Evaluation of Left Ventricular Global and Regional Myocardial Work Quantitatively by Pressure-strain Loop in Young Strength Athletes With Different Heart Rates

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Abstract

Objective:

The present study aimed to investigate the difference in left ventricular (LV) global and regional myocardial work (MW) of strength athletes with different heart rates (HR) through non-invasive LV pressure-strain loop (PSL) and further address the effect of athlete's resting heart rate variability on the LV systolic function.

Methods:

Fifty young professional wrestlers were collected randomly and divided into two groups in accordance with their different heart rates: the low HR group (45~60 bpm, \(n_1=25\)) and the high HR group (60~80 bpm, \(n_2=25\)). Thirty individuals with gender- and age-matched healthy volunteers served as controls (\(n_3=30\)). Global and regional MW parameters were evaluated using LV-PSL derived from speckle tracking echocardiography (STE) and brachial artery pressure. The differences between three groups of data and the linear relationship among conventional echocardiographic parameters and global myocardial work efficiency (GWE) were analyzed.

Results:

The indicators of global and regional MW did statistically significantly differ between the athlete and control groups. Compared with the control group, Peak strain dispersion (PSD) and global myocardial wasted work (GWW) increased while GWE reduced all in the low HR and high HR groups, and global myocardial work index (GWI), global myocardial constructive work (GCW), global longitudinal strain (GLS) decreased in the low HR group (all \(P<0.05\)). In comparison to the low HR group, GWI, GCW, GWW, PSD increased in the high HR group and GWE reduced (\(P<0.05\)). According to the regional MW analysis, the mean regional myocardial work index (RMWI) was increased gradually from basal to apical levels, which were similar across the three groups, but regional myocardial work efficiency (RMWE) did not. Multiple linear regression analysis indicated that the HR, posterior wall thickness (PWT), interventricular septal thickness (IVST), GLS, and PSD were well correlated with GWE (\(b=-0.247, -0.390, -0.370, 0.340, \) and -0.554, respectively, \(P<0.05\)).

Conclusions:

The sub-clinical changes of LV contractile performance were more obvious in young strength athletes with high resting heart rates and PSL could be used to assess LV GMW and RMW quantitatively and accurately in reflecting LV systolic function.

Introduction

Continuous exercise with various levels of training intensity and burden was typically termed as “Athlete's heart”, which might be subjected to morphological, functional, and electrophysiological myocardial...
adaptations and even increase the risk of adverse cardiovascular-related outcomes[1]. Left ventricular phenotype differences were largely determined by exercise type and training time, and of which strength athletes were performed as wall thickening, cardiac chamber dilation and so on. Additional change that the resting heart rate is lower of athletes than that of normal subjects could be easily observed[2]. Previous studies described that the resting heart rates of athletes were generally 45 ~ 80 beats/min, and even less than 30 beats/min in some elite ones[3]. String correlation exists between heart rate and exercise intensity, body oxygen uptake and energy metabolism[4], whereas the influence of resting heart rate on the changes of athlete's cardiac function is still unclear.

Compared with cardiac catheterization, computed tomography angiography and cardiac magnetic resonance, echocardiography, as a non-invasive method with advantages being convenient, inexpensive, and reproducible, has played an indispensable role in the pre-participation cardiovascular screening of athletes[5]. Left ventricular ejection fraction (LVEF) was a traditional established method in evaluating cardiac systolic function but susceptible to the influence of LV pre- and after-load and ventricular wall motion[6, 7]. In recent years, LV myocardial strain with no angle-dependent has been a promising alternative in characterizing LV systolic function to conventional methods as above[8]. However, strain is predisposed by LV after-load and image quality, which will limit the accuracy on estimating LV performance[9].

To accurately assess LV function, pressure-strain loop (PSL) has been established by considering myocardial strain and after-load through speckle tracking echocardiography (STE) and aortic pressure as a reproducible method[10]. Moreover, its rationality and effectiveness to quantitatively assess left ventricular myocardial work (LVMW) have been proven by much research[11]. LV-PSL can not only obtain the global MW but the 17-segment MW bull’s eye diagram of the LV, which benefits to understand the global systolic function as well as local myocardial contractile function of LV.

The objectives of this research were to: (i) describe the difference in LV global myocardial work (GMW) and regional myocardial work (RMW) of strength athletes with different resting heart rates; (ii) investigate the influence of resting heart rates on LV contractile performance of athletes.

**Materials And Methods**

**Study population**

A total of fifty young professional athletes dedicated to wrestling recruited from the Athletics Center were enrolled in the athlete group. According to their different heart rates, they were split up into the Low HR group (HR of 45 ~ 60 bpm, average 52.38 ± 4.72 bpm, \( n_1 = 25 \)) and the High HR group (HR of 60 ~ 80 bpm, average 66.68 ± 6.45 bpm, \( n_2 = 25 \)). The inclusion criteria were as following: (i) years of training ≥ 5, time of training per week ≥ 30 hours; (ii) never stopping intensive strength exercise; (iii) without records of stimulant use; (iv) sinus rhythm. The exclusion criteria included: (i) without good image quality for offline analysis; (ii) coronary heart disease, myocardial infarction, or arrhythmia; (iii) valve disease such as stenosis
or regurgitation; (iv) hypertension, diabetes, kidney disease and other systemic disease. Meanwhile, thirty sedentary individuals who underwent physical examination at the same period in the First Affiliated Hospital of Zhengzhou University were collected as the control group (HR of 60 ~ 96 bpm, average 71.65 ± 10.17 bpm, \( n_3 = 30 \)). The ones have no history of continuous training were included and the exclusion conditions were performed as described above. The study protocol has obtained the review and approval by the ethics committee and informed written consent was provided by all participants.

Echocardiography

Transthoracic echocardiography image acquisition was compiled by using a Vivid E95 color Doppler ultrasound diagnostic apparatus (GE Vingmed Ultrasound, Horten, Norway), equipped with a M5S transducer (frequency of 2.0 ~ 4.0mHz). Brachial artery pressure including systolic and diastolic blood pressure detected by electronic sphygmomanometer in a quiet state before examination and then recorded. The subjects were instructed to place in the left lateral decubitus position and breath calmly, with simultaneous electrocardiogram displaying. The standard LV long-axis, apical two-chamber, and four-chamber views of the gray-scale dynamic images at frame rates ≥ 60 frames/sec for three consecutive cardiac cycles were collected and restored in the offline analysis workstation of Echo PAC software (ver. 203, GE Vingmed Ultrasound, Norway).

LV diameter in diastolic (LVD), posterior wall thickness (PWT) and diastolic interventricular septum (IVST) on the LV long-axis view were measured using 2DE, and the relative ventricular wall thickness (RWT) was calculated by the equation: \(( \text{IVSTd} + \text{PWTd})/\text{LVDd} \). Measuring LV ejection fraction (EF), end-systolic volume (ESV), end-diastolic volume (EDV) and stroke volume (SV) utilized Simpson's biplane method.

Myocardial strain and work analysis

Import the images into the Echo PAC workstation and determine the three points of the mitral valve annulus and the apex on the long-axis, apical two-chamber, and four-chamber views respectively. Then, the system automatically traced the LV entire myocardial movement trajectory after identifying the endocardial borders, and the region of interest could be manually adjusted if necessary. Next, the brachial artery systolic and diastolic pressure value were entered, and the aortic valve closure time was automatically defined by the software on the long-axis view to obtain the LV-PSL and LVMW 17-segment bull's eye diagram (Fig. 1).

The 17-segment of LV was included three levels, the basal, middle and apical level, and several wall compartments, anterior, anterolateral, inferolateral, inferior, postseptal and anteroseptal, among which, the basal and middle level with six parts and the apical level with five parts. The regional myocardial work index (RMWI) and regional myocardial work efficiency (RMWE) were automatically calculated form three levels of LV and get the average for three times. According to our previous research, GWE was considered as the best predictor of LV contractility, so we analysis the linear relationship between GWE with general parameters to better understand the work efficiency of athletes. Global and regional MW parameters were compared between three groups and the 17-segment RMWI and RMWE were showed (Fig. 2).
Non-invasive PSL combined 2D-STE and arterial pressure to acquire the dynamic changes of LV pressure and strain during the mitral valve closing to opening process, which had been proven to have good consistency with invasive cardiac catheterization measurements[12]. Among them, a non-invasive LV pressure curve was constructed by the system using the brachial artery pressure that based on the period of LV isovolumetric and ejection obtained by echocardiography. Global myocardial work (GWI) presented the area of PSL, that was the total work calculated by the LV from mitral valve closure to mitral valve opening. Global myocardial constructive work (GCW) represented the work that conductive to LV ejection, including myocardial contracting in systole and elongating in isovolumic relaxation. Global myocardial wasted work (GWW) was constructed by lengthening myocytes in systole adding shortening myocytes in isovolumic relaxation, which was not conductive to LV ejection. Global myocardial work efficiency (GWE) was the ratio of constructive work divided by the sum of constructive and wasted work.

**Statistical analysis**

Statistical analysis was carried out with the aid of SPSS (ver. 24.0, IBM, Chicago, IL). All measurement data conforming to a normal distribution were presented as mean ± standard deviation (SD). Comparison among the three groups were conducted by one-way ANOVA which was followed by Tukey-Kramer test when variances were homogeneous or Games-Howell test when not. Multiple linear regression analysis was applied for relations of GWE. Intra- and inter-observer reliability of MW parameters measurement was interpreted using intraclass correlation coefficient (ICC) with 10 randomly selected athletes. \( P \)-values \( \leq 0.05 \) were identified statistically significant.

**Results**

General clinical data and echocardiographic characteristics of participants

The general and echocardiographic data of participants in the three groups were shown in Table 1. Statistically significant difference were found between the three groups except age and LVEF \( (P > 0.05) \). Compared with the control group, the body surface area (BSA), weight, body mass index (BMI), IVST, PWT, LVD, EDV, ESV and SV were all increased and heart rate decreased in the Low HR and High HR group (all \( P \leq 0.05 \)). However, it did not show statistically differences between the two athlete groups in terms of the above indices (\( P > 0.05 \)).
Table 1
Clinical and echocardiographic data of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control group (n&lt;sub&gt;3&lt;/sub&gt; = 30)</th>
<th>Low HR group (n&lt;sub&gt;1&lt;/sub&gt; = 25)</th>
<th>High HR group (n&lt;sub&gt;2&lt;/sub&gt; = 25)</th>
<th>F-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>19.06 ± 1.03</td>
<td>19.19 ± 1.12</td>
<td>19.25 ± 1.06</td>
<td>0.168</td>
<td>0.846</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>71.65 ± 10.17</td>
<td>52.38 ± 4.72*</td>
<td>66.68 ± 6.45*,**</td>
<td>36.853</td>
<td>0.000</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.76 ± 0.02</td>
<td>1.79 ± 0.07</td>
<td>1.77 ± 0.06</td>
<td>1.904</td>
<td>0.157</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.86 ± 7.31</td>
<td>82.95 ± 11.20*</td>
<td>85.87 ± 18.69*</td>
<td>13.316</td>
<td>0.000</td>
</tr>
<tr>
<td>BSA (m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>1.82 ± 0.09</td>
<td>2.02 ± 0.16*</td>
<td>2.03 ± 0.22*</td>
<td>12.813</td>
<td>0.000</td>
</tr>
<tr>
<td>BMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>22.63 ± 2.23</td>
<td>25.72 ± 2.25*</td>
<td>27.02 ± 5.02*</td>
<td>11.790</td>
<td>0.000</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>125.44 ± 8.59</td>
<td>122.57 ± 8.76</td>
<td>129.75 ± 8.06**</td>
<td>3.223</td>
<td>0.046</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>83.89 ± 6.07</td>
<td>77.47 ± 6.01*</td>
<td>80.75 ± 5.31</td>
<td>7.294</td>
<td>0.001</td>
</tr>
<tr>
<td>IVST (mm)</td>
<td>8.41 ± 0.34</td>
<td>10.38 ± 0.36*</td>
<td>10.65 ± 0.56*</td>
<td>209.236</td>
<td>0.000</td>
</tr>
<tr>
<td>PWT (mm)</td>
<td>8.59 ± 0.46</td>
<td>10.55 ± 0.47*</td>
<td>10.84 ± 0.50*</td>
<td>158.712</td>
<td>0.000</td>
</tr>
<tr>
<td>LVD (mm)</td>
<td>45.28 ± 2.57</td>
<td>50.42 ± 1.31*</td>
<td>50.90 ± 2.46*</td>
<td>47.178</td>
<td>0.000</td>
</tr>
<tr>
<td>RWT</td>
<td>0.37 ± 0.02</td>
<td>0.41 ± 0.01*</td>
<td>0.42 ± 0.01*</td>
<td>45.815</td>
<td>0.000</td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>107.23 ± 15.57</td>
<td>167.07 ± 29.75*</td>
<td>156.87 ± 28.90*</td>
<td>43.294</td>
<td>0.000</td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>39.87 ± 6.28</td>
<td>61.47 ± 13.56*</td>
<td>57.91 ± 11.74*</td>
<td>30.571</td>
<td>0.000</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>67.35 ± 10.50</td>
<td>105.60 ± 17.91*</td>
<td>98.95 ± 18.43*</td>
<td>44.726</td>
<td>0.000</td>
</tr>
<tr>
<td>EF (%)</td>
<td>62.87 ± 3.00</td>
<td>63.39 ± 3.47</td>
<td>63.04 ± 2.80</td>
<td>0.170</td>
<td>0.844</td>
</tr>
</tbody>
</table>

HR heart rate; BSA body surface area; BMI body mass index; SBP systolic blood pressure; DBP diastolic blood pressure; IVST diastolic interventricular septal thickness; PWT diastolic posterior wall thickness; LVD left ventricular end-diastolic diameter; RWT relative wall thickness; EDV end-diastolic volume; ESV end-systolic volume; SV stroke volume; EF ejection fraction.

*P < 0.05 vs Control group, **P < 0.05 vs Low HR group

Myocardial strain and work analysis

Comparisons of LV myocardial strain and MW indicators between groups were presented in Table 2–3. No statistically significant difference was identified among the three groups regarding GLS. The GWW, PSD and GWE reduced in the two athlete groups in relation to the control group, and GWI, GCW increased...
in the Low HR group, all with statistical difference ($P\leq0.05$). Compared with the Low HR group, increased GWI, GCW, GWW, PSD and decreased GWE achieving statistically significant difference in the High HR group. In the regional MW analysis, the basal and middle GWE in the High HR group decreased than that of the other two groups, and the apical GWI increased compared to the Low HR group. The three groups all exhibited the same results that the mean RMWI was increased from basal to apical, and the mean RGWE in middle and apical were increased compared to the basal but there was no statistical difference between the first two. The multiple linear regression analysis summarized in Table 4 showed that HR, IVST, PWT, GLS and PSD were well associated with GWE (Fig. 3), and the standardized regression coefficient ($b'$) were $-0.247$, $-0.390$, $-0.370$, $0.340$ and $-0.554$ respectively (all $P\leq0.05$).

### Table 2

Left ventricular strain and myocardial work parameters analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control group ($n_3 = 30$)</th>
<th>Low HR group ($n_1 = 25$)</th>
<th>High HR group ($n_2 = 25$)</th>
<th>$F$-value</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GWI (mmHg%)</td>
<td>2062.13 ± 161.10</td>
<td>1923.85 ± 165.95$*$</td>
<td>2144.29 ± 231.05$**$</td>
<td>7.143</td>
<td>0.002</td>
</tr>
<tr>
<td>GCW (mmHg%)</td>
<td>2359.03 ± 238.80</td>
<td>2176.77 ± 169.81$*$</td>
<td>2454.23 ± 344.98$**$</td>
<td>6.064</td>
<td>0.004</td>
</tr>
<tr>
<td>GWW (mmHg%)</td>
<td>44.74 ± 19.62</td>
<td>61.03 ± 14.05$*$</td>
<td>87.49 ± 31.07$**$</td>
<td>20.333</td>
<td>0.000</td>
</tr>
<tr>
<td>GWE (%)</td>
<td>97.36 ± 0.82</td>
<td>96.55 ± 0.97$*$</td>
<td>95.34 ± 1.35$**$</td>
<td>20.199</td>
<td>0.000</td>
</tr>
<tr>
<td>GLS (%)</td>
<td>-21.41 ± 1.49</td>
<td>-20.37 ± 1.76</td>
<td>-21.18 ± 2.22</td>
<td>2.176</td>
<td>0.122</td>
</tr>
<tr>
<td>PSD (ms)</td>
<td>30.16 ± 6.95</td>
<td>35.69 ± 5.23$*$</td>
<td>39.88 ± 6.25$**$</td>
<td>13.122</td>
<td>0.000</td>
</tr>
<tr>
<td>Basal GWI (mmHg%)</td>
<td>1649.69 ± 178.11</td>
<td>1635.01 ± 187.62</td>
<td>1634.09 ± 187.80</td>
<td>0.048</td>
<td>0.953</td>
</tr>
<tr>
<td>Middle GWI (mmHg%)</td>
<td>1922.30 ± 186.82</td>
<td>1911.60 ± 222.94</td>
<td>2015.37 ± 215.22</td>
<td>1.461</td>
<td>0.240</td>
</tr>
<tr>
<td>Apical GWI (mmHg%)</td>
<td>2317.91 ± 300.78</td>
<td>2190.29 ± 302.66</td>
<td>2456.75 ± 342.30$**$</td>
<td>3.252</td>
<td>0.045</td>
</tr>
<tr>
<td>Basal GWE (%)</td>
<td>95.85 ± 1.42</td>
<td>95.35 ± 1.69</td>
<td>94.42 ± 2.24$***$</td>
<td>3.585</td>
<td>0.034</td>
</tr>
<tr>
<td>Middle GWE (%)</td>
<td>98.32 ± 0.70</td>
<td>98.19 ± 0.52</td>
<td>97.53 ± 0.85$***$</td>
<td>6.680</td>
<td>0.002</td>
</tr>
<tr>
<td>Apical GWE (%)</td>
<td>98.25 ± 1.07</td>
<td>97.54 ± 1.45</td>
<td>97.67 ± 0.96</td>
<td>2.353</td>
<td>0.103</td>
</tr>
</tbody>
</table>

GWI global myocardial work index; GCW global constructive myocardial work; GWW global wasted myocardial work; GWE global myocardial work efficiency; GLS global longitudinal strain; PSD peak strain dispersion

* $P \leq 0.05$ vs Control group, ** $P \leq 0.05$ vs Low HR group.
Table 3
Comparison of mean regional myocardial work at different segments ($n_1 = 25$, $n_2 = 25$, $n_3 = 30$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Basal</th>
<th>Middle</th>
<th>Apical</th>
<th>$F$-value</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean RMWI (mmHg%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1649.69 ± 178.11</td>
<td>1922.30 ± 186.82#</td>
<td>2317.91 ± 300.78##</td>
<td>62.519</td>
<td>0.000</td>
</tr>
<tr>
<td>Low HR group</td>
<td>1635.01 ± 187.62</td>
<td>1911.60 ± 222.94#</td>
<td>2190.29 ± 302.66##</td>
<td>26.202</td>
<td>0.000</td>
</tr>
<tr>
<td>High HR group</td>
<td>1634.09 ± 187.80</td>
<td>2015.37 ± 215.22#</td>
<td>2456.75 ± 342.30##</td>
<td>40.930</td>
<td>0.000</td>
</tr>
<tr>
<td>Mean RMWE (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>95.85 ± 1.42</td>
<td>98.32 ± 0.70#</td>
<td>98.25 ± 1.07#</td>
<td>46.615</td>
<td>0.000</td>
</tr>
<tr>
<td>Low HR group</td>
<td>95.35 ± 1.69</td>
<td>98.19 ± 0.52#</td>
<td>97.54 ± 1.45#</td>
<td>25.268</td>
<td>0.000</td>
</tr>
<tr>
<td>High HR group</td>
<td>94.42 ± 2.24</td>
<td>97.53 ± 0.85#</td>
<td>97.67 ± 0.96#</td>
<td>24.219</td>
<td>0.000</td>
</tr>
</tbody>
</table>

RGWI regional myocardial work index; RGWE regional myocardial work efficiency

# $P \leq 0.05$ vs Basal level, ##$P \leq 0.05$ vs Middle level.

Table 4
Multiple linear regression analysis related to GWE

<table>
<thead>
<tr>
<th>Variables</th>
<th>Regression coefficient ($b$)</th>
<th>Standard error</th>
<th>Standard regression coefficient ($b'$)</th>
<th>$t$-value</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>102.669</td>
<td>5.571</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>-0.035</td>
<td>0.015</td>
<td>-0.247</td>
<td>-2.382</td>
<td>0.024*</td>
</tr>
<tr>
<td>IVST (mm)</td>
<td>-1.067</td>
<td>0.342</td>
<td>-0.390</td>
<td>-3.119</td>
<td>0.004*</td>
</tr>
<tr>
<td>PWT (mm)</td>
<td>-0.953</td>
<td>0.355</td>
<td>-0.370</td>
<td>-2.688</td>
<td>0.012*</td>
</tr>
<tr>
<td>RWT</td>
<td>-18.223</td>
<td>10.543</td>
<td>-0.187</td>
<td>-1.728</td>
<td>0.095</td>
</tr>
<tr>
<td>EF (%)</td>
<td>0.082</td>
<td>0.045</td>
<td>0.201</td>
<td>1.831</td>
<td>0.078</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>-0.009</td>
<td>0.015</td>
<td>-0.066</td>
<td>-0.643</td>
<td>0.526</td>
</tr>
<tr>
<td>GLS (%)</td>
<td>0.220</td>
<td>0.066</td>
<td>0.340</td>
<td>3.324</td>
<td>0.002*</td>
</tr>
<tr>
<td>PSD (ms)</td>
<td>-0.119</td>
<td>0.022</td>
<td>-0.554</td>
<td>-5.506</td>
<td>0.000*</td>
</tr>
</tbody>
</table>
*P < 0.05, indicating statistically significant of the linear relationship.

Repeatability

There were excellent intra- and inter-observer agreement for the MW parameters. The correlation coefficients of intra-observer in GWI, GWW, GCW, GWE were 0.981, 0.899, 0.990, 0.802, and those of the inter-observer were 0.904, 0.937, 0.988 and 0.811, respectively (all $P < 0.001$).

Discussion

After long-term professional and systematic high-intensity training, athletes would develop with different cardiac structural remodeling and functional changing due to different types of exercise[13, 14]. Strength athletes were mainly characterized by static anaerobic exercise for energy supply when training, which caused an increase in peripheral vascular resistance[15]. Compensatory hypertrophy of the LV myocardium was stimulated in response to the pressure overload, according to the process of Frank-Starling mechanism, which eventually lead to LV concentric remodeling[16]. The results of the study showed that the resting heart rates of the athlete group were reduced compared with the control group. In fact, it was possible due to the decrease in sympathetic nervous system activity and the increase in vagal tone caused by training, as well as the increased sensitivity of the myocardium to the vagal nerve[3, 17].

Based on the finding of the study, GWI and GCW were increased in the High HR group than the Low HR group. It could be explained that higher sympathetic nerve excitability was corresponding to faster heart rates, which stimulated increased myocardial contractility and peripheral vasoconstriction, resulting in increased cardiac afterload[4]. Meanwhile, the increased GWW in High HR group might be attributed to the increase of myocardial wall stress following the added LV stiffness and concentric remodeling[18]. Elevated heart rates during training or competing of athletes would reduce the fraction of time spent in diastolic coronary perfusion, with rapid increase of local myocardial oxygen consumption or metabolic demands but without proportional blood supply, easily driving myocardial ischemia especially the subendocardial[19, 20]. Hoffernan et al[21]. believed that vascular remodeling would be occur in athlete’s hearts after high-intensity training. Due to the continuous rise in cardiac output and incremental inflammation indicators after strenuous exercise, adversely effect on endothelial cells was made while vascular stiffness augmented.

Dyssynchronous ventricular wall motion, that was impaired synchronization of myocardial contraction evidenced by increased PSD in the two athlete groups resulted in the mechanical efficiency of LV ejection reduced[22]. LV remodeling and decreased synchronization of myocardial contraction could cause electrophysiological abnormalities of myocardium, not only leading to cardiac conduction disturbances but also reducing the effectiveness of myocardial coordination in contraction and relaxation[23, 24]. The evidence stated as above were more obvious in athletes with faster heart rates.

Detection of heart rate and heart rate variability were commonly used to reflect the changes of athletes, which presented dynamic balance among parasympathetic and sympathetic activity[25, 26]. A lower
resting heart rate of athletes could reduce myocardial oxygen consumption, improve work efficiency, and increase heart reserve[27]. However, the athletes with higher heart rates showed more pronounced subclinical changes in LV contractile performance as demonstrated in present study.

Limitations

This study was subject to several limitations that should be stated. The type of exercise was relatively single only including young male wrestlers with a small simple size. Moreover, it just shed light on the influence of athlete's heart rates on LV systolic function at rest, but the changes before and after exercise were not mentioned, which would be further investigated in the later stage.

Conclusions

The present study confirmed earlier findings that non-invasive LV-PSL could quantitively assess LV global and regional MW of athletes with different resting heart rates, and more accurately evaluate the changes of LV systolic function in an early time. After long-term special training, the increased LV wasted work and decreased work efficiency suggested that young strength athletes had experienced sub-clinical changes in LV contractile function, and this was more pronounced in those with a faster resting heart rate, indicating that, to a certain extent, the athletes with faster resting heart rate would suffer from an increased incidence of cardiovascular events. Therefore, the resting heart rate has certain reference significance in the screening of athletes before competition and the selection of elite ones.

Abbreviations

LV: left ventricular; PSL: pressure-strain loop; MW: myocardial work; GWI: global myocardial work index; GCW: global constructive myocardial work; GWW: global wasted myocardial work; GWE: global myocardial work efficiency; GLS: global longitudinal strain; PSD: peak strain dispersion; RMWI: regional myocardial work index; RMWE: regional myocardial work efficiency.

Declarations

Acknowledgements

Not applicable.

Author’s contributions

Shaohua Hua conceived of the study design and provided project oversight. Pengge Li and Yonggao Zhang compiled the data and drafted the manuscript. Lijin Li and Zhen Li collected the images and revised the article. Mengjiao Sun and Mengmeng Liu participated in the design of the article structure.

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Availability of data and materials

All data generated or analyzed during the study are included in this published article.

Ethical approval and consent to participate

This study was authorized by the ethics committee of Zhengzhou University First Affiliated Hospital (2020-KY-205), and written informed consent was obtained from all the participants.

Consent for publication

Consent for publication was obtained from all the participates.

Competing interests

The authors declare that they have no competing interests.

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References


Figures
Figure 1

Global and regional myocardial work parameters estimated by left ventricular pressure-strain loop and 17-segment bull’s-eye diagram. A. The control group. B. The Low HR group. C. The High HR group. MVC, mitral valve closure; AVO, aortic valve open; AVC, aortic valve closure; MVO, mitral valve open.
Figure 2

(A-B) Left ventricular mean regional myocardial work values including RMWI and RMWE in 17-segment at different levels between the athlete and control groups. (C-D) The comparison between basal, middle, and apical level in left ventricular mean regional myocardial work among the three groups. A-C: RMWI. B-D: RMWE. RMWI, regional myocardial work index; RMWE, regional myocardial work efficiency. *P < 0.05 and **P < 0.001 indicating significantly different between two levels; ns, indicating no significance.
Figure 3

Normal P-P plot regression standardized residual dependent variable: GWE.