

# Tricuspid valve regurgitation: still struggling with the who and when

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Article Litwiński et al., see p. 731

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Tricuspid valve regurgitation (TR) is common being at least moderate in severity in 15% of the population [1] and is independently associated with worse prognosis [2]. Despite this, there is very little understood about when and whom to intervene upon. There are no randomised trials to guide management and all guideline recommendations are based on expert opinions given the lack of evidence [3]. Part of this evidence gap relates to current outcome studies combining heterogeneous aetiologies of severe TR which can be either primary, related to i) valve pathology (endocarditis, flail, pacemaker leads) or ii) atrial enlargement (atrial fibrillation [AF] related TR); or secondary, related to right ventricular (RV) remodelling from pulmonary hypertension or heart failure [4]. Related to this aetiological heterogeneity, and the difficulty in determining RV function in the presence of severe TR, reported operative mortality has varied drastically in the literature averaging 8.8% [5] but ranging as high as 30% to 50% in some cohorts [6].

Adding to this literature, Litwinski et al. [7] present their single-centre experience of patients undergoing tricuspid valve replacement (TVR) between 2000 and 2010. In this study of 86 patients, with a mean age of 58.5 and high mean EUROSCORE (8.75), who underwent TVR, in-hospital mortality was very high at 20.9% similar to other single-centre experiences. Only 42% underwent TVR as the primary option with the remaining undergoing concomitant TVR with another operation. Most patients (58%) were undergoing a reoperation and there was a high intraoperative complication rate (23%). The vast majority of patients had tricuspid regurgitation with 69% having AF, 70% having pulmonary hypertension and 26% having a low ejection fraction thus indicating a significant proportion of secondary TR in this cohort. Patients had advanced congestive symptoms with an New York Heart Association (NYHA) class of III or IV in 70% and ascites in 77%.

In-hospital mortality was related to advanced NYHA class with no mortality in patients with no or mild symptoms (NYHA class I and II). Other preoperative factors associated with mortality including anaemia, symptoms of RV failure, pulmonary hypertension, high EUROSCORE, were also indicative of increased comorbidity and disease severity. The authors highlight that patients with TR, advanced RV failure and symptoms have poor outcomes with surgery, and suggest that early surgery when patients are only minimally symptomatic can be associated with good outcomes using TVR.

Overall, this study adds to the growing body of evidence that waiting to intervene on the tricuspid valve until patients have right heart failure refractory to diuretics is associated with a very high operative mortality. There is accumulating evidence that worse symptom status and RV dysfunction are associated with worse operative mortality [8–12]. Given that TVR is not technically complex and the very high mortality reported is not related to surgical volumes [5], it is likely that patient rather than operative factors drive this high mortality. Delaying surgery until patients have symptoms that are refractory to diuretics, as suggested by current guidelines, likely leads to worse underlying RV remodelling, left ventricular atrophy from chronic underfilling due to ventricular interdependence [13] and congestive liver and renal failure at the time of eventual surgery. There is an urgent need for randomised trials of early intervention for severe tricuspid regurgitation to address this critical knowledge gap.

There are some limitations to consider which are similar to most of the literature in this field. First, it is a retrospective single-centre study with only 86 cases over 10 years, which exemplifies the difficulty with performing randomised trials in this field. Operative experience with tricuspid surgery and post-operative management of RV failure has changed over 10 years, which introduces heterogeneity. Moreover, this

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analysis only included patients undergoing valve replacement, which may have selected for an even higher risk cohort. Although there is no randomised trial to guide tricuspid valve repair versus replacement, typically replacement is reserved for patients with severe annular dilation, ventricular remodeling, or patients at high risk for repeat operation [14]. Also, there is no gender-specific analysis, and further analysis to understand differences between men and women may be warranted. Finally, the lack of a clear diagnosis for the tricuspid valve disease and the inclusion of many patients with secondary TR reiterates the need for better phenotyping of these patients prior to surgery. Accurately delineating primary valvular abnormalities causing TR versus secondary TR from pulmonary hypertension or left-sided heart failure can be difficult, and in secondary TR outcomes may be more driven by underlying ventricular function and pulmonary hypertension [15]. Clearly categorising patients as primary TR, AF-related TR, and secondary TR from heart failure will enable more homogenous comparison across studies to better inform clinical practice.

Thus, while there has been some important work in the area of tricuspid valve surgery, the question of how to best manage patients with isolated tricuspid valve surgery continues to be an important consideration. Future research will likely need to delve into identifying patients that would be good candidates for isolated tricuspid valve surgery, and compare outcomes in these patients to those on optimal medical therapy. A better understanding of the natural pathophysiology of isolated tricuspid valve disease, outcomes in these patients, and the optimal timing of valve repair or replacement will be important to best stratify and manage patients with tricuspid valve disease. As Litwinski et al. [7] suggest, the timing of tricuspid valve surgery may be critical to prevent RV dysfunction, heart failure, and adverse outcomes. Additional research to better understand the timing of ventricular dysfunction after development of TR will be an important step to optimising outcomes in these patients.

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