Physical Exercise, Aortic Blood Pressure, and Aortic Wall Elasticity and Composition in Rats

Nathalie Niederhoffer, Pascal Kieffer, Dominique Desplanches, Isabelle Lartaud-Idjouadiene, Marie-Hélène Sornay, Jeffrey Atkinson

Abstract—With a training schedule (8 weeks’ treadmill running at 30 m/min up a 10% incline 5 d/wk for 90 min/day), we investigated whether exercise modifies aortic wall dimensions, composition (calcium and elastin content), or stiffness in normotensive 6-month-old male Wistar WAG/Rij rats. Maximal oxygen uptake was measured in half of the rats (n=10 per group). Wall stiffness was evaluated in the other half (9 trained and 10 untrained) on the basis of changes in thoracoabdominal pressure pulse wave velocity and differences in amplitude between the peripheral and central aortic pressure signals. Experiments were performed in nonanesthetized, unrestrained rats and then after pithing. The impact of exercise on the oxidative capacity of the plantaris muscles was evaluated with the measurement of citrate synthase activity. Training increased maximal oxygen uptake by 34% and citrate synthase activity by 40%. Mean peripheral aortic pressure increased by 6% and 19% in trained rats, under awake and pithed conditions, whereas mean central aortic pressure increased by 16%, after pithing only. All indexes of aortic stiffness were similar in trained and control rats, as were aortic wall dimensions, composition, cardiac mass, and heart rate. In conclusion, physical exercise in young rats appears to have no effect on aortic stiffness. (Hypertension. 2000;35:919-924.)

Key Words: exercise ■ aorta ■ elasticity ■ calcium ■ rats

Medial calcification of the aortic wall leads to the destruction of elastic fibers and an increase in stiffness.1,2 This process can be reversed with pharmacological (chronic treatment with an angiotensin I–converting enzyme inhibitor) and with nonpharmacological (exercise) interventions. Matsuda et al3 showed that forced swimming in rats decreased the calcium content of elastin, increased the elastin content of the aortic wall, and improved the aortic wall elastic properties. Exercise training may not be the only element involved. Forced swimming was used, and other components, such as excitement, prolonged submergence (and hence stimulation of the diving reflex), and the maintenance of temperature homeostasis, may have been involved in the cardiovascular changes that were observed. Treadmill running may be a more suitable model that overcomes these difficulties.4

The objective of this study was therefore to measure aortic wall elasticity and composition in nonanesthetized, freely moving rats after an 8-week period of treadmill running. Maximal O2 uptake (Vo2 max) was measured in the first experiment, and aortic wall stiffness was measured in the second.

Methods

Animals and Training

Six-month-old normotensive male Wistar rats (WAG/Rij; Center d’Etudes Atomiques de Saclay) were divided into 2 groups: 21 untrained rats (mean weight 309±10 g) and 20 trained rats (mean weight 316±11 g). Exercise training consisted of running on a motor-driven treadmill (Gymrol SA) at 30 m/min up a 10% incline for 5 days a week. During the first 4 weeks, the duration of exercise was gradually increased from 10 to 90 min/day and then maintained at this level for an additional 4 weeks. At the end of the training period, 10 rats from each group were used for Vo2 max measurement. The remainder were transferred to the facility in Nancy, where aortic wall mechanics and composition were evaluated. Experiments were performed in accordance with the guidelines of the European Union and the French Ministry of Agriculture.

Maximal Oxygen Uptake

The treadmill used for the measurement of maximal oxygen uptake (Vo2 max) was placed in an airtight Plexiglas chamber. Air was drawn through the system with a vacuum pump; flow rate, which was maintained at 4 L/min, was controlled daily with a gas meter. A smaller pump delivered airflow to an O2 analyzer (model OA 137; Servomex) and a CO2 analyzer (UNOR S.2; Schlumberger) through a flowmeter (200 mL/min). The treadmill exercise protocol consisted of a 3-minute warm-up at 10 m/min on a 0% gradient followed by increases in treadmill speed, gradient, or both every 3 min. Vo2 max was defined as the point at which O2 uptake did not increase with increasing workload or when the rat stopped running.

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Experiments were performed from the third to the fifth day after the end of the training period. Experiments in untrained rats were performed 1 week later.
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Animals were anesthetized with 2% halothane/O2. A femoral venous cannula was implanted for the continuous infusion of the α1-adrenoceptor agonist phenylephrine. Animals were pithed and immediately ventilated with a rodent respirator (1.2 mL/100 g, 50 strokes/min; rodent respirator 601; Harvard Apparatus).

Arterial cannulas were connected to the recording system, and baseline aortic blood pressures and pulse wave velocity were measured. Aortic cannulas were then replaced with 2 Millar Mikro-Tip pressure transducers (SPR 407). Pulse wave velocity was similar in both cases (polyethylene 598±99 cm/s, Millar 573±53 cm/s; P>0.05, n=60 observations). Pulse wave velocity was used for the calculation of 2 indexes of wall stiffness: elastic modulus and isobaric elasticity (see later).

A third index of wall stiffness, aortic pressure wave amplification, was calculated as the ratio of peripheral aortic pulse pressure to central aortic pulse pressure. This reflects the progressive increase in aortic pulse pressure from central to distal sites, with the abdominal aorta being stiffer than the thoracic aorta. Aortic pressure wave amplification decreases with central aortic stiffening.2 Pulse amplification may also be modified simply because of alterations in peripheral wave reflection, independent of changes in stiffness of the central aorta. To distinguish between the 2 mechanisms, we studied pressure transfer function and wave reflection analysis.

A fourth index of wall stiffness was the pressure transfer function. Fourier analysis was performed on the central and peripheral pressure waveforms recorded during a 4-second period at a mean arterial blood pressure of 100 mm Hg. An arbitrary level of 100 mm Hg, which is lower than the mean of the groups, was chosen so that all animals would be included in the analysis. Power spectra were calculated over the first 5 harmonics. Frequency-dependent amplification was determined by dividing the amplitude of the peripheral pressure signal by the amplitude of the corresponding harmonic of the central pressure signal and plotted versus the harmonic. This relationship is shifted upward and to higher frequency values with central aortic stiffening.

The possible impact of wall stiffness on end-systolic pressure was evaluated by measuring wave reflections on the central pressure signal. Ninety-five percent of the pressure waveforms were of the type A as defined by Murog et al,8 and the following parameters were calculated on type A waveforms during a 1-second period (~6 heartbeats) and averaged: (1) the height from the foot of the reflected wave to the systolic peak (ΔP; mm Hg), (2) the augmentation index (the ratio of ΔP to pulse pressure, %), (3) the travel time of the reflected wave (time from the foot of the pressure wave to the shoulder [Δt], ms), and (4) the left ventricular ejection time (time from the foot of the pressure wave to the diastolic incisure [LVET], ms). In a separate experiment (see earlier), the timings of arterial wave reflections were similar whether they were measured with polyethylene cannulas or Millar Mikro-Tip pressure transducers (Δt 30±2 and 35±2 ms, respectively). ΔP was lower when measured with Millar Mikro-Tip pressure transducers (2.0±0.3 mm Hg; P<0.05 versus cannula), but augmentation index was not significantly different, because there was a simultaneous decrease in central aortic pulse pressure (Millar, 13±4 and 22±2 mm Hg, P>0.05 versus cannula, 21±4 and 27±2 mm Hg, for augmentation index and central aortic pulse pressure, respectively).

**Aortic Blood Pressure, Pulse Wave Velocity, and Pulse Amplification in Pithed Rats**

Figure 1. Frequency responses of the Millar Mikro-Tip pressure sensor and polyethylene cannula pressure recording systems (cannulas were previously implanted for 24 hours in a rat aorta), expressed as variations in amplitude (top: A, %) and phase lag (bottom: φ, °). *P<0.05 vs A=100% or φ=0° (ie, vs the Millar Mikro-Tip pressure sensor).

Polyethylene cannulas (0.96/0.58 mm OD/ID) were implanted under 2% halothane/O2 anesthesia, into the descending thoracic aorta, abdominal aorta, and abdominal vena cava. During the 24-hour recovery period, rats lost 4±1% of their body weight. There were 2 deaths after surgery (1 per group).

At 24 hours after catheterization, the aortic cannulas of nonanesthetized, unrestrained rats were filled with heparinized (5 IU/ml), gas-free 0.15 mol/L NaCl solution and connected to a low-volume pressure transducer (Baxter; Bentley Laboratories Europe) via 15 cm of polyethylene cannula. The frequency response of the whole recording systems (3 cannulas, previously implanted for 24 hours in vivo in a rat aorta, filled with gas-free saline solution plus transducer and amplifier) was evaluated up to 30 Hz with a sinusoidal wave generator and compared with that of a Millar Mikro-Tip pressure transducer (0.67 mm, SPR 407; Millar Instruments). The frequency response of the cannula system was flat up to 25 Hz and then slightly underdamped from 25 to 30 Hz (maximal value +7.2±1.2% at 30 Hz, Figure 1, top). Phase lag was slightly but significantly different from 0 (Figure 1, bottom).

The pressure signals were converted into digital form and recorded online at a sampling rate of 256 Hz. After a 30-minute habituation period, baseline parameters were determined on a beat-to-beat basis and averaged over periods of 4 s/min for 1 hour. An algorithm was used to detect the maximal and minimal values of each pressure signal and to calculate the mean aortic blood pressure (mm Hg) on the basis of the waveform area, pulse pressure as the diastolic pressure minus the systolic pressure, and heart rate (bpm) on the basis of a count of the entire number of cycles during the 4-second period.

Pulse wave velocity (cm/s) was calculated as the distance between the 2 cannula tips, measured in situ after postmortem fixation by sticking a damp cotton thread onto the aorta (7.6±0.2 cm [24±1 cm/kg] in untrained rats, 7.5±0.2 cm [24±1 cm/kg] in trained rats, P>0.05), divided by transit time. Transit times (ms) were measured online for each 4-second period (1024 sampling points, 25 heartbeats) with an algorithm that systematically shifted in time the peripheral pressure waveform with respect to the central pressure waveform and determined the value of the time shift that provided the highest correlation. This is based on least-squares analysis of the differences in the amplitudes of the central and peripheral pressure signals at a given point in time; the analysis was repeated following increments of the peripheral sampling points and the creation of intermediate points with linear interpolation. Because the sampling rate was 1/3.9 ms and 10 intermediate points were created, the theoretical resolution of the calculated transit time was 0.059 ms (ie, ±2.5% error for a wave traveling at 455 cm/s). In a separate experiment performed on 3 anesthetized adult male Wistar rats, transit times were determined with the same method after the chronic implantation of polyethylene cannulas, which were then replaced with 2 Millar Mikro-Tip pressure transducers (SPR 407). Pulse wave velocity was similar in both cases (polyethylene 598±99 cm/s, Millar 573±53 cm/s, P>0.05, n=60 observations). Pulse wave velocity was used for the calculation of 2 indexes of wall stiffness: elastic modulus and isobaric elasticity (see later).
measured. Phenylephrine was infused (400 to 800 nmol/kg) so as to raise central aortic mean blood pressure from baseline to the level measured in the nonanesthetized, unrestrained state. Arterial blood pressure and transit time were recorded every 30 seconds for 30 minutes. Values for aortic pulse wave velocity (dependent variable) were expressed as a function of central aortic mean blood pressure (independent variable) according to a linear model; we previously showed that the linear model gives similar results to an exponential model over a range of pressure of 40 to 120 mm Hg (results not shown). “Isobaric elasticity” is defined as the slope relating pulse wave velocity to central aortic mean blood pressure.1,5,9,10

### Aortic Wall Composition and Structure, Wall Stress, Elastic Modulus, and Cardiac Mass

Rats were perfused for 30 minutes at their normotensive central aortic mean blood pressure levels (measured before pithing) with 10% formol containing PBS. A 1-cm sample of the thoracic descending aorta (just below the aortic arch) was excised, immersed in 10% formol, dehydrated in graded ethanol solutions, and embedded in paraffin. Sixteen sections (thickness 20 μm) were stained with hematoxylin-eosin for the determination of aortic internal diameter and medial thickness (Saisam algorithm; Microvision Instruments). Elastic modulus and wall stress (106 dyne/cm2, from the Moens-Korteweg and Lamé equations, respectively, where PWV is baseline pulse wave velocity in awake rats (cm/s), D is internal diameter (cm), h is medial thickness (cm), ρ is blood density (1.05 g/cm3), and CMABP is mean central aortic blood pressure measured in awake rats (dyne/cm2).

A 0.1-cm sample of the abdominal aorta was removed, and the aortic wall content of the elastin-specific cross-linking amino acids desmosine and isodesmosine (μg/g aortic wet wt) was determined with capillary zone electrophoresis and UV detection after acid hydrolysis.11 Tissue calcium content (μmol/g aortic dry wt) was determined on a 0.5-cm sample of the thoracic descending aorta with atomic absorption spectrophotometry (AA10; Varian Ltd) after mineralization and acid digestion of the tissue.12 The heart was removed, and the left ventricle was dissected free and weighed.

### Muscle Enzyme Analysis

The plantaris muscles from both legs were removed from all rats of both series and processed for citrate synthase (EC 4.1.3.7) activity. This enzyme is responsive to endurance exercise and was selected as a marker of the oxidative capacity of the muscle. Tissue samples (10 mg) were homogenized at 4°C in 0.3 mol/L phosphate buffer containing 0.05% bovine serum albumin (pH 7.7) with a glass Potter-Elvehjem homogenizer. The samples were frozen at −80°C and thawed 3 times to disrupt the mitochondrial membrane. Citrate synthase activity (μmol substrate · min−1 · g−1) was measured spectrophotometrically at 30°C according to Sree.13

### Statistics

Values are given as mean±SEM. Linear regression ANOVA was performed with standard parametric techniques, and results are expressed as slopes and intercepts. For any given parameter, missing values per group were ≥2. Differences between groups were evaluated with ANOVA plus the Bonferroni test. A value of P<0.05 was chosen to indicate statistical significance.

### Results

**VO2 max and Muscle Citrate Synthase Activity**

VO2 max increased by 34%, from 83±3 to 111±3 mL O2 · kg−1 · min−1 in control rats to 111±3 mL O2 · kg−1 · min−1 in trained rats (P<0.05). Citrate synthase activity of plantaris muscles in trained rats from the VO2 max series of experiments was increased by 44% (25±2 versus 17±1 μmol · min−1 · g−1 in untrained rats, P<0.05). A similar increase was observed in the series in which aortic mechanics was measured (+39%, from 19±1 μmol · min−1 · g−1 in untrained rats to 26±2 μmol · min−1 · g−1 in trained rats, P<0.05).

<table>
<thead>
<tr>
<th>Group</th>
<th>Untrained Rats (n=10)</th>
<th>Trained Rats (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean aortic blood pressures, mm Hg</td>
<td>110±2</td>
<td>114±1</td>
</tr>
<tr>
<td>Systolic</td>
<td>127±2</td>
<td>130±1</td>
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<tr>
<td>Diastolic</td>
<td>93±2</td>
<td>96±1</td>
</tr>
<tr>
<td>Pulse</td>
<td>34±1</td>
<td>34±1</td>
</tr>
<tr>
<td>Peripheral aortic blood pressures, mm Hg</td>
<td>Mean</td>
<td>107±2</td>
</tr>
<tr>
<td>Systolic</td>
<td>133±2</td>
<td>140±2‡</td>
</tr>
<tr>
<td>Diastolic</td>
<td>88±2</td>
<td>94±1†</td>
</tr>
<tr>
<td>Pulse</td>
<td>44±1*</td>
<td>46±1*</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>341±11</td>
<td>322±14</td>
</tr>
<tr>
<td>Pulse amplification</td>
<td>1.31±0.02</td>
<td>1.35±0.04</td>
</tr>
<tr>
<td>Pulse wave velocity, cm/s</td>
<td>453±13</td>
<td>457±11</td>
</tr>
</tbody>
</table>

*P<0.05, peripheral vs central aortic blood pressures. †P<0.05, trained vs untrained rats.

### Aortic Blood Pressures, Indexes of Aortic Wall Stiffness, Wave Reflection, and Heart Rate in Nonanesthetized, Unrestrained Rats

During the recording period, rats were calm, and the values for aortic blood pressure were stable (intraobserver and interobserver coefficients of variability were <4%). Central aortic blood pressure values were similar in both groups (Table 1). Peripheral aortic mean, systolic, and diastolic blood pressures were significantly increased in trained rats (+6%, +5%, and +7%, respectively); central and peripheral pulse pressures were not modified.

Pulse wave velocity was unchanged after training. Pulse pressure increased by 30% to 35% between central and peripheral aortic recording sites in both groups; pulse amplification values were similar in trained and untrained rats. Frequency-dependent amplification increased up to a maximum at the second harmonic and then decreased at higher harmonics (Figure 2). There were no differences between the 2 groups, and there were no significant differences in intensity or timing of arterial wave reflection (Table 1). The heart rate was unchanged after training.

### Aortic Blood Pressures and Isobaric Elasticity in Phenylephrine-Infused Pithed Rats

Baseline postpithing values for aortic blood pressures, pulse amplification, heart rate, and pulse wave velocity were...
Group Velocity in Pithed Untrained and Trained Rats

Pressures; Heart Rate; Pulse Amplification; and Pulse Wave

TABLE 2. Baseline Aortic Mean, Systolic, Diastolic, and Pulse

and wall thickness/internal diameter ratio) was similar in the

Tableau aortic geometry (internal diameter, wall thickness,

The continuous infusion of phenylephrine produced a

The 4 indicators of aortic elasticity (elastic modulus, pulse wave amplification, pressure transfer function, and isobaric elasticity) were similar in trained and untrained rats. The relationship between elastic modulus and wall stress was unchanged.

Aortic Wall Composition and Structure, Wall Stress, Elastic Modulus; and Cardiac and Left Ventricular Mass in Untrained and Trained Rats

TABLE 3. Body Weight; Thoracic Aortic Wall Geometry and Composition, Wall Stress, Elastic Modulus; and Cardiac and Left Ventricular Mass in Untrained and Trained Rats

Figure 2. Amplification of pressure wave harmonics along the aorta in nonanesthetized, freely moving untrained (●) and trained (○) rats.

significantly lower than those measured before pithing in all

2 groups (Table 3), as were wall stress and elastic modulus. There was a significant linear relationship between elastic modulus (dependent variable) and wall stress (independent variable) that was similar in both groups (slopes 2.7±0.8 and 2.0±0.7, intercepts 0.22±0.96×10⁶ and 0.98±0.79×10⁶
dyne/cm² in trained and untrained rats, respectively). Physical training had no effect on aortic calcium or desmosine-plus-isodesmosine content. Neither cardiac nor left ventricular mass was significantly modified with physical training.

Discussion

Aortic Wall Structure and Elasticity

Training had no effect on aortic dimensions (length, internal diameter, or wall thickness) or on wall elastin content (as judged from the lack of change in the content of desmosines) or calcium content. The 4 indicators of aortic elasticity (elastic modulus, pulse wave amplification, pressure transfer function, and isobaric elasticity) were similar in trained and untrained rats. The relationship between elastic modulus and wall stress was unchanged.

These results do not confirm the hypothesis of Matsuda et al (see the introduction). The length of the training period in their experiments (16 weeks) was longer than that of the present study (8 weeks). In the present study, an 8-week period of treadmill running was used because such a forced running program is used to induce modifications of the skeletal muscle function and aerobic capacity in our laboratory.14,15 Albeit, changes in the structure and mechanical properties of central arteries can occur relatively rapidly and over a time span similar to that of the present study. Structural and functional arterial alterations can develop in ≈2 months after the induction of hypertension16 or elastocalcinosis,1,2 and these alterations can be reversed equally rapidly.2,16 Furthermore, our training program produced marked increases in VO₂ max (+34%) and skeletal muscle citrate synthase activity. These increases are similar to those obtained in previous studies, showing that aerobic capacity of the skeletal muscle is increased after the same training program14 and that

<table>
<thead>
<tr>
<th>Group</th>
<th>Untrained Rats (n=10)</th>
<th>Trained Rats (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight, g</td>
<td>319±8</td>
<td>319±10</td>
</tr>
<tr>
<td>Thoracic aortic geometry</td>
<td></td>
<td></td>
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<tr>
<td>Internal diameter, mm</td>
<td>1.35±0.04</td>
<td>1.40±0.05</td>
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<tr>
<td>Medial thickness, µm</td>
<td>95±6</td>
<td>87±4</td>
</tr>
<tr>
<td>Medial thickness/internal diameter</td>
<td>0.071±0.005</td>
<td>0.063±0.005</td>
</tr>
<tr>
<td>Thoracic aortic wall composition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium content, µmol/g</td>
<td>19±1</td>
<td>21±2</td>
</tr>
<tr>
<td>Desmosine + isodesmosine, µg/g</td>
<td>694±88</td>
<td>876±75</td>
</tr>
<tr>
<td>Wall stress, 10⁶ dyne/cm²</td>
<td>1.1±0.1</td>
<td>1.2±0.1</td>
</tr>
<tr>
<td>Elastic modulus, 10⁶ dyne/cm²</td>
<td>3.2±0.3</td>
<td>3.5±0.3</td>
</tr>
<tr>
<td>Cardiac mass, g</td>
<td>0.90±0.02</td>
<td>0.92±0.03</td>
</tr>
<tr>
<td>Left ventricular mass, g</td>
<td>0.69±0.01</td>
<td>0.70±0.02</td>
</tr>
</tbody>
</table>

*P<0.05, trained vs untrained rats.
such a program is able to modify the mRNA expression of uncoupled proteins related to energy dissipation in skeletal and heart muscles.\textsuperscript{15}

A more plausible explanation for the difference between our results and those of Matsuda et al\textsuperscript{3} resides in their use of forced swimming as exercise training. As stated in the introduction, such a program has elements other than physical training that may induce cardiovascular changes. Moreover, Matsuda et al\textsuperscript{3} also reported results obtained after 16 weeks of forced running (at 30 m/min, 60 min/day, 6 d/wk), and in this case, as in the present study, the calcium content of elastin, the elastin content, and the elastic properties of the aortic wall were unaltered.\textsuperscript{3} All of these observations lead to the conclusion that a forced running program, as currently used to evaluate skeletal muscle conditioning and adaptation of aerobic capacity, does not modify aortic function and mechanics, at least not in young animals.

The lack of an effect of physical training on elasticity and the structure of large elastic arteries agrees with some reports in young or adult humans\textsuperscript{17,18} but not all.\textsuperscript{19,20} In the latter reports, however, because athletes were compared with untrained subjects, subjects with certain genetically determined vascular features may be selected out of the general population. Thus, a greater aortic elasticity may be causal rather than an adaptive response to physical exercise.

Cardiovascular Fitness

In the present study, physical exercise did not improve cardiovascular fitness; heart rate and central aortic blood pressures were not changed in trained awake rats. In the pithed preparation, both central and peripheral aortic mean blood pressures increased after physical training. This suggests that physical training produced an increase in peripheral resistance, because perfusion pressure at maximal dilation (which is the case in the pithed rat) is directly related to the wall-to-lumen ratio of resistance vessels.\textsuperscript{21} This merits further investigation because although some reports suggest that physical training produces a decrease in vascular resistance,\textsuperscript{22} other reports do not.\textsuperscript{23} In the unanesthetized baseline condition, peripheral aortic mean and diastolic pressures were also greater in trained rats. Because aortic dimensions were unaltered, the drop in mean pressure along the aorta was therefore smaller in trained rats. This may indicate a smaller cardiac output in trained rats. In the light of this observation, the lack of resting bradycardia after exercise may represent an adaptation to maintain stroke volume. Gleeson et al\textsuperscript{24} used a program that increases VO\textsubscript{2 max} by 16% and citrate synthase activity by 38% and found no resting bradycardia in young rats. In older animals (18-month-old normotensive WAG/Rij rats), we observed that 6 weeks’ treadmill running induced resting bradycardia (377 ± 30 bpm in awake trained rats [n=4], P<0.05 versus 442 ± 3 bpm in control rats [n=6]) and lowered central aortic pulse pressure (36±4 mm Hg in trained rats, P<0.05 versus 48±3 mm Hg in control rats) with no change in the elastic properties of the aortic wall (elastic modulus 8.6±4.7 10\textsuperscript{6} dyne/cm\textsuperscript{2} in trained rats, P>0.05 versus 3.4±0.5 10\textsuperscript{6} dyne/cm\textsuperscript{2} in control rats). This pattern of reduced heart rate and pulse pressure with no change in elasticity suggests a fall in stroke volume.

Finally, our physical training did not induce cardiac hypertrophy. Some authors have reported an increase in cardiac or left ventricular mass, or both, relative to body weight after physical training.\textsuperscript{25,26} This has not been confirmed by others.\textsuperscript{24} It has been suggested that this may be due to a substantial decrease in body weight with relatively little change in cardiac mass.\textsuperscript{4,27}

Wave reflections were similar in trained and untrained rats. It should be noted, however, that in 95% of waveforms of the central aortic pulse pressure contour in the untrained rat are characterized by rapid wave reflection and a large augmentation index such that the wave is of type A as defined by Murgo et al.\textsuperscript{8} Therefore, modification of the reflected wave may be difficult to obtain in rats. One also cannot exclude the possibility that the pressure recording system that was used does not allow accurate detection of the inflection point, which requires the presence of at least the seventh or eighth harmonic (ie, a frequency component of ≈50 Hz). If a second-order underdamped response of the pressure recording system is assumed (as predicted by the frequency response measured up to 30 Hz), the components at 50 Hz may well be attenuated, and this may preclude accurate detection of the inflection point, and explain (not statistically significant) differences in augmentation index values obtained with the polyethylene cannula or Mikro-Tip transducer. All of these arguments may temper our comments regarding wave reflection on the basis of the augmentation index calculation.

In conclusion, a forced running program that conditions skeletal muscle and increases aerobic capacity does not modify aortic mechanics or improve cardiovascular fitness in young rats.

Acknowledgments

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