Evaluation of Left Atrial Function in Physiological and Pathological Left Ventricular Myocardial Hypertrophy by Real-time Tri-plane Strain Rate Imaging

Address for correspondence:
Zhi-Bin Wang, MD
Department of Cardiac Ultrasound
Qingdao University
Medical College Hospital
No.16 Jiangsu Road
Qingdao City, P.R. China
qdecholab@163.com

Pin Sun, MD; Zhi-Bin Wang, MD; Jian-Xing Li, MD; Jing Nie, MD; Yan Li, MD;
Xiang-Qin He, MD, Xiao-Ting Su, MD
Department of Cardiac Ultrasound, Qingdao University Medical College Hospital
(Sun, Wang, Li, Nie, Li, He, Su) Qingdao City, China

ABSTRACT

Background: We investigated the difference between left ventricular (LV) hypertrophy caused by primary hypertension and physiological LV hypertrophy in athletes as seen in left atrial (LA) function by real-time tri-plane strain rate imaging.

Hypothesis: A real-time tri-plane imaging technique using the same phase of the same cardiac circle was used to synchronously demonstrate the section of apical 4-chamber, 2-chamber, and apical left ventricle long axis.

Methods: We measured standard Doppler echocardiographic quantitative analysis and the strain rate peak values of each LA wall in the systolic phase, in the early stage of diastole, and in the advanced stage of diastole and made a comparison of these values.

Results: The alteration of configuration and function of the left atrium in hypertensive patients is an early sign of the myocardial damage caused by hypertension. Strain rate imaging could sensitively reflect LA function changes in the early stages of hypertension. While physiological, myocardial hypertrophy is a benign reaction, LA function is significantly different from that of hypertension.

Conclusions: Real-time tri-plane strain rate imaging techniques could simultaneously analyze 3 sections, which shortens scanning time and depletes the influence of variations of different cardiac cycles on quantitative analysis of local myocardial segments of the left atrium. This would improve the comparability of myocardial movement of different segments so that we could more comprehensively and accurately evaluate the systolic and diastolic function of the left atrium in primary hypertension and physiological LV hypertrophy in athletes.

Introduction

Real-time tri-plane strain rate imaging techniques can display 3 sections simultaneously at the same phase of the same cardiac cycle and give direct quantitative evaluation of the systolic function of local cardiac muscle. This reflects the spatial distribution of the velocity gradient or shear rate of the unit myocardial length, which is related to relative velocity, insusceptible to the bodily movement of the heart and the traction of the neighboring segment, and therefore could more veritably reflect the functional status of the local cardiac muscle.[1] Studies show that there are differences in left ventricular (LV) function in physiological and pathological LV myocardial hypertrophy.[2] Nevertheless, the function of the left atrium, which is an important part of heart function and plays a significant role in maintaining it, is often neglected. Physiological LV hypertrophy in athletes has a different prognosis from that of myocardial hypertrophy in primary hypertensive cardiopathy. This study discusses the application value of strain rate imaging techniques in evaluating left atrial (LA) function in physiological and pathological LV myocardial hypertrophy by comparing the disparity of strain rate parameters of the left atrium in athletes, hypertensive patients, and normal healthy people.

Data and Methods

General Data

A total of 30 patients with primary hypertension (hypertension group) who came to our clinic or were admitted to our hospital from January 2008 to August 2008 were chosen; among them were 22 males and 8 females, with an age range of 33 to 58 years (41 ± 6 yrs). The inclusion criteria were: (1) a diagnosis of hypertension in accordance with the criteria made by the World Health Organization's International Hypertension Conference which is systolic pressure ≥ 140 mm Hg (1 mm Hg = 0.133 kPa) and/or diastolic pressure ≥ 90 mm Hg; (2) LV myocardial hypertrophy with left ventricular mass index (LVMI) calculated through cardiac ultrasonic examination) >134 g/m^2 for males, >110 g/m^2 for females;[3] (3) without arrhythmia and cardiac conduction anomaly; (4) secondary hypertension, hypertrophic cardiomyopathy, diabetes, and other cardiovascular diseases were excluded; (5) good-quality sonograms. A total of 25 professional athletes (athletes' group) with myocardial hypertrophy were chosen; among them were 20 males and 5 females with an age range from 25

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to 40 years (31 ± 6 yrs). All subjects in the athletes' group had received professional training and had been engaged in professional sports for more than 8 years. Left ventricular mass index was $>134\ g/m^2$ in males and $>110\ g/m^2$ in females; a family history of hypertension and hypertrophic cardiomyopathy was excluded. Most sports involve both endurance and strength training and there is no need to clearly divide them. Therefore, we did not separate sports into types for this study. In addition, 30 healthy volunteers, whose age, gender, and surface area were a match with both the athletes' group and the hypertension group, were also chosen as the control group.

Equipment

A Duplex Color Doppler Ultrasonoscope GE Vivid 7 (GE, Fairfield, CT), equipped with a 2-dimensional (2D) detecting head (M3S; frequency: 1.5–4.3 MHz) and a 3-dimensional (3D) detecting head (V3; frequency: 1.5–4.0 MHz) could perform strain rate imaging and off-line quantitative analysis based on real-time tri-plane imaging. The subject took a left lateral position, breathing steadily, and the ultrasound image was collected in end expiration when breath was held. At least 3 continuous cardiac cycles were measured by each ultrasound index and an average value was calculated; an electrocardiogram (ECG) was recorded simultaneously.

Research Method

Routine ultrasonic cardiogram (UCG): After ideal 2D ultrasound images were taken from all subjects, the anteroposterior diameter of the left atrium, end-diastolic and end-systolic diameter of the left ventricle, end-diastolic thickness of the interventricular septum, and the LV posterior wall were measured at the parasternal left ventricle long axis section. The LV ejection fraction, as well as the LVMI was calculated. The supra-inferior diameters and transverse diameters of the left atrium were measured at the sections of the apical 4-chamber. Left atrial volume (LAV) was calculated according to the LAV formula: $LAV = \pi D_1D_2D_3/6$ (D_1, D_2, and D_3 refer to the anteroposterior, transverse, and supra-inferior diameters of the left atrium respectively). Blood flow frequency spectrum at the mitral orifice during diastolic phase, including flow rate peak value in early stage (E) and advanced stage (A) of diastole, as well as the ratio of E/A, were measured at the sections of the 4 cavities in the cardiac apex under pulse Doppler mode.

Real-time tri-plane strain rate imaging: After obtaining ideal 2D ultrasound images by placing the V3 detecting head on the apex of the heart, images of the sections of the apical 4-chamber were recorded. The tri-plane imaging technique was applied simultaneously to obtain the 3 sections of the apical 4-chamber, apical 2-chamber, and apical left ventricle long axis. The principle is to use a 2D standard section as a reference section and form an incision angle of 60-degrees, thereby obtaining 2 sections that form an angle of 60-degrees and 120-degrees respectively, with the model section. This is so that 3 sections could be displayed synchronously at the same phase of the same cardiac cycle. The strain rate imaging analytical system is started, placing the sample volume into the central part of all segments and setting the sampling area to $4\ mm \times 2\ mm$. The system would demonstrate the strain rate curve of this segment. Every index measures at least 3 successive cardiac cycles. We calculated the average value and then analyzed the data off-line. After the lateral wall, anterior wall, inferior wall, and posterior wall of the left atrium has been displayed in real-time tri-plane imaging, the Doppler sound beam should be placed as parallel to the cardiac atrium wall as possible to reduce errors and increase the image quality of the region being looked at. We have not analyzed the results of the atrium septum because it is susceptible to the size and function status of the right heart. The commonly used strain rate indexes of the left atrium are: strain rate peak value of systolic phase, strain rate peak value of early stage of diastole, strain rate peak value of advanced stage of diastole, the unit of which is s^{-1}.

Statistical Analysis

Statistic software SPSS 11.0 (SPSS, Chicago, IL) was applied; measurement data was denoted by mean $\pm$ SD. Multifactor variance analysis was conducted to compare interblock variables; $P < 0.01$ was regarded as a difference and had statistical significance.

Results

Routine Cardiac Ultrasound Parameter

The anteroposterior, supra-inferior, and transverse diameter of the left atrium, end-diastolic and end-systolic diameter of the left ventricle, end-diastolic thickness of the interventricular septum, and the LV posterior wall were measured on 25 athletes, 30 primary hypertensive patients, and 30 healthy volunteers. The LAV, LV ejection fraction, and LVMI were calculated. An index of 95% of LVMI in the control group was stipulated as the top limit of normal value range, which was $116\ g/m^2$ for males and $105\ g/m^2$ for females. The differences of gender composition, age, and heart rate among the normal control group, athletes' group, and hypertension group were not statistically significant ($P > 0.05$). Left ventricular mass index in the athletes' group and the hypertension group was $>134\ g/m^2$ for males and $>110\ g/m^2$ for females. Compared with the control group, the anteroposterior, supra-inferior, and transverse diameter of the left atrium, as well as the LAV were all significantly increased in the athletes' group and the hypertension group ($P < 0.01$). The E/A ratio was < 1 in the hypertension group and it

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Table 1. Standard Doppler Echocardiographic Quantitative Analysis ($\bar{x} \pm s$)

	Controls (n = 30)	Hypertensives (n = 30)	Athletes (n = 25)
LA anteroposterior diameter (cm)	3.2 ± 0.25	3.82 ± 0.28[a]	3.85 ± 0.25[b,c]
LA transverse diameter (cm)	3.3 ± 0.33	4.22 ± 0.35[a]	4.13 ± 0.42[b,c]
LA supra-inferior diameter (cm)	4.2 ± 0.23	4.82 ± 0.21[a]	4.85 ± 0.29[b,c]
LA maximum volume (cm^2)	23.21 ± 0.25	39.66 ± 0.33[a]	39.51 ± 0.33[b,c]
LV end-diastolic diameter (cm)	4.60 ± 0.15	4.24 ± 0.11	4.52 ± 0.13
Septal wall end-diastolic thickness (cm)	0.82 ± 0.25	1.23 ± 0.31[a]	1.16 ± 0.35[b,c]
LV posterior wall end-diastolic thickness (cm)	0.90 ± 0.21	1.25 ± 0.27[a]	1.23 ± 0.31[b,c]
LV mass index (g/m^2)	85.18 ± 20.9	143.3 ± 22.2[a]	138.2 ± 21.7[a,c]
LV ejection fraction (%)	63.1 ± 5.08	65.25 ± 4.22	67.7 ± 4.21
E (cm/s)	91.0 ± 16.7	62.14 ± 3.95	92.5 ± 11.6
A (cm/s)	65.28 ± 4.7	95.2 ± 12.9	70.55 ± 2.78
E/A	1.40 ± 0.17	0.70 ± 0.26[d]	1.31 ± 0.15

Abbreviations: LA, left atrial; LV, left ventricle.
[a] Patients with hypertension vs controls: $P < 0.01$.
[b] Athletes vs controls: $P < 0.01$.
[c] Athletes vs patients with hypertension: $P > 0.05$.
[d] Patients with hypertension vs controls and athletes: $P < 0.01$.

was normal in the athletes' group and the control group (Table 1).

Real-time Tri-plane Strain Rate Parameter

The strain rate peak value of the lateral wall, anterior wall, inferior wall, and posterior wall of the left atrium in systolic phase, in the early stage of diastole, and in the advanced stage of diastole were measured in all subjects. Results showed that the strain rate peak value in the athletes' group (Figure 1) approximated that in the control group (Figure 2) and was not statistically significant ($P > 0.05$). Compared with the athletes group and the control group, the hypertension group (Figure 3) had a clearly lowered strain rate peak value and there was a significant difference ($P < 0.01$; Table 2). During the study, we found that the strain rate of the left atrium in the hypertension group was also lowered before the diameter of the left atrium was not significantly different from that in the control group. This was especially obvious in patients with an enlarged left atrium. The situation indicated that even if there was no change in the left atrium revealed in a routine UCG in patients with hypertensive LV myocardial hypertrophy, the deformation function of the LA muscle was still reduced. On the contrary, the strain rate peak value in athletes had no significant difference compared with that of the control group.

Discussion

Reports on an athlete's heart first appeared in the work of Henschen, who described an enlarged heart through physical examination and percussion.[4] With the development of modern UCG and other imaging examining methods, these changes were further revealed and confirmed. In 1975, Morganroth[5] and fellow researchers first found evidence of a thickened LV wall, increased weight, and end-diastolic volume of the left ventricle in athletes by UCG. The essential difference between physiological and pathological LV hypertrophy is that the former functions well and is reversible; there is no change in strain rate peak values of the walls in the left atrium. The change in an athlete's heart will recover after training has stopped. Alteration of ECG and UCG disappears slowly, too, which also gives evidence that this is a physiological hypertrophy.[6–8] Having a different prognosis from physiological LV hypertrophy, hypertensive hypertrophy is not a beneficial compensation mechanism. It could be an independent risk factor that increases the morbidity and mortality of a cardiovascular accident.[3] Long-term endurance training causes structural change of the ventricles, increased left atrium inner diameter, a thickened ventricular wall, and elevated left ventricle weight, the extent of these changes is related to the intensity and duration of the training.[9–11] Statistics in China show that approximately 40% of an athlete's heart has been enlarged by over 10%.[12–13]

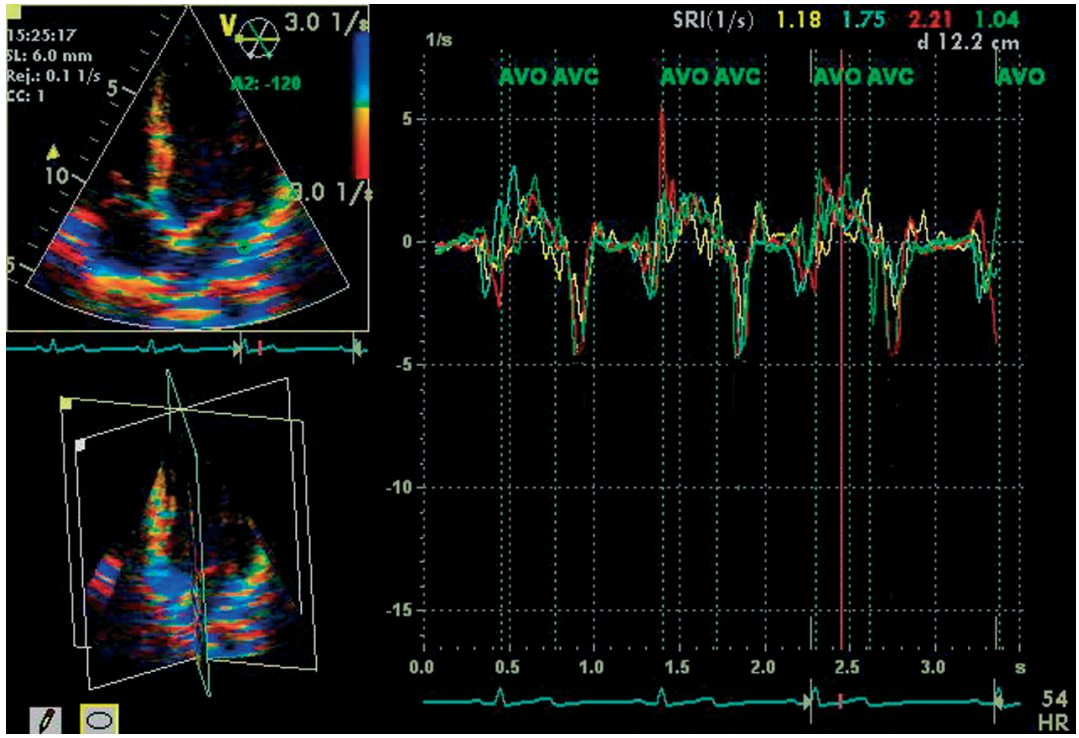

Figure 1. The strain rate peak value in the athletes' group.

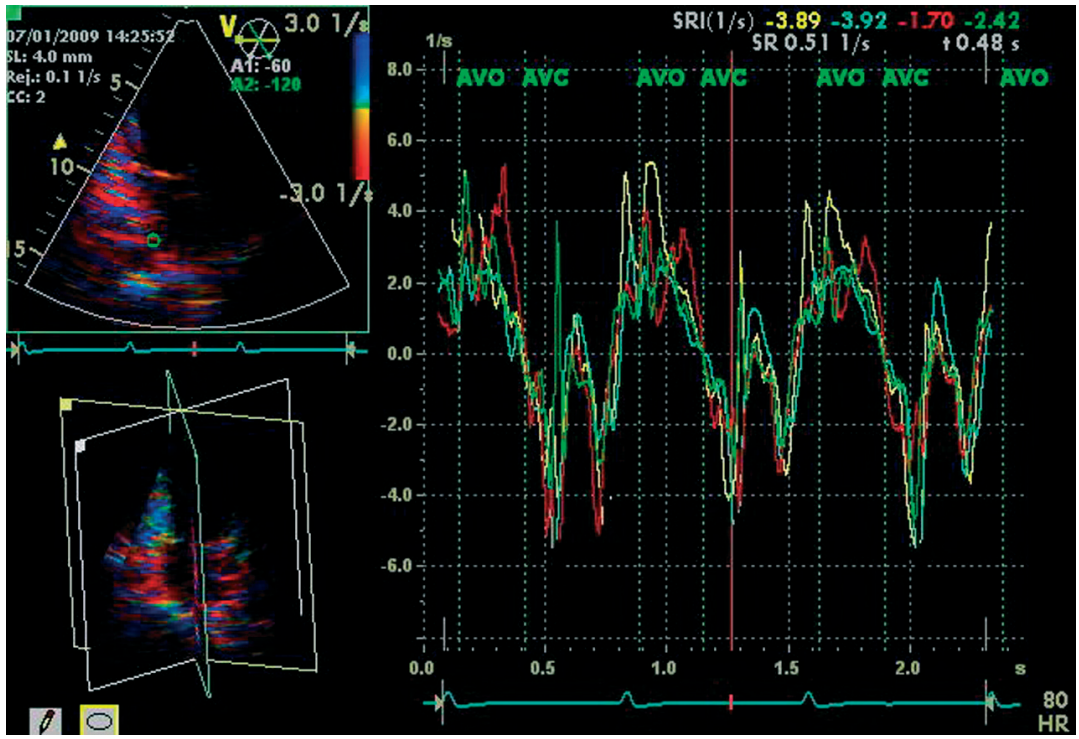

Figure 2. The strain rate peak value in the control group.

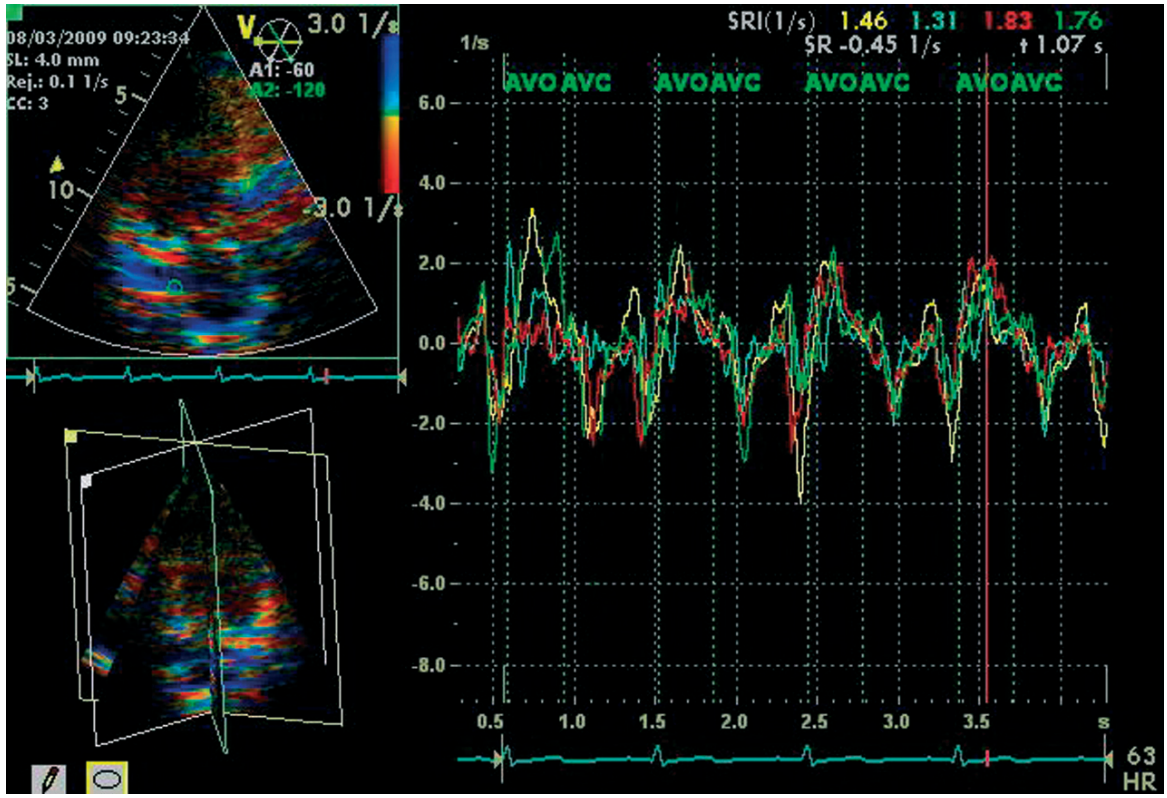

Figure 3. The strain rate peak value in the hypertension group.

The left atrium myocardium is distributed in 2 layers: deep and surface. The surface layer muscle is a transversal muscle bundle that encircles the atrium, while the deep layer is inherent muscle in the left atrium. They have 3 major functions: reservoir function, pipeline function, and assistant pump function (increasing LV filling during atrial systole period as an active ejection pump). Patients with hypertensive LV myocardial hypertrophy have weakened pipeline function of the left atrium and a strengthened reservoir function. The assistant pump function of the left atrium has been enhanced before LV hypertrophy appears and the enhancement is especially significant after the hypertrophy appears. The configuration and function change of the atrium is an early sign of hypertensive myocardial damage, and the change in the left atrium gets more significant along with aging and aggravation of disease. The pathological fundament is, the increased pressure load causes volume enlargement of myocardial cells, proliferation of interstitial cells, as well as fibroplasia, which results in the decrease of ventricular compliance; elevated LA pressure and tension of the atrial wall induces the prolonging of myocardium, the enlargement of the left atrium, and the reduction of the strain rate peak value.

Function change of the left atrium indicates the preload and afterload of it and plays an important role in maintaining normal heart function,[14–15] therefore, accurate evaluation of the left atrium is clinically very necessary. At present, evaluation of the left atrium mainly includes the area, volume, diameter, and pressure of the left atrium, which is mainly based on the conception of entirety. However, because of the irregularity of the left atrium's shape and the effect of the complicated movement of the heart, the evaluation has many limits. The major problems are the complication of measurement and the influence of LV function. Nevertheless, strain rate imaging could overcome these problems and provide a method of quantitative analysis for the assessment of the size and function of the left atrium. The real-time tri-plane strain rate imaging technique could simultaneously analyze in 3 sections, which shortens scanning time and depletes the influence of variation of different cardiac cycle on quantitative analysis of local myocardial segments in the left atrium, thereby improving the comparability of myocardial movement of different segments.

As a new ultrasound technique, strain rate imaging has certain limits, for example, it could be affected by noise, frame rate, resolution, angle of incidence, and so forth.

Table 2. Strain Rate Peak Value Baseline Measurements of the Left Atrial ($\bar{x} \pm s$)

	Controls (n = 30)	Hypertensives (n = 30)	Athletes (n = 25)
Left atrial anterior wall			
Systolic phase (s^{-1})	3.24 ± 0.30	2.52 ± 0.42^{a}	$3.35 \pm 0.47^{b,c}$
Early stage of diastole (s^{-1})	4.07 ± 0.32	1.94 ± 0.40^{a}	$4.18 \pm 0.34^{b,c}$
Advanced stage of diastole (s^{-1})	2.94 ± 0.36	2.43 ± 0.49^{a}	$2.78 \pm 0.45^{b,c}$
Left atrial inferior wall			
Systolic phase (s^{-1})	2.97 ± 0.27	2.35 ± 0.24^{a}	$2.88 \pm 0.17^{b,c}$
Early stage of diastole (s^{-1})	3.83 ± 0.17	1.39 ± 0.24^{a}	$3.77 \pm 0.22^{b,c}$
Advanced stage of diastole (s^{-1})	2.08 ± 0.33	2.46 ± 0.37^{a}	$2.11 \pm 0.31^{b,c}$
Left atrial posterior wall			
Systolic phase (s^{-1})	3.64 ± 0.31	2.66 ± 0.38^{a}	$3.55 \pm 0.42^{b,c}$
Early stage of diastole (s^{-1})	4.57 ± 0.30	2.19 ± 0.40^{a}	$4.68 \pm 0.34^{b,c}$
Advanced stage of diastole (s^{-1})	2.91 ± 0.33	2.55 ± 0.41^{a}	$2.89 \pm 0.43^{b,c}$
Left atrial lateral wall			
Systolic phase (s^{-1})	3.14 ± 0.27	2.55 ± 0.34^{a}	$3.03 \pm 0.26^{b,c}$
Early stage of diastole (s^{-1})	4.08 ± 0.32	2.17 ± 0.32^{a}	$3.97 \pm 0.35^{b,c}$
Advanced stage of diastole (s^{-1})	2.56 ± 0.33	3.08 ± 0.45^{a}	$2.36 \pm 0.31^{b,c}$

[a] Patients with hypertension vs controls: $P < 0.01$.
[b] Athletes vs controls: $P > 0.05$.
[c] Patients with hypertension vs athletes: $P < 0.01$.

We have reduced errors as much as possible during our operative procedure. Since frame rate has a big influence on strain rate, we adjusted the frame rate to 70–100 frames per second to precisely measure the strain rate in the systolic phase, and to 120–150 frames per second in the diastolic phase. We placed the Doppler sound beam as parallel to the cardiac atrium wall as possible to decrease the influence of the incidence angle. In addition, we will continue to increase the sample size of the athletes' group in future studies and compare statistics of different types of sports, hopefully to disclose more distinctly and accurately the function change of the left atrium in physiological and pathological LV hypertrophy.

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