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EDITORIAL
Valvular heart disease

Transcatheter tricuspid valve replacement: defining the sweet spot for intervention

Nina Ajmone Marsan and Jeroen J. Bax *

Department of Cardiology, Leiden University Medical Center, Albinusdreef 2, 2333 ZA Leiden, the Netherlands

This editorial refers to 'Tricuspid valve replacement and outcomes by baseline tricuspid regurgitation severity: the TRISCEND II trial', by P. Lurz *et al.*, https://doi.org/10.1093/eurheartj/ehaf676.

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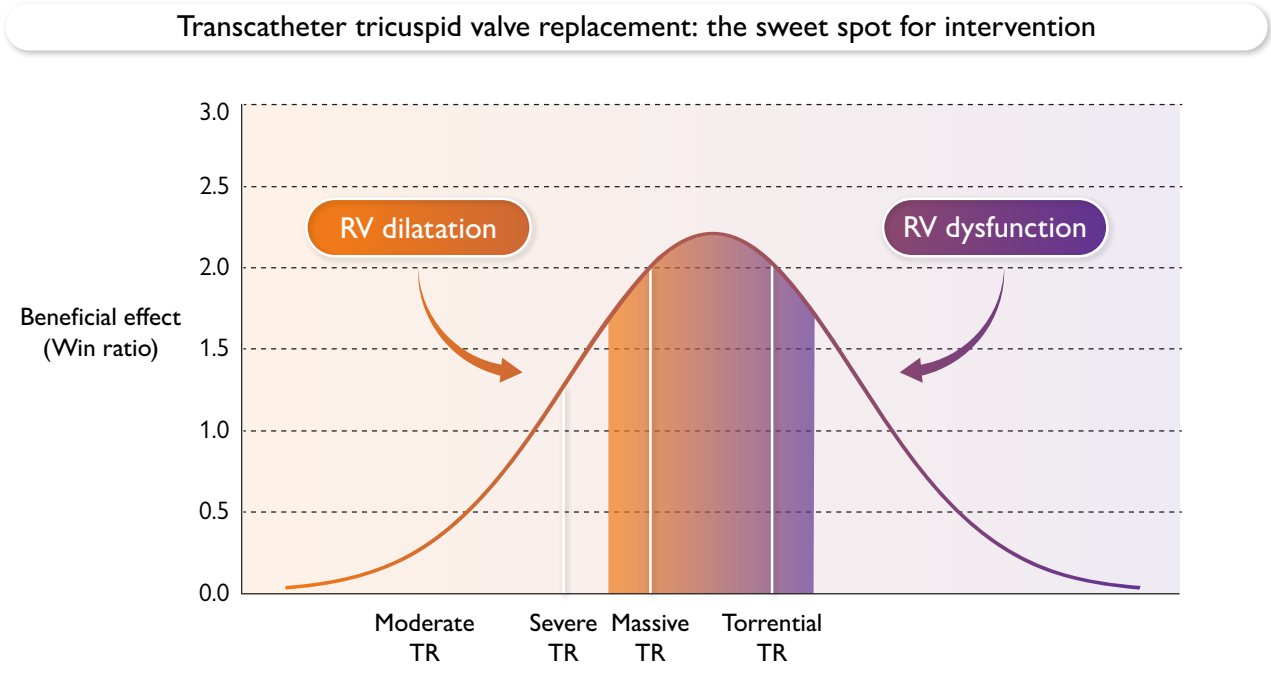
Graphical Abstract



A conceptual framework illustrating the optimal therapeutic window of TTVR across the spectrum of TR severity. A schematic curve plotting expected clinical benefit (y-axis) against TR severity (x-axis) demonstrates a peak around massive TR, declining as torrential TR is accompanied by RV dysfunction. A shaded region marks the 'sweet spot for TTVR', with annotations highlighting the distinction of reversible RV dilatation vs irreversible RV dysfunction.

* Corresponding author. Tel: +31 71 526 2020, Fax: +31 71 526 6809, Email: BAXEHJ@lumc.nl, j.j.bax@lumc.nl

Tricuspid regurgitation (TR) is now recognized as an important therapeutic target given its demonstrated association with poor prognosis, heart failure (HF) hospitalizations, and impaired quality of life.[1] Over the last decade, the treatment of TR has therefore significantly evolved, and transcatheter therapies have filled a critical gap for patients deemed at high surgical risk.[2] Transcatheter edge-to-edge repair (TEER) has paved the way, being the first transcatheter intervention for TR to show clinical benefit in randomized clinical trials. However, its clinical efficacy has been related to the severity of residual TR post-intervention, which was less likely to be reduced to moderate or less when patients presented with more than severe (massive or torrential) TR at baseline.[3]

Transcatheter tricuspid valve replacement (TTVR), by contrast, offers the possibility of the almost complete elimination of TR, and therefore a clearer distinction of the true effect of the treatment. As a result, the degree of disease severity at baseline becomes the main determinant of outcome after TTVR, and of the extent of the biological benefit.

In the TRISCEND II trial, TTVR with the EVOQUE system (Edwards Lifescience, Irvine, CA, USA) was shown to significantly improve patients' quality of life at 1 year.[4] The post-hoc analysis by Lurz *et al*., published in the current issue of the *European Heart Journal* presents crucial insights into the magnitude of the clinical benefit of TTVR in relation to baseline TR severity, distinguishing severe TR vs massive or torrential TR.[5]

The authors confirmed the results of the pivotal trial, expanding the analysis to 18 months follow-up, and showing an overall win ratio of 2.02 for the composite hierarchical endpoint, reflecting a double likelihood of clinical benefit in the treatment group compared with controls. This advantage persisted across subgroups of TR severity, but in patients with severe TR, the win ratio was 1.6, while in those with massive or torrential TR, it increased to 2.2. Also, patients with massive or torrential TR showed significantly lower rates of HF hospitalizations (combined with all-cause mortality) as compared with controls, further supporting a significant interaction between treatment allocation and baseline TR severity. These findings reinforce the efficacy of TTVR but also suggest a potential therapeutic window in which clinical benefit may be maximized. However, this post-hoc analysis was not powered for interaction testing, and the results should be considered hypothesis-generating and interpreted with caution, with a critical appraisal of the specific findings.

For the overall comparison of the TTVR group vs controls, it is important to note that, due to the 2:1 randomization ratio and the option for crossover treatment after 12 months, the control group of this extended analysis was relatively small, hampering the statistical power. In particular, 53 control patients crossed over (although with no significant difference between patients with baseline severe or massive/torrential TR), of which 22 were within the visit window, and therefore were included in the control group for the win ratio calculations (while the others were censored). Also, the temporal pattern of observed events highlights the biological trade-off of this therapy, which may carry higher procedural risk but confer longer term benefit: adverse events occurred earlier and more frequently in the TTVR arm, largely related to the procedure itself, while late events were more frequent in the control arm, reflecting progressive right heart failure and persistent TR.

When looking at the subanalyses based on baseline TR severity, the distribution of demographics and comorbidities was relatively balanced between groups, but patients with massive/torrential TR presented with more severe right ventricular (RV) dilatation and central venous congestion. Of note, no significant differences among the groups were observed in RV function or stroke volume parameters, which were only mildly depressed. Importantly, patients with massive/torrential TR confirmed an overall higher risk profile, as the highest adverse event rate (and in particular HF hospitalization) was observed in the control patients with baseline massive/torrential TR, while these patients, when undergoing TTVR, showed a higher incidence of early cardiovascular mortality. However, the higher win ratio favouring treatment in patients with baseline massive/torrential TR suggests that the longer term beneficial effect was the largest in these patients and compensated for the higher peri-procedural risk.

Interestingly, among the controls, several patients (26%) with severe baseline TR showed a spontaneous TR improvement to mild to moderate at 1 year, while only a few patients with massive/torrential TR showed a similar behaviour. These observations may be explained by the statistical phenomenon of regression to the mean and have led to the lower HF hospitalization rate in these patients, but most importantly emphasize the need for a thorough assessment of TR severity with robust and repeated imaging. Ideally in such trials, severe (or more) TR should be confirmed with a run-in period to ensure that the disease is not reducible with optimized medical therapy, before proceeding to randomization or intervention. Besides the description of the TR severity over time, follow-up echocardiography to assess the changes in RV remodelling was also lacking, which might be influenced not only by the resolution of TR after intervention, but also by valve haemodynamics (i.e. prosthesis gradient, not reported), the need for chamber pacing, or the variations in pulmonary pressures.

Equally important, the TRISCEND II trial population was highly selected, raising questions about the generalizability of the findings. Broader patient cohorts (for example with reduced left ventricular and RV function), such as those anticipated in TRISCEND III post-market surveillance and international TTVR registries, will be critical to validate these findings in real-world practice. These will clarify whether procedural risks, anticoagulation management, and device durability are acceptable across a less selected population.

Despite these caveats, the study by Lurz *et al.* provides new important data which highlight the need for careful baseline characterization, and bring us one step closer to a refined, individualized strategy for managing TR patients. It confirms TTVR clinical benefit across the spectrum of TR severity (if at least severe), but also suggests a probable window of maximal effectiveness. A therapeutic 'sweet spot' for TTVR might be that of patients with significant symptoms, high surgical risk, unfavourable anatomy for repair, including a higher degree of TR severity, and evidence of RV remodelling without overt RV dysfunction (*Graphical Abstract*). Therefore, integrated clinical and echocardiographic phenotypization should be performed for every patient with severe TR to ensure timely referral to intervention and to guide individualized choice of treatment.

Declarations

Disclosure of Interest

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References

1. Wang N, Fulcher J, Abeysuriya N, McGrady M, Wilcox I, Celermajer D, *et al.* Tricuspid regurgitation is associated with increased mortality independent of pulmonary pressures and right heart failure: a systematic review and meta-analysis. *Eur Heart J* 2019;**40**:476–84. https://doi.org/10.1093/eurheartj/ehy641

2. Praz F, Borger MA, Lanz J, Marin-Cuartas M, Abreu A, Adamo M, *et al.* 2025 ESC/EACTS Guidelines for the management of valvular heart disease. *Eur Heart J* 2025;**46**:4635–736. https://doi.org/10.1093/eurheartj/ehaf194
3. Sorajja P, Whisenant B, Hamid N, Naik H, Makkar R, Tadros P, *et al.* Transcatheter repair for patients with tricuspid regurgitation. *N Engl J Med* 2023;**388**:1833–42. https://doi.org/10.1056/NEJMoa2300525
4. Hahn RT, Makkar R, Thourani VH, Makar M, Sharma RP, Haeffele C, *et al.* Transcatheter valve replacement in severe tricuspid regurgitation. *N Engl J Med* 2025;**392**:115–26. https://doi.org/10.1056/NEJMoa2401918
5. Lurz P, Hahn RT, Kodali S, Makkar R, Sharma RP, Davidson CJ, *et al.* Tricuspid valve replacement outcomes by baseline tricuspid regurgitation severity: the TRISCEND II trial. *Eur Heart J* 2026;**47**:2059–73. https://doi.org/10.1093/eurheartj/ehaf676