is definitely faster in patients with functional or ischemic mitral regurgitation, and I think that has to do with concomitant right ventricular dysfunction in those patients versus a patient with mitral valve prolapse. But you are still going to have to create a study that spans many years, as well as choose an appropriate clinical end point. Unfortunately, in my opinion New York Heart Association functional class is a poor metric for right heart function. I did not have data on diuretic use, liver transaminases, or kidney function. I believe that in a clinical trial a surrogate end point, such as right ventricular dimensions or function in addition to grade of tricuspid regurgitation, would need to be used.

However I definitely agree that a randomized prospective trial would be ideal to help inform the cardiac surgery community on this controversial issue.

Dr Harold L. Lazar (Boston, Mass). Did you measure pulmonary artery (PA) pressures either by echo or by cath and have you found that to be a determinant of what ultimately happens

to the TR? I have seen that patients who have minimal TR but have high PA pressures at the time of mitral valve replacement or repair tend to be the ones who tend to have increased TR in the future.

Dr Goldstone. We did look at PA pressures. They were measured by right heart catheterization, and preoperative mean PA pressure was not a significant predictor of progression of TR postoperatively in univariate analysis. That is consistent with the Mayo paper, which did not find pulmonary artery pressure to be a significant predictor either. I agree that it makes sense that pulmonary hypertension would influence TR progression. Perhaps the reason why different studies identify different predictors of TR progression alludes to the multifactorial nature of functional TR.

Dr Lazar. In those patients who had progressive TR, did their PA pressures go up or did they stay the same?

Dr Goldstone. I do not have longitudinal data on PA pressure.

EDITORIAL COMMENTARY

The role of cognitive dissonance in the management of functional tricuspid regurgitation at the time of degenerative mitral valve repair

Rakesh M. Suri, MD, DPhil,^a and Yan Topilsky, MD^b

See related article on pages 2802-10.

Cognitive dissonance: Psychologic conflict resulting from incongruous beliefs and attitudes held simultaneously.

In this issue of the *Journal*, Goldstone and colleagues¹ from the University of Pennsylvania and Stanford have assembled the third largest series (n = 495) in the literature of patients with mitral valve prolapse undergoing mitral valve repair with coexistent functional tricuspid valve regurgitation (TR) not surgically managed. Forty-three percent of patients had anywhere between trivial to moderately severe TR at mitral valve repair, and freedom

from greater than moderate TR was approximately 60% at 9 years. Older age and grade of preoperative TR predicted progression; and “some degree” of right ventricular dysfunction was more frequent in those with severe TR during follow-up.

What does this study tell us? The findings reaffirm those published from the Mayo Clinic in 2011 and in 2014.^{2,3} Our institution’s first series studied 699 patients who underwent mitral valve repair for isolated degenerative leaflet prolapse and moderate or less coexistent functional TR. We identified that moderate TR actually regressed with time in most cases after successful and durable repair of degenerative mitral valve disease. Indeed, only 1 patient out of the entire series required reoperation for progressive TR 4.5 years after mitral valve repair. Our most recent report expanded on this analysis, studying 747 adult patients with mitral valve prolapse among whom 683 underwent mitral valve repair and 64 had replacement. We found no difference in the occurrence of moderately severe or severe TR after mitral valve repair versus replacement and found the univariate predictors of TR progression to be older age, female sex, higher pulmonary arterial pressure, and larger left atrial size. Only 2 patients required reoperation for late functional TR. The report of Goldstone and colleagues¹ thus adds to

From the Division of Cardiovascular Surgery,^a Mayo Clinic, Rochester, Minn; and the Tel Aviv Sourasky Medical Center,^b Tel Aviv, Israel.

Disclosures: Authors have nothing to disclose with regard to commercial support.

Received for publication Aug 25, 2014; accepted for publication Aug 25, 2014; available ahead of print Oct 3, 2014.

Address for reprints: Rakesh M. Suri, MD, DPhil, Division of Cardiovascular Surgery, Mayo Clinic College of Medicine, 200 First St SW, Rochester, MN 55905 (E-mail: suri.rakesh@mayo.edu).

J Thorac Cardiovasc Surg 2014;148:2810-2

0022-5223/\$36.00

Copyright © 2014 by The American Association for Thoracic Surgery

<http://dx.doi.org/10.1016/j.jtcvs.2014.08.040>

FIGURE 1. Kaplan-Meier estimates of the rates of cardiovascular events in patients with normal ejection fraction, normal pulmonary pressure, and no other significant comorbidity stratified by tricuspid regurgitation severity. Values in parentheses are event rates at 5 years, which differed significantly between patients with moderate tricuspid regurgitation (TR; effective regurgitant orifice [ERO] <40 mm²) and severe tricuspid regurgitation (effective regurgitant orifice >40 mm²) but not between patients with trivial tricuspid regurgitation and those with moderate tricuspid regurgitation.

an already significant body of evidence in existence reassuring us that the performance of early correction of mitral regurgitation is important not only for its own well-known benefits⁴ (preservation of survival and minimization of late heart failure risk) but also to diminish the late occurrence of functional TR.

Although the data of Goldstone and colleagues¹ are entirely consistent with our own, their conclusions are incongruous and puzzling in stating that “less than severe TR is likely to progress after surgery if uncorrected.” They use “RV [right ventricular] dysfunction” to justify concomitant tricuspid valve repair, even though symptoms, survival, and reoperation were not affected by TR according to their own data. Unfortunately, the fatal flaw lies in reliance on semi-quantitative assessments of right ventricular function, which undermines the validity of the intended conclusion. Qualitative assessment of RV size and function is no longer recommended by current guidelines⁵ because it is associated with wide interreader variability.⁶ Importantly, in the presence of TR, RV afterload decreases and right atrial pressure increases, making qualitative assessment of RV function even more inaccurate.

Although the specter of an at times high-risk reoperation for progressive TR in the setting of severe mitral regurgitation caused by ischemic disease, rheumatic valvulopathy, congenital heart disease, or radiation heart disease no doubt merits careful avoidance, all available scientific evidence has proved this to be a very low likelihood event in the setting of degenerative mitral valve disease. We recently have shown that lesser degrees of quantified TR are in fact associated with

neither excess mortality (Figure 1) nor excess morbidity in the context of normal ejection fraction, normal pulmonary pressure, and absence of significant comorbidities.⁷ Reassuringly, patients with incidentally discovered less than moderate functional TR undergoing early degenerative mitral valve repair are most likely to fulfil these low risk criteria. Although the article of Goldstone and colleagues¹ proposes that, by default, concomitant tricuspid repair “may be warranted,” tricuspid valve repair was not, in fact, studied in the current report at all, and thus this extrapolated and nuanced conclusion reaches beyond the scope of the presented data.

On the basis of available long-term outcomes data, the psychologic stress of not intervening upon the tricuspid valve should therefore be diminished when specifically considering patients with mitral valve prolapse, particularly those undergoing early mitral repair before the onset of guideline-based triggers (such as symptoms, ejection fraction <60%, left ventricular end-systolic diameter >40 mm, pulmonary pressure >50 mm Hg, or atrial fibrillation). Proponents of routine concomitant TR repair often ask, “What could be simpler, snare the venae cavae, open the right atrium, and place a cerclage stitch or annuloplasty band?” Although as surgeons we may find this technically appealing and philosophically satisfying, the simple fact remains that the weight of evidence does not support this practice in all settings. Rationally, the performance of concomitant tricuspid repair at the time of degenerative mitral valve repair should be strongly considered in the presence of (1) greater than moderate TR, (2) right heart dysfunction or right heart failure symptoms, (3) pulmonary hypertension, (4) reduced left ventricular systolic function or cardiomyopathy, and (5) organic tricuspid valve pathology. In contrast, the decision to pursue conservative management of less than moderate functional TR in patients who undergo effective degenerative mitral regurgitation correction and who are unlikely ever to have difficulty with the tricuspid valve or the right ventricle should not engender cognitive dissonance due to the fact that it is congruous with all currently available scientific evidence.

References

- Goldstone AB, Howard JL, Cohen JE, MacArthur JW Jr, Atluri P, Kirkpatrick JN, Woo YJ. Natural history of coexistent tricuspid regurgitation in patients with degenerative mitral valve disease: Implications for future guidelines. *J Thorac Cardiovasc Surg.* 2014;148:2802-10.
- Yilmaz O, Suri RM, Dearani JA, Sundt TM III, Daly RC, Burkhart HM, et al. Functional tricuspid regurgitation at the time of mitral valve repair for degenerative leaflet prolapse: the case for a selective approach. *J Thorac Cardiovasc Surg.* 2011;142:608-13.
- Rajbanshi BG, Suri RM, Nkomo VT, Dearani JA, Daly RC, Burkhart HM, et al. Influence of mitral valve repair versus replacement on the development of late functional tricuspid regurgitation. *J Thorac Cardiovasc Surg.* April 23, 2014 [Epub ahead of print].

4. Suri RM, Vanoverschelde JL, Grigioni F, Schaff HV, Tribouilloy C, Avierinos JF, et al. Association between early surgical intervention vs watchful waiting and outcomes for mitral regurgitation due to flail mitral valve leaflets. *JAMA*. 2013; 310:609-16.
5. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr*. 2010;23:685-713. quiz 86-8.
6. Ling LF, Obuchowski NA, Rodriguez L, Popovic Z, Kwon D, Marwick TH. Accuracy and interobserver concordance of echocardiographic assessment of right ventricular size and systolic function: a quality control exercise. *J Am Soc Echocardiogr*. 2012;25:709-13.
7. Topilsky Y, Nkomo VT, Vatury O, Michelena HI, Letourneau T, Suri RM, et al. Clinical outcome of idiopathic tricuspid regurgitation. *JACC Cardiovasc Imaging*. November 5, 2014 [Epub ahead of print].