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

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Evaluation of left ventricular 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 (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 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 HR: the low HR (45~60 bpm, n_1=25) and the high HR (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, and then compared between the above three groups.

Results:

The indicators of global and regional MW did statistically significantly differ between the athlete and control groups. Peak strain dispersion (PSD) and global myocardial wasted work (GWW) increased while global myocardial work efficiency (GWE) reduced in LHR and HHR groups compared with the control group, and global myocardial work index (GWI), global myocardial constructive work (GCW), global longitudinal strain (GLS) decreased in LHR group ($P<0.05$). In comparison to the LHR

group, GWI, GCW, GWW, PSD increased in HHR group and GWE reduced ($P<0.05$). According to the regional MW analysis, the mean regional myocardial work index (RMWI) increasing gradually from basal to apical levels were similar across the three groups and 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 correlated with GWE (b'= -0.247, -0.390, -0.370, 0.340, and -0.554, respectively, $P<0.05$).

Conclusions:

The LV contractile performance was more impaired in young strength athletes with high heart rates and PSL can be used to assess LV GMW and RMW quantitatively and accurately in reflecting LV systolic function.

Key words:

Echocardiography; Pressure-strain loop; Strength athlete; Heart rate; Myocardial work.

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 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 heart rates; (ii) investigate the influence of heart rate 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 age 19.19±1.12 years, n_1=25) and the High HR group (HR of 60~80 bpm, average age 19.25±1.06 years, 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 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 (average age 19.06±1.03 years, n_3=30). The one has 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 for three consecutive cardiac cycles were collected and restored in the offline analysis workstation of Echo PAC software (ver. 202, 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: (IVSTd+PWTd)/LVDd. Measuring LV ejection fraction (EF), end-systolic volume (ESV), end-diastolic volume (EDV) and stroke volume (SV) utilized Simpon'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 manually adjusted the region of interest if necessary. Next, the brachial artery pressure value was 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). 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.

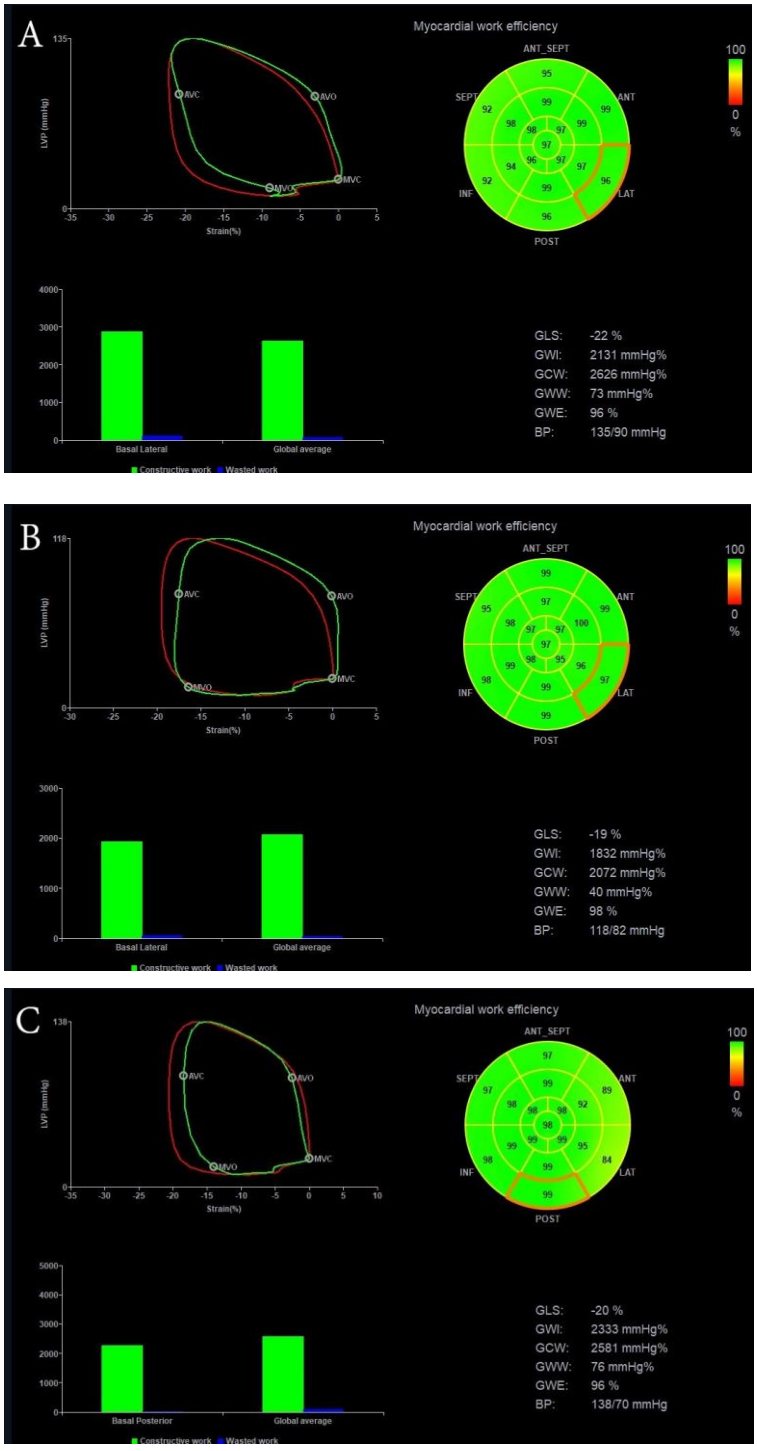


Fig. 1 Global and regional myocardial work parameters estimated by left ventricular pressure-strain loop and 17-segment bull's-eye diagram. A. The control group. B. The Low HR group. C. The High HR group. MVC, mitral valve closure; AVO, aortic valve open; AVC, aortic valve closure; MVO, mitral valve open.

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 $<$ 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 $P<0.05$). 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

Variable	Control group (n_3=30)	Low HR group (n_1=25)	High HR group (n_2=25)	*F*-value	*P*-value
Age (years)	19.06±1.03	19.19±1.12	19.25±1.06	0.168	0.846

Heart rate (bpm)	71.65±10.17	52.38±4.72*	66.68±6.45*, **	36.853	0.000
Height (m)	1.76±0.02	1.79±0.07	1.77±0.06	1.904	0.157
Weight (kg)	68.86±7.31	82.95±11.20*	85.87±18.69*	13.316	0.000
BSA (m^2)	1.82±0.09	2.02±0.16*	2.03±0.22*	12.813	0.000
BMI (kg/m^2)	22.63±2.23	25.72±2.25*	27.02±5.02*	11.790	0.000
SBP (mmHg)	125.44±8.59	122.57±8.76	129.75±8.06**	3.223	0.046
DBP (mmHg)	83.89±6.07	77.47±6.01*	80.75±5.31	7.294	0.001
IVST (mm)	8.41±0.34	10.38±0.36*	10.65±0.56*	209.236	0.000
PWT (mm)	8.59±0.46	10.55±0.47*	10.84±0.50*	158.712	0.000
LVD (mm)	45.28±2.57	50.42±1.31*	50.90±2.46*	47.178	0.000
RWT	0.37±0.02	0.41±0.01*	0.42±0.01*	45.815	0.000
EDV (ml)	107.23±15.57	167.07±29.75*	156.87±28.90*	43.294	0.000
ESV (ml)	39.87±6.28	61.47±13.56*	57.91±11.74*	30.571	0.000
SV (ml)	67.35±10.50	105.60±17.91*	98.95±18.43*	44.726	0.000
EF (%)	62.87±3.00	63.39±3.47	63.04±2.80	0.170	0.844

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 ($P<0.05$). 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 $P<0.05$).

Table 2 Left ventricular strain and myocardial work parameters analysis

Variable	Control group (n_3=30)	Low HR group (n_1=25)	High HR group (n_2=25)	F-value	P-value
GWI (mmHg%)	2062.13±161.10	1923.85±165.95*	2144.29±231.05**	7.143	0.002
GCW (mmHg%)	2359.03±238.80	2176.77±169.81*	2454.23±344.98**	6.064	0.004
GWW (mmHg%)	44.74±19.62	61.03±14.05*	87.49±31.07*,**	20.333	0.000
GWE (%)	97.36±0.82	96.55±0.97*	95.34±1.35*,**	20.199	0.000
GLS (%)	-21.41±1.49	-20.37±1.76	-21.18±2.22	2.176	0.122
PSD (ms)	30.16±6.95	35.69±5.23*	39.88±6.25*,**	13.122	0.000
Basal GWI	1649.69±178.11	1635.01±187.62	1634.09±187.80	0.048	0.953

(mmHg%)					
Middle GWI (mmHg%)	1922.30±186.82	1911.60±222.94	2015.37±215.22	1.461	0.240
Apical GWI (mmHg%)	2317.91±300.78	2190.29±302.66	2456.75±342.30**	3.252	0.045
Basal GWE (%)	95.85±1.42	95.35±1.69	94.42±2.24*,**	3.585	0.034
Middle GWE (%)	98.32±0.70	98.19±0.52	97.53±0.85*,**	6.680	0.002
Apical GWE (%)	98.25±1.07	97.54±1.45	97.67±0.96	2.353	0.103

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

$^{*}P$ ＜0.05 vs Control group, $^{**}P$ ＜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)

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

RGWI regional myocardial work index; RGWE regional myocardial work efficiency

$^{\#}P$ ＜0.05 vs Basal level, $^{\#\#}P$ ＜0.05 vs Middle level.

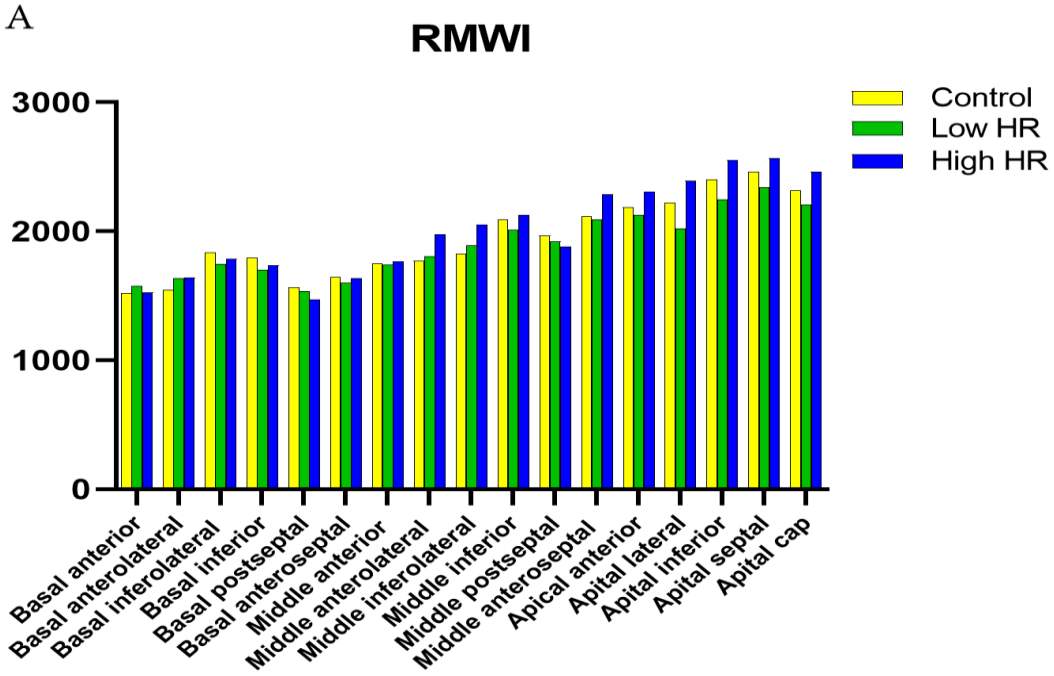
Table 4 Multiple linear regression analysis related to GWE

Variables	Regression coefficient (*b*)	Standard error	Standard regression coefficient (*b′*)	*t*-valve	*P*-valve
Constant	102.669	5.571	-	-	-
HR (bpm)	-0.035	0.015	-0.247	-2.382	0.024*
IVST (mm)	-1.067	0.342	-0.390	-3.119	0.004*
PWT (mm)	-0.953	0.355	-0.370	-2.688	0.012*

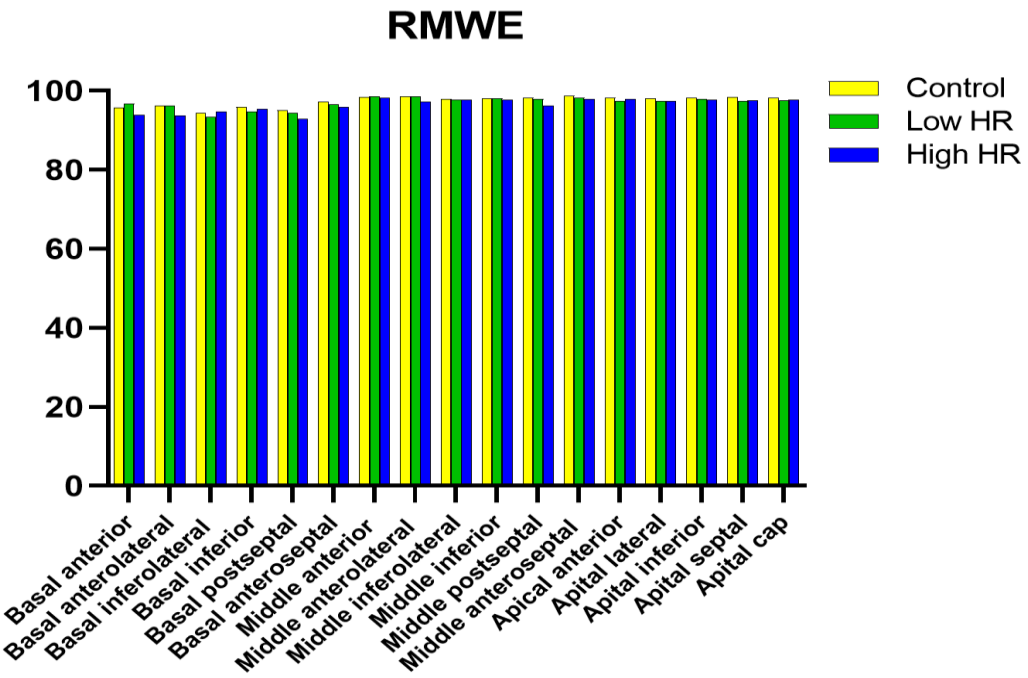
RWT	-18.223	10.543	-0.187	-1.728	0.095
EF (%)	0.082	0.045	0.201	1.831	0.078
SBP (mmHg)	-0.009	0.015	-0.066	-0.643	0.526
GLS (%)	0.220	0.066	0.340	3.324	0.002*
PSD (ms)	-0.119	0.022	-0.554	-5.506	0.000*

*P＜0.05, indicating statistically significant of the linear relationship.

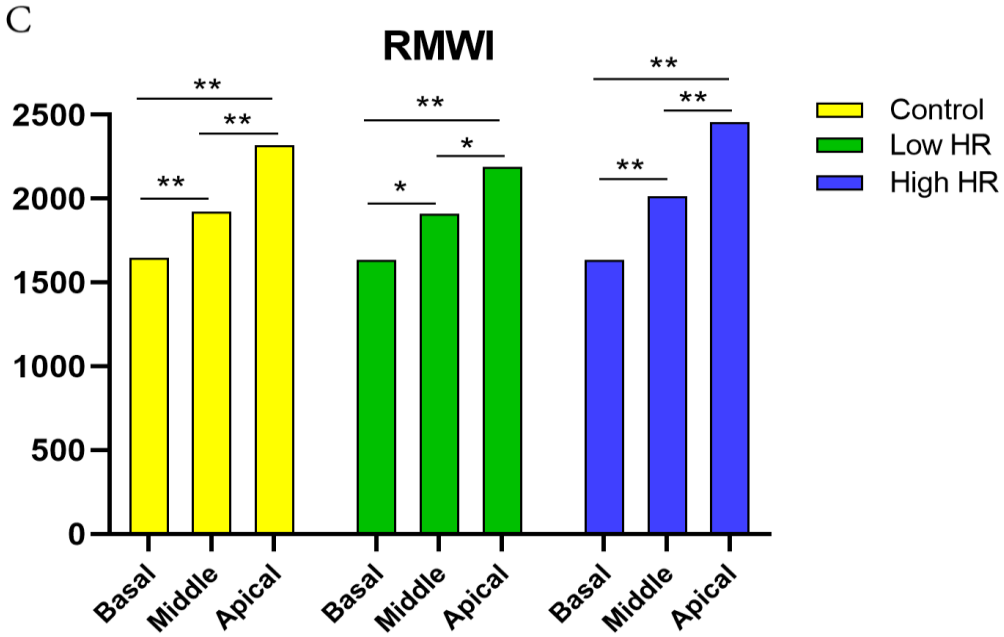
A



B


RMWE
100
80
60
40
20
0
Control
Low HR
High HR
Basal anterior
Basal anterolateral
Basal inferolateral
Basal inferior
Basal posteptal
Basal anteroseptal
Middle anterior
Middle anterolateral
Middle inferolateral
Middle inferior
Middle postseptal
Middle anteroseptal
Apical anterior
Apital lateral
Apital inferior
Apital septal
Apital cap

C


RMWI
2500
2000
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Control
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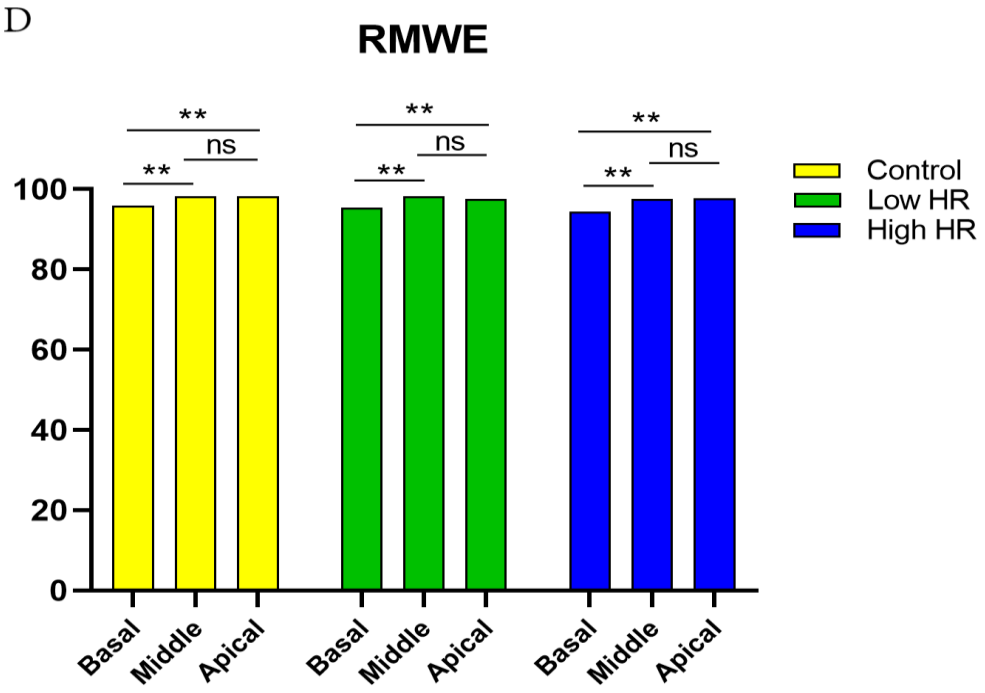

Fig. 2 (A-B) Left ventricular mean regional myocardial work values including RMWI and RMWE in 17-segment at different levels between the athlete and control groups. (C-D) The comparison between basal, middle, and apical level in left ventricular mean regional myocardial work among the three groups. A-C: 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.

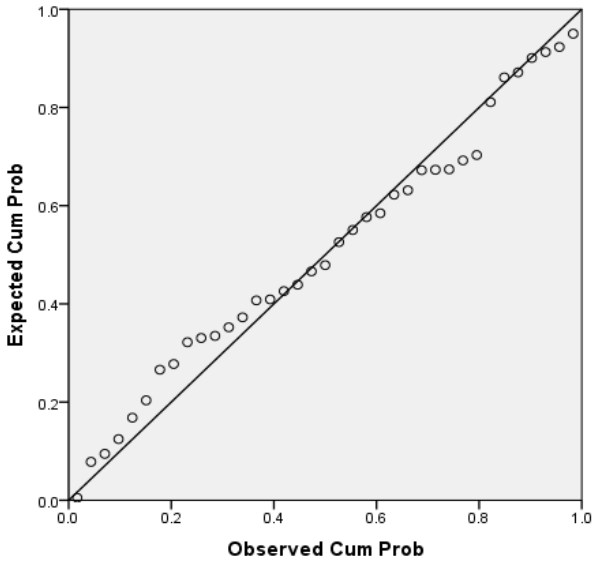

Fig. 3 Normal P-P plot regression standardized residual dependent variable: GWE.

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

vasocontraction, 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 hearts 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 non-invasive LV-PSL could quantitively assess LV global and regional MW of athletes with different 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 subclinical changes in LV contractile function, and this was more pronounced in those with a faster 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.

Acknowledgements

Not applicable.

Availability of data and materials

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

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Author's contributions

Shaohua Hua conceived of the study design and provided project oversight. Pengge Li 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.

Ethics approval

This study was authorized by the ethics committee of the First Affiliated Hospital of Zhengzhou University (2020-KY-205).

Consent to participates

The written informed consent was obtained from all the participates.

Consent to publication

Consent to publication was obtained from all the authors.

Conflicts of interest

The authors declare that they have no competing interests.

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Figures

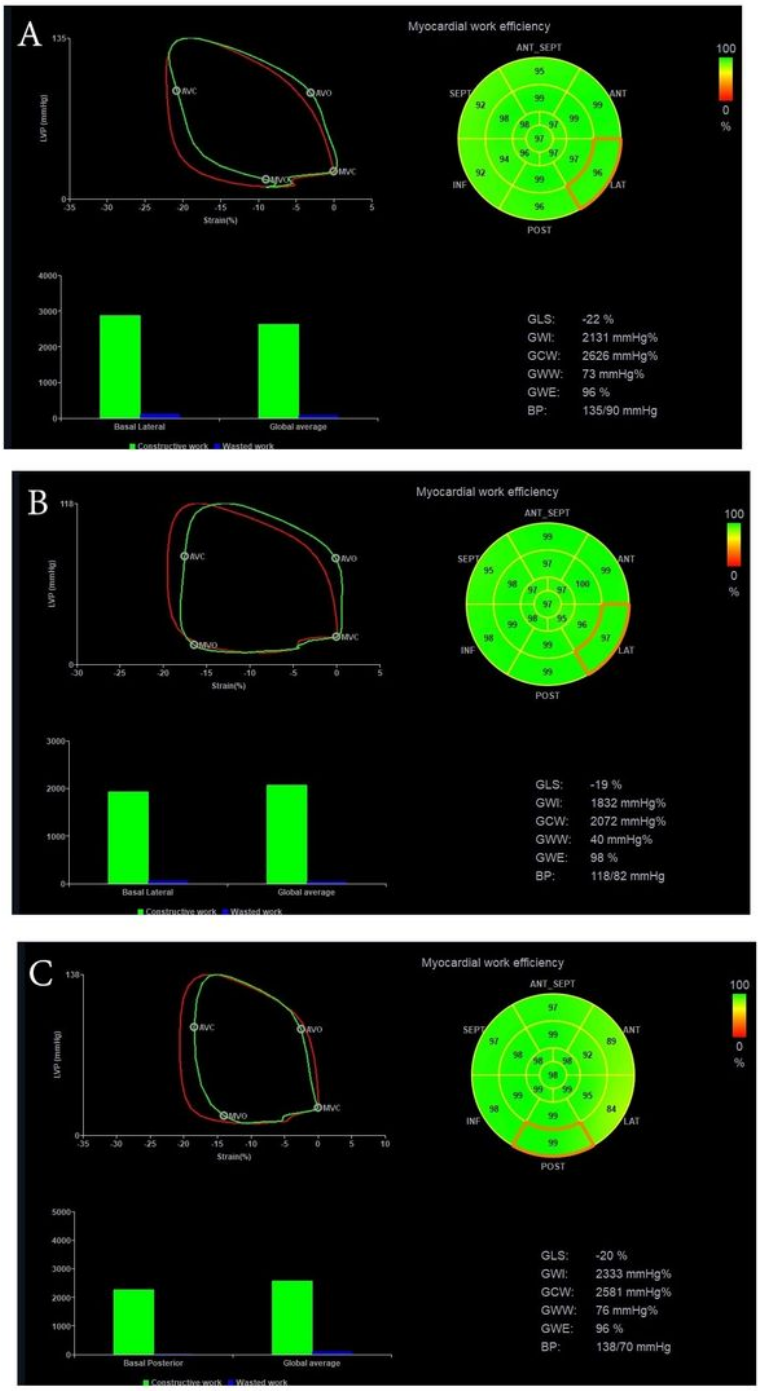

Figure 1

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.

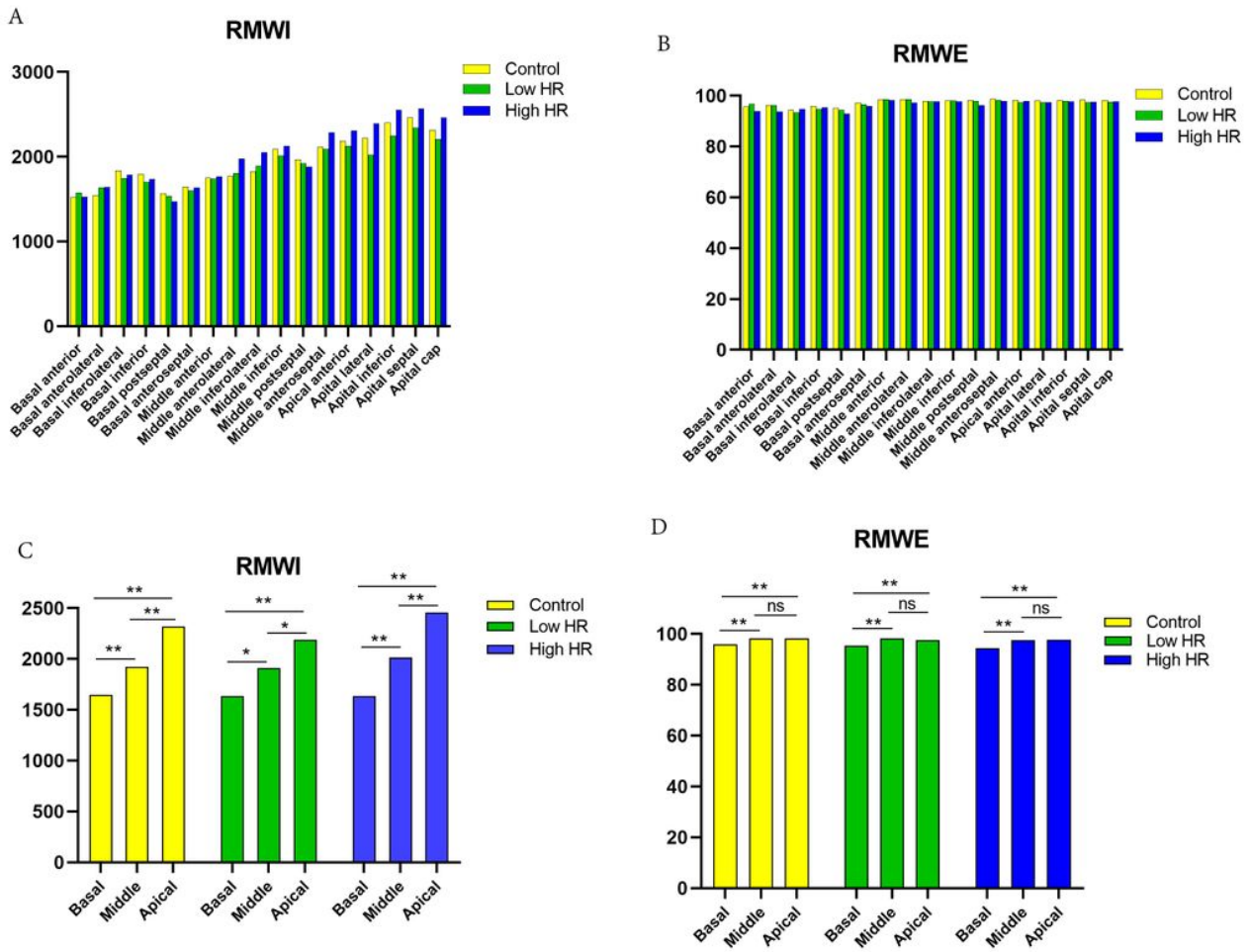

Figure 2

(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.

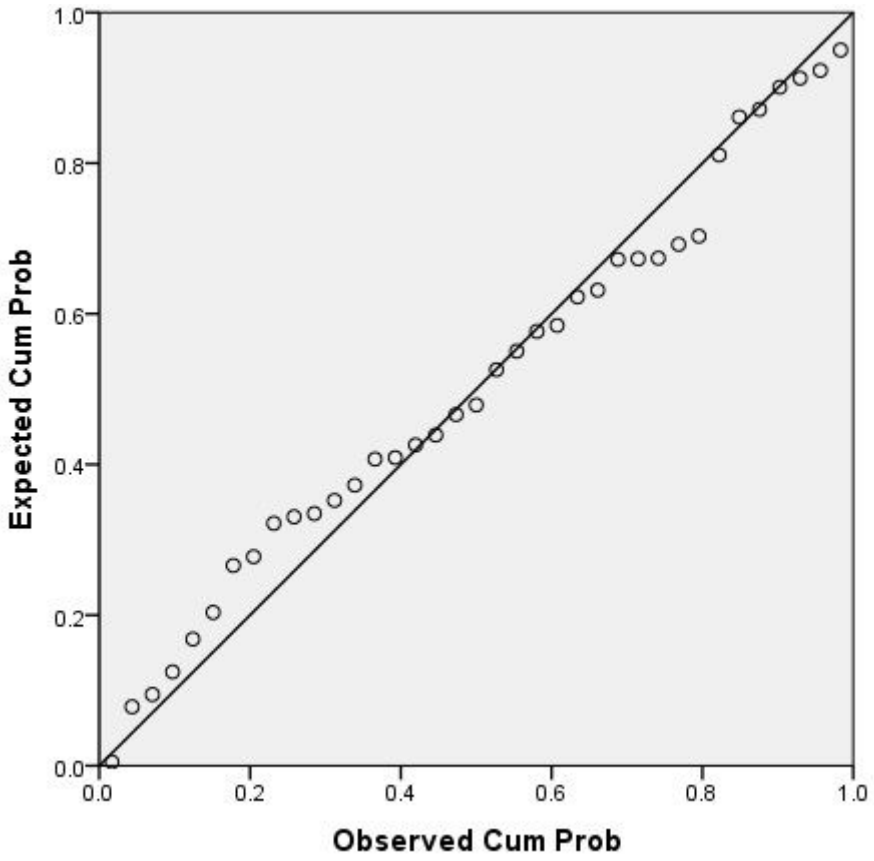

Figure 3

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