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Heterogeneous Distribution of Left Ventricular Contractile Injury in Chronic Aortic Insufficiency

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Abstract

Background—Global systolic strain has been described previously in patients with chronic aortic insufficiency (AI). This study explored regional differences in contractile injury.

Methods—Tagged magnetic resonance images of the left ventricle (LV) were acquired and analyzed to calculate systolic strain in 42 patients with chronic AI. Multiparametric systolic strain analysis was applied to relate cardiac function in AI patients to a normal strain database (N = 60). AI patients were classified as having normal or poor function based on their results. A two-way repeated-measures analysis of variance was applied to analyze regional differences in injury.

Results—The mean and standard deviation of raw strain values (circumferential strain, longitudinal strain, and minimum principal strain angle) are presented over the entire LV in our normal strain database. Of the 42 patients with AI, 15 could be defined as having poor function by multiparametric systolic strain analysis. In AI patients with poor function, statistical analysis showed significant differences in injury between standard LV regions ($F_{369,44.33} = 3.47$, $p = 0.017$) and levels ($F_{1,49,17.88} = 4.41$, $p = 0.037$) of the LV, whereas no significant differences were seen in the group with normal cardiac function.

Conclusions—Patients with poor function, as defined by multiparametric systolic strain z scores, exhibit a consistent, heterogeneous pattern of contractile injury in which the septum and posterior regions at the base are most injured.

Heart failure secondary to left ventricular (LV) contractile dysfunction is the final common pathway of primary or secondary contractile diseases of the myocardium. In ischemic cardiomyopathy, the pattern of distribution of contractile dysfunction is regionally heterogeneous, being dependent on region-specific coronary arterial blood supply. In contrast, myocyte disease mediated by valvular insufficiency/stenosis or autoimmune/infectious (viral) processes would reasonably be expected to affect all LV myocytes in a uniform manner, resulting in a homogeneous pattern of contractile injury distribution. However, we and others have shown a consistent pattern of heterogeneous injury in patients with dilated cardiomyopathy [1–3].

Many patients with chronic aortic insufficiency (AI) still retain normal LV function. Over time, the LV dilates with subsequent hypertrophy and progresses in some patients to ventricular dysfunction and symptoms of heart failure [4]. Aortic valve replacement inconsistently results in a return of normal cardiac function [4–6].

Advanced noninvasive imaging techniques, such as magnetic resonance imaging (MRI) (eg, phase-contrast MRI [7, 8], tagged MRI [9–12], displacement encoding with stimulated echoes MRI [13], and harmonic phase MRI [14]) and ultrasound imaging (eg, tissue Doppler [15] and speckle tracking [16]) provide quantitative measures of systolic strain in the heart. Regional strain patterns in patients with AI are significantly different than in controls, as detected using velocity vector imaging [17], tissue Doppler imaging [18,19], and tagged MRI [20–22]. These studies have characterized regional differences between patients with chronic AI and control individuals, and we conducted this study to explore regional differences within this population.

Systolic circumferential and longitudinal strains vary between regions in the normal human heart. For this reason, multiparametric systolic strain analysis [23] relates 3-dimensional (3D) strain data in an individual to a normal strain database (N = 60), which provides a normalized, local measure of contractile injury over the entire LV. We successfully applied this approach to describe a consistent, regionally heterogeneous pattern of cardiac dysfunction in patients with dilated cardiomyopathy [1] and in sheep after ligation of blood supply to the anterior and apical LV walls [24].

In this investigation, we apply multiparametric systolic strain analysis to characterize regional injury in patients with chronic AI. We divided our AI study group patients into two subgroups: normal cardiac function and poor cardiac function (as defined by multiparametric systolic strain analysis). Further subgroup analysis allows the testing of the hypothesis that a consistent pattern of heterogeneous contractile injury distribution characterizes chronic AI patients with poor LV contractile function.

Material and Methods

The Human Research Protection Office at Washington University, St. Louis, Missouri, approved this study. All participants gave informed written consent.

Patient Characteristics

Sixty healthy volunteers (28 men, 32 women), age 33 ± 11 years, with no known cardiac disease, underwent strain analysis and contributed complete LV systolic strain information to a normal human strain database. A group of 42 patients with isolated grade 2+ to 4+ chronic AI also underwent multiparametric systolic strain analysis. Two of the 42 AI patients were excluded because of additional cardiac pathologies (mitral regurgitation and coronary artery disease), and 6 patients were excluded because of poor image quality, leaving 34 AI patients. Select clinical data from the AI patients are presented in Table 1.

Magnetic Resonance Imaging

Electrocardiogram-gated short-axis and long-axis tagged MRIs were acquired from end-diastole through systole using a 1.5-T Sonata scanner (Siemens Medical Systems, Malvern, PA). In each imaging plane, a spatial modulation of magnetization (SPAMM) radio-frequency tissue-tagging preparation [9, 25] was applied, followed by 2D-balanced steady-state free precession cine image acquisition. Short-axis images covered the entire heart, and long-axis images were acquired in four radially oriented planes. Typical imaging parameters were tag spacing, 8 mm; slice thickness, 8 mm; repetition time, 30.3 ms; echo time, 2.2 ms; field of view, 306×350 mm; and image matrix, 168×256 . In the same breathhold, anatomic and tagged images were acquired at corresponding slice positions.

Strain Analysis

Strain is a measure of deformation that relates a deformed configuration to a reference configuration. A detailed account of our strain analysis has been previously reported [26, 27]; however, a brief description is provided below. A semiautomated algorithm was applied to identify the endocardial and epicardial walls of the LV in the short-axis anatomic images (Fig 1A). Long-axis walls were identified manually (Fig 1A).

A semiautomated tag-finding algorithm, based on pixel intensity, identified the intersections of tag lines at each time point in the short-axis and long-axis tagged images (Fig 1B). Displacements were calculated from end-diastole to end-systole at each intersection point within the myocardium (Fig 1C). For registration, the user selected the posterior and anterior boundaries of the septum using the right ventricular free wall endocardial intersection points with the septum on the most basal short-axis image (Fig 1D). These two points and information from the LV geometry were used to register each heart to a standard *p*-version finite-element mesh (Fig 1E). Within each element of the finite-element mesh, displacements were fit in the least-squares sense to basis functions [28]. Continuity of displacement components was enforced at the element boundaries. The fitting of basis functions to approximate the displacement data and the calculation of strain was performed using Stress-Check (ESRD Inc, St. Louis, MO).

MRI-Based Multiparametric Strain Z-Score Analysis

Owing to the normally heterogeneous pattern of LV strain, raw values must be normalized to supply clinically relevant information. Our laboratory previously described and tested a method (multiparametric strain analysis) to relate an individual's function to a normal strain database [1, 23]. The *z* scores generated from raw strain values represent the number of standard deviations each raw value is from the mean of the group. Our composite multiparametric systolic strain *z* score combines three strain metric *z* scores (circumferential strain, longitudinal strain, and minimum principal strain angle) into one average *z* score. The minimum principal strain angle is the angle between the plane formed by the circumferential and longitudinal axes and the axis of minimum principal strain [29].

The mean and standard deviation for each of the three strain measures were calculated at each point of an encompassing grid of 15,300 discrete LV points for the 60 normal volunteers. By comparison with this normal strain database, patient-specific *z* scores for each of the three strain measures were calculated and averaged into one multiparametric strain *z* score that represents LV point-specific contractile function relative to normal. For each AI patient, *z* scores were averaged over six regions (posterior septum, anterior septum, anterior, anterior lateral, posterior lateral and posterior) and three levels (base, mid, and apex).

Poor Function vs Normal Function in Patients With AI

AI patients were split into two groups: normal and poor cardiac function. Those who had at least 2 of the 18 LV regions with a multiparametric *z* score of 1 or higher, which according to previous analyses is a good estimate of contractile injury, were placed in the poor cardiac-function group (*n* = 15) and the remainder in the normal-function group (*n* = 19).

Statistical Analysis

For statistical analyses, a two-way repeated-measures analysis of variance was applied to test the null hypothesis that injury, as measured by multiparametric systolic strain *z* scores, was uniform over the entire LV. The alternative hypothesis was that a consistent pattern of heterogeneous injury existed in AI patients with poor function. Because of our previous experience in analyzing other cardiac diseases in which the septum is generally more injured

than the lateral wall, planned contrasts were defined to test for significant differences between these regions.

The data were analyzed as a whole, then split into the normal-function and poor-function groups and analyzed again. *F* statistics were calculated to compare the null and alternative hypotheses. SPSS software (SPSS Inc, Chicago, IL) was used for all statistical computations.

Results

Raw Strain in Normal Controls

Figure 2 contains contour plots on a standard heart mesh of the mean and standard deviation of raw circumferential strain (Fig 2A), longitudinal strain (Fig 2B), and minimum principal strain angle (Fig 2C). Average circumferential strain is higher in magnitude at the endocardial wall (−0.25 to −0.35) compared with the epicardial wall (−0.05 to −0.15), with a smooth transition between the two. The standard deviation is less than 0.10 over most of the heart.

Average longitudinal strain is more constant, varying between −0.10 and −0.20 over most of the heart, with slightly higher values of −0.15 to −0.30 in the apex. Again, the standard deviation is mostly less than 0.10 over most of the heart, with the lower half of the apex having higher values (0.10 to 0.20).

Average minimum principal strain angles range from 10 to 20 degrees over most of the heart. The lower half of the apex contains much higher angles (20 to 35 degrees). The standard deviation is less than 15 degrees over most of the heart, with the lower half of the apex having a higher deviation.

Multiparametric Systolic Strain Z Scores

Figure 3 shows the mean and standard deviation of multiparametric systolic strain *z* scores for the total AI group (Fig 3A), the normal-function group (Fig 3B), and the poor cardiac-function group (Fig 3C). In the entire AI group, average multiparametric *z* scores ranged between −1.0 and 1.0. The endocardial wall tended to have lower values than the epicardial wall. Higher values appeared in the posterior septum, anterior septum, and posterior regions compared with the anterior lateral and posterior lateral regions, particularly at the base.

The normal-function group has many average multiparametric *z* scores that are less than 0, indicating hyper-contractility in those regions. No major differences between regions are seen qualitatively. In the poor-function group, average multiparametric *z* scores are higher (worse contractile function) than the normal-function group, with the highest values present at the base in the anterior septum, posterior septum, and posterior regions.

Heterogeneous Regional Injury

A two-way repeated-measures analysis of variance was applied to test for differences in multiparametric systolic strain *z* scores of the six LV regions and three LV levels. The means of the regionally averaged multiparametric *z* scores are provided in Table 2.

When the entire group of AI patients was considered, the Mauchly test indicated the assumption of sphericity was violated for the levels ($\chi^2 = 10.51$, $p = 0.005$) and the regions ($\chi^2 = 61.32$, $p < 0.001$). We corrected the number of degrees of freedom using the Huynh-Feldt estimates of sphericity ($\epsilon_{levels} = 0.78$, $\epsilon_{regions} = 0.61$). No significant main effect was seen between the levels ($F_{1.56,40.52} = 2.07$, $p > 0.05$) or regions ($F_{3.03,78.81} = 2.61$, $p > 0.05$) and the amount of injury.

For the normal-function group, the Mauchly test indicated the assumption of sphericity was violated for the regions ($\chi^2 = 33.34$, $p = 0.003$) but not the levels ($\chi^2 = 1.73$, $p > 0.05$). We corrected the number of degrees of freedom using the Huynh-Feldt estimates of sphericity ($\epsilon_{regions} = 0.57$). No significant main effect was seen between the levels ($F_{2,26} = 3.29$, $p > 0.05$) or regions ($F_{2,86,37.13} = 0.575$, $p > 0.05$) and the amount of injury.

For the poor-function group, the Mauchly test indicated the assumption of sphericity was violated for the levels ($\chi^2 = 8.16$, $p = 0.017$) and the regions ($\chi^2 = 37.12$, $p = 0.001$). We corrected the number of degrees of freedom using the Huynh-Feldt estimates of sphericity ($\epsilon_{levels} = 0.73$, $\epsilon_{regions} = 0.66$). A significant main effect was seen between the regions and the amount of injury ($F_{3,69,44.33} = 3.47$, $p = 0.017$). Contrasts revealed a significant difference between the posterior and anterior lateral regions ($F_{1,12} = 8.15$, $p = 0.014$) and the anterior lateral and posterior septum regions ($F_{1,12} = 6.43$, $p = 0.026$). Additional contrasts were tested between the anterior and posterior regions ($F_{1,12} = 3.84$, $p = 0.074$) and the posterior septum and posterior lateral regions ($F_{1,12} = 4.53$, $p = 0.055$) that trended towards significance. A significant main effect was also seen between the levels and the amount of injury ($F_{1,49,17.88} = 4.41$, $p = 0.037$). Contrasts revealed a significant difference between the base and the apex ($F_{1,12} = 5.46$, $p = 0.038$) but no significant difference between the base and mid ($F_{1,12} = 2.61$, $p > 0.05$).

Comment

Heterogeneous Distribution of Contractile Injury

AI patients in the normal-function group had average multiparametric z scores that were negative, indicating greater contractility than normal averages. Factors contributing to this finding may include the Starling effect or an increased adrenergic state. In the patients with poor function, statistical analysis showed significant main effects between the regions and levels. In particular, the posterior and posterior septal regional contractile function was significantly worse than that in the lateral regions, and the basal was significantly worse than the apical, suggesting a predilection toward basal septal injury. These results are surprising given that the volume-overload physiology of AI is thought to affect all LV myocytes in a homogeneous manner.

Clinical Significance

The injurious influences of the volume-overload physiology imposed on LV myocytes by chronic AI should have no predilection for basal level myocytes over those in the apical level, nor should they have a preference for the septal myocytes over those in the LV free wall. This application of MRI-based multiparametric strain analysis in patients with chronic AI suggests precisely such a predilection in the subgroup of patients with reduced LV contractile function. This pattern of heterogeneous contractile injury distribution mirrors what we have previously reported in patients with dilated cardiomyopathy [1].

The mechanism by which a global and, presumably, uniform distribution of the injurious influence translates into a consistent pattern of heterogeneous contractile injury distribution is unclear. Although it is the various associated pathophysiologic influences that injure the contractile capabilities of the human cardiac myocyte, our results suggest that it is not the injurious vectors themselves that determine the distribution of the resulting contractile injury. The heterogeneous distribution of injury seems to have a relatively consistent pattern, suggesting that factors that are consistently present—perhaps inherent—in every heart, such as regionally varying geometric influences, may contribute to the susceptibility of one region over another. These geometric influences, such as the LV chamber wall tethering by the mitral subvalvular apparatus or the junction points of the right ventricular free wall to the

LV, almost assuredly have a significant influence on stress distribution in the LV and are worthy of further investigation.

In regard to mechanistic definition, however, we must remember that this study is focused solely on the quantification of regional LV normalized strain injury in chronic AI. Although our results offer no specific insight into the mechanism of injury, if confirmed by others, they may provide a valuable springboard for further mechanistic investigation.

This study has some limitations. Strains are calculated using tagged MRI by tracking the intersection of tag planes in 3D. Although 3D displacements were measured, only two of the six strain components (circumferential and longitudinal strain) were used in our multiparametric analysis, which was due to limited-displacement measurement in the radial direction. Recent advanced MRI methods to estimate strain in the human heart, such as 3D harmonic phase [30], 3D oblique tag lines [31], and 3D displacement encoding with stimulated echoes [32], may provide higher resolution of 3D displacement measurements within a reasonable scan time for patients. A higher density of accurate displacement data may improve the estimation of all six components of strain and allow for more sensitive comparisons.

Image sets from different imaging planes are acquired during separate breath holds, which can lead to registration errors. To minimize these errors, patients are instructed to be as consistent as possible with their breath holds, and practice breath holds are performed.

Tag lines in SPAMM images tend to fade over time due to T1 relaxation. To reduce tag fading, sequences using complementary SPAMM have been developed [11, 12] that allow the tags to persist throughout the cardiac cycle. In our experience with the sequence used for this study, tags consistently persist for the entire systolic period. Thus, we do not believe that significant error was introduced into our measurements as a result of tag fading associated with SPAMM imaging.

A major assumption in our analysis is the determination of a point-to-point correspondence between the LV of different individuals. Although not exact, our approach provides a reasonable match between individuals. The small and uniform standard deviation values of the strain components seen over the mid and base levels of the LV in our normal strain database reassure us that the assumptions made during registration are reasonable.

In conclusion, multiparametric systolic strain analysis provides a normalized characterization of regional LV contractile injury. In this investigation, a consistent, heterogeneous pattern of contractile dysfunction was demonstrated in patients with poor cardiac function due to chronic AI, despite the presumed homogeneous LV distribution of the injurious pathophysiology of volume overload. Because the pattern of contractile injury distribution is similar to that demonstrated in patients with dilated cardiomyopathy, we are encouraged to extend future investigation to other patient subsets where a homogeneous pattern of myocyte injury is expected (eg, aortic stenosis, mitral regurgitation). The potential diagnostic and therapeutic consequences of this new paradigm of contractile injury distribution, such as earlier irreversible injury detection in valvular heart disease by the focusing of contractile metrics upon “sentinel” LV regions (where injury occurs earliest), deserve further clinical investigation.

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Fig 1.

Image acquisition and postprocessing steps: Long-axis and short-axis anatomic and tagged magnetic resonance images of the left ventricle were acquired between (A) end-diastole and (B) end-systole. Myocardial boundaries and tag lines were identified in each image. (C) Displacement vectors were calculated in 3 dimensions. (D) A standard p-version finite-element mesh was registered to the individual's left ventricle geometry. The p-version finite-element analysis was used to fit the components of displacement over the entire left ventricle and calculate the strain. (E) The z scores relative to the normal strain database (N = 60) were calculated for three strain measures and combined into one multiparametric systolic strain z score.

Fig 2.

Mean and standard deviation of raw strain in normal controls of (A) circumferential strain (E_{CC}), (B) longitudinal strain (E_{LL}), and (C) minimum principal strain angle. More deviation is seen at the cap of the apex than the rest of the left ventricle. The model is divided into three levels, base, mid, and apex, and into six regions, comprising posterior (P), posterior lateral (PL), anterior lateral (AL), anterior (A), anterior septum (AS), and posterior septum (PS).

Fig 3. Mean and standard deviation of multiparametric z scores are shown for the total (A) aortic insufficiency (AI) group, and the AI groups with (B) poor cardiac function and (C) normal cardiac function. Note the difference in the scale bars between the mean and standard deviation plots. The model is divided into three levels, base, mid, and apex, and into six regions, comprising posterior (P), posterior lateral (PL), anterior lateral (AL), anterior (A), anterior septum (AS), and posterior septum (PS).

Table 1
Patient Characteristics

Variable ^a	AI	Cardiac Function	
		Poor	Normal
Sex			
Male	28	15	13
Female	6	0	6
Age, years	44 ± 15	46 ± 15	42 ± 14
Height, meters	1.8 ± 0.1	1.8 ± 0.1	1.8 ± 0.1
Weight, kg	82 ± 12.8	84.9 ± 13.5	79.9 ± 12.2
Ejection fraction	0.597 ± 0.117	0.555 ± 0.127	0.630 ± 0.127
EDV, mL	187.6 ± 82.1	212.9 ± 74.5	167.7 ± 81.9
ESV, mL	75.6 ± 46.8	94.7 ± 56.3	62.1 ± 30.5
AI degree, 1+ to 4+	3.4 ± 0.6	3.7 ± 0.4	3.2 ± 0.6

^aCategoric data are presented as number and continuous data as mean ± standard deviation.

AI = aortic insufficiency; EDV = end diastolic volume; ESV = end systolic volume.

Table 2
Regional Multiparametric Systolic Strain z Scores^a

Level ^b	Region	AI	Cardiac Function	
			Poor ^c	Normal
Base	PS	0.38 ± 0.91	1.18 ± 0.78	-0.25 ± 0.34
	AS	0.47 ± 0.92	1.14 ± 0.97	-0.06 ± 0.39
	A	0.27 ± 0.68	0.75 ± 0.64	-0.11 ± 0.44
	AL	0.17 ± 0.62	0.54 ± 0.63	-0.12 ± 0.44
	PL	0.24 ± 0.59	0.72 ± 0.40	-0.13 ± 0.41
	P	0.39 ± 0.85	1.19 ± 0.55	-0.24 ± 0.35
Mid	PS	0.25 ± 0.71	0.81 ± 0.56	-0.19 ± 0.47
	AS	0.19 ± 0.63	0.63 ± 0.61	-0.15 ± 0.40
	A	0.21 ± 0.61	0.66 ± 0.62	-0.14 ± 0.29
	AL	0.06 ± 0.57	0.51 ± 0.53	-0.29 ± 0.27
	PL	0.15 ± 0.59	0.63 ± 0.48	-0.25 ± 0.29
	P	0.29 ± 0.70	0.86 ± 0.52	-0.16 ± 0.45
Apex	PS	0.23 ± 0.88	0.56 ± 1.07	-0.05 ± 0.58
	AS	0.19 ± 0.88	0.63 ± 0.98	-0.15 ± 0.48
	A	0.36 ± 0.85	0.63 ± 0.98	0.02 ± 0.63
	AL	0.21 ± 0.76	0.72 ± 0.58	-0.16 ± 0.66
	PL	0.28 ± 0.87	0.82 ± 0.88	-0.14 ± 0.59
	P	0.27 ± 0.97	0.64 ± 0.98	-0.04 ± 0.87

^aData are presented as mean ± standard deviation.

^bLevels: base vs apex, $p = .038$.

^cPoor function group: regional: P vs AL, $p = .014$; AL vs PS, $p = .026$.

A = anterior; AI = aortic insufficiency; AL = anterior; AS = anterior septum; P = posterior; PL = posterior lateral; PS = posterior septum.