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## **Characteristic Systolic Waveform of Left Ventricular Longitudinal Strain Rate in Patients with Hypertrophic Cardiomyopathy**

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## **ABSTRACT**

We analyzed the waveform of systolic strain and strain-rate curves to find a characteristic left ventricular (LV) myocardial contraction pattern in patients with hypertrophic cardiomyopathy (HCM), and evaluated the utility of these parameters for the differentiation of HCM and LV hypertrophy secondary to hypertension (HT). From global strain and strain-rate curves in the longitudinal and circumferential directions, the time from mitral valve closure to the peak strains (T-LS and T-CS, respectively) and the peak systolic strain rates (T-LSSR and T-CSSR, respectively) were measured in 34 patients with HCM, 30 patients with HT and 25 control subjects. The systolic strain-rate waveform was classified into 3 patterns (“V”, “W”, and “√” pattern). In the HCM group, T-LS was prolonged, but T-LSSR was shortened; consequently, T-LSSR/T-LS ratio was distinctly lower than in the HT and control groups. The “√” pattern of longitudinal strain-rate waveform was more frequently seen in the HCM group (74%) than in the control (4%) and HT (20%) groups. Similar but less distinct results were obtained in the circumferential direction. To differentiate HCM from HT, the sensitivity and specificity of the T-LSSR/T-LS ratio  $<0.34$  and the “√”-shaped longitudinal strain-rate waveform were 85% and 63%, and 74% and 80%, respectively. In conclusion, in patients with HCM, a reduced T-LSSR/T-LS ratio and a characteristic “√”-shaped waveform of LV systolic strain rate was seen, especially in the longitudinal direction. The timing and waveform analyses of systolic strain rate may be useful to distinguish between HCM and HT.

**Key words:** speckle tracking echocardiography, hypertrophic cardiomyopathy, hypertension, strain-rate waveform

## **INTRODUCTION**

Although the left ventricular (LV) ejection fraction is usually preserved or increased in patients with hypertrophic cardiomyopathy (HCM) [1-3], reduced myocardial contraction, especially in the longitudinal direction, has been observed in HCM patients by tissue Doppler imaging (TDI) [4-6], Doppler strain-rate imaging [7] and 2-dimensional speckle tracking echocardiography (STE) [8-10]. On the other hand, there have been few reports on alterations in the timing of the systolic peak and the systolic waveform of the LV myocardial strain rate in patients with HCM. Thus, we analyzed the waveform of systolic strain/strain-rate curves to find a characteristic LV myocardial contraction abnormality in patients with HCM and evaluated the usefulness of these parameters for the differentiation between HCM and LV hypertrophy secondary to hypertension (HT).

## **SUBJECTS AND METHODS**

### **Subjects**

The present study examined 34 patients with HCM, 30 age-matched patients with hypertension (HT) having an increased LV mass index ( $>115 \text{ g/m}^2$  for males and  $95 \text{ g/m}^2$  for females), and 25 age-matched normal control subjects in whom good quality echocardiographic images could be obtained. HCM was defined as LV hypertrophy (maximum wall thickness of  $\geq 15 \text{ mm}$ ) with an interventricular septal thickness/posterior wall thickness ratio of  $>1.3$  by 2-dimensional echocardiography that could not be explained by any other cardiac or systemic abnormalities [1,2]. Among the 34 patients with HCM, LV outflow tract (LVOT) obstruction with a pressure

gradient >30 mmHg was detected in 7 (20%), and LV cavity obliteration defined as the disappearance of the LV cavity more than the half of the long axis was observed in 8 (24%) patients. The diagnosis of HT was made when the blood pressure measurements at physical and mental rest on 2 or more subsequent visits was consistently >140 mmHg in systole or >90 mmHg in diastole, or if patients were taking anti-hypertensive medications and with history of hypertension. We excluded patients with obvious systolic dysfunction whose echocardiographic LV ejection fraction was <50%, pulmonary hypertension with tricuspid regurgitant velocity >3.4 m/s [11], or those with cardiac rhythm disturbances such as atrial fibrillation, frequent premature beats and artificial pacing. Patients with congestive heart failure, diabetes mellitus, coronary heart disease, congenital cardiac anomaly, valvular heart disease, and other systemic diseases that cause LV hypertrophy were also excluded.

Medications for patients with HT were  $\beta$ -blocker in 7 patients, angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker in 17, calcium antagonist in 11, diuretics in 7 and an anti-arrhythmic drug in 1; those for HCM patients were  $\beta$ -blocker in 18 patients, angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker in 12, calcium antagonist in 7, diuretics in 3 and anti-arrhythmic drugs in 7. The control group consisted of healthy volunteers without any clinical or echocardiographic evidence of cardiovascular disease.

This study was approved as a retrospective observational study by the Research Ethics Committee of Hokkaido University Hospital. All the study patients recruited for this study were those examined using GE machines (Vivid 7 or Vivid E9) for clinical purposes and met the above criteria.

### **Basic Echocardiographic Measurements**

Echocardiography was performed using Vivid 7 or Vivid E9 ultrasonographic machines (GE Healthcare, Buckinghamshire, UK) with M4S or M5S transducers, respectively. The LV end-diastolic dimension, LV end-systolic dimension and left atrial (LA) end-systolic dimension were measured in the parasternal long-axis image. The thicknesses of the interventricular septum and LV posterior wall were measured in the end-diastolic parasternal short-axis image at the chordal level. LV ejection fraction was calculated from apical 2-chamber and 4-chamber images using the biplane disk-summation method following the guidelines of the American Society of Echocardiography [12].

Pulsed-Doppler echocardiography was performed to measure peak early and late diastolic transmitral flow velocities (E and A, respectively) and their ratio (E/A). Tissue Doppler imaging of the mitral annulus was performed in the apical 4-chamber view, and the peak early-diastolic annular velocity ( $e'$ ) was measured at the interventricular septal annulus, and E/ $e'$  was calculated.

### **Two-dimensional Speckle Tracking Echocardiography**

Apical 4-chamber image and parasternal short-axis image at the mid-LV level were recorded, and digital cine loops of 3 cardiac cycles were stored on the hard disk of the echocardiographic machine. They were transferred to a workstation (EchoPAC PC version 113, GE Healthcare, Buckinghamshire, UK) after the examination for off-line analysis. LV myocardial strain and strain rate in the longitudinal direction were assessed in the apical 4-chamber view and those in the circumferential direction were in the parasternal short-axis view at the mid-LV level. The highest-quality image among 3 cardiac cycles was selected for each speckle tracking analysis. In each view, the LV endocardial surface of the end-systolic frame was traced manually, and the speckle tracking width was modified so as to cover the whole LV wall thickness to obtain the

so-called ‘global’ strain/strain-rate curves.

From the global longitudinal strain/strain-rate curves, peak longitudinal strain (LS), peak systolic longitudinal strain rate (LSSR), and the times from mitral valve closure to LS and LSSR (T-LS and T-LSSR, respectively) were measured (**Figure 1-A, B and C**). Also, in the circumferential direction, peak circumferential strain (CS), peak circumferential strain rate (CSSR), and the times from mitral closure to CS and CSSR (T-CS and T-CSSR, respectively) were measured (**Figure 1-D, E and F**). Additionally, we classified systolic strain-rate waveforms into 3 patterns as follows. A “V”-shaped pattern was defined as a simple monophasic pattern with a single negative peak (**Figure 2A**). A “W”-shaped pattern was defined as a curve with 2 or more distinct negative peaks during the ejection phase and the magnitude of second peak being more than half that of the first peak (**Figure 2B**). And a “√” (square root) pattern was defined as a curve with a single sharp negative peak and mid-systolic inflection point followed by a low velocity plateau (**Figure 2C**).

### **Statistical Analysis**

Statistical analysis was performed using standard statistical software (SPSS version 22 for Windows, SPSS Inc., Chicago, USA). All numerical data are represented as means  $\pm$  standard deviations. Differences among the 3 groups were tested using Fisher’s LSD test when there was a significant difference among groups by one-way analysis of variance. Categorical variables were compared by Chi-square test at first, and differences in pairs of groups were tested using Ryan’s method [13]. Receiver operating characteristic (ROC) analysis was used to evaluate the accuracy of various echocardiographic parameters for distinguishing HCM from LV hypertrophy secondary to HT. The reproducibility of the strain/strain-rate analysis was assessed in 20 randomly selected study subject patients. Two independent blinded observers analyzed the

same cine-loop and one of them repeated the analysis on a separate day. For all statistical tests, a p value <0.05 was considered to indicate significance.

## **RESULTS**

### **Clinical and Echocardiographic Features of Study Subjects**

As summarized in **Table 1**, interventricular septal thickness, LV posterior wall thickness, LA dimension and E/e' were significantly greater, and e' was significantly smaller in both the HT and HCM groups compared to the control group. LA dimension, interventricular septal thickness and E/e' were significantly greater, and e' was significantly lower in the HCM group than in the HT group. LV ejection fraction was significantly greater in the HCM group than the control group. Heart rate was significantly lower in the HCM group compared to the other 2 groups. Systolic and diastolic blood pressures were significantly greater in the HT group compared to the other 2 groups, and E/A was significantly lower in the HT group compared to the control group. Other parameters did not differ among the 3 groups.

### **Peak Systolic Strain and Strain Rate and Time to the Peaks**

As summarized in the upper part of **Table 2**, LS and LSSR were significantly reduced in the HCM and HT groups compared to the control group, and in the HCM group compared to the HT group. T-LS was prolonged, but T-LSSR was shortened in the HCM group compared to the other 2 groups, T-LSSR/T-LS ratio was significantly reduced in the HCM group compared to the control and HT groups.

As also shown in the lower part of **Table 2**, CS was significantly reduced in the HCM

group compared to the other 2 groups, but no significant difference was observed between the HT and control groups. T-CSSR was significantly shorter in the HCM group than in the control group, and did not differ between the HT and HCM groups. T-CS, CSSR and T-CSSR/T-CS ratio did not differ among the 3 groups.

All the timing parameters of strain and strain rate did not differ between HCM patients with LVOT obstruction and those without. They also did not correlate with LV end-diastolic dimension, interventricular septal thickness, E/e' or LA diameter except for the significant correlation between T-LS and E/e' ( $r=0.39$ ,  $p<0.05$ ).

The utilities of strain and strain-rate parameters for discriminating between patients with HT and those with HCM are summarized in **Table 3**. The areas under the curves were greater for LS and T-LSSR/T-LS ratio than for the other parameters. A T-LSSR/T-LS ratio  $<0.34$  showed better sensitivity than LS,  $>-16.3\%$ , despite its relatively low specificity.

### **Waveform of Systolic Strain Rate**

As shown in **Figure 3**, in the longitudinal direction, the “√” pattern was significantly more frequently seen in the HCM group (74%) compared to the control (4%) and HT (20%) groups ( $p<0.001$  for both). The “W” pattern was significantly less frequently seen in the HCM (12%) compared with the control (36%) and HT (47%) groups ( $p<0.05$  and  $p<0.01$ , respectively). The “V” pattern was significantly less frequently seen in HCM (15%) compared to the control (60%,  $p<0.001$ ) group. In the circumferential direction, the “√” pattern was significantly more frequently seen in the HCM than in the others. The prevalence of the “V” and “W” patterns did not differ significantly among the 3 groups.

Among the 25 HCM patients showing “√”-shaped longitudinal strain-rate waveform, 4 (16%) had LVOT obstruction and 7 (28%) had LV cavity obliteration, and 17 (68%) had neither

of these abnormalities. Among the 17 HCM patients showing the “√”-shaped circumferential strain-rate waveform, 2 (12%) had LVOT obstruction and 3 (18%) had LV cavity obliteration while 14 (82%) had neither.

The performance of the strain-rate waveform for discriminating between patients with HT and those with HCM is summarized in **Table 4**. The “√”-shaped strain-rate waveform in the longitudinal direction showed good sensitivity and specificity, while that in the circumferential direction had excellent specificity but low sensitivity. The presence of the “√”-shaped waveform in either direction had excellent sensitivity and acceptable specificity, and that in the both directions had excellent specificity but low sensitivity.

### **Reproducibility**

Intra- and Interobserver variability was 5% and 9% for LS, 3% and 4% for T-LS, 9% and 11% for LSSR, 13% and 14% for T-LSSR, 7% and 6% for CS, 6% and 6% for T-CS, 6% and 3% for CSSR, and 10% and 11% for T-LCSSR. Cohen’s  $\kappa$  coefficient for intra- and interobserver agreements of the strain-rate waveforms were 0.84 and 0.79, respectively.

## **DISCUSSION**

In the present study, we evaluated the time-amplitude waveform of systolic LV myocardial strain and strain rate in patients with HCM compared with normal subjects and patients with hypertensive LV hypertrophy. The timing of the peak longitudinal strain and strain rate in the HCM patients showed interesting conflicting abnormalities; T-LS was prolonged and T-LSSR was shortened; thus, T-LSSR/T-LS ratio was distinctly reduced in HCM. Moreover, patients

with HCM characteristically showed the “√”-shaped systolic strain-rate waveform.

### **Mechanism of Abnormal Systolic Strain Rate Waveform in HCM**

Shortening of the time to peak strain rate, prolongation of the time to peak strain, and the characteristic “√”-shaped strain-rate waveform shown in our HCM patients were considered to be associated with mid-systolic abrupt interruption of myocardial contraction and faint but prolonged contraction thereafter. LVOT obstruction and/or LV cavity obliteration can not explain the mechanism because the majority of our HCM patients with the “√”-shaped strain-rate waveform had neither. In patients with HCM, it is reported that myocardial ischemia can be caused by an imbalance between myocardial oxygen supply and demand associated with prominent hypertrophy [14,15]. Myocardial ischemia and the resultant fibrosis can induce a delay in contraction, but it seems unlikely to cause the mid-systolic interruption of myocardial contraction.

In the HCM patients of the present study, the strain/strain-rate abnormalities were more clearly seen in the longitudinal direction than in the circumferential direction. It was reported that myocardial disarray occurs most strongly in the inner layer of LV myocardium, next in the midwall and the least in the outer layer [16]; hence myocardial disarray in the subendocardial layer, where the myocardial fiber runs longitudinally, may be associated with the mid-systolic interruption of the longitudinal contraction. On the other hand, myocytes in the subepicardial layer are less affected by disarray [17], and this may explain the faint but prolonged longitudinal contraction. Moreover the preserved subepicardial myocardium may also explain the less frequent strain/strain-rate abnormalities in the circumferential direction. An abnormal shortening of the inner and middle layers can be masked by the contraction of the outer layer myocardium due to the small curvature radius of the LV wall in the short-axis plane [18]. Thus,

myocardial disarray might be the most probable cause of the strain/strain-rate abnormalities predominantly seen in the longitudinal direction.

Sengupta et al. reported that there was an apex-to-base order of contraction in normal pig hearts using sonomicrometry [19]. Duchateau et al. reported the apex-to-base sequence of contraction was seen in healthy human, and there are hypersynchrony or invert asynchrony between the apex and base in patients with HCM based on the circumferential strain analysis [20]. They also discussed that LV contraction sequence alterations could be explained by abnormal fiber orientation including myocardial fiber disarray. We consider that the invert asynchrony may be one of the possible mechanisms of the contraction timing abnormality shown in the present study.

### **Comparison with the Previous Studies**

In the present study, global T-LS of the LV myocardium derived from the speckle tracking technique was prolonged in HCM patients compared to HT patients and normal controls. Prolongation of T-LS in HCM patients has already been reported using Doppler strain imaging [21,22]. Ito et al. first reported the prolongation of T-LS of almost all the segments in HCM patients [21]. Ganame et al. have also reported the prolongation of regional T-LS, especially in the more severely hypertrophied segments of HCM patients with or without LV outflow obstruction [22]. Both investigators speculated that myocardial ischemia may be associated with the prolongation of T-LS.

In the present study, we found that T-LSSR was shortened while T-LS was conversely prolonged; thus, the T-LSSR/T-LS ratio was distinctly reduced in patients with HCM. To our knowledge, there has been no report investigating abnormalities in the timing of systolic strain rate in patients with HCM while a few reports have indirectly assessed the timing of

longitudinal myocardial contraction using tissue Doppler echocardiography. Tabata et al. and D'Andrea et al. reported the prolongation of the time from the Q-wave to peak systolic mitral annular velocity [5,23]. In contrast, Cardim et al. reported the shortening of the time from mitral valve closure to the peak systolic mitral annular velocity [6]. The time between the onset of electrical systole and mechanical systole was shown to be prolonged in HCM patients [23], and this might be the cause of the above discrepancy. The present study used the mitral valve closure as the start point of the measurement, similarly with Cardim et al., and found that the actual time from the onset of the mechanical systole to the peak contraction is shortened in patients with HCM. In addition, we analyzed the timing of the myocardial contraction in the circumferential direction, which could not be assessed using the tissue Doppler technique. In the circumferential direction, the time to peak strain rate was shortened in HCM patients, and the time to peak circumferential strain was also shortened; thus, the ratio between them was not significantly reduced. Moreover, as far as we know, no previous studies have focused on the characteristic systolic strain-rate waveform in patients with HCM. In the present study, the “√”-shaped systolic strain-rate waveform was characteristically observed in HCM patients, especially in the longitudinal direction.

### **Clinical Implication of Timing and Pattern Analysis of Strain Rate Waveform**

In the present study, the T-LSSR/T-LS ratio and “√”-shaped strain-rate waveform were shown to be useful for the differentiation between HCM and HT, while the more classical and simple parameter, LS, also showed good sensitivity and specificity as has been reported by several other investigators [7,10]. However, the amplitudes of strain and strain rate depend profoundly upon the analysis algorithm, which may differ among ultrasound manufacturers. In contrast, the analysis of the timing and waveform pattern of the strain-rate curve is considered to be less

dependent on the algorithm incorporated in the ultrasound machine. Moreover, the visual inspection of the global strain-rate waveform may contribute to at-a-glance diagnosis in routine echocardiographic examinations. Thus, the timing and waveform analyses of myocardial strain and strain rate may provide a useful and reliable practical tool for the diagnosis of HCM.

### **Limitations**

The study has several limitations. First, we did not perform cardiac MRI, which was reported to be useful to assess the extent of myocardial fibrosis and disarray [9,24,25] and might have been effective to elucidate the mechanism of the strain/strain-rate abnormalities shown in the present study. Secondly, we did not perform layer-by-layer strain/strain-rate analyses because the software algorithm of our machine did not support layer-specific analysis; such analysis may also contribute to clarifying the mechanism of the strain/strain-rate abnormalities. Thirdly, the frame rate used for the speckle tracking analysis was not very high, and it might have influenced the accuracy of the timing analysis in this study. Fourthly, we did not perform segmental strain/strain-rate analysis, in which noise signals may interfere with the accurate timing analysis. Finally, the study population was relatively small, and only included patients with HCM of the asymmetrical septal hypertrophy type.

### **CONCLUSION**

A reduced T-LSSR/T-LS ratio and a “√”-shaped waveform of LV strain rate were characteristically seen in patients with HCM. The timing and waveform analyses of systolic strain and strain rate may be helpful to distinguish between HCM and LV hypertrophy

secondary to HT.

### **CONFLICT OF INTEREST**

The authors declare that they have no conflict of interest.

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## FIGURE LEGENDS

### **Figure 1 Measurements of strain and strain-rate parameters using speckle tracking echocardiography**

A region of interest for speckle tracking analysis was set on the entire LV myocardium in the apical 4-chamber view (**A**) and parasternal short axis view at the mid-ventricular level (**D**). The peak longitudinal strain (LS) and the time to LS (T-LS) from the mitral valve closure (MVC) were measured from the global strain curve (**B**). The peak systolic longitudinal strain-rate (LSSR) and the time to LSSR (T-LSSR) from the MVC were measured from the global strain-rate curve (**C**). Similarly, the peak circumferential strain (CS), the time to CS (T-CS), the peak systolic circumferential strain-rate (CSSR), and the time to CSSR (T-CSSR) were measured (**E,F**).

### **Figure 2 Pattern analysis of the systolic longitudinal strain-rate waveform**

A waveform with a single mid-systolic peak during the ejection phase and with a linear or convex envelope was defined as “V” pattern (**A**); that with 2 distinct peaks was defined as “W” pattern (**B**); and that with a single sharp peak and a mid-systolic inflection point followed by a low-velocity plateau was defined as “√” pattern (**C**).

### **Figure 3 Percentages of “V”-, “W”- and “√”-shaped strain-rate waveforms in each group**

Control group shown with white bars, hypertensive left ventricular hypertrophy (HT) group with light gray bars and hypertrophic cardiomyopathy (HCM) group with dark gray bars.

**Table 1 Clinical characteristics and echocardiographic features**

	Control (n=25)	HT (n=30)	HCM (n=34)	p value (ANOVA)
Age (years)	53.4 ± 6.2	57.9 ± 11.4	59.4 ± 13.5	0.12
Male (%)	12 (48%)	16 (53%)	22 (63%)	0.50
Body surface area (m <sup>2</sup> )	1.65 ± 0.14	1.59 ± 0.17	1.66 ± 0.19	0.20
Heart rate (bpm)	63.1 ± 9.2	64.9 ± 11.7	58.1 ± 6.6 * ††	0.013
Systolic blood pressure (mmHg)	111.3 ± 12.7	138.3 ± 18.4 ***	117.9 ± 10.8 †††	<0.001
Diastolic blood pressure (mmHg)	69.3 ± 9.2	83.8 ± 10.3 ***	67.6 ± 11.0 †††	<0.001
LV end-diastolic dimension (mm)	47.7 ± 2.9	46.2 ± 3.5	46.1 ± 4.9	0.25
LV end-systolic dimension (mm)	29.4 ± 2.3	27.7 ± 3.9	27.3 ± 4.6	0.10
LV ejection fraction (%)	66.1 ± 3.9	67.6 ± 5.6	70.7 ± 8.4 **	0.023
LA dimension (mm)	34.6 ± 4.8	38.7 ± 4.5 **	45.1 ± 6.3 *** †††	<0.001
Interventricular septal thickness (mm)	9.1 ± 1.2	12.4 ± 1.6 ***	20.6 ± 4.7 *** †††	<0.001
LV posterior wall thickness (mm)	8.7 ± 1.1	11.0 ± 1.0 ***	10.5 ± 1.1 ***	<0.001
E (cm/s)	74.2 ± 12.8	69.8 ± 13.6	70.8 ± 19.8	0.58
E/A	1.22 ± 0.31	0.91 ± 0.23 **	1.11 ± 0.57	0.018
e' (cm/s)	10.6 ± 2.8	7.0 ± 1.7 ***	4.7 ± 2.5 *** †††	<0.001
E/e'	7.4 ± 2.3	10.4 ± 2.5 *	17.3 ± 7.8 *** †††	<0.001

HT, hypertension; HCM, hypertrophic cardiomyopathy; LV, left ventricle; LA, left atrium; E, peak early-diastolic transmitral flow velocity; E/A, ratio of E to peak late-diastolic transmitral flow velocity; e', peak early-diastolic mitral annulus velocity at the interventricular septum

\*p<0.05, \*\* p<0.01, \*\*\* p<0.001 versus Control; † p<0.05, †† p<0.01, ††† p<0.001 versus HT

**Table 2 Strain and strain-rate parameters**

	Control (n=25)	HT (n=30)	HCM (n=38)	p value (ANOVA)
<b>Longitudinal parameters</b>				
LS (%)	-21.5 ± 2.4	-18.6 ± 3.8 **	-13.7 ± 3.4 *** ††	<0.001
T-LS (ms)	347 ± 41	361 ± 56	422 ± 67 *** ††	<0.001
LSSR (s <sup>-1</sup> )	-1.11 ± 0.17	-0.99 ± 0.21 *	-0.85 ± 0.21 *** ††	<0.001
T-LSSR (ms)	167 ± 41	152 ± 50	123 ± 32 *** ††	<0.001
T-LSSR/T-LS	0.48 ± 0.10	0.43 ± 0.16	0.30 ± 0.07 *** ††	<0.001
<b>Circumferential parameters</b>				
CS (%)	-21.5 ± 2.9	-20.6 ± 3.0	-18.0 ± 3.7 *** ††	<0.001
T-CS (ms)	340 ± 42	331 ± 46	315 ± 48	0.11
CSSR (s <sup>-1</sup> )	-1.15 ± 0.20	-1.17 ± 0.23	-1.12 ± 0.25	0.72
T-CSSR (ms)	166 ± 35	151 ± 41	133 ± 36 **	0.005
T-CSSR/T-CS	0.49 ± 0.11	0.46 ± 0.12	0.42 ± 0.09	0.054

HT, hypertension; HCM, hypertrophic cardiomyopathy; LS, peak longitudinal strain; T-LS, the time to LS from mitral valve closure; LSSR, peak systolic longitudinal strain rate; T-LSSR, the time to LSSR from mitral valve closure; CS, peak circumferential strain; T-CS, the time to CS from mitral valve closure; CSSR, peak systolic circumferential strain rate; T-CSSR, the time to CSSR from mitral valve closure

\* p<0.05, \*\* p<0.01, \*\*\*p<0.001 versus Control; † p<0.05, †† p<0.01, ††† p<0.001 versus HT

**Table 3 Performance of strain and strain-rate parameters for discriminating between patients with HT and those with HCM**

	AUC	P value	Cut off value	Sensitivity	Specificity	PPV	NPV	Accuracy
<b>Longitudinal parameters</b>								
LS (%)	0.84	<0.001	-16.3%	79%	77%	79%	77%	78%
T-LS (ms)	0.76	<0.001	390 ms	71%	67%	71%	67%	69%
LSSR (s <sup>-1</sup> )	0.68	0.012	-0.85 s <sup>-1</sup>	59%	73%	71%	61%	66%
T-LSSR (ms)	0.67	0.020	127 ms	65%	67%	69%	63%	66%
T-LSSR/T-LS	0.77	<0.001	0.34	85%	63%	73%	79%	75%
<b>Circumferential parameters</b>								
CS (%)	0.70	0.007	-18.7%	59%	75%	73%	62%	67%
T-CS (ms)	0.59	0.22						
CSSR (s <sup>-1</sup> )	0.56	0.44						
T-CSSR (ms)	0.64	0.06						
T-CSSR/T-CS	0.59	0.24						

PPV= positive predictive value; NPV, negative predictive value. Other abbreviations are the same in **Table 2**.

**Table 4 Performance of strain rate waveform analysis for discriminating between patients with HT and those with HCM**

	<b>Sensitivity</b>	<b>Specificity</b>	<b>PPV</b>	<b>NPV</b>	<b>Accuracy</b>
Longitudinal SR “√” pattern	74%	80%	81%	73%	77%
Circumferential SR “√” pattern	53%	89%	85%	63%	70%
Longitudinal or circumferential SR “√” pattern	84%	71%	77%	80%	78%
Longitudinal and circumferential SR “√” pattern	44%	96%	93%	60%	68%

SR, strain rate. Other abbreviations are the same in **Table 2 and 3**.

Figure 1

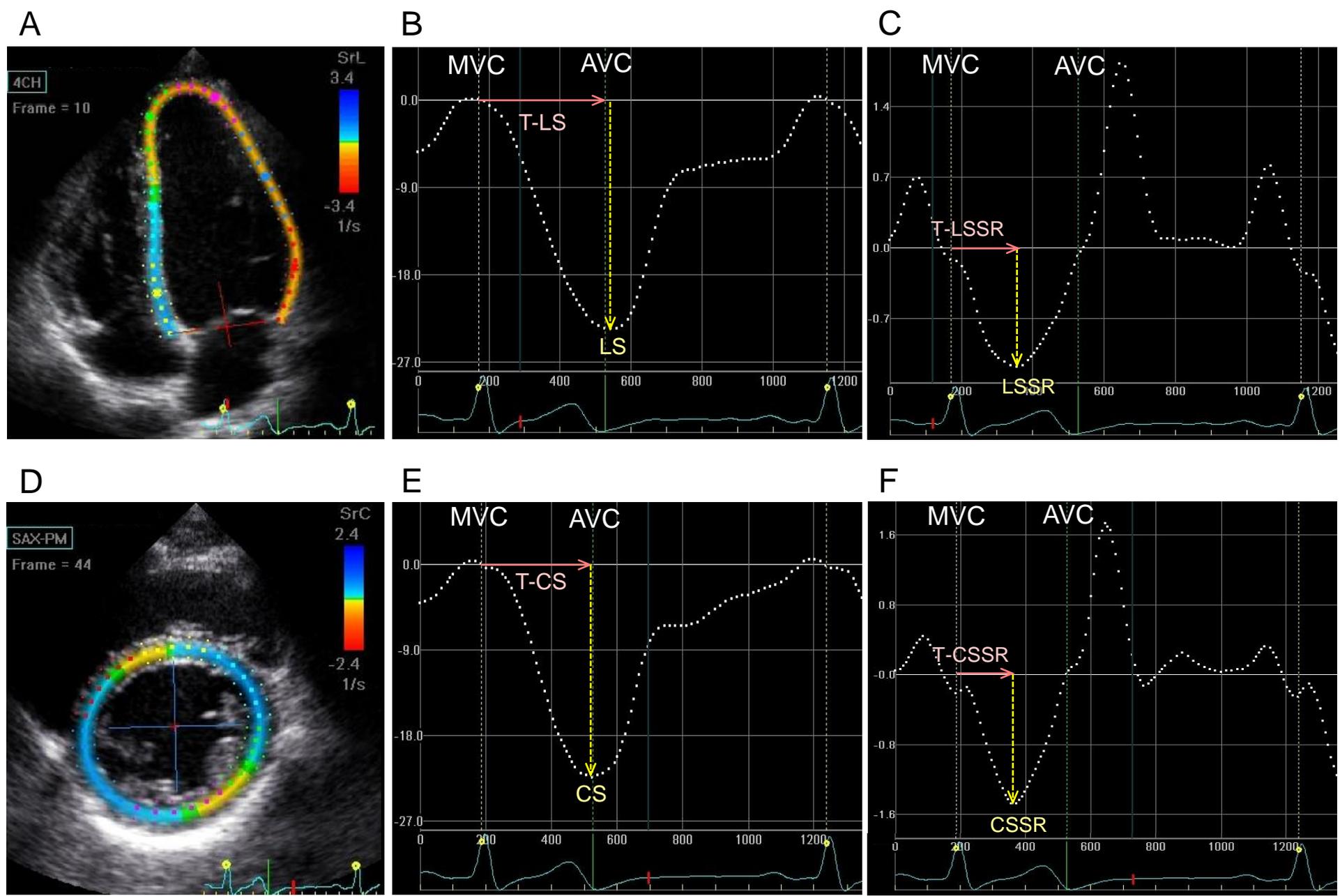


Figure 2

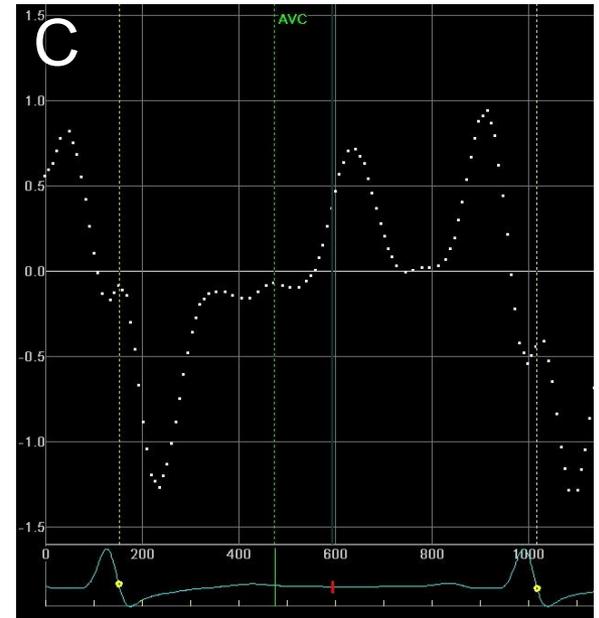
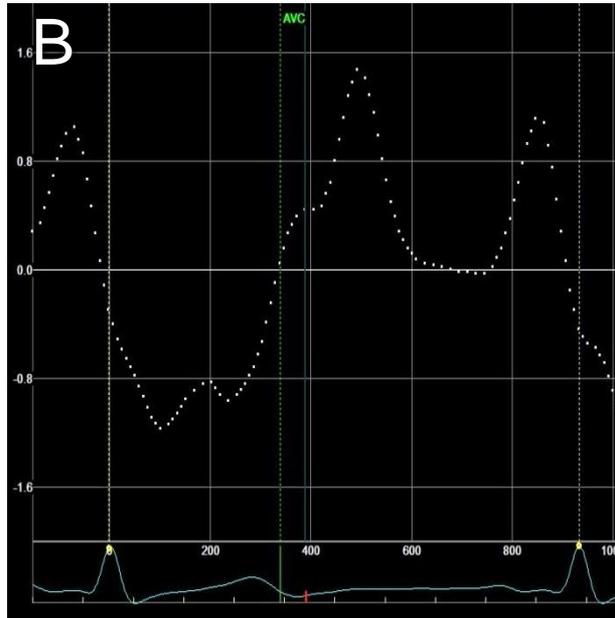
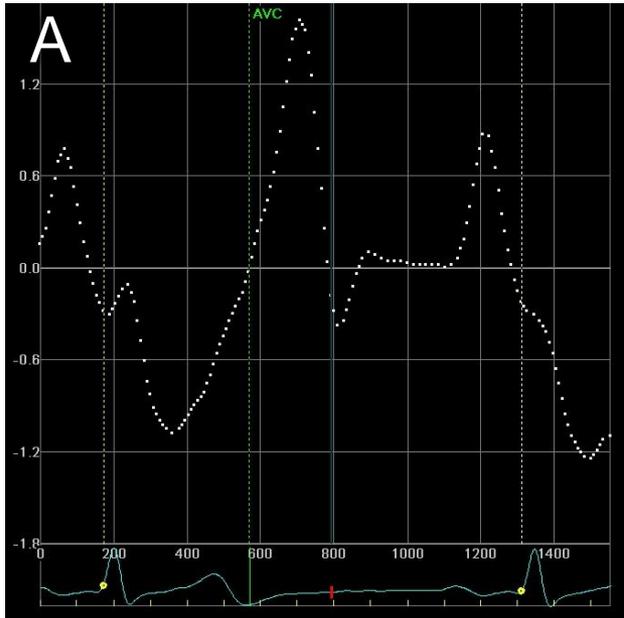


Figure 3

