Functional and Metabolic Evaluation of the Athlete’s Heart By Magnetic Resonance Imaging and Dobutamine Stress Magnetic Resonance Spectroscopy

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Background—The question of whether training-induced left ventricular hypertrophy in athletes is a physiological rather than a pathophysiological phenomenon remains unresolved. The purpose of the present study was to detect any abnormalities in cardiac function in hypertrophic hearts of elite cyclists and to examine the response of myocardial high-energy phosphate metabolism to high workloads induced by atropine-dobutamine stress.

Methods and Results—We studied 21 elite cyclists and 12 healthy control subjects. Left ventricular mass, volume, and function were determined by cine MRI. Myocardial high-energy phosphates were examined by 31P magnetic resonance spectroscopy. There were no significant differences between cyclists and control subjects for left ventricular ejection fraction (59 ± 5% versus 61 ± 4%), left ventricular cardiac index (3.4 ± 0.4 versus 3.4 ± 0.4 L · min⁻¹ · m⁻²), peak early filling rate (562 ± 93 versus 535 ± 81 mL/s), peak atrial filling rate (315 ± 93 versus 333 ± 65 mL/s), ratio of early and atrial filling volumes (3.0 ± 1.0 versus 2.6 ± 0.6), mean acceleration gradient of early filling (5.2 ± 1.4 versus 5.8 ± 1.9 L/s²), mean deceleration gradient of early filling (−3.1 ± 0.9 versus −3.2 ± 0.7 L/s²), mean acceleration gradient of atrial filling (3.6 ± 1.8 versus 4.5 ± 1.7 L/s²), and atrial filling fraction (0.23 ± 0.06 versus 0.26 ± 0.04, respectively). Cyclists and control subjects showed similar decreases in the ratio of myocardial phosphocreatine to ATP measured with 31P magnetic resonance spectroscopy during atropine-dobutamine stress (1.41 ± 0.20 versus 1.41 ± 0.18 at rest to 1.21 ± 0.20 versus 1.16 ± 0.13 during stress, both P=NS).

Conclusions—Left ventricular hypertrophy in cyclists is not associated with significant abnormalities of cardiac function or metabolism as assessed by MRI and spectroscopy. These observations suggest that training-induced left ventricular hypertrophy in cyclists is predominantly a physiological phenomenon. (Circulation. 1998;97:666-672.)

Key Words: hypertrophy n magnetic resonance imaging n metabolism n spectroscopy n stress

Athlete’s heart is the term used for training-induced left ventricular (LV) hypertrophy in athletes.1–3 The question of whether the enlarged heart of athletes is a purely physiological phenomenon or should be considered a risk factor comparable to pathological LV hypertrophy induced by hypertension or hypertrophic cardiomyopathy is still unresolved.4–6 Concern has been raised by the reports of sudden deaths occurring in athletes, possibly related to LV hypertrophy and impaired LV filling.7,8 Echocardiographic studies have demonstrated reduced diastolic properties in several older elite cyclists, suggesting that in the long run, extreme physical training may have a negative effect on the heart, manifested by partly irreversible LV hypertrophy and impaired LV filling.9,10 Previous studies of the morphological and functional aspects of the athlete’s heart used echocardiography.11–13 In recent years, magnetic resonance techniques with the capability of assessing cardiac mass, function, and metabolism (31P magnetic resonance spectroscopy) became available.14–16 A recent study from our institution demonstrated normal systolic function parameters and a normal high-energy phosphate metabolism in elite cyclists at rest.17 In that study, however, we did not investigate diastolic function or high-energy phosphate metabolism during stress. Because abnormalities of diastolic filling may precede abnormalities of systolic function in the early stages of disease, we considered it important to evaluate diastolic function in cyclists as well.18 In addition, we acquired 31P magnetic resonance spectra at rest and during atropine-dobutamine stress conditions. The aim of the present study was (1) to detect the presence of abnormalities in systolic and diastolic function at rest in the hypertrophic hearts of elite cyclists and (2) to investigate the response of myocardial high-energy phosphate metabolism in these hearts to high workloads induced by...
atropine-dobutamine stress. To this purpose, we used MRI and 31P magnetic resonance spectroscopy.

Methods

Study Population
The study group consisted of 21 male elite cyclists (mean age, 42±8 years; range, 28 to 52 years) and 12 male healthy volunteers (mean age, 47±8 years; range, 33 to 56 years) matched for height and weight. The athletes cycled 12,000 km/y; their duration of athletic history was 23±7 years (range, 13 to 35 years). The 12 control subjects were healthy individuals, none of whom were engaged in sporting activities other than recreation. All individuals were free of known cardiovascular disease and were normal by physical examination. They were all nonsmoking, normotensive, and had a negative family history of coronary heart disease. A standard 12-lead ECG was recorded at rest, which was normal in the control subjects. In 14 cyclists (67%), the resting ECG met the Sokolow-Lyon voltage criteria of LV hypertrophy (sum of amplitudes of S wave in V1 and R wave in V5 or V6 >3.5 mV). The study was approved by the Human Research Committee at our institution, and all individuals gave informed consent.

Magnetic Resonance Imaging

Anatomic and Functional Assessment
MRI was performed with a Philips Gyroscan ACS–NT system (Philips Medical Systems) operating at 1.5 T and ECG gating. Magnetic resonance images were acquired with breath-hold multishot echoplanar imaging as described previously.19 Flow-Velocity Measurements
Volume flow was assessed with magnetic resonance velocity mapping of flow across the mitral orifice and through the aorta.20 Velocity maps were acquired with a flip angle of 30°, an echo time of 10 to 12 ms, a section thickness of 8 mm, a field of view of 350 mm, and two measurements of a 128×128 pixel acquisition matrix. The number of time frames varied according to heart rate, resulting in a temporal resolution of 25 to 30 ms. The maximum phase shift of 180° was set to occur with a velocity of 100 cm/s for mitral flow and 150 cm/s for aortic flow. Velocity aliasing was not encountered. To acquire data during late diastole, velocity mapping was performed with retrospective gating. Data were collected continuously during the cardiac cycle. Retrospectively, each measurement was attributed to the point in time of the cardiac cycle it represented, with the simultaneously recorded ECG used as a reference. From the recorded data, a series of velocity maps was reconstructed that represented an average cardiac cycle during the acquisition interval.

Magnetic Resonance Image Analysis

Anatomic and Functional Assessment
Multislice, multiphase, short-axis image analysis was performed with the MR analytical software system MASS21 and a SUN IPX workstation (Sun Microsystems Computer Corp). End-diastolic and end-systolic epicardial and endocardial contours of the stack of short-axis image sections were traced manually. Papillary muscles were outlined and included in the LV wall. LV mass and LV ejection fraction were calculated as described previously by our institution.22 Flow-Velocity Measurements
Volume flow was calculated by tracing a region of interest along the borders of the mitral valve and the borders of the aorta in all time frames of a velocity map series. For each time frame, instantaneous volume flow was calculated by a computer algorithm by multiplying spatial average flow velocity and the area of the region of interest. Summation of all instantaneous volume flow data yielded total flow per cardiac cycle. All measurements were performed twice by one observer on two separate occasions at least 2 weeks apart and once by a second observer. The following parameters of diastolic function were measured or derived (Fig 1): LV stroke volume (mL), peak early filling rate (mL/s), early filling volume (mL), peak atrial filling rate (mL/s), atrial filling volume equals area under second peak (mL); atrial filling time is duration of second peak (ms); and atrial filling fraction is flow volume during atrial filling (area under second peak) divided by stroke volume (total area under curve). B, Schematic transmural filling pattern in a cyclist. Mean acceleration of early filling is indicated by α; mean deceleration of early filling by β; and mean acceleration of atrial contraction by γ.

31P Magnetic Resonance Spectroscopy
Highly reproducible 31P magnetic resonance spectra of the anterior wall of the left ventricle were acquired at rest and during atropine-dobutamine stress in 18 cyclists and 11 control subjects.23 A 1.5-T Gyroscan S15 (Philips Medical Systems) was used, with a 10-minute, three-dimensional, image-selected in vivo spectroscopy protocol as described previously.24 31P magnetic resonance spectra were transferred to a remote SUN-SPARC workstation to be quantified automatically by model function analysis in the time domain, with a priori spectroscopic knowledge used to improve the accuracy of the spectral parameters.24 The ATP level in the spectra was corrected for the relative contribution of ATP present in the blood.25 Signal modeling, prior knowledge, and correction for partial saturation effects were applied as in a previous study.25

Atropine-Dobutamine Infusion Protocol
After the baseline spectrum was recorded, 0.03 mg/kg atropine sulfate was administered in three separate doses of 0.01 mg/kg to achieve complete cholinergic blockade.26 Thereafter, myocardial stress was induced by administration of incremental intravenous doses of dobutamine, which increases myocardial oxygen consumption by positive
TABLE 1. Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Cyclists</th>
<th>Control Subjects</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>21</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>42±8</td>
<td>47±8</td>
<td>.14</td>
</tr>
<tr>
<td>Height, cm</td>
<td>181±6</td>
<td>182±7</td>
<td>.77</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>77±8</td>
<td>76±10</td>
<td>.73</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>2.0±0.1</td>
<td>2.0±0.2</td>
<td>.73</td>
</tr>
<tr>
<td>DBPrest, mm Hg</td>
<td>71±9</td>
<td>72±5</td>
<td>.61</td>
</tr>
<tr>
<td>SBPrest, mm Hg</td>
<td>122±13</td>
<td>121±11</td>
<td>.83</td>
</tr>
<tr>
<td>Training, km/y</td>
<td>12 400±0.8</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>Training history, y</td>
<td>23±7</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>Resting heart rate, bpm</td>
<td>52±6</td>
<td>61±4</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

BSA indicates body surface area; DBP, diastolic blood pressure; and SBP, systolic blood pressure.

Results

Subject Characteristics

The anthropometric characteristics of the 21 cyclists and 12 control subjects are presented in Table 1. Athletes and control subjects were similar with regard to anthropometric parameters such as age, height, body mass, and body surface area. Both groups had similar blood pressures under resting conditions. Only resting heart rate was significantly lower in cyclists than in control subjects (52±6 versus 61±4 bpm, P<.0001).

LV Anatomy and Function

LV anatomic and functional parameters of the 21 cyclists and 12 control subjects are presented in Table 2. Cyclists had a 45% increase in LV mass compared with control subjects (P<.001). In addition, indexed LV mass, absolute and indexed LV end-diastolic volume, and ratio of LV mass to volume were also significantly increased in cyclists compared with control subjects (all P<.001). Also, LV stroke index was significantly larger in cyclists than in control subjects (65±7 versus 56±6 mL/m² P<.005), but because of the significantly lower heart rate of the cyclists, this did not result in a significantly increased cardiac index under resting conditions. There was no significant difference in LV ejection fraction between the two groups (59±5 in cyclists versus 61±4% in control subjects).

Flow Velocity Measurements

LV diastolic function parameters are presented in Table 3 and Fig 1. There were no significant differences between cyclists and control subjects in peak early filling rate, peak atrial filling rate, early filling volume, atrial filling volume, ratio of early to atrial filling volumes, mean acceleration and mean deceleration gradients of early filling, mean acceleration gradient of atrial filling, and atrial filling fraction. The peak ratio of the early to atrial filling rates was significantly higher in athletes than in control subjects (1.9±0.5 versus 1.6±0.2, P=.043), but after

TABLE 2. Left Ventricular Anatomic and Functional Parameters

<table>
<thead>
<tr>
<th></th>
<th>Cyclists</th>
<th>Control Subjects</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>21</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>LV mass, g</td>
<td>200±23</td>
<td>138±14</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LV mass index, g/m²</td>
<td>102±10</td>
<td>69±6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LV end-diastolic volume, mL</td>
<td>222±32</td>
<td>184±21</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LV end-diastolic volume index, mL/m²</td>
<td>112±14</td>
<td>93±9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LV mass-to-volume ratio, g/mL</td>
<td>0.9±0.08</td>
<td>0.75±0.03</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LV stroke index, mL/m²</td>
<td>65±7</td>
<td>56±6</td>
<td>.004</td>
</tr>
<tr>
<td>LV cardiac index, L·min⁻¹·m⁻²</td>
<td>3.4±0.4</td>
<td>3.4±0.4</td>
<td>.77</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>59±5</td>
<td>61±4</td>
<td>.25</td>
</tr>
</tbody>
</table>

LV indicates left ventricular.

TABLE 3. Flow Velocity Measurements

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Cyclists</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak early filling rate, mL/s</td>
<td>562±93</td>
<td>535±81</td>
<td>.41</td>
</tr>
<tr>
<td>Peak atrial filling rate, mL/s</td>
<td>315±93</td>
<td>333±65</td>
<td>.54</td>
</tr>
<tr>
<td>Ratio of peak early and atrial filling rate</td>
<td>1.9±0.5</td>
<td>1.6±0.2</td>
<td>.043</td>
</tr>
<tr>
<td>Early filling volume, mL</td>
<td>82±14</td>
<td>73±11</td>
<td>.056</td>
</tr>
<tr>
<td>Atrial filling volume, mL</td>
<td>29±8</td>
<td>29±6</td>
<td>.94</td>
</tr>
<tr>
<td>Ratio of early and atrial filling volume</td>
<td>3.0±1.0</td>
<td>2.6±0.6</td>
<td>.14</td>
</tr>
<tr>
<td>Left ventricular stroke volume, mL</td>
<td>124±18</td>
<td>109±11</td>
<td>.005</td>
</tr>
<tr>
<td>Atrial filling fraction, mL/mL</td>
<td>0.23±0.06</td>
<td>0.26±0.04</td>
<td>.14</td>
</tr>
<tr>
<td>Mean acceleration gradient of early filling, L/s²</td>
<td>5.2±1.4</td>
<td>5.8±1.9</td>
<td>.32</td>
</tr>
<tr>
<td>Mean deceleration gradient of early filling, L/s²</td>
<td>-3.1±0.9</td>
<td>-3.2±0.7</td>
<td>.81</td>
</tr>
<tr>
<td>Mean acceleration gradient of atrial filling, L/s²</td>
<td>3.6±1.8</td>
<td>4.5±1.7</td>
<td>.17</td>
</tr>
<tr>
<td>Early filling time, ms</td>
<td>311±40</td>
<td>284±48</td>
<td>.08</td>
</tr>
<tr>
<td>Atrial filling time, ms</td>
<td>177±27</td>
<td>163±26</td>
<td>.18</td>
</tr>
</tbody>
</table>

See also Fig 1 for explanation.
correction for age and heart rate, this difference was no longer significant ($P=.23$).

**Hemodynamic Data**

The responses of the hemodynamic variables to the administration of atropine-dobutamine are shown in Table 4. Significant increases in heart rate, blood pressure, and rate-pressure product were observed in cyclists and control subjects ($P<.001$). There was no significant difference in rate-pressure product at rest between the two groups. However, during stress, the rate-pressure product of the cyclists was significantly higher than that of control subjects ($25.9\pm2.4\times10^3$ versus $23.3\pm3.0\times10^3$ mm Hg/min, $P=.016$).

**Metabolic Data**

The myocardial phosphocreatine (PCr)/ATP ratio decreased significantly during atropine-dobutamine stress in athletes and in control subjects (Table 4). In cyclists, the PCr/ATP ratio decreased from $1.41\pm0.20$ to $1.21\pm0.20$ ($P<.001$); in control subjects, the PCr/ATP ratio diminished from $1.41\pm0.18$ to $1.16\pm0.13$ ($P<.001$). The decrease, however, was similar in both groups ($0.20\pm0.21$ versus $0.24\pm0.15$, $P=NS$). Examples of in vivo $^{31}$P magnetic resonance spectra acquired at rest and during atropine-dobutamine stress in a cyclist and a healthy volunteer are shown in Fig 2.

**Intraobserver and Interobserver Variability in Mitral Flow Measurements**

Intraobserver variability for the mitral flow measurements, representing LV stroke volume, was 4.8%. The interobserver variability was slightly higher (5.9%).

**Discussion**

The primary aims of our study were to assess systolic and diastolic function at rest in the hypertrophic hearts of highly trained cyclists and to investigate the response of myocardial high-energy phosphate metabolism during pharmacological stress. To this purpose, we used MRI, velocity mapping, and $^{31}$P magnetic resonance spectroscopy.

The main observation of the present study was the demonstration that highly trained cyclists with LV hypertrophy have normal LV systolic and diastolic properties. In addition, we found a similar decrease in myocardial PCr/ATP ratio during high workloads caused by atropine-dobutamine stress in cyclists and sedentary control subjects. This study extends our previous findings in cyclists, in whom we demonstrated a

**TABLE 4. Hemodynamic and Metabolic Data in 18 Cyclists and 11 Control Subjects During Atropine-Dobutamine Stress**

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Stress</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, bpm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyclists</td>
<td>$53\pm7$</td>
<td>143$\pm9$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Controls</td>
<td>$61\pm5$</td>
<td>147$\pm10$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>$P$</td>
<td>&lt;.001</td>
<td>.30</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyclists</td>
<td>$122\pm13$</td>
<td>181$\pm18$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Controls</td>
<td>$121\pm11$</td>
<td>158$\pm18$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>$P$</td>
<td>.83</td>
<td>.002</td>
<td></td>
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<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyclists</td>
<td>$71\pm10$</td>
<td>85$\pm11$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Controls</td>
<td>$72\pm5$</td>
<td>78$\pm12$</td>
<td>.17</td>
</tr>
<tr>
<td>$P$</td>
<td>.60</td>
<td>.10</td>
<td></td>
</tr>
<tr>
<td>Rate-pressure product, mm Hg/min$\times10^3$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyclists</td>
<td>$6.5\pm1.3$</td>
<td>25.9$\pm2.4$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Controls</td>
<td>$7.3\pm1.0$</td>
<td>23.3$\pm3.0$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>$P$</td>
<td>.076</td>
<td>.016</td>
<td></td>
</tr>
<tr>
<td>PCr/ATP ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>$1.41\pm0.18$</td>
<td>1.21$\pm0.20$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cyclists</td>
<td>$1.41\pm0.20$</td>
<td>1.16$\pm0.13$</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>$P$</td>
<td>1.00</td>
<td>.44</td>
<td></td>
</tr>
</tbody>
</table>
normal systolic function and normal cardiac metabolism at rest. The present findings therefore support the concept that LV hypertrophy in cyclists is a physiological adaptation without significant adverse effects on cardiac function and metabolism.

Physiological Versus Pathological LV Hypertrophy

Intense long-term physical training is well known to cause LV hypertrophy. Endurance cycling ranks among the type of sports with the highest impact on LV mass. In the present study, a substantial increase in LV mass was found in cyclists compared with control subjects (45%), which is in agreement with previous magnetic resonance and echocardiographic studies on athlete’s heart. Whether the athlete’s heart carries an increased risk for cardiovascular events remains an intriguing clinical problem, because LV hypertrophy in the general population has been shown to be an independent risk factor for cardiovascular morbidity and sudden cardiac death. An early characteristic of LV hypertrophy resulting from disease states such as hypertension, hypertrophic cardiomyopathy, and other hypertrophic conditions is a change in diastolic properties. Diastolic dysfunction can therefore serve as a sensitive and early indicator of disease and may be used to differentiate between physiological and pathological hypertrophy. In addition, profound changes in cardiac metabolism may discriminate between physiological and pathological hypertrophy.

Diastolic Function

LV diastolic function is commonly assessed by study of the pattern of ventricular filling through the mitral valve. Doppler echocardiography is generally regarded as the noninvasive technique of choice for the assessment of ventricular filling because of its clinical utility, wide availability, and low cost. Cine magnetic resonance velocity mapping is a relatively new method that may add important information to Doppler echocardiographic data, because flow is obtained three-dimensionally. It therefore makes possible the calculation of both average velocity and flow in the great arteries and across cardiac valves. This method has been validated previously at our institution. Manifestations of abnormal diastolic relaxation are, among other things, a diminished extent and rate of LV filling in early diastole, an increased contribution of atrial contraction to diastolic filling, and a decreased ratio of flow in the early rapid filling phase to that in the atrial contraction phase.

However, interpretation of these measurements can be difficult, because they are influenced not only by changes in the intrinsic LV diastolic properties but also by factors such as age, heart rate, body mass index, blood pressure, valvular disease, loading conditions, and contractility of both ventricular and atrial myocardium. Conclusions about LV diastolic function should therefore be made only after correction for these parameters.

In our study, the differences in blood pressure or body mass index between the two groups of subjects were negligible. The cyclists had a significantly lower resting heart rate and were slightly younger than the control subjects, although this difference in age did not reach statistical significance.

With advancing age, the relative contribution of early diastolic filling diminishes, whereas the atrial filling becomes more prominent, resulting in a decreased ratio of early to atrial filling. With increasing heart rate, the diastolic filling pattern changes in favor of atrial contribution, together with a decrease of the velocity area of the early diastolic filling time and a decrease of the deceleration time, resulting in a lower ratio of the early to late peak filling rates and a lower ratio of early to late filling volumes.

Our study found a significantly higher ratio of peak early to atrial filling volumes in cyclists compared with control subjects, corresponding to slightly altered LV diastolic properties in cyclists. However, after correction for age and heart rate, the cyclists demonstrated a normal diastolic filling pattern, with no significant differences in parameters such as peak early filling rate, peak atrial filling rate, early filling volume, atrial filling volume, ratio of early to atrial filling volumes, mean acceleration gradient of early filling, mean deceleration gradient of early filling, mean acceleration gradient of atrial filling, and atrial filling fraction. Also, there was no significant difference in LV ejection fraction between cyclists and control subjects. These data indicate that LV hypertrophy in cyclists is not associated with significant changes in systolic or diastolic LV function. We could not confirm the findings of Nishimura et al., who found significantly depressed resting systolic LV function in 40- to 49-year-old cyclists, or the findings of Miki et al., who demonstrated a decreased ratio of early to atrial filling velocities in 40- to 60-year-old cyclists. Our findings are, however, in agreement with other previous Doppler echocardiographic studies in cyclists and other athletes that demonstrated normal or enhanced LV function parameters. To the best of our knowledge, our study is the first to use MRI in the assessment of LV systolic and diastolic function in the athlete’s heart.

Myocardial Metabolism With Increased Work State

31P magnetic resonance spectroscopy can be used to study myocardial metabolism noninvasively by measuring the relative proportions of high-energy phosphates. Abnormalities of high-energy phosphate metabolism have been described in studies of failing hypertrophied human hearts and in animal hearts with severe LV hypertrophy. However, imbalances in energy production and utilization in less severely hypertrophied hearts may become apparent only under stress conditions. In addition, existing abnormalities have been observed to become manifest during stress, with larger changes in hypertrophied than in normal hearts.

Compared with control subjects, the cyclists of the present study showed a similar decrease in myocardial PCr/ATP ratio in response to a more than threefold increment in rate-pressure product caused by atropine-dobutamine infusion. This finding argues against a greater vulnerability to myocardial ischemia in the athlete’s heart than in control hearts under stress conditions. This lack of evidence for stress-induced ischemia in the athlete’s heart is particularly noteworthy because the cyclists achieved an even higher rate-pressure product than the control subjects. A decline in myocardial PCr/ATP ratio with a threefold increment in rate-pressure product has not been observed previously in normal human hearts. The increase in rate-pressure product from rest to stress was 1.3 in the study
of Conway et al. and was not fully described by Kuno et al. The results obtained in our control group are consistent with previous experimental studies regarding myocardial responses to increased stress induced by dobutamine infusion. This consistency provides further evidence that the metabolic changes observed in the present study are more closely related to work state than to LV hypertrophy.

Potential Limitations

Exercise-induced cardiac stress is the most physiological and, therefore, most desirable form of stress. However, spectral acquisition during exercise-induced cardiac stress is subject to several limitations, such as motion artifacts due to movements and increased respiration of the patient and the low level of exercise presently attainable. Pharmacological stress appears to be the most suitable alternative for conventional exercise, because it is characterized by absence of motion artifacts induced by patient motion and excessive ventilation during exercise.

A relative drawback of spectroscopy is the relatively large voxel size required for an adequate signal (60\(\times\)70\(\times\)70 mm\(^3\)). Although we carefully avoided inclusion of skeletal muscle, diaphragm, or liver tissue, we could not completely avoid contamination of the voxel by contribution of blood. Blood correction was performed, because blood contains only ATP and no PCr, which may therefore alter the observed PCr/ATP ratio. However, voxels obtained from hypertrophied hearts are expected to be less contaminated with blood because of increased myocardial wall thickness in the athletes.

Last, we can of course not fully exclude the presence of coronary artery disease in our population, with a mean age of 42 years in the cyclists and a mean age of 47 years in the control subjects. However, because of complete absence of any cardiac complaints at rest and during exercise and the lack of major coronary risk factors, the estimated prevalence of coronary artery disease is \(\leq 5.5\%\) in both groups of subjects.

Conclusions

LV hypertrophy in cyclists is not associated with significant abnormalities of systolic or diastolic function. During dobutamine-atropine stress, the PCr/ATP ratio determined in the LV myocardium of cyclists decreased, but this decrease was smaller than the decrease observed in healthy volunteers. These findings demonstrate that strenuous training is not associated with pathological changes in cardiac function or myocardial high-energy phosphate metabolism, suggesting that the athlete’s heart is predominantly a physiological adaptation of the heart without negative consequences.

Acknowledgments

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MRI Evaluation of Athlete’s Heart


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