athletes, making this possibility unlikely. The difference in BMI between the groups must also be considered; however, studies in both our own laboratory and others⁴⁸ have not found an association between BMI and stiffness of the large elastic arteries. Furthermore, neither arterial compliance nor systolic blood pressure was correlated with BMI in this study.

Consistent with the lower arterial compliance of the athletic group was the higher pulse pressure, measured both peripherally at the brachial artery and centrally at the common carotid artery. The difference in pulse pressure was attributable to both greater systolic and lower diastolic arterial blood pressure in the athletes. While previous studies have reported that muscular strength-trained athletes have similar or lower pressures than the sedentary population, such studies have been confounded by a number of factors. These have included absence of control data,²⁵ nonsedentary control groups,³⁰ small sample sizes,³¹ steroid use,²⁵ and nonobjective methods of blood pressure assessment.^{22,25,30–32} Therefore, our study has revealed a significant difference in resting arterial pulse pressure but not mean pressure in exclusively strength-trained individuals not previously identified. Furthermore, the higher systolic pressure of the athletic group was maintained at maximal exercise, indicating a greater afterload during aerobic exercise in the strength-trained group. Our findings do not, however, preclude a blood pressure-lowering effect of more moderate levels of dynamic resistance training, as reported by Kelley⁴⁹ in a recent meta-analysis.

The afterload presented to the heart during each cardiac cycle can be quantified by the determination of aortic input impedance. This is dependent on the intrinsic physiological characteristics of the arterial tree, the characteristic impedance, and the magnitude and timing of reflected pressure waves from the periphery. Aortic input impedance and both of its components, characteristic impedance and wave reflection, along with regional aortic stiffness (β -index and E_p) and leg pulse wave velocity, were used to determine whether the observed difference in whole body arterial compliance was more likely to be due to structural differences in the proximal arterial system or to differences in reflection properties. Aortic input impedance was shown to be greater in the athletic group compared with controls and was associated with a greater characteristic impedance in the absence of a difference in the wave reflection component. These data suggesting that the strength-trained athletes had structurally stiffer aortas were further supported by our regional measures of aortic stiffness, β -index and E_p , which were higher in the trained group. Directly measured carotid to femoral pulse wave velocity was not, however, different between the 2 groups. This method measures the temporal separation of the occurrence of the pressure wave in the carotid and femoral arteries. Since we assume that at the time the pressure is measured in the carotid artery, the wave moving toward the femoral artery will have traversed the same distance, the corresponding length of proximal aorta is not incorporated in the pulse

wave velocity calculation. The appropriate transit distance is the manubrium sternum–femoral distance minus the carotid–manubrium sternum distance.¹⁷ Thus, central pulse wave velocity does not incorporate the most proximal part of the aorta. Since all our other measures of arterial mechanical properties do include the proximal aorta, the data suggest that the effects of strength training may be specific to this region.

Leg pulse wave velocity was higher in athletes, indicating that the more muscular peripheral arteries were also stiffer than those in sedentary controls. A previous study in hammer throwers reported higher compliance in the radial artery of the dominant arm relative to both the contralateral arm and to an inactive control group.⁵⁰ The difference between these findings and our own may relate to the arm-specific and dual static and dynamic components of hammer throwing.

Consistent with previous studies, our echocardiographic data support the notion that static exercise increases left ventricular mass in absolute terms but not in relation to skeletal muscle mass.^{22,51} In competitive weight lifters, the increase in left ventricular mass results in a concentric left ventricular hypertrophy assessed by either the ratio of mass to volume or the ratio of wall thickness to lumen diameter.²² The athletic group of the present study included some competitive weight lifters and body builders, but most were amateur and, consistent with previous data relating to athletes of this caliber, did not show an increase in ratio of wall thickness to lumen.²² The mechanism linking increased skeletal muscle mass to increased cardiac mass might plausibly be related to the intermittent elevations in arterial pressures experienced during muscular strength-training exercise.^{22–24} In the long term, however, our data suggest that stiffening of the proximal aorta may contribute to left ventricular hypertrophy through elevation in resting systolic blood pressure and by augmenting the pressor responses experienced during exercise.

The cross-sectional nature of this study does not permit investigation of the mechanisms by which regular muscular strength training may decrease proximal aortic and leg compliance. It is likely that the acute elevations in arterial blood pressure associated with resistance exercise lead to long-term changes in the smooth muscle content of the arterial wall and the load-bearing properties of collagen and elastin. There is increasing evidence, from animal studies, that the relative proportions and properties of collagen and elastin within the arterial wall change after periods of aerobic exercise training.^{52,53} Although these studies investigated the effects of aerobic endurance exercise, the results remain relevant, since they indicate that changes in intrinsic arterial wall characteristics can occur after relatively short periods of exercise training.

This study has demonstrated that a regular resistance-training program designed to promote skeletal muscle strength in healthy young men is associated with lower proximal aortic and leg compliance than those in an age-matched control group. These vascular changes were associated with higher central and brachial pulse pressures at rest and higher brachial systolic pressure at maximum

aerobic exercise. The clinical implications of these findings with regard to cardiovascular risk warrant further investigation.

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References

- Rajkumar C, Cameron JD, Christophidis N, Jennings GL, Dart AM. Reduced systemic arterial compliance is associated with left ventricular hypertrophy and diastolic dysfunction in older people. *J Am Geriatr Soc*. 1997;45:803–808.
- Watanabe H, Ohtsuka S, Kakihana M, Sugishita Y. Decreased aortic compliance aggravates subendocardial ischaemia in dogs with stenosed coronary artery. *Cardiovasc Res*. 1992;26:1212–1218.
- Kass DA, Saeki A, Tunin RS, Recchia FA. Adverse influence of systemic vascular stiffening on cardiac dysfunction and adaptation to acute coronary occlusion. *Circulation*. 1996;93:1533–1541.
- Benetos A, Safar M, Rudnicki A, Smulyan H, Richard JL, Ducimetiere P, Guize L. Pulse pressure: a predictor of long-term cardiovascular mortality in a French male population. *Hypertension*. 1997;30:1410–1415.
- Dart AM, Lacombe F, Yeoh JK, Cameron JD, Jennings GL, Laufer E, Esmore D. Aortic distensibility in patients with isolated hypercholesterolaemia, coronary artery disease, or cardiac transplant. *Lancet*. 1991;338:270–273.
- Mohiaddin RK, Underwood SR, Bogren HG, Firmin DN, Klipstein RH, Rees SRO, Longmore DB. Regional aortic compliance studied by magnetic resonance imaging: the effects of age, training and coronary artery disease. *Br Heart J*. 1989;62:90–96.
- Lacombe F, Dart A, Dewar E, Jennings G, Cameron J, Laufer E. Arterial elastic properties in man: a comparison of echo-Doppler indices of aortic stiffness. *Eur Heart J*. 1992;13:1040–1045.
- Avolio AP, Chen S-G, Wang R-P, Zhang C-I, Li M-F, O'Rourke MF. Effects of ageing on changing arterial compliance and left ventricular load in a northern Chinese urban community. *Circulation*. 1983;68:50–58.
- Gatzka CD, Cameron JD, Kingwell BA, Dart AM. Relation between coronary artery disease, aortic stiffness and left ventricular structure in a population sample. *Hypertension*. 1998;32:575–578.
- Cameron JD, Jennings GL, Dart AM. The relationship between arterial compliance, age, blood pressure and serum lipid levels. *J Hypertens*. 1995;13:1718–1723.
- Stefanidis C, Stratos C, Boudoulas H, Kourouklis C, Toutouzas P. Distensibility of the ascending aorta: comparison of invasive and non-invasive techniques in healthy men and in men with coronary artery disease. *Eur Heart J*. 1990;11:990–996.
- Hirai T, Sasayama S, Kawasaki T, Yagi S. Stiffness of systemic arteries in patients with myocardial infarction. *Circulation*. 1989;80:78–86.
- Li JK-J, Zhu Y. Arterial compliance and its pressure dependence in hypertension and vasodilation. *Angiology*. 1994;45:113–117.
- Dart AM, Silagy C, Dewar E, Jennings G, McNeil J. Aortic distensibility and left ventricular structure and function in isolated systolic hypertension. *Eur Heart J*. 1993;14:1465–1470.
- Avolio AP, Fa-Quan D, Wei-Qiang L, Yao-Fei L, Zhen-Dong H, Lian-Fen X, O'Rourke MF. Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. *Circulation*. 1985;71:202–210.
- Kingwell BA, Cameron JD, Gillies KJ, Jennings GL, Dart AM. Arterial compliance may influence baroreflex function in athletes and hypertensives. *Am J Physiol*. 1995;268(Heart Circ Physiol. 37):H411–H418.
- Vaitkevicius PV, Fleg JL, Engel JH, O'Connor FC, Wright JG, Lakatta LE, Yin FCP, Lakatta EG. Effects of age and aerobic capacity on arterial stiffness in healthy adults. *Circulation*. 1993;88(pt 1):1456–1462.
- Cameron JD, Dart AM. Exercise training increases total systemic arterial compliance in humans. *Am J Physiol*. 1994;266(Heart Circ Physiol. 35):H693–H701.
- Kingwell BA, Arnold PJ, Jennings GL, Dart AM. Spontaneous running increases aortic compliance in Wistar-Kyoto rats. *Cardiovasc Res*. 1997;35:132–137.
- Kingwell BA, Arnold PJ, Jennings GL, Dart AM. The effects of voluntary running on cardiac mass and aortic compliance in Wistar-Kyoto and spontaneously hypertensive rats. *J Hypertens*. 1998;16:181–185.
- MacDougall JD, Tuxen D, Sale DG, Moroz JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol*. 1985;58:785–790.
- Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Echocardiographic left ventricular masses in distance runners and weight lifters. *J Appl Physiol*. 1980;48:154–162.
- Colan SD, Sanders SP, Borow KM. Physiologic hypertrophy: effects of left ventricular systolic mechanics in athletes. *J Am Coll Cardiol*. 1987;9:776–783.
- Morganroth J, Maron BL, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. *Ann Intern Med*. 1975;82:521–524.
- Pearson AC, Schiff M, Mrosek D, Labovitz AJ, Williams GA. Left ventricular diastolic function in weight lifters. *Am J Cardiol*. 1986;58:1254–1259.
- Van Hoof R, Macor F, Lijnen P, Staessen J, Thijs L, Vanhees L, Fagard R. Effect of strength training on blood pressure measured in various conditions in sedentary men. *Int J Sports Med*. 1996;17:415–422.
- Wiley RL, Dunn CL, Cox RH, Hueppchen NA, Scott MS. Isometric exercise training lowers resting blood pressure. *Med Sci Sports Exerc*. 1992;24:749–754.
- Stone MH, Wilson GD, Blessing D, Rozenek R. Cardiovascular responses to short-term Olympic style weight-training in young men. *Can J Appl Sport Sci*. 1983;8:134–139.
- Hurley BF, Hagberg JM, Goldberg AP, Seals DR, Ehsani AA, Brennan RE, Holloszy JO. Resistive training can reduce coronary risk factors without altering VO_2max or percent body fat. *Med Sci Sports Exerc*. 1988;20:150–154.
- Colan SD, Sanders SP, MacPherson D, Borow KM. Left ventricular diastolic function in elite athletes with physiologic cardiac hypertrophy. *J Am Coll Cardiol*. 1985;6:545–549.
- Fleck SJ, Dean LS. Resistance-training experience and the pressor response during resistance exercise. *J Appl Physiol*. 1987;63:116–120.
- Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Cardiovascular responses to static exercise in distance runners and weight lifters. *J Appl Physiol*. 1980;49:676–683.
- Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Chronic training with static and dynamic exercise: cardiovascular adaptation, and response to exercise. *Circ Res*. 1981;48(suppl 1):I-171–I-178.
- O'Driscoll SW, Horii E, Ness R, Cahalan TD, Richards RR, An K. The relationship between wrist position, grasp size, and grip strength. *J Hand Surg*. 1992;17A:169–177.
- Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, Reichek N. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol*. 1986;57:450–458.
- Liu Z, Brian KP, Yin FCP. Estimation of total arterial compliance: an improved method and evaluation of current methods. *Am J Physiol*. 1986;251(Heart Circ Physiol. 20):H588–H600.
- Kingwell BA, Berry KL, Cameron JD, Jennings GL, Dart AM. Arterial compliance increases after moderate-intensity cycling. *Am J Physiol*. 1997;273:H2186–H2191.
- Chen C-H, Ting C-T, Nussbacher A, Nevo E, Kass DA, Pak P, Wang S-P, Chang M-S, Yin FCP. Validation of carotid tonometry as a means of estimating augmentation index of ascending aortic pressure. *Hypertension*. 1996;27:168–175.
- Chiu YC, Arand PW, Shroff SG, Feldman T, Carroll JD. Determination of pulse wave velocities with computerized algorithms. *Am Heart J*. 1991;121:1460–1470.
- Kelly R, Fitchett D. Noninvasive determination of aortic input impedance and external left ventricular power output: a validation and repeatability study of a new technique. *J Am Coll Cardiol*. 1992;20:952–963.
- Westerhof N, Sipkema P, van den Bos GC, Elzinga G. Forward and backward waves in the arterial system. *Cardiovasc Res*. 1972;6:648–656.
- Chang KC, Tseng YZ, Kuo TS, Chen HI. Impaired left ventricular relaxation and arterial stiffness in patients with essential hypertension. *Clin Sci*. 1994;87:641–647.
- Kingwell BA, Sherrard B, Jennings GL, Dart AM. Four weeks of cycle training increases basal production of nitric oxide from the forearm. *Am J Physiol*. 1997;272:H1070–H1077.

44. Alen M, Rahkila P. Reduced high-density lipoprotein-cholesterol in power athletes: use of male sex hormone derivatives, an atherogenic factor. *Int J Sports Med.* 1984;5:341–342.
45. Alen M, Rahkila P, Marniemi J. Serum lipids in power athletes self-administering testosterone and anabolic steroids. *Int J Sports Med.* 1985; 6:139–144.
46. Alen M, Rahkila P, Reinila M, Vihko R. Androgenic-anabolic steroid effects on serum thyroid, pituitary and steroid hormones in athletes. *Am J Sports Med.* 1987;15:357–361.
47. Alen M, Reinila M, Vihko R. Response of serum hormones to androgen administration in power athletes. *Med Sci Sports Exerc.* 1985;17: 354–359.
48. Matthews PG, Wahlqvist ML, Marks SJ, Myers KA, Hodgson JM. Improvement in arterial stiffness during hypolipidaemic therapy is offset by weight gain. *Int J Obes Relat Metab Disord.* 1993;17: 579–583.
49. Kelley G. Dynamic resistance exercise and resting blood pressure in adults: a meta-analysis. *J Appl Physiol.* 1997;82:1559–1565.
50. Giannattasio C, Cattaneo BM, Mangoni AA, Carugo S, Sampieri L, Cuspidi C, Grassi G. Changes in arterial compliance induced by physical training in hammer-throwers. *J Hypertens.* 1992;10(suppl 6):S53–S55.
51. Fleck SJ. Cardiovascular adaptations to resistance training. *Med Sci Sports Exerc.* 1988;20:S146–S151.
52. Koutis G, Kadi F, Vandewalle H, Lechat P, Hadjiisky P, Monod H. Effects of an endurance training programme on the passive and noradrenaline-activated compliances of rat aorta. *Eur J Appl Physiol.* 1995;71: 173–179.
53. Matsuda M, Nosaka T, Sato M, Iijima J, Ohshima N, Fukushima H. Effects of exercise training on biochemical and biomechanical properties of rat aorta. *Angiology.* 1989;40:51–57.

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