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Value of layer-specific strain distribution patterns in hypertrophied myocardium from different etiologies

Mu-shiang Huang [a,b,1], Wen-Huang Lee [b,1], Huey-Ru Tsai [c,1], Yen-Wen Liu [b,1], Ping-Yen Liu [b,1], Wei-Chuan Tsai [b,*,1]

[a] Division of Cardiology, Department of Internal Medicine, Tainan Hospital, Ministry of Health and Welfare, Taiwan
[b] Division of Cardiology, Department of Internal Medicine, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Taiwan
[c] Division of Cardiology, Department of Internal Medicine, Madou Sin-Lau Hospital, Tainan, Taiwan

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ABSTRACT

Background: Intrinsic myocardial mechanics might have different patterns because of the different etiologies of myocardial hypertrophy. We used layer-specific strain to compare those with aortic stenosis (AS) and hypertrophic cardiomyopathy (HCM) and examined the differences in strain distribution pattern and for their clinical implications.
Methods: Comprehensive echocardiography was done in 3 groups: 129 with moderate-to-severe AS, 172 consecutive patients with HCM, and 58 healthy controls. Left ventricle (LV) layer-specific deformation parameters were obtained using two-dimensional speckle tracking echocardiography. The transmural strain gradient was defined as the strain difference between subendocardial and subepicardial myocardium. Both diseased groups were further divided based on the median value of transmural strain gradient for the hemodynamics correlation.
Results: Compared with the HCM group, the AS group had more preserved transmural longitudinal strain gradient ($4.49 \pm 1.3\%$ vs. $3.61 \pm 1.2\%$, $p < 0.001$), which was not significantly different from that of the healthy controls ($4.49 \pm 1.3\%$ vs. $4.54 \pm 1.0\%$, $p = 0.975$). And only in AS group the transmural circumferential strain correlated with myocardium mass index ($r = -0.237$, $p = 0.008$), and the hemodynamic profiles (LV ejection fraction and LA pressure) were correlated well with transmural strain gradient, in that the lower subgroup had a significantly lower LV ejection fraction and higher average E/E′.
Conclusions: Myocardium hypertrophy from different etiology resulted in different layer-specific strain distribution pattern. The loss of an adequate transmural strain gradient correlated with hemodynamics and might reflect intrinsic myocardial dysfunction.

1. Introduction

Myocardial hypertrophy can be caused by intrinsic cardiomyopathy such as hypertrophic cardiomyopathy (HCM), or by extrinsic pressure overload such as aortic stenosis (AS). Pathologically, the hypertrophied myocardium of HCM is displayed in more of a full-thickness distribution of generalized cardiomyopathy. The pressure overload of AS, however, has a relatively heterogenous influence on myocardium sequentially extending from the sub-endocardial layer toward the sub-epicardial layer sequentially [1–6]. Echocardiography-derived myocardial strain has been used to study myocardial mechanics for many years [7]. A novel technique called layer-specific myocardial strain allows further investigation of myocardial function at sub-layer levels [8]. It has been widely used in ischemic heart disease [9–12], but less used in other categories.

We hypothesized that hypertrophied myocardium had different remodeling in response to alternative etiologies, and that subendocardial and subepicardial myocardium would have different degrees of remodeling based on the diseased. We intended to use layer-specific myocardium strain analysis to show the significant remodeling differences. Previous study [13] has already reported that, in general, that the absolute value of subendocardial myocardial strain was greater than that of subepicardial strain and that there is a natural gradient. In studies on AS, the longitudinal and radial strain ratios of subendocardial and subepicardial myocardium were significantly correlated with AS severity [2,5], and better subendocardial myocardium longitudinal strain recovery after AS unloading by trans-catheter aortic valve replacement (TAVR) [6]. In a HCM study [14], this ratio was also significantly correlated with left ventricle (LV) size. However, these studies had relatively small samples and lacked head-to-head comparison.

* Corresponding author at: Division of Cardiology, Department of Internal Medicine, National Cheng Kung University Hospital, College of Medicine, 138 Sheng-Li Road, Tainan 704, Taiwan.
E-mail address: wctsai@seed.net.tw (W.-C. Tsai).
[1] This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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Table 1
Echocardiographic parameters of AS and HCM patients.

Variable	Group		p*
	AS	HCM	
	129	172	
LV myocardium mass index (mg/m^2)	133.4 ± 47.7	152.3 ± 49.3	0.001
Mitral inflow E velocity (m/s)	1.1 ± 0.8	0.7 ± 0.3	0.000
Mitral inflow deceleration time (ms)	236.7 ± 85.3	231.5 ± 95.6	0.627
Average E/E′	18.2 ± 9.3	14.4 ± 6	0.000
Left atrium volume (ml)	116 ± 45.6	111.6 ± 42.9	0.394
LV end diastolic volume (ml)	74.5 ± 32.3	67.1 ± 22.6	0.028
LV end systolic volume (ml)	27.4 ± 19.2	22.3 ± 11.7	0.009
LV ejection fraction (%)	66.5 ± 10.8	67.4 ± 9.1	0.446
Longitudinal strain			
Global (%)	−15.6 ± 3.9	−13.4 ± 3.9	0.000
Subepicardial (%)	−13.5 ± 3.5	−11.6 ± 3.5	0.000
Subendocardial (%)	−18.0 ± 4.4	−15.3 ± 4.3	0.000
Transmural gradient[+] (%)	4.5 ± 1.3	3.6 ± 1.2	0.000
Ratio[$]	1.3 ± 0.1	1.3 ± 0.2	0.559
Circumferential strain			
Global (%)	−14.8 ± 5.1	−14.3 ± 4.9	0.447
Subepicardial (%)	−7.0 ± 3.9	−7.4 ± 3.5	0.351
Subendocardial (%)	−27.9 ± 9.2	−26.6 ± 8.7	0.241
Transmural gradient[+] (%)	20.8 ± 8.8	19.2 ± 8.0	0.094
Ratio[$]	4.3 ± 2.3	3.9 ± 1.9	0.148

LV: Left ventricle.
* p value for comparison between HCM and AS group using Student's *t*-test for normally distributed continuous data.
+ |Subendocardial - subepicardial|.
$ Subendocardial/subepicardial myocardial strain.

We didn't intend to make the differential diagnosis between these two etiologies, since they can usually be differentiated in clinical, but wanted to (1) identify and compare whether the deformation distribution pattern distinct in hypertrophied myocardium from AS and HCM, and to (2) further analyze its correlation with hemodynamics and further clinical implications.

2. Methods

2.1. Ethical considerations

The protocol for this study was approved by the Institutional Review Board of National Cheng Kung University Hospital (B-ER-106-162).

2.2. Patients

In this cross-sectional retrospective study, from October 2010 to November 2015, we enrolled consecutive patients with HCM and those with moderate-to-severe AS from outpatient clinic refer for echocardiography. We excluded patients with other significant valvular heart disease: over moderately severe, history of pacemaker implantation or bundle branch block, or history of valvular repair or replacements.

HCM was diagnosed as significant muscle hypertrophy over 15 mm that could not be explained solely by loading condition [15,16]. Severe AS was defined as an estimated indexed aortic valve area (AVAi) < 0.6 cm^2/m^2, a maximal trans-aortic valve flow velocity (Vmax) > 4.0 m/s, and a mean pressure gradient (mPG) > 40 mm Hg. Moderate AS was defined as an AVAi > 0.6 cm^2/m^2, a Vmax 3.0 to 4.0 m/s, and a mPG 25 to 40 mm Hg [17].

Healthy controls (n = 58) were enrolled from health examinations between October 2012 and April 2013. The controls were apparently healthy and without structural heart disease, hypertension or coronary artery disease.

We further divided both groups into two equal subgroups using the median value of transmural longitudinal and circumferential strain gradient as the cutoff to demonstrate its correlation with hemodynamics.

2.3. Echocardiography

Image acquisition and strain analysis were using standard methods [18]. We examined all patients, who were in left lateral decubitus position, using an ultrasound system with a 3.5 MHz probe (Vivid series; GE Healthcare, Horten, Norway). The cardiac cycles from 3 standard apical views (4-chamber, 2-chamber, and long-axis) and from parasternal short-axis views were obtained using conventional 2-dimensional gray-scale echocardiography. All images were stored in digital format for offline analysis (Echopac PC version 113; GE Healthcare, TelAviv, Israel). Based on American Society of Echocardiography recommendations [19], left atrial (LA) volume, LV end-diastolic volume, LV end-systolic volume, and LV ejection fraction (LVEF) were measured in the apical two- and four-chamber views. LA volume was calculated using an area-length approximation, and the LV mass index using linear measurement of LV diastolic diameter and wall thickness in the parasternal long-axis M-mode view at the end diastole. Peak early diastolic (E) velocity, peak late diastolic (A) velocity of the mitral inflow and deceleration time of the early filling velocity were obtained using pulsed-wave Doppler after placing a sample volume (1–3 mm axial size) between mitral leaflet tips in the apical four-chamber view. Septal and lateral early diastolic mitral annular velocities (E′) were acquired using tissue pulsed-wave Doppler with placing a sample volume (usually 5–10 mm axial size) over the septal and lateral mitral annulus. Mitral peak E velocity divided by the average of septal and lateral E′ gave us the value of average E/E′. Two-dimensional gray-scale images were acquired in three standard apical views (apical four-chamber, apical two-chamber, and apical long-axis) for three cardiac cycles and stored digitally with a frame rate of 50–70 frames/s for subsequent off-line analysis.

Longitudinal and circumferential strain were assessed using two-dimensional speckle tracking echocardiography in 3 standard apical views, and parasternal short axis views at the mid-ventricular level. The region of interest was adjusted to ensure optimal analysis of most LV wall based on the ASE/European Association of Echocardiography consensus statement [20]. Each apical view assessment produced 6 segmental values of peak systolic longitudinal strain. For each segment, the software automatically separated the myocardium into subendocardial and subepicardial layers. Peak systolic longitudinal strain of subendocardial myocardium and subepicardial myocardium were derived (Supplementary Fig. 1). We defined "transmural strain gradient" as absolute difference between subendocardial and subepicardial myocardium, in circumferential or longitudinal vectors, and "strain ratio" as subendocardial myocardial strain divided by subepicardial myocardial strain.

2.4. Reproducibility

Twenty patients were randomly selected for an evaluation of interobserver variability. Subendocardial myocardium longitudinal strain, subepicardial myocardium longitudinal strain, and global longitudinal strain (GLS) were measured by two independent observers. For intraobserver variability, we repeated the same measurements 1 month apart. The intraclass correlation coefficient and mean percentage error (absolute difference divided by the average of the two observations) were also calculated.

2.5. Statistical analysis

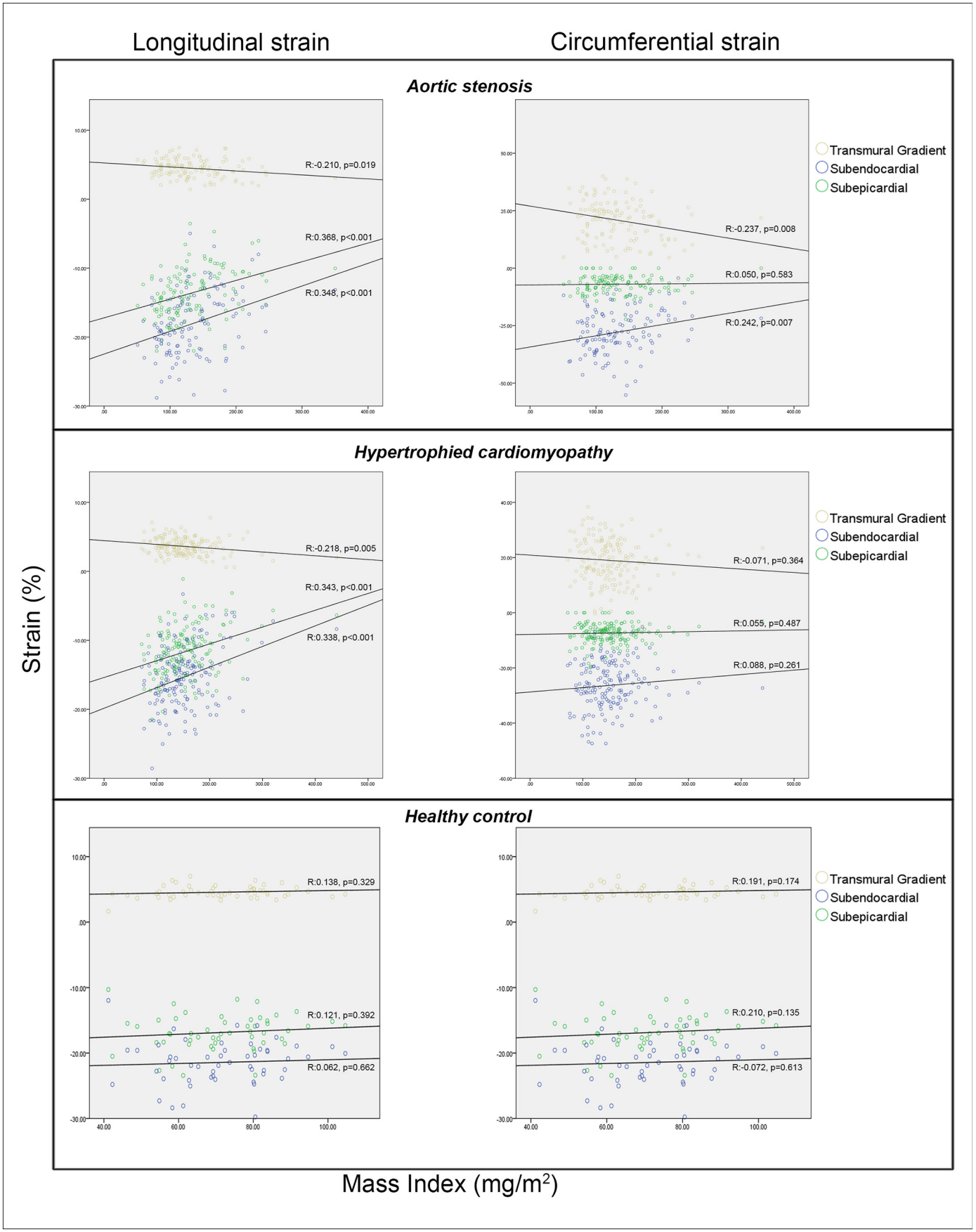
Continuous variables are presented as the mean ± SD (standard deviation), and Pearson correlation coefficient was applied to exam its correlation between LV mass index, each layer-specific strain values, LVEF and average E/E′ in figures. Dichotomous data are presented as numbers (percentage). By using the χ^2 test for categorical variables and the independent samples *t*-test for continuous variables, we first identified factors with significant correlations to enter multivariate linear regression analysis ($p < 0.10$). p values < 0.05 were considered to indicate statistical significance. The All statistical analysis was done using SPSS 21 for windows.

3. Results

3.1. Basic characteristics

Of the 178 HCM patient records reviewed, 6 were excluded because: the image quality was poor in 4 medical records, and because 2 patients had pacemakers. The remaining 172 patients (113 men; mean age: 64.6 ± 14 years) were assigned to HCM group. Of the 138 patients diagnosed as moderate-to-severe AS, 9 were excluded because the image quality was poor. The remaining 129 patients (59 men, mean age 71.9 ± 12 years) were assigned to the AS group. There were 58 medical records (29 men, mean age 49.1 ± 13.1 years) in the Healthy controls group. The mean age was significantly higher in the AS than in the HCM group (71.9 ± 12 vs. 64.6 ± 14 years; $p < 0.001$), there were significantly more men in the HCM group (65.7% vs. 45.7%; $p = 0.001$), and diastolic blood pressure was found lower in the AS group (76.8 ± 12.9 vs. 73.5 ± 13 mm Hg, $p = 0.031$) (Supplementary Table 1).

Fig. 1. The correlation of layer-specific strains and transmural gradient with the left ventricle (LV) mass index in AS, HCM and healthy control groups. The absolute value of both layers longitudinal strain and transmural gradient declined significantly as the mass index increased in the two diseased groups compare to healthy control. However, the circumferential subepicardial myocardium strain has no correlation with LV mass in all groups, but the absolute value of subendocardial myocardium and transmural gradient in AS group declined and correlated strongly with LV myocardium mass index but not in HCM and healthy control groups.


Longitudinal strain
Circumferential strain
Aortic stenosis
R:-0.210, p=0.019
R:0.368, p<0.001
R:0.348, p<0.001
R:-0.237, p=0.008
R:0.050, p=0.583
R:0.242, p=0.007
Transmural Gradient
Subendocardial
Subepicardial
Hypertrophied cardiomyopathy
R:-0.218, p=0.005
R:0.343, p<0.001
R:0.338, p<0.001
R:-0.071, p=0.364
R:0.055, p=0.487
R:0.088, p=0.261
Transmural Gradient
Subendocardial
Subepicardial
Healthy control
R:0.138, p=0.329
R:0.121, p=0.392
R:0.062, p=0.662
R:0.191, p=0.174
R:0.210, p=0.135
R:-0.072, p=0.613
Transmural Gradient
Subendocardial
Subepicardial
Strain (%)
Mass Index (mg/m2)

3.2. Echocardiography

The LV mass index of the HCM group was significantly greater than that of the AS group (152.3 ± 49.3 vs. 133.4 ± 47.7 g/m^2; $p < 0.001$) (Table 1). Although the LVEFs of the 2 groups were non-significantly different (67.4 ± 9.1 vs. 66.5 ± 10.8%; $p = 0.446$), both LV end diastolic and end systolic volume were significantly lower in the HCM group (67.1 ± 22.6 vs. 74.5 ± 32.3 ml, $p = 0.028$; and 22.3 ± 11.7 vs. 27.4 ± 19.2 ml, $p = 0.009$, respectively). The average E/E′ and mitral inflow E velocity that indicate LA pressure were significantly higher in the AS group (Average E/E′ 14.4 ± 6 vs. 18.2 ± 9.3; $p < 0.001$).

Neither the layer-specific circumferential strains between the HCM and the AS groups nor the transmural circumferential strain gradient were significantly different. There were, however, significant differences both in layer-specific longitudinal strains (subepicardial myocardium −11.6 ± 3.5 vs. −13.5 ± 3.5%, $p < 0.001$; subendocardial myocardium −15.3 ± 4.3 vs. −18.0 ± 4.4%, $p < 0.001$) and in transmural strain gradient (3.6 ± 1.2 vs. 4.5 ± 1.3%, $p < 0.001$) (Table 1 and Supplementary Fig. 2). The transmural strain gradient still remained significantly ($p < 0.001$) different after it had been adjusted for global longitudinal strain, age, gender, LV mass index, diastolic blood pressure, and average E/E′ in multivariate analysis (Supplementary Table 2).

To see the layer-specific strain distribution of normal myocardium, we analyzed the Healthy controls (Supplementary Fig. 2). Subepicardial and subendocardial layer-specific longitudinal strain values were significantly lower in the HCM and AS groups than in the Healthy controls group, as was the transmural longitudinal strain gradient remained unchanged in the AS group (4.49 ± 1.3% vs 4.54 ± 1.0%, $p = 0.975$) but significantly decreased in the HCM group (3.61 ± 1.2% vs 4.54 ± 1.0%, $p < 0.001$). In contrary to difference in longitudinal strain distribution, the circumferential strain seems less affected between groups.

We illustrated the correlation of layer-specific strain values with the LV myocardium mass index (Fig. 1). The absolute value of both layers longitudinal strain and transmural gradient declined significantly as the mass index increased. In the other hand, the circumferential subepicardial myocardium strain has no correlation with LV mass in all groups, but the absolute value of subendocardial myocardium and transmural gradient in AS group declined and correlated strongly with LV myocardium mass index but not in HCM and normal groups.

The transmural longitudinal strain gradient was strongly correlated with LVEF and average E/E′ in both groups (Fig. 2). Meanwhile, the correlation between transmural circumferential strain gradient only existed in AS group and more prominent in relationship with LVEF.

We divided the both groups into 2 subgroups based on the transmural strain gradient above or below the median value (longitudinal: 4.6% and 3.5%; circumferential 22.2% and 19.3%, respectively). In AS, results showed that the trans-aortic peak velocity (Vmax) and indexed aortic valve area (AVAi) were not significantly different between these two subgroups, but the LVEF was significantly lower (62.8 ± 12.2% vs. 70.2 ± 7.6%, $p < 0.001$; 62.8 ± 12.3% vs. 69.9 ± 7.6%, $p < 0.001$) and the average E/E′ was significantly higher (20.6 ± 10.7 vs. 15.8 ± 7.0, $p = 0.003$; 20.3 ± 11.2 vs. 16.2 ± 6.9, $p = 0.017$) in the low transmural strain gradient subgroup (Table 2). Compared to AS, in HCM, results showed only the LV ejection fraction was significantly lower in lower longitudinal gradient subgroup (64.6 ± 9.9% vs. 70.2 ± 7.4%, $p = 0.002$). Other hemodynamic profiles were no significant different between subgroups (Supplementary Table 3).

3.3. Reproducibility

The inter- and intra-observer mean percentage errors were 9.27 and 6.74% for subendocardial myocardium longitudinal strain, respectively; 7.21 and 6.86% for subepicardial myocardium longitudinal strain, respectively; and 4.48 and 5.14% for GLS, respectively. The intraclass correlation coefficient of inter- and intra-observer reliability were 0.950 (95% CI: 0.871–0.981) and 0.972 (95% CI: 0.927–0.989) for subendocardial myocardium longitudinal strain, respectively; 0.943 (95% CI: 0.852–0.978) and 0.972 (95% CI: 0.928–0.989) for subepicardial myocardium longitudinal strain, respectively; and 0.915 (95% CI: 0.779–0.967) and 0.77 (95% CI: 0.939–0.991) for GLS, respectively.

4. Discussion

Our study confirmed that the layer-specific strains existed different distribution pattern between AS and HCM groups, both in longitudinal strain and circumferential aspects. The layer-specific strain and derivative transmural gradient may also reflect patient's hemodynamic status. When we compared the two subgroups of AS group - divided by transmural longitudinal or circumferential strain, the low strain gradient subgroup, with similar AS severity, were associated with lower LVEF, and higher predicted LV filling pressure. This is the first study with a large sample to directly compare the layer-specific strains in patients with hypertrophied myocardium from different etiologies and to explore its clinical application.

4.1. Different strain distribution pattern in hypertrophied myocardium

In longitudinal strain, both the AS group and HCM group had significantly lower layer-specific strain values than did the Healthy control group, but the transmural longitudinal strain gradient was more preserved in the AS group (Supplementary Fig. 2). The difference remained significant even after adjusted for global longitudinal strain, age, gender, LV mass index, diastolic blood pressure, and average E/E′ in multivariate analysis.

In circumferential strain, compared to the significance of longitudinal strain, all layer-specific strain values seem less affected, and only the subendocardial myocardium had statistical difference from the healthy control. However, when we observe the correlation between LV mass index and layer-specific strain values (Fig. 1), we can still tell the difference exited between these two groups. Unlike longitudinal strain that affected all layers in both groups significantly, the subepicardial myocardium circumferential strain had no correlation with the mass index, and only the subendocardial myocardium in AS group declined significantly as the myocardium get more hypertrophied. The subendocardial circumferential seems more susceptible to pressure overload condition, but less affected by intrinsic myocardium disease in HCM group.

4.2. Transmural strain gradient as a marker and correlation with hemodynamics

In both diseased groups, the transmural longitudinal strain gradient correlated significantly with the LVEF, and the average E/E′ (Fig. 2). Although E/E′ is less reliable for assessing LV filling pressure in HCM then in AS group, this correlation shown in individual group could represent more advanced hemodynamic deterioration in the same population. We still used E/E′ as an important parameters in our study due to E/E′ was correlated with worse outcomes in many situations

Fig. 2. The correlation of transmural longitudinal and circumferential strain gradient with the left ventricular ejection fraction (LVEF) and the average early mitral velocity with the early diastolic annular velocity ratio (E/E′) in the HCM and the AS groups. A: The top left and top right panels show a significantly positive correlation of LVEF with the transmural longitudinal strain gradient (top left and top right). The bottom left and bottom right panels show a significantly inverse correlation of the average E/E′ with the longitudinal strain gradient. B: In contrary to longitudinal, the correlation between transmural circumferential strain gradient only existed in AS group and more prominent in relationship with LVEF.

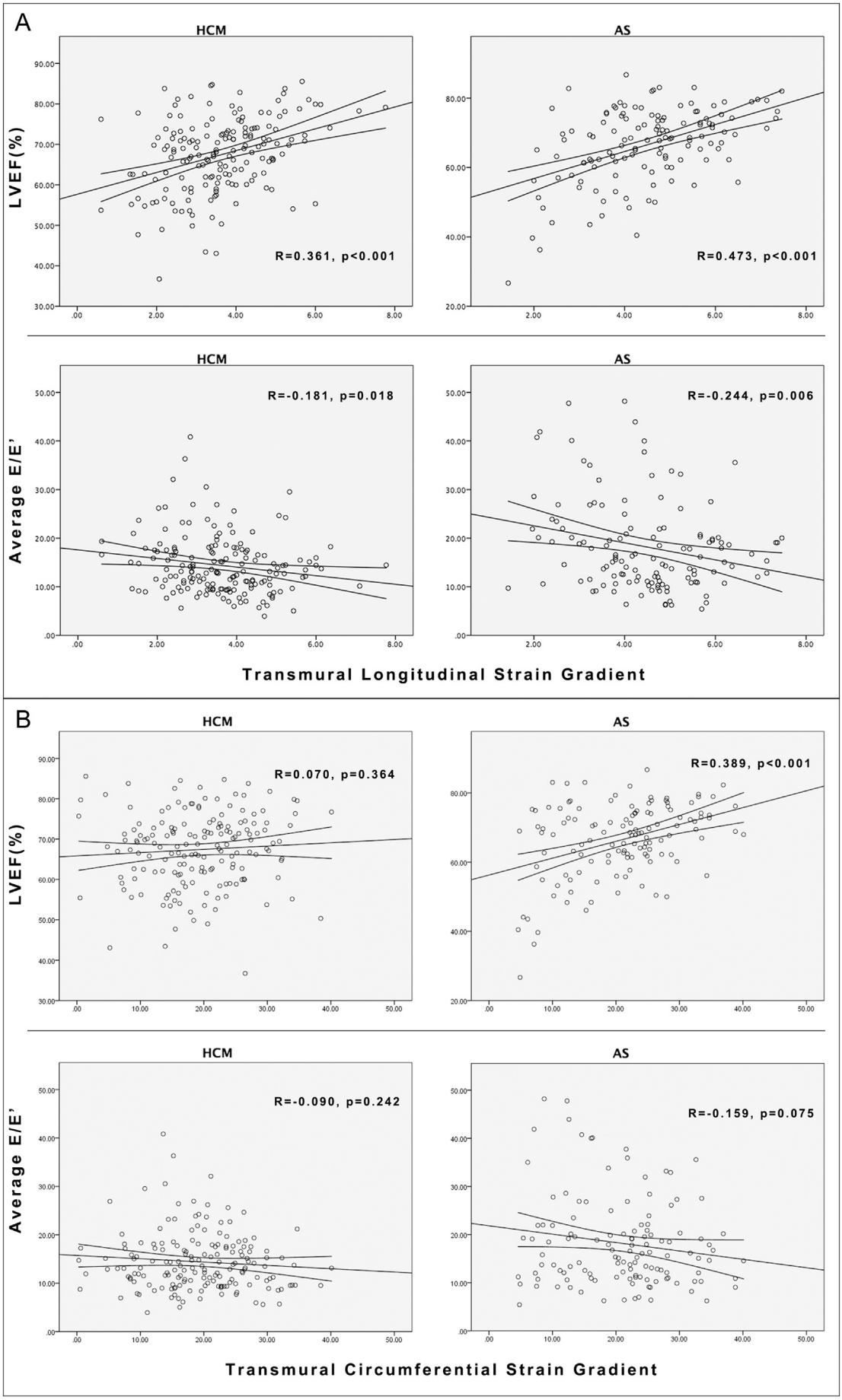

A
HCM
AS
LVEF(%)
R=0.361, p<0.001
R=0.473, p<0.001
HCM
AS
Average E/E'
R=-0.181, p=0.018
R=-0.244, p=0.006
Transmural Longitudinal Strain Gradient
B
HCM
AS
LVEF(%)
R=0.070, p=0.364
R=0.389, p<0.001
HCM
AS
Average E/E'
R=-0.090, p=0.242
R=-0.159, p=0.075
Transmural Circumferential Strain Gradient

Table 2
Parameters of the two AS subgroups: transmural strain gradient above and below the median (longitudinal: 4.6%; circumferential: 22.2%).

Variable	Transmural longitudinal strain gradient		p	Transmural circumferential strain gradient		p
	<4.6% (n = 64)	>4.6% (n = 65)		<22.2% (n = 62)	>22.2% (n = 65)	
LV myocardium mass index (mg/m^2)	143.1 ± 53.1	123.9 ± 40.0	0.025	144.1 ± 54.7	123.7 ± 38.7	0.020
Left atrium volume	120.1 ± 47.4	111.9 ± 43.8	0.310	124.5 ± 48.9	108.7 ± 41.6	0.052
Average E/E′	20.6 ± 10.7	15.8 ± 7.0	0.003	20.3 ± 11.2	16.2 ± 6.9	0.017
LV ejection fraction (%)	62.8 ± 12.2	70.2 ± 7.6	<0.001	62.8 ± 12.3	69.9 ± 7.6	<0.001
Trans-aortic valve maximal velocity (m/s)	4.4 ± 0.93	4.3 ± 0.92	0.454	4.5 ± 0.95	4.3 ± 0.86	0.274
Aortic valve area index (cm^2/m^2)	0.85 ± 0.40	0.97 ± 0.42	0.123	0.90 ± 0.40	0.92 ± 0.42	0.691

LV: Left ventricle.

and reserved as an important diastolic function parameter according to current guideline [21]. These findings supported that transmural longitudinal strain can serve as a marker for severity of both groups. In circumferential aspect, comparable to Fig. 2, the transmural circumferential strain gradient had no significant correlation in HCM group, but do have significant correlation in AS group of LVEF ($p < 0.001$) and trend in LV filling pressure ($p = 0.075$). The rationale we choose transmural gradient instead of subendocardial circumferential strain as the marker was that, by subtracting the subepicardial strain, transmural strain gradient might be less affected by aging since both subendocardial and subepicardial strain would be influenced by age. The circumferential transmural strain gradient may also be applied as a marker of severity in AS group.

4.3. Transmural strain gradient: possible hint of myocardium failure in AS

The transmural longitudinal strain gradient was more preserved in the AS group compared to HCM group. A possible explanation was HCM affects entire layers of myocardium with intrinsic myocardial failure. In contrast to the pathological myocardium in HCM, the hypertrophied myocardium in AS patients might be able to maintain the transmural longitudinal strain gradient as the primary response to the pressure-overloaded environment. Therefore, the transmural longitudinal strain gradient might reflect the compensatory mechanism of myocardium, and the loss of that might indicate intrinsic myocardial dysfunction. In the other hand, the transmural circumferential strain gradient was more susceptible in the AS group than in HCM, hence the decline of the gradient may also indicate myocardium dysfunction in AS.

We, like Koya Osawa et al., found a positive correlation between subendocardial and subepicardial myocardium strain ratio and the AS severity (Vmax and AVAi) [5]. Additionally, we found that the transmural longitudinal strain gradient was associated with LV performance instead of AS severity. An earlier study by Ozawa et al., which compared longitudinal and circumferential layer-specific strain ratio between heart failure HCM patients with preserved LVEF and Healthy controls [14], reported that all layers of longitudinal strain and transmural strain gradient were lower in HCM patients. Kim et al. described layer-specific myocardial strain in AS patient before and after pressure unloading by TAVR [6]. All layer-specific longitudinal strain of myocardium improved after TAVR, especially in the subendocardial myocardium. Furthermore, the transmural longitudinal strain gradient increased after unloading. The tendency of the transmural strain gradient to increase or normalize after unloading hypertrophied myocardium in AS patients indicates that the gradient can be used as a marker for intrinsic myocardial properties.

4.4. Limitations

This study has the first limitation of a single-center design. To strengthen and underpin this technique, further validations of the prognostic findings with efforts to find optimal cut points at multiple centers are required. Future outcome studies, both in AS and HCM arms incorporating the transmural strain gradient are also of interest. Second, the measurement of LV mass index from 2D M-mode based on geometric formulas to calculate the volume of LV myocardium, which would be relatively imprecise in HCM group considering asymmetric distribution. For most precise condition we may need cardiac magnetic resonance imaging or 3D echocardiography for asymmetric myocardium, however they are not practical in clinical practice, since cardiac magnetic resonance imaging is expensive and the 3D echocardiography measurement of LV mass are not yet well validated, and relies on sufficient spatial and temporal resolution for analysis [19]. Third, all of the subjects were from outpatient clinics and most of them were in stable condition, and in functional classes I and II. Still, this population is closer to real-world circumstances, because in Taiwan, most asymptomatic patients opt for conservative treatment and wait until symptoms develop. Finally, our strain values are vendor specific, so the cutoff value derived here may not be applicable to other systems.

5. Conclusion

Myocardium hypertrophy from different etiology resulted in different layer-specific strain distribution pattern. AS patients had more preserved transmural longitudinal strain gradient and more susceptible transmural circumferential strain than did HCM patients. The loss of an adequate transmural strain gradient correlated with hemodynamics and might reflect intrinsic myocardial dysfunction in AS, and deserve long term prognosis investigation.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2019.01.044.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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