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The Effect of Trabeculae Carneae on Left Ventricular Diastolic Compliance: Improvement in Compliance With Trabecular Cutting

The role of trabeculae carneae in modulating left ventricular (LV) diastolic compliance remains unclear. The objective of this study was to determine the contribution of trabeculae carneae to the LV diastolic compliance. LV pressure–volume compliance curves were measured in six human heart explants from patients with LV hypertrophy at baseline and following trabecular cutting. The effect of trabecular cutting was also analyzed with finite-element model (FEM) simulations. Our results demonstrated that LV compliance improved after trabecular cutting ($p < 0.001$). Finite-element simulations further demonstrated that stiffer trabeculae reduce LV compliance further, and that the presence of trabeculae reduced the wall stress in the apex. In conclusion, we demonstrate that integrity of the LV and trabeculae is important to maintain LV stiffness and loss in trabeculae leads to more LV compliance. [DOI: 10.1115/1.4035585]

Keywords: trabeculae carneae, LV compliance, wall stress, stiffness, hypertrophy, surgery

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Manuscript received May 25, 2016; final manuscript received December 13, 2016; published online January 24, 2017. Assoc. Editor: Thao (Vicky) Nguyen.

Table 1 Donor information on six trabecular cutting hearts, and two control hearts

Control heart #	Trabecular cutting							Control	
	1	2	3	4	5	6	Mean $\pm$ SD	7	8
Sex	M	M	M	M	M	M	M	M	M
Age (yr)	46	64	65	68	53	74	61.7 $\pm$ 10.3	68	71
Heart weight (g)	561	614	445	755	687	664	621 $\pm$ 108	829	545
LV wall thickness (mm)	17.0	18.0	18.8	20.5	19.0	16.3	18.3 $\pm$ 1.5	20.0	15.5
Trabeculae cut at apex	26	12	9	42	21	58	28.0 $\pm$ 18.8	—	—
Trabeculae cut at septum	5	8	9	10	9	16	9.5 $\pm$ 3.6	—	—
Trabeculae cut at free wall	30	23	13	11	14	10	16.8 $\pm$ 7.9	—	—
Total trabeculae cut	61	43	31	63	44	84	54.3 $\pm$ 18.9	—	—
BMI (kg/m ²)	35.3	32.5	31.4	27.1	23.7	29.5	29.9 $\pm$ 4.1	34.6	25.1
History of diabetes	—	+	+	—	—	+	—	—	—
Index of hypertension	+	+	+	+	—	+	—	+	+
Cause of death	CA	CA	ESLF	CA	CA	CA	—	CA	ARF

Note: Abbreviations: BMI = body mass index; ARF = acute renal failure; CA = cardiac arrest; ESLF = end-stage liver failure; F = female; HF = heart failure; LV = left ventricular; M = male; SD = standard deviation.

Introduction

Left ventricular trabeculae carneae (henceforth trabeculae) have been generally considered embryologic remnants that play no functional role in the adult heart [1]. Trabeculae and papillary muscles account for more than 20% of LV mass [2–5] (trabeculae ~13% [4]). During embryologic morphogenesis of the heart, trabeculae are among the first features to arise in the developing cardiac tube, later undergoing a process known as compaction in which trabeculae condense to form the myocardium [1,6]. However, free-running trabeculae remain in the ventricles of human and mammalian hearts [6,7]. The free-running trabeculae occur mostly in the apex of mammalian hearts [7,8] where myocardium is thinnest and wall stress is therefore anticipated to be the highest.

There have been few proposed functional theories about trabeculae in the literature, but some suggested that trabeculae improve nourishment of myocardium during development [6,9], may help buffer the kinetics of blood flow in the ventricle [9,10], or assist in emptying blood more completely during systole [11]. Furthermore, excessive numbers of free-running trabeculae occur in pathological conditions, most notably left ventricular noncompaction, characterized by a layer of prominent trabeculae and deep intertrabecular recesses at least twice as thick as the compacted myocardium, which results in heart failure (HF) [12–14]. Prominent trabeculae occur in dilated cardiomyopathy, acquired LV hypertrophy due to hypertension, and congenital heart disease [6,12,15]. Trabecular hypertrophy also occurs in hypertrophic cardiomyopathy [5,16–18]. Recently, Gati et al. showed that increased LV trabeculation occurs in response to LV preload due to pregnancy [3], in patients with anemia [19], and in highly trained athletes [20]. Trabeculae are also known to become fibrotic in systolic heart failure; the fibrotic content of trabeculae carneae has been found to be up to 2.1 fold greater in failing hearts [21]. However, there have been no reports of how trabeculae affect LV function, especially LV diastolic compliance or wall stress in normal and hypertrophy hearts.

Accordingly, we hypothesize that trabeculae in human hearts are not merely embryologic remnants, but instead provide structural support. We suspect that trabeculae serve a mechanical role in affecting wall stress and diastolic compliance. The *objective* of this study is to determine the contribution of trabeculae carneae to LV wall stress and diastolic compliance. We compared the passive pressure–volume relationship of hypertrophic human hearts before and after trabecular cutting and used computational models to illustrate that a different extent of trabeculae cutting could lead to differences in LV chamber stiffness and wall stress.

Materials and Methods

Human Hearts Specimen Collection. Human hearts were obtained postmortem within 24 h of death from six male organ donors who died suddenly (Table 1). Hearts were de-identified in accordance with IRB requirements, and informed consent for research was obtained from the donors' families. Hearts were placed in ice-cold phosphate-buffered saline (PBS) solution to transfer to laboratory for testing within 24 h postmortem.

Trabeculae Carneae Definitions. For the purpose of this study, only free-running trabeculae (i.e., the body of trabeculae which were not fused to the ventricle wall) were examined. Delineation was not made between trabeculae carneae and trabeculae septo-marginalis sinistralis [22]. Thus, all trabeculations within the LV aside from papillary muscles and chordae tendineae were considered trabeculae carneae. Trabeculae were defined as bundles of muscle projecting from the inner surfaces of ventricle of the heart, or bundles of muscle connecting to other bundles of muscle in the lumen of the ventricle (Fig. 1). Many trabeculae connected only to other trabeculae to form “trabecular nets.” The location of trabeculae was defined into three regions: apical (lower 25% of the ventricle), septal (upper 75% of the ventricular septum), or free wall (upper 75% of the ventricular free wall) region.

Fig. 1 Longitudinal cross section of a typical human LV illustrating the trabeculae (examples marked by arrowheads). A chordae tendineae is marked by the arrow.

Fig. 2 Ex vivo experimental setup for human hearts. (a) A custom cap was attached to the heart above the mitral valve. A latex balloon was inserted through the cap into the LV chamber. The cap included a hemostasis valve to allow a high fidelity micromanometer pressure catheter to be placed within the balloon, and a luer lock for volume input. (b) Measurements of LV compliance were conducted with the heart suspended by the cap inside a custom housing chamber which was surrounded by 37°C water circulation to mimic in vivo conditions.

Baseline Compliance Measurement. The compliance of the LV was determined through pressure–volume loop measurement [23]. Briefly, each heart was placed in a physiologic PBS solution used in patients at 37°C to relax for 30 min before the testing. The left atrium of each heart was opened, and a custom cap (Fig. 2(a)) was sutured just above the mitral orifice. A thin-walled, tubular latex balloon was inserted through the cap into the LV and secured using polytetrafluoroethylene tape to create a fluid-tight seal. The heart was suspended by the cap inside of a 37°C temperature-controlled chamber (Fig. 2(b)). A high fidelity micromanometer pressure catheter (Transonic, Ithaca, NY) was inserted through a hemostasis valve in the cap into the balloon within the LV. The coronary arteries were perfused 20 min before and continuing throughout measurements, using a peristaltic pump with identical flow rate for all tests. To measure baseline compliance, saline was injected through the cap into the balloon in increments of 5 or 10 ml depending on heart size, and the pressure was

Fig. 3 Three-dimensional sectional view of the computational model of the left ventricle with three layers of trabeculae. All dimensions are in mm. The wall thickness is 13.5 mm at the equator and 5.5 mm at the apex.

recorded by a data acquisition system (ML750 Powerlab/4SP, AD Instruments, Colorado Springs, CO) until a pressure of 50 mm Hg was achieved.

Trabecular Cutting Procedure. Trabecular cutting was performed on six human hearts. Following baseline compliance measurements, the balloon was removed and the heart was suspended by the cap using a vise clamp. Through the cap, custom-made tools were used to sever all visible free-running trabeculae which connected to the ventricle wall. The number of trabeculae was determined by hand counting. The number and location (apical, septal, or free wall region) of the severed trabeculae were recorded.

Postprocedural Compliance Measurement. Following trabecular cutting, compliance was measured again as described above. Interference with the inflation of the balloon by uncut trabeculae was determined to be negligible, as the starting volumes where pressure develops were the same for each heart before and after trabecular cutting. Thus, differences between the compliance curves measured before and after trabecular cutting were interpreted as changes in the compliance of the LV chamber.

Two control hearts (#7–8) were examined to validate the compliance measurement procedure. In one control, the baseline measurement was repeated to determine whether repeated inflating the latex balloon in the LV would affect diastolic compliance (no trabeculae were cut). In the second control, a sham operation was done wherein the measurement system was removed from the heart after the initial measurement, the trabecular cutting procedure was mimicked using a blunt tool in which no trabeculae were actually cut, and the measurement system was then reintroduced to the heart. Furthermore, the baseline stiffness of the balloon alone was confirmed to be negligible (orders of magnitude lower) compared to the heart.

Heart Dimension Measurements. Following the postprocedural compliance measurement, hearts were weighed and LV wall thicknesses were measured midway between the apex and base at four locations along the circumference, at the two insertion points for the right ventricle (RV) wall and two symmetrical locations on the LV free wall. Average LV thickness was defined as the mean of these four measurements.

Quantification of Ventricular Compliance. To quantify the compliance and its change due to trabecular cutting, we fitted the pressure–volume curves with an exponential function

Fig. 4 A comparison of a typical LV wall region before (top panel) and after (bottom panel) trabecular cutting

$$P = A[e^{\beta(V-V_0)} - 1] \quad (1)$$

where P and V represent the pressure and lumen volume, A and β are constants, and V_0 is the initial volume at zero pressure. The parameter β represents the stiffness of the P – V response and was compared before and after the cutting to quantify the compliance changes.

Finite-Element Model (FEM) Simulations. To better understand the effects of different trabeculae cutting plan on the diastolic compliance and end-diastolic wall stress distribution in the myocardium, FEM was performed on a model LV. An ellipsoidal solid model of the LV was created based on previously reported shapes of human and canine hearts [7,24], but the dimensions were modified in the range of human heart used in this study. Briefly, the wall thickness was 13.5 mm at the equator and 5.5 mm at the apex with a cavity volume of 30 ml (Fig. 3). The myocardium was assumed to behave as a nonlinear anisotropic material with the Fung's exponential strain energy function W [25,26]

$$W = \frac{C}{2}(e^Q - 1) \quad (2)$$

$$Q = b_r E_{rr}^2 + b_f E_{ff}^2 + b_c E_{cc}^2 + 2b_{rf} E_{rc}^2 + 2b_{fc} E_{fc}^2 + 2b_{fs} E_{rf}^2$$

where b_r , b_f , and b_{fs} are material constants; E_{ij} ($i, j = r, f, c$) are strains and subscripts; and r, f , and c correspond to radial, fiber, and cross-fiber directions, respectively. The material constants and the fiber directions were obtained from a previous study on dog heart ($b_f = 26.7$, $b_r = 2$, $b_{fs} = 14.7$, $C = 1.2$ kPa) due to the lack of human heart data [25]. The myocardial wall was divided

into eight layers of equal thickness with different fiber orientations. The orientation of fibers with respect to the circumferential direction varied from -37° at epicardium to 98° at endocardium [25]. We used three layers of radially running trabeculae and one layer of longitudinal trabeculae that connects the three radially oriented layers in our model as it most closely matches the observed orientation of trabecular nets, which our results indicate as having the greatest impact on wall stress and compliance (Fig. 3). The trabeculae were assumed to have a diameter 2 mm (to mimic hypertrophic trabecular carneae muscles) and the same Fung's nonlinear anisotropic material with fibers aligned in the axial direction. The stiffness of trabeculae was also increased to determine the impact of hypertrophied/fibrotic versus normal trabeculae on LV diastolic compliance.

The finite-element analysis was performed using the commercial finite-element analysis package ABAQUS[®]. The myocardial wall was meshed using linear hexahedral elements (C3D8H), whereas the trabeculae were meshed using quadratic tetrahedral elements (C3D20H) to better fit their geometric shapes. Mesh size was selected after a pilot study on mesh sensitivity. The passive diastolic response of the LV was simulated by applying an internal pressure of up to 50 mm Hg on the endocardial wall to match the experimental load range. To prevent rigid body motion, kinematic boundary conditions were applied on the base of the LV by constraining the longitudinal displacement (being zero) of all the nodes of the base. In addition, the circumferential displacements of the nodes of the epicardial-basal edge were also constrained [27]. The variation of the internal cavity volume with the internal pressure was determined. Four simulations were run for comparison—two models with the same three layers of intact trabeculae but of different stiffness, a third model with only one layer of intact trabeculae near the apex (i.e., two layers of trabeculae most distant from the apex were cut off from the endocardial wall), and a fourth model without trabeculae (i.e., all the layers of trabeculae were cut off from the endocardial wall). The first and second simulations were run with the trabeculae intact but at normal stiffness ($b_f = 14.7$) or higher stiffness ($b_f = 26.7$) to illustrate the effect of stiffness of the trabeculae. The circumferential wall stress and fiber strain were reported at the apical and midventricle region of the LV.

Statistical Analysis. For LV wall thickness, whole-heart weight, and end-diastolic LV volume, we tested statistical differences between two groups with two-sided Student's t -test and analyzed statistical differences between more than two groups with one-way analysis of variance (ANOVA). For changes in LV passive compliance following trabecular cutting, we computed within-subject changes in pressure (uncut minus cut) and assessed the significance of the relation between changes in pressure and volume using a repeated measures mixed-effects cubic model and assuming an autocorrelation matrix. We used SAS Version 9.3 and all statistics testing was two-sided with a significance level of 5%.

Results

Heart Mass and Dimensions. For the six fresh human ex vivo hearts (Table 1), the donor's average age was 61.7 ± 10.3 yr and average BMI was 29.9 ± 4.1 kg/m². Of these donors, 50% had diabetes and 83.3% had hypertension. Overall, donor hearts had LV hypertrophy based on their wall thickness, whole heart weight, and chamber volumes [28,29]. The average LV wall thickness was 18.3 ± 1.5 mm (normal = 6–10 mm), [29] the average whole-heart weight was 621.0 ± 108.4 g (normal = 319 g), [30] and the average end-diastolic volume was 34.4 ml (normal = 67–155 ml) [29] and was defined as the LV chamber volume measured at an LV pressure of 10 mm Hg.

The Effects of Trabecular Cutting on LV Diastolic Compliance. For each human heart, all visible trabeculae were cut (Fig. 4). For the six experimental hearts that underwent trabecular

Fig. 5 Comparison of pressure–volume relationships of human hearts before and after trabecular cutting. Six human heart (a)–(f) and their average (G) pressure–lumen volume curves before (• = uncut) and after (○ = cut) trabecular cutting. For the average, volume was normalized to the highest volume measured for each heart. R^2 values correspond to cubic fits in all panels. For all six cases, trabecular cutting increased the compliance of the left ventricle.

cutting, we cut a total of 54.3 ± 17.2 trabeculae per heart, an average of 28.0 ± 17.2 trabeculae in the apical region, 9.5 ± 3.3 trabeculae in the septal region, and 16.8 ± 7.2 trabeculae in the free wall region. The pressure volume relationships of these six hearts before and after trabecular cutting are summarized in Fig. 5. As is visibly evident, there was an acute increase in passive LV compliance following trabecular cutting. The LV volumes at a diastolic pressure of 10 mm Hg were interpolated from the data in Fig. 5 and the results showed that the LV volume increased significantly ($p < 0.05$) from 34.4 ± 20.1 ml to 49.0 ± 33.0 ml (or an average of 37% increase in volume) after trabecular cutting. The stiffness parameter (β) decreased significantly from 0.036 ± 0.024 to 0.029 ± 0.019 ($p < 0.05$, paired t-test) following trabeculae cutting.

LV Compliance of Control Hearts. Tests from the two control hearts demonstrated that inflating the PV balloon in the LV did not affect diastolic compliance after repeated measurements. There was also no difference in the PV curve after the sham operation (Fig. 6).

LV Compliance and Wall Stress From Model Simulations. Finite-element simulations demonstrated that trabeculae shield the mechanical stress in the LV and trabecular cutting affects the wall stress and LV diastolic compliance. The LV diastolic compliance curve was steeper for the model with three intact layers of trabeculae compared to the model without trabeculae, anticipating the decrease in compliance of the LV with trabecular cutting (Fig. 7). The LV diastolic compliance curve was similar for the model

with only one intact layer (bottom apex layer) of trabeculae compared to the model without trabeculae. The stiffness parameter β for the LV models with all trabeculae, with only one apical layer of trabeculae, and without trabeculae were 0.065, 0.058, and 0.057 for the LV model with all trabeculae, with only one apical layer of trabeculae, and without trabeculae, respectively. These results demonstrated that a similar increase in compliance (i.e., a decrease in stiffness) of the LV could be achieved by severing the trabeculae network near the midventricle but retaining a few trabeculae near the apex. These results also suggest that cutting trabeculae closer to midventricle region would have larger effect on LV compliance than cutting trabeculae near the apex. Increasing the stiffness of the trabeculae resulted in an even steeper LV pressure-lumen volume relationship and therefore a further reduction in compliance (Fig. 7). In other words, in hearts with stiffer trabeculae, cutting trabeculae would result in a more significant effect on LV diastolic compliance.

In addition, our simulations demonstrated that trabeculae cutting has little effect on the circumferential stress in the midventricular region. The peak circumferential wall stress was at the apex for all models, and was highest in absence of all trabeculae (Fig. 8 and Table 2). Among the three models, the model without trabeculae had the highest LV compliance and the highest apical wall stress and strain. In the model with three intact layers of trabeculae, apical wall stress and strain were the lowest, with stress redistributed from the apex into the trabeculae and the LV compliance was the lowest as well. When the two upper layers of trabeculae were removed, and one layer was maintained at the apex, the model showed an increase in LV compliance similar to removing

Fig. 6 The pressure–volume relationship of control hearts without trabecular cutting. (a) Comparison of measurements of repeating saline filling in heart #7. The pressure measurement system remains in the LV during the tests. (b) Comparison of measurements after sham operation in heart #8. The pressure measurement system was removed from the heart after the first measurement, and trabecular cutting was mimicked with a blunt tool, but no trabeculae were cut. The pressure measurement system was then placed back into the heart for the second measurement. R^2 values correspond to cubic fits of the pressure–volume data. In both cases, there was no significant change in compliance in the absence of trabecular cutting.

all trabeculae, but with reduced apical wall stress and strain. These results suggest that partial cutting may achieve improved LV compliance without significantly increasing stress and strain in the apex.

Discussion

In ex vivo human hearts with LV hypertrophy, we demonstrated that severing trabeculae caused an acute increase in passive LV compliance. Finite-element simulations further demonstrated that trabeculae affect the LV compliance (pressure–volume relationship) and wall stress distribution within the LV. These results support our hypotheses that (A) trabeculae serve the functional role of providing mechanical support to the LV chamber during diastolic filling in hearts, (B) hypertrophy and fibrosis of trabeculae can contribute to decreased LV compliance in hearts with LV hypertrophy, and (C) trabecular cutting could increase LV compliance.

The finite-element analysis allowed us to examine the effect of removing selected trabeculae on LV compliance and wall stress, which is difficult to perform experimentally. Our results demonstrated that severing the network of free-running trabeculae could improve LV diastolic compliance. Though severing all trabeculae may lead to large stresses near the apex, which could result in long-term apical bulging and adverse LV remodeling [31,32] it can be mitigated by retaining some of the trabeculae near the apex. Maintaining the layer of trabeculae near the apex achieved the beneficial increase on LV compliance while reducing the possible stress and strain changes that would occur if all the trabeculae were severed.

The mass (size) of trabeculae can play a role in reducing the LV compliance. Though the change of trabeculae size was not

Fig. 7 Pressure–volume relationship obtained from the FEM simulations of the LV model without trabeculae, with three layers of intact and normal trabeculae, with three layers of intact and stiffer trabeculae, and with one layer of intact and normal trabeculae. These results demonstrate that severing trabeculae increases LV compliance and the presence of stiffer trabeculae decreases LV compliance.

simulated, we expect that an increase in trabeculae size is expected to increase the structural stiffness of the trabeculae and thus increase the stiffness of the LV. This is because our simulations showed that increased trabeculae stiffness would increase the LV stiffness. Therefore, an increase in the mass of trabeculae can lead to an increase in the LV stiffness and thus cutting these hypertrophic trabeculae would lead to more reduction in LV stiffness.

The current study is the first to propose both a physiologic role and the pathologic impact of trabeculae in LV diastolic function. Previously, Jöbsis et al. investigated a procedure of “scoring” the visceral pericardium and reported a minimal decrease in the passive stiffness of the LV [33]. Schaff et al. reported an apical cutting procedure which enlarges the LV cavity to improve diastolic compliance [34]. However, this procedure is a very traumatic procedure with apical incision and myectomy. In a pilot study, we found that a similar procedure of “endocardial scoring” that makes four equally distributed endocardial cuts of 20% wall thickness from the base to apex would also improve the passive LV compliance. While apical cutting, endocardial scoring, and trabecular cutting could lead to a shift in the LV pressure–volume curve and increased compliance, trabecular cutting is the least traumatic with little likelihood of scar formation.

Reduced left ventricular (LV) diastolic compliance is a feature of heart failure (HF) with preserved ejection fraction (HFpEF), which is responsible for half of all HF hospitalizations and is increasing in prevalence each year [35]. Since diastolic dysfunction was first shown to contribute to HF, there have been many clinical trials that have attempted to improve morbidity and mortality [36,37]. Despite these efforts, there have been no drugs discovered or operations described to date which acutely improve diastolic compliance in patients [35–38]. Therefore, it would be of clinical interest to develop a surgical approach to treat diastolic heart failure. Our results suggest that a surgical procedure of severing free-running trabeculae could make the LV structurally more compliant during passive filling and serve as a potential new treatment for decreased LV compliance. Through the approach could be highly invasive, it can be done through endovascular procedures through mitral orifice as an alternative for patient with severe diastolic heart failure. Further in vivo studies are needed to explore this approach. While this study was not intended to

Fig. 8 Color map of circumferential stress distribution obtained from FEM simulations of LV model during a chamber pressure of 10 mm Hg. The stress was obtained for the cases without trabeculae, with three layers of trabeculae, and with only one apical layer of trabeculae. Each color represents the range of stress shown in the scale.

Table 2 Maximum circumferential stress and fiber strain developed at the midventricle and apex regions at distending pressure of 10 mm Hg for the three finite-element models of the LV

	Maximum stress (kPa)		Maximum strain	
	Apex	Midventricle	Apex	Midventricle
With three layers	6.0	0.7	−0.11	0.055
With one layer	6.3	0.7	−0.15	0.055
No trabeculae	9.6	0.7	−0.20	0.055

demonstrate the feasibility of this approach in patients, the current results provides a fundamental understanding of the potential role of trabeculae in LV diastolic function and serves as a basis for future studies.

There are several limitations to the current study. First, for human hearts in this study, ventricular wall thickness and volume are known to be largely influenced by the timing in the cardiac cycle in which the ventricle wall was set by rigor mortis [30]. Since the measurements for pressure volume relations were performed before and after the trabecular cutting under the same conditions (temperature, pH, calcium and potassium concentrations), we expect the differences in the results were due to the trabecular cutting. Echocardiographic and clinical data are not available to us since the identities of these patients were not known as required by the IRB. The postmortem measurements were all that were available to define these patients as having LV hypertrophy. However, given a doubling of heart weight in the experimental hearts compared to normal hearts in the literature, LV hypertrophy is expected to be present.

The finite-element model was based on generic geometric model. Thus, while the pressure volume curve trends are comparable between the model results and experimental measurement, the data should not be compared directly. Also, the papillary muscles and chordae tendineae were not modeled for simplicity since they remain the same prior and post-trabeculae cutting. Though they may also contribute to the structural stiffness, they cannot be severed since they are critical for the functioning of the heart valves. While computational models have been shown as powerful tools to analyze LV function [39,40], due to a lack of human data, we used material properties for a canine myocardium, which have its limitations and cannot be directly extrapolated to the human heart. We did not perform parameter estimation based on the measured pressure volume relationships due to our lack of geometric details of individual LVs during measurement of passive LV pressure volume relations. With the LV geometry measured in future studies, this approach can be used to estimate material parameters by

matching the pressure–volume results of FE with the experiments. That said, the current model is reasonable for qualitative illustration of the effect of trabeculae carnea on the passive compliance and wall stress distribution of the heart. In addition, effects of trabecular cutting on systolic function were beyond the scope of this study and need to be investigated in the future. Recently, Messas et al. demonstrated that cutting some collagenous chordae do not adversely affect the left ventricular contractile function [41]. As some trabeculae are more fibrotic in nature than the myocardial wall, we expect that the contraction force generated by trabeculae in the systolic phase would be much smaller than the normal myocardium and thus the effect of cutting trabeculae on systolic function would be minimal. The long-term effects of trabecular cutting on the LV wall remodeling need further study as well.

In conclusion, we demonstrate that integrity of the LV and trabeculae is important to maintain LV stiffness and loss in trabeculae leads to more LV compliance.

Acknowledgment

This work was supported by the South Texas Veterans Health Care System (VA Merit Grant, San Antonio, TX), the Janey and Dolph Briscoe Center for Cardiovascular Research (San Antonio, TX), Grant No. HL095852 from the National Heart, Lung, and Blood Institute, the Clayton Foundation, and an AHA National Innovation Award (15IRG23320009). This project was also funded in part by the GREAT program from the Office of the Vice President for Research of the University of Texas at San Antonio.

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