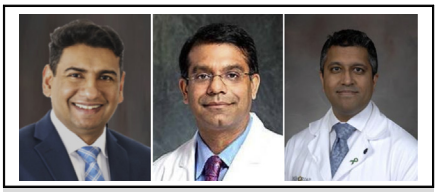
See Article page 1788.

Commentary: Treating "functional" tricuspid valve regurgitation—why, when, and how?

Faisal H. Cheema, MD,[a,b,c] Pranav Loyalka, MD,[a] and Keshava Rajagopal, MD, PhD[a,c]



Faisal H. Cheema, MD, Pranav Loyalka, MD, and Keshava Rajagopal, MD, PhD

CENTRAL MESSAGE

The indications, outcomes, and optimal techniques for the treatment of functional tricuspid valve regurgitation remain unclear.

ADULT

Tricuspid valve (TV) regurgitation (TR), like its more-studied and -understood left-sided counterpart, mitral valve regurgitation (MR), is the consequence of interactions between TV apparatus structure and the physiological conditions affecting systolic TV function: intrinsic right ventricular (RV) systolic function and RV loading conditions. In turn, the relationship between TV structure and function is not one-way: structure impacts function, but function impacts structure as well. In individual cases, the inciting insult responsible for the end-result of TR may be intrinsic to the valve structure, or may be extrinsic—due to decreased RV contractility, increased RV afterload, and/or increased RV preload. What is often termed "functional" TR (or MR) may be defined as regurgitation occurring as a consequence of these pathologic extrinsic stimuli.

Whether functional or nonfunctional in origin, the rationale for TR treatment is that by decreasing TR, a vicious cycle of TR → RV volume overload → RV systolic dysfunction → worsening functional TR is interrupted. However, because functional TR does not originate from intrinsic valvular pathology, it is less clear whether treatment of TR will be durable and effective in preventing the aforementioned sequelae. Moreover, when to treat TR in general is far less clear than MR, for which much greater data motivating and supporting indications and guidelines are present. This is made even more challenging by the fact that unlike MR, the majority of TR is functional, and more specifically, is driven by RV afterload mismatch. Finally, the best approach to treat either TR in general or functional TR in particular is unknown.

In this issue of the *Journal*, Sohn and colleagues[1] conducted a propensity score–matched retrospective analysis of rigid incomplete ring versus De Vega suture annuloplasty to treat functional TR. The vast majority of patients had important left-sided cardiac valvular disease (mitral >> aortic) that typically required/was treated via valve replacement. The sizing approaches to the TV were different depending on the repair technique employed, introducing potential bias. Residual/recurrent moderate or greater TR was more common in the De Vega group. However, this did not translate to differential survival or other complications, with a relatively long follow-up.

Does residual/recurrent moderate functional TR matter? These findings would suggest that the answer is no. Although the landmark Cardiothoracic Surgical Trials Network study[2] of coronary artery disease–related functional MR showed that residual/recurrent moderate or greater MR does not adversely impact survival, it does relate to recurrent heart failure and cardiovascular rehospitalizations. For the TV, we now have data to suggest that TR recurrence is not associated with inferior survival or greater incidences of complications. This prompts further questions. Most importantly, should severe TR even be treated at the time of the index left-sided procedure (particularly since TR is driven by group II pulmonary hypertension in these cases), or should percutaneous approaches such as the MitraClip (Abbott Laboratories, Chicago, Ill) in the tricuspid position be used selectively in the postoperative setting? Are there even longer-term adverse sequelae of residual/recurrent TR? When it comes to when and how to treat functional TR, little is clear.

From the [a]HCA Houston Healthcare, Gulf Coast Division, Houston, Tex; [b]HCA Research Institute, Nashville, Tenn; and [c]Department of Clinical Sciences, University of Houston College of Medicine, Houston, Tex.

Disclosures: Authors have nothing to disclose with regard to commercial support.
Received for publication Dec 4, 2019; revisions received Dec 4, 2019; accepted for publication Dec 4, 2019; available ahead of print Dec 23, 2019.

Address for reprints: Keshava Rajagopal, MD, PhD, HCA Houston Healthcare, 1200 Binz St, Suite 900, Houston, TX 77004 (E-mail: krajago2@central.uh.edu).

J Thorac Cardiovasc Surg 2021;161:1799-800
0022-5223/$36.00

https://doi.org/10.1016/j.jtcvs.2019.12.026

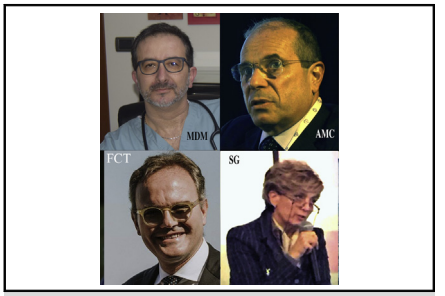
References

1. Sohn SH, Kim KH, Lee Y, Choi JW, Hwang HY. Long-term outcomes of rigid ring versus DeVega annuloplasty for functional tricuspid regurgitation: a propensity score-matching analysis. *J Thorac Cardiovasc Surg*. 2021;161:1788-98.e5.
2. Goldstein D, Moskowitz AJ, Gelijns AC, Ailawadi G, Parides MK, Perrault LP, et al. Two-year outcomes of surgical treatment of severe ischemic mitral regurgitation. *N Engl J Med*. 2016;374:344-53.

See Article page 1788.

Commentary: Opportunity knocks for every heart surgeon, but you have to give a tricuspid a ring

Michele Di Mauro, MD, PhD, MSc,[a] Sabina Gallina, MD,[a] Fabrizio Tancredi, MD,[a] and Antonio M. Calafiore, MD[b]



Michele Di Mauro, MD, PhD, MSc (MDM), Antonio M. Calafiore, MD (AMC), Fabrizio Tancredi, MD (FCT), and Sabina Gallina, MD (SG)

CENTRAL MESSAGE

The long-term results of the paper by Sohn and colleagues add a new, valid piece to the complex puzzle of evidence reported in the literature.

The long-term results of the paper entitled "Long-Term Outcomes of Rigid Ring Versus DeVega Annuloplasty for Functional Tricuspid Regurgitation: A Propensity Score Matching Analysis" by Sohn and colleagues[1] add a new, valid piece to the complex puzzle of evidence reported in the literature.[2-4] Parolari and colleagues,[5] pooling the data from 9 studies, reported the freedom from recurrent moderate or greater tricuspid regurgitation (TR) at 8 and 15 years in patients who underwent TR repair with a prosthetic ring was 88.5% (95% confidence interval [CI], 84.5-92.5) and 78.9% (95% CI, 69.7-89.3), significantly greater than patients who underwent TR repair without a prosthetic ring, 81.8% (95% CI, 78.0-85.8) and 50.5% (95% CI, 40.2-63.6) ($P = .0107$).

Conversely, a very recent meta-analysis[3] reported the pooled effect of the technique on late recurrence of TR; 4 studies in which function TR was treated with either ring or suture failed to show any difference (risk ratio, 0.98; CI, 0.72-1.33). The ring cohort involved either a flexible or rigid ring, so the authors compared the effect of the type of ring used on late TR recurrence. Hence, pooling the results of 4 studies, the rigid ring was found to provide more stable results over time rather than the flexible. The latter finding was confirmed also by the meta-analysis reported by Wang and colleagues[3]; the authors pooled the results from 5 studies and concluded that a rigid ring had significantly better freedom from moderate or more TR at 5 years (odds ratio, 0.44; 95% CI, 0.20-0.99), even if there was no significant difference in overall rates of reoperation ($P = .232$) and survival ($P = .086$) between a flexible band and rigid ring. These differences are found in the fact that most of the studies compared heterogenous groups of rings.

Sohn and colleagues[1] compared 2 matched groups of patients with functional TR undergoing a De Vega or rigid ring. They did not find any difference in terms of long-term all-cause mortality, cardiac mortality, and tricuspid valve–related events, but when a rigid ring was used, the cumulative incidence of TR recurrence at 10 years was significantly lower (6.3% vs 19.1%, $P < .001$), and this finding was confirmed, by means of a longitudinal analysis, in all periods.

From the [a]Department of Heart Disease, SS Annunziata Hospital, Chieti; and [b]Department of Cardiac Surgery, Pope John Paul II Foundation, Campobasso, Italy.

Disclosures: Authors have nothing to disclose with regard to commercial support.
Received for publication Dec 1, 2019; revisions received Dec 1, 2019; accepted for publication Dec 3, 2019; available ahead of print Dec 23, 2019.

Address for reprints: Michele Di Mauro, MD, PhD, MSc, Biostat, Heart Disease Department, SS Annunziata Hospital, Via dei Vestini, 66100, Chieti, Italy (E-mail: mdimauro1973@gmail.com).

J Thorac Cardiovasc Surg 2021;161:1800-1
0022-5223/$36.00

https://doi.org/10.1016/j.jtcvs.2019.12.016