gurgitation (IMR) by cutting the strut chordae of the anterior mitral leaflet, and they combined this approach with ring annuloplasty and left ventricular volume reduction surgery. The technique reported by Yamamoto and colleagues is an effective modality, but there are some concerns that should be clarified. As stated previously, IMR is a ventricular disease and not a valvular disease. Although the tethering of the mitral leaflet might play role in the development of chronic IMR, it is not the cause but rather the result of a pathophysiologic process. Chordal cutting increases leaflet coaptation area by abolishing the bending of the anterior mitral leaflet, but it does not address all underlying mechanisms of functional IMR. We also know that the underlying cause of chronic IMR might differ between individual patients. Because the underlying mechanism might differ between patient groups, the chordal cutting technique might not have the same effectiveness in all patients.

Lastly, I think that there was also a misinterpretation of the successful result. The authors used the combination of 3 different techniques, which addressed all the underlying mechanisms, but only emphasized the importance of chordal cutting. I believe that the successful result of this surgical intervention could not be attributed to only the chordal cutting procedure. Therefore the authors should comment on how they draw such a result from the data presented. Additionally, instead of chordal cutting, I also believe that the main factor for the disappearance of leaflet restriction is the left ventricular volume reduction surgery, which decreases the interpapillary distance.

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Reference

1. Yamamoto H, Iguro Y, Sakata R, Arata K, Yotsumoto G. Effectively treating ischemic mitral regurgitation with chordal cutting in combination with ring annuloplasty and left ventricular reshaping approach. *J Thorac Cardiovasc Surg.* 2005;130:589-90.
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Reply to the Editor:

We thank Dr Basaran for raising important questions concerning our report of chordal cutting in combination with ring annuloplasty and overlapping cardiac volume reduction (OLCVR) operation, which is a new clinical therapeutic approach for the treatment of ischemic mitral regurgitation (IMR).

We think that IMR associated with myocardial infarction and ischemic cardiomyopathy is a progressive disease. There is a possibility of recurrence of IMR after the operation because of the persistent left ventricular (LV) remodeling. Therefore we paid attention to not only a disappearance of mitral regurgitation immediately after the operation but also to the long-term prevention of IMR recurrence after surgical intervention.

We also think that LV plasty should be able to not only reduce the LV volume but also to correct the displaced papillary muscles (PMs), and such LV plasty will thus contribute greatly to reducing mitral tethering.

In some cases of IMR, chordal cutting is a possible additional technique, depending on the patterns of chordal attachment, and this technique will surely increase the coaptation of the mitral leaflets in comparison with LV plasty alone. In addition, we expect this technique will also help to prevent postoperative IMR recurrence.

The current surgical approach for IMR mainly focuses on annular size reduction with an annuloplasty ring, which is usually effective. However, mitral annuloplasty (MAP) is not a fundamental therapy for IMR. Zhu and colleagues¹ reported that MAP causing anterior displacement of posterior mitral annulus augments the tethering of posterior mitral annulus with an impaired mobility and coaptation, and this mechanism is also related to persistent IMR after MAP. Therefore additional mitral valve plasty will be necessary to reduce such tethering.

Several methods related to the reduction of the tethering of IMR have been reported. Two approaches have been considered. The first is to correct the displaced PMs, and the second is to perform chordal cutting. The reduction of the distance between PMs by using PM imbrication and PM sling and the reduction of the distance between the mitral annulus and PMs by relocating the posterior PM and LV plication can lead to a decrease in the degree of tethering and the disappearance of MR. The chordal cutting method, which cuts tethered

strut chordae, is a direct correction of the tethering. However, whether chordal cutting is possible depends on the attachment of strut chordae to the anterior mitral leaflet. Only in cases in which the bifurcated strut chordae are attached to the leaflet edge and the marginal portion between the rough and clear zone can chordal cutting spare the chorda attached to the leaflet edge.

In this case, with severely dilated and dysfunctional LV after anterior myocardial infarction, we thought it necessary to reduce the LV volume for end-stage ischemic cardiomyopathy. The OLCVR operation is thus considered to be an effective procedure to correct the displaced PM dimensions, but there is a risk that an LV volume reduction sufficient to correct the PM displacement might result in too small of an LV volume. Matsui and coworkers² reported OLCVR with PM plication to enhance the remodeling effect of the ventricle.

We used 3 techniques to treat IMR in this case. We also think that MAP and LV plasty (OLCVR) contributed to our successful results and that chordal cutting can therefore reinforce mitral coaptation. We hope that IMR recurrence can thus be prevented long term after surgical intervention by means of such a strategy.

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References

1. Zhu F, Otsuji Y, Yotsumoto G, Yuasa T, Ueno T, Yu B, et al. Mechanism of persistent ischemic mitral regurgitation after annuloplasty: importance of augmented posterior mitral leaflet tethering. *Circulation.* 2005;112(suppl): I396-401.
2. Matsui Y, Fukada Y, Naito Y, Sasaki S. Integrated overlapping ventriculoplasty combined with papillary muscle plication for severely dilated heart failure. *J Thorac Cardiovasc Surg.* 2004;127:1221-3.
doi:10.1016/j.jtcvs.2006.01.034

Residual strain in the aorta To the Editor:

We were excited to see yet another seminal contribution to the literature on aortic aneurysmal disease arising from the labora-

tories of Dr Elefteriades and his colleagues.¹ Their work continues to broaden our fundamental understanding of this lethal but treatable disease. The recently reported work demonstrates a remarkable correlation between the predicted behavior of the aorta based on mechanical properties and the natural history of this disorder as observed previously by this same group of investigators. In the course of their discussion of the significance of their work, the authors graciously referenced our previous work touching on the mechanical properties of the ascending aorta, with particular emphasis on bicuspid aortic valve disease and changes with aging.² An additional finding from our study relevant to the author's work, however, perhaps should be made. As one might have anticipated, we observed changes in the intrinsic mechanical properties of the aorta, as defined by the elastic properties determined by means of biaxial testing. Of greater interest, perhaps, were our observed changes with age in the opening angle as a reflection of residual strain.

Residual stress and strain are critical concepts in understanding the mechanical properties of the arterial wall. Absent residual strain, intraluminal pressure within a thick-walled tube will result in an uneven distribution of stress across the wall, with the greatest stress born by the inner portion and the least stress by the outer. The pioneering work by Chuong and Fung³ demonstrated over 2 decades ago that the distribution of stresses and strains within a vessel wall are nonuniform, both circumferentially and longitudinally. Calculations based on the assumption that the unloaded vessel is at zero stress result in remarkably high stress concentrations at the inner wall. The unloaded vessel, however, is not physiologic. The vessel wall exhibits residual strain to effect a more even distribution of stress across the thickness of media and adventitia in the loaded state. Just as a concrete beam can be prestressed in anticipation of the loaded state, the effect of residual strain in a vessel wall is to make more uniform the distribution of circumferential wall stress under physiologic conditions with intraluminal pressure. Experimental observations confirm this phenomenon in vivo. Variations in residual strain have been observed in differing segments of the aorta in the rat, just as one would anticipate given differences in pressure wave forms.⁴ Liu

and Fung⁵ further demonstrated that residual strain is subject to remarkably rapid changes with cellular remodeling caused by altered hemodynamics.

The existence of residual strain is easily demonstrated in the operating room by using a test that is, in fact, familiar to all cardiovascular surgeons. If one resects a tubular segment of a vessel and then cuts the resulting ring open, the vessel will spring open (the opening angle) to a zero-stress state.⁶ This reflects residual tensile stress in the outer wall, while the inner wall has residual compressive stress. Accordingly, the ring will tend to turn itself inside out.

Of what possible clinical relevance is this phenomenon? Aneurysm rupture and dissection represent structural failures of the aortic wall. We also know that, despite arguments over the relative contributions of cellular apoptosis and fiber degradation, all would agree that rather dramatic changes in the structure of the wall itself can be observed with aging or against the backdrop of connective tissue disease. One can well imagine that concomitant changes in residual strain and other material properties might occur, as well as changes in vessel diameter and thickness in response to aging and hemodynamic forces. Could these alterations affect stress distribution across the wall of the aorta? It is tempting to hypothesize that alterations in vessel wall structure leading to disproportionate loading of the outer portion of the aortic wall could lead to primary failure of the subadventitia. Could this be an alternative explanation for the phenomenon of intramural hematoma? Could primary mechanical failure of the adventitia lead to creation of a potential space that fills with blood as a secondary phenomenon?

Although this theory is unsubstantiated by experimental work, it demonstrates the potential importance of accounting for residual strain among the mechanical properties of the aortic wall. In this regard we must respectfully disagree with the authors' statement that "a full profile of the mechanical properties of the aorta can be gleaned by measuring six specific physical characteristics: blood pressure (systolic and diastolic), aortic diameter (systolic and diastolic), and thickness of the aortic wall (systolic and diastolic)." We believe there is more to it than that. The intrinsic properties of this highly complex amalgam of

connective tissue and cellular elements must enter into the equation.

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References

1. Koullias G, Modak R, Tranquilli M, Korkolis DP, Barash P, Elefteriades JA. Mechanical deterioration underlies malignant behavior of aneurysmal human ascending aorta. *J Thorac Cardiovasc Surg*. 2005;130:677-83.
2. Okamoto ref Okamoto RJ, Xu HD, Kouchoukos NT, Moon MR, Sundt TM. The influence of mechanical properties on wall stress and distensibility of the dilated ascending aorta. *J Thorac Cardiovasc Surg*. 2003;126:842-50.
3. Chuong CJ, Fung YC. Three-dimensional stress distribution in arteries. *J Biomech Eng*. 1983;105:268-74.
4. Liu SQ, Fung YC. Zero-stress states of arteries. *J Biomed Eng*. 1988;110:82-4.
5. Liu SQ, Fung YC. Relationship between hypertension, hypertrophy, and opening angle of zero-stress state of arteries following aortic constriction. *J Biomed Eng*. 1989;111:325-35.
6. Chuong CJ, Fung YC. On residual stress in arteries. *J Biomed Eng*. 1986;108:189-92. doi:10.1016/j.jtcvs.2005.12.070

Reply to the Editor:

Our team wishes to thank Dr Sundt for his extremely kind comments regarding our scientific investigations at the Center for Thoracic Aortic Disease at Yale University. We agree entirely with Dr Sundt that "residual strain" represents yet another facet of the complex mechanical behavior and measurable properties of the aortic wall. He and his colleagues, in the important work they cite in their letter, have measured this phenomenon in human aortas. From an engineering standpoint, residual strain is considered more important in thick-walled tubes (ratio of wall thickness to vessel diameter >1:10). The aneurysmal aorta is often thin-walled (thickness of 2-3 mm in a vessel with a diameter $\geq$ 5 cm) and often becomes even more so as the aneurysmal process advances (Figure 1). Thus the effect of residual strain, although an important inherent design characteristic of the human aorta, might diminish as the aneurysmal state advances.

We also have observed regional differences in stress distribution and resultant