Mid-Term Valve-Related Outcomes After Transcatheter Tricuspid Valve-in-Valve or Valve-in-Ring Replacement

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ABSTRACT

BACKGROUND Transcatheter aortic and pulmonary valves have been used to treat stenosis or regurgitation after prior surgical tricuspid valve (TV) replacement or repair. Little is known about intermediate-term valve-related outcomes after transcatheter tricuspid valve replacement (TTVR), including valve function, thrombus, and endocarditis.

OBJECTIVES The authors sought to evaluate mid-term outcomes in a large cohort of patients who underwent TTVR after surgical TV repair or replacement, with a focus on valve-related outcomes.

METHODS Patients who underwent TTVR after prior surgical TV replacement or repair were collected through an international registry. Time-related outcomes were modeled and risk factors assessed.

RESULTS Data were collected for 306 patients who underwent TTVR from 2008 through 2017 at 80 centers; 52 patients (17%) had a prior history of endocarditis. Patients were followed for a median of 15.9 months after implantation (0.1 to 90 months), with 64% of patients estimated to be alive without TV reintervention or a valve-related event at 3 years. The cumulative 3-year incidence of death, reintervention, and valve-related adverse outcomes (endocarditis, thrombosis, or significant dysfunction) were 17%, 12%, and 8%, respectively. Endocarditis was diagnosed in 8 patients 2 to 29 months after TTVR, for an annualized incidence rate of 1.5% per patient-year (95% confidence interval: 0.45% to 2.5%). An additional 8 patients were diagnosed with clinically relevant valve thrombosis, 3 in the short term, 2 within 2 months, and 3 beyond 6 months. Only 2 of these 8 patients received anticoagulant therapy before thrombus detection (p = 0.13 vs. patients without thrombus). Prior endocarditis was not a risk factor for reintervention, endocarditis, or valve thrombosis, and there was no difference in valve-related outcomes according to TTVR valve type.

CONCLUSIONS TV dysfunction, endocarditis, and leaflet thrombosis were uncommon after TTVR. Patients with prior endocarditis were not at higher risk for endocarditis or other adverse outcomes after TTVR, and endocarditis occurred with similar frequency in different valve types. Though rare, leaflet thrombosis is an important adverse outcome, and further study is necessary to determine the appropriate level of prophylactic therapy after TTVR. (J Am Coll Cardiol 2019;73:148–57) © 2019 by the American College of Cardiology Foundation.
Transcatheter valve replacement has altered the therapeutic landscape for individuals with native and post-operative stenosis or regurgitation of the aortic, pulmonary, mitral, and tricuspid valves (1–11). In recent reports of data from the international VIVID registry (Valve-in-Valve International Database Registry), it was shown that transcatheter tricuspid valve (TV) implantation after prior surgical repair or bioprosthesis valve replacement can be performed successfully and safely, with good short-term outcomes in most patients (6–8). Although those early findings were encouraging, there is relatively little information about intermediate outcomes or associations between patient-related factors and valve-related outcomes after transcatheter TV replacement (TTVR). In patients who have undergone transcatheter aortic valve replacement, long-term durability, valve thrombosis, and to a lesser degree, endocarditis have emerged as important concerns (12–15), whereas recurrent obstruction and endocarditis are the most prominent adverse outcomes that have been observed after transcatheter pulmonary valve replacement (16,17). These observations are not surprising, insofar as all patients with a prosthetic valve are at risk for endocarditis (18–20). Given these findings, it is important to understand valve-related outcomes after TTVR, including the epidemiology of post-intervention endocarditis and the impact of prior endocarditis. Therefore, we undertook the present study to evaluate mid-term outcomes in a large international cohort of patients who underwent TTVR after surgical TV repair or replacement.

METHODS

PATIENTS AND PROCEDURES. Data were collected through the voluntary unsponsored Valve-in-Valve International Database Registry, for patients with acquired or congenital heart disease (CHD) who underwent TTVR after prior TV repair (valve-in-ring) or replacement (valve-in-valve) surgery, as described previously (6–8). TTVR procedures were performed clinically at the discretion of the implanting physician. Post-procedural antiplatelet or antithrombotic therapy was also determined by the treating clinicians. Data collected included basic demographic, historical, and diagnostic variables, procedural details, and follow-up information, as reported in prior studies (6–8). Follow-up data were actively collected through January 2018, when the database was locked for analysis. Patients with <3 years follow-up who were alive, reintervention-free, and had no follow-up visit entered in our database within the past 2 years were defined as lost to follow-up for this study. Patients who underwent transcatheter valve implantation in the tricuspid position of a modified Bjork Fontan (i.e., right atrium-to-right ventricle connection) were reported separately (21) and were not included in this study. For this follow-up outcomes study, we excluded patients who underwent catheterization with intention, but no attempt, to implant a valve (n = 5) or an unsuccessful attempt to deliver a valve to the implant site (n = 1), which were included in a prior report (6). Institutional review boards at participating centers approved submission of data.

OUTCOMES AND DATA ANALYSIS. The primary outcomes evaluated for this study were TV function,

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reintervention (surgical or transcatheter) on the TV, and endocarditis or valve thrombus after TTVR. Early post-implantation TV function was assessed using transesophageal or transthoracic echocardiographic according to methods described previously, with significant tricuspid regurgitation (TR) defined as moderate or greater in severity or reintervention for a stated indication of TR, and significant tricuspid stenosis (TS) defined as a mean Doppler gradient \( \geq 10 \text{ mm Hg} \) or reintervention for a stated indication of TS (6,7). Reinterventions included surgical or transcatheter intervention on the TTVR or for paravalvar regurgitation, or removal of the TTVR during surgery for another indication, as a separate procedure (i.e., implantation of a second valve or closure of paravalvular leaks during the primary TTVR procedure were not considered reinterventions). Indications for reintervention were at the discretion of treating physicians and were not standardized. Diagnosis of endocarditis was also determined by the treating physicians. Valve function and valve thrombosis were also reported. A composite valve-related adverse outcome metric was also defined as reintervention, endocarditis, thrombus, or significant TS or TR. Valve-related outcomes were compared according to prior history and valve-related factors.

Data were presented as frequency (%), median (minimum to maximum), or mean \( \pm \text{ SD} \). Differences between groups were assessed using chi-square analysis, Fisher exact test, independent samples Student’s t-test, or Wilcoxon rank sum test as appropriate. Cumulative incidence functions were estimated from competing risk data using Gray’s method (22), and time-related outcomes were depicted with cumulative incidence competing outcome curves. Survival analysis was performed with Cox regression, and univariable analysis of time-related outcomes other than death was performed by fitting proportional subdistribution hazards regression model with death as a competing risk using the method of Fine and Gray (23). Multivariable modeling was performed only for analysis of death, with \( \leq 1 \) variable included for every 10 events. In analyses of time-related outcomes, several early post-implantation variables (immediate post-TTVR mean TS gradient, significant residual TR/TS or paravalvular leak, and type of anticoagulation/antiplatelet therapy) were considered in addition to historical pre-implantation data. For Cox and Fine-Gray regression, hazard ratios (HRs) were presented with 95% confidence intervals (CIs). Cumulative incidence estimates were presented as proportion with 95% CI. All p values were 2-sided and were considered significant if \( p < 0.05 \). R (R Foundation for Statistical Computing, Vienna, Austria) and SPSS version 22 (IBM, Armonk, New York) were used for statistical analyses.

RESULTS

PATIENTS. From 2008 through 2017, 306 patients from 80 centers underwent TTVR after prior surgical TV replacement (valve-in-valve \( n = 284 \); 93%) or repair (valve-in-ring \( n = 22 \)) and were included in this analysis. Fifty-two of these patients (17%) had a prior history of endocarditis, which was the underlying cause of TV dysfunction that ultimately led to replacement or repair in most cases (Table 1). In 11 of the 52 patients with prior endocarditis, CHD with TV involvement or intervention-related TV injury was the underlying reason for TVR or repair, and in 8 others, it was endocarditis in the setting of nonvalvar CHD. TTVR was performed with a Melody valve in 138 patients (45%) (Medtronic, Dublin, Ireland) or a Sapien valve in 168 (55%; Sapien in 19, Sapien XT in 82, and Sapien 3 in 67) (Edwards Lifesciences, Irvine, California). Intraprocedural echocardiography was performed in 84% of patients, including transesophageal in 164 (54%), intracardiac in 57 (19%), transthoracic in 26 (8%), and multiple modalities (usually intracardiac and transesophageal) in 9 (3%). Additional interventions were performed at the same catheterization in 47 patients (15%) (Online Table 1).

SHORT-TERM OUTCOMES. In 7 patients, the implanted valve (Sapien in 3 patients, Sapien XT in 3 patients, Melody in 1 patient) was malpositioned or embolized; 4 of these patients had a second valve implanted during the same catheterization resulting in elimination of paravalvular regurgitation, 2 had the valve repositioned to the right atrium or inferior vena cava in the catheterization lab and underwent surgery emergently (\( n = 1 \)) or several months later (\( n = 1 \)), and 1 with a stable valve but paravalvular leakage underwent surgical TVR later during the same hospitalization.

After TTVR, the mean TV inflow gradient and TR grade measured by echocardiography improved significantly (both \( p < 0.001 \)). The mean Doppler TV gradient was reduced from a median of 9 mm Hg (0 to 29, 9.0 \( \pm 4.5 \) mm Hg) to 4 mm Hg (0 to 16, 3.8 \( \pm 2.0 \) mm Hg). In the 298 patients with a valve in place and a reported measurement, TR was trivial or none in 83% (\( n = 251 \)), mild in 16% (\( n = 49 \)), and moderate in 1% (\( n = 3 \)). Five patients (1.3%) had important residual TV dysfunction: moderate TR in 3 patients and significant TS in 2. There was no difference in the early post-intervention gradient according to
The median post-catheterization hospital stay was 2 days (1 to 81 days). All but 9 patients were placed on some form of antiplatelet/anticoagulant therapy, with 135 on aspirin or another antiplatelet agent only, 61 treated with warfarin alone, 9 on another anticoagulant, and 82 on both antiplatelet and anticoagulant agents (15 unknown). Of the 135 patients with a history of atrial fibrillation or flutter, 93 were discharged on anticoagulant therapy.

**Follow-up.** Survival. Patients were followed for a median of 15.9 months after implantation (0.1 to 90 months), with a total of 541 patient-years of follow-up. During that time, 36 patients died, 8 within 30 days (2.6%). The cumulative incidence of death on competing outcomes analysis, accounting for all of the competing risks depicted in the Central Illustration, was 0.18. Death was related to procedural complications in 2 patients, cardiovascular causes unrelated to the procedure in 20, and noncardiovascular causes in 14. All but 5 of the patients who died were in New York Heart Association functional class III or IV pre-TTVR, and 11 had been hospitalized and acutely ill before the procedure. By multivariable Cox regression analysis, older age (HR: 1.03 per year; 95% CI: 1.02 to 0.05 per year; p < 0.001) and patient acutely ill and hospitalized before TTVR (HR: 4.4; 95% CI: 2.1 to 9.1; p < 0.001) were associated with higher risk of death over time. When patients hospitalized pre-TTVR were excluded from the analysis, only older age (HR: 1.03; 95% CI: 1.01 to 1.05; p < 0.001) was significant. Prior history of endocarditis and other baseline or procedural factors were not associated with shorter survival.

Among the 244 patients who were alive and had not undergone reintervention, 184 had been followed for <3 years, 56 of whom were considered lost to follow-up according to the definition used for this study. Thirty-nine patients who were alive and reintervention-free had been followed for <1 year.

**Valve-related outcomes.** Valve-related adverse outcomes, including TV reintervention, endocarditis, thrombosis, and significant TS or TR, are depicted in the competing outcomes curve shown in the Central Illustration.

**TV reintervention.** Thirty-one patients (10%) underwent reintervention on the TV during follow-up: surgical TVR in 18 patients, a second TTVR within the first for malposition or dysfunction in 8 patients, redilation of the original TTVR in 3 patients, and device closure of paravalvar leak in 2 patients. Two other patients had the TTVR removed due to heart transplantation. Indications for reintervention are summarized in Table 2. Four reinterventions were within the first month after TTVR and 10 were during the first 6 months. Five patients died after reintervention, 3 within 30 days and 2 at 6 to 7 months later. The cumulative incidence of reintervention was 5% at 1 year, 12% at 3 years, and 19% at 5 years (Central Illustration). By Fine-Gray regression, higher early post-TTVR inflow gradient was associated with shorter freedom from reintervention (HR: 1.14 per mm Hg; 95% CI: 1.04 to 1.25 per mm Hg; p = 0.005).

**Endocarditis.** During follow-up, endocarditis was diagnosed in 8 patients 2 to 29 months after TTVR. Six of these patients had underlying CHD (4 with Ebstein...
anomaly), and 1 had a prior history of endocarditis associated with intravenous drug use. Causative organisms are listed in Table 3. By competing risk analysis, the estimated cumulative incidence of endocarditis was 0.017 (0.006 to 0.040) at 1-year post-TTVR and 0.042 (0.019 to 0.079) at 3 years and beyond, and the annualized incidence rate was 1.5% per patient-year (95% CI: 0.4% to 2.5%). On univariable analysis, there were no significant associations between endocarditis and potential risk factors, including prior history of endocarditis, method of anticoagulant/antiplatelet therapy, and immediate post-TTVR right ventricular inflow gradient. One of the 8 patients had a pre-existing transvenous pacing system, and 2 with a prosthetic pulmonary valve or conduit had also undergone transcatheter pulmonary valve replacement, but none had evidence of lead or pulmonary valve involvement. TTVR had been performed with a Melody valve in 5 of these patients and a Sapien valve in 3 (Sapien XT in 2, Sapien 3 in 1). Four of these patients had been discharged on antiplatelet therapy only, 1 on warfarin only, and 3 on both.

No patient died as a result of or in the context of an endocarditis episode. Four patients underwent reintervention for endocarditis, including surgical TVR in 3 patients and balloon TV valvuloplasty for moderate TS in 1 patient. In the other 4 patients, the endocarditis was not associated with deterioration of valve function and was treated medically without reintervention.
Among the 184 patients who had been followed for <3 years, the proportion with a prior history of endocarditis (i.e., as a potential risk factor for post-TTVR endocarditis) did not differ significantly from those with >3 years follow-up (p = 0.23). Of the 56 patients defined in the preceding text as lost to follow-up, 29% had a prior history of endocarditis, which was less than in the cohort not lost to follow-up (17%; p = 0.056), and 1 had been diagnosed with post-TTVR endocarditis before being lost to follow-up.

**VALVE THROMBOSIS.** An additional 8 patients (Melody n = 5, Sapien n = 3) were diagnosed with clinically relevant valve thrombosis (n = 4) or presumed thrombus (immobility or thickening of the leaflets; n = 4), 3 within several days of TTVR, 2 within 2 months (early), and 3 beyond 6 months (late). The cumulative incidence of thrombosis on competing risk analysis was 0.033 (0.015 to 0.061) at 3 years and beyond, and higher immediate post-TTVR inflow gradient was associated with increased risk of thrombosis (HR: 1.38 per mm Hg; 95% CI: 1.13 to 1.70 per mm Hg; p = 0.002). Examples are depicted in Figures 1 and 2 and Online Videos 1A and 1B. None of these patients had a prior history of endocarditis, 3 had a transvenous pacing system, 3 had atrial fibrillation/flutter, and 2 had other cardiac valve prostheses (1 mitral, 1 pulmonary)—these factors were not significantly associated with thrombosis. All 3 patients with acute thrombosis were treated with anti-coagulation after diagnosis, with resolution of the thrombus in 2 patients; 1 patient also had a balloon valvuloplasty at the time, and the other 2 patients underwent surgical TVR 8 and 41 months later for TR or mixed TR and TS, respectively. Of the 2 patients with early thrombus, 1 underwent surgical TVR, and the other (who had a second valve implanted at the time of TTVR due to distal implantation and moderate regurgitation around the first) was started on warfarin, with resolution of thrombus and good TV function 48 months later. Notably, the patient who underwent surgical TVR for early thrombosis also developed early thrombosis of the surgical valve, which was treated medically and left with severe TR and moderate TS. All 3 patients with late thrombosis underwent reintervention, 1 patient at the time of diagnosis and 2 patients later for progressive TS or TR. Thus, at the most recent follow-up, 2 of the 8 had the original TTVR in place with good function.

The 3 patients with acute thrombus were discharged on both anticoagulant and antiplatelet therapy after the immediate treatment, but 2 of the 3 patients had not been on post-procedural anticoagulant before the thrombus; 4 of 5 patients with subacute or late thrombus were discharged from the original TTVR procedure on antplatelet therapy alone and 1 on a nonwarfarin oral anticoagulant. Thus, only 2 of the patients who developed a thrombus (p = 0.13 by log-rank test), and 1 of the 5 with an acute or early (within 2 months) thrombus (p = 0.15 by Fisher exact test), were on anticoagulant therapy.

**TTVR function.** Follow-up echocardiographic data were available for all but 25 of the surviving patients who had not undergone reintervention, 17 patients of whom had been followed for <1 year. In addition to the patients who underwent TV reintervention, significant TR (moderate or severe, n = 7), TS (mean Doppler gradient >10 mm Hg; n = 6), or combined TS and TR (n = 1) was diagnosed in 14 patients after TTVR, 4 within 6 months of implantation (immediate n = 1). The mechanisms of TS or TR were not reported in detail. Missing data limited the analysis, but only 32% of patients who were diagnosed with significant TR or TS during follow-up were discharged on antithrombotic agents, compared with 52% of those who did not develop significant TV dysfunction. There was no apparent difference in TV dysfunction.
according to transcatheter valve type; of the valves with TR, 4 were Melody and 3 were Sapien, and of the valves with TS or mixed dysfunction, 6 were Sapien and 1 was Melody.

**DISCUSSION**

**TTVR OUTCOMES AND IMPLICATIONS.** In prior reports from this international multicenter registry, we showed that TTVR after prior surgical TV replacement or repair was associated with a high
rate of technical success and few serious procedural complications, restoration of good TV function, and clinical improvement in most patients (6-8). In particular, given the importance of endocarditis and thrombosis after other transcatheter valve therapies (12-16, 18, 19, 24, 25), we undertook this study to assess the burden of these outcomes after TTVR, to evaluate whether there were patient-related or procedural factors associated with greater risk of endocarditis and thrombosis after TTVR, and to determine whether prior endocarditis history was associated with post-TTVR outcomes.

TV ENDOCARDITIS. Compared with left-sided endocarditis, relatively little is known about endocarditis affecting the right-sided valves. In studies of surgical TV replacement, endocarditis was a common comorbidity and underlying cause of TV dysfunction (20, 26). However, there was no information in those studies regarding the causes and details of the original endocarditis episode. Similarly, in the present cohort, a prior history of endocarditis was reported in 17% of patients, most of whom underwent TV replacement as a consequence. In patients undergoing surgery for endocarditis, investigators have observed several pathological differences between right- and left-sided endocarditis (27), and it has been reported that TV involvement is associated with worse survival (28). Although there is limited information on endocarditis after TV repair or replacement, regardless of the underlying pathology, it appears to be an uncommon, but nontrivial, late complication that more often affects mechanical than bioprosthetic valves (29-32).

In this large unselected cohort, endocarditis was reported in 8 of 306 patients after TTVR, with various organisms affecting both valves similarly. Diagnostic criteria for tricuspid or pulmonary valve endocarditis are less clear than for the more common left-sided infection, and it was recently reported that right-sided endocarditis is less likely to be invasive than left-sided disease (27), so there may be some ambiguity about the certainty of diagnosis. As also noted in this study, noninfected thrombus may be observed after TTVR, and may be difficult to distinguish from endocarditis with echocardiography. Two of the 8 cases in this series were presumed to have endocarditis but without an identified organism. Although the available data are insufficient to draw firm conclusions, on the basis of the published reports and this study, there does not appear to be an obvious difference between endocarditis risk after surgical TVR and TTVR. Although small numbers precluded robust assessment of risk factors, only 1 of the 8 cases of endocarditis (16%) had a prior history of endocarditis, similar to the 17% prior prevalence in the overall cohort. Thus, prior endocarditis does not appear to confer substantial risk of post-TTVR endocarditis and should not exclude patients from this therapy. Given the pathogenesis of endocarditis and experimental data suggesting a benefit of thromboprophylaxis (33, 34), it will be important to assess whether and what type of anticoagulant/antithrombotic therapy and hemodynamic factors after TTVR are associated with the risk of endocarditis.

TV THROMBUS. The issue of leaflet thrombosis after transcatheter aortic valve replacement, often subclinical, has emerged as an important consideration in that population (15), and there is ongoing debate about the proper approach to post-implantation thromboprophylaxis. In this study, valve thrombus was not investigated systematically with computed tomography to detect subtle or minor cases, which may have been missed, but 8 patients were found to have hemodynamically significant thrombus confirmed by transesophageal or intracardiac echocardiography, leading to surgical TVR or redo TTVR in 6. Three of these cases were acute, and may have been related to hemodynamic factors unique to transcatheter valve implantation in the TV position, including large dilated right atrial and ventricular chambers, impaired right ventricular function, and consequently, sluggish flow across the new valve. A higher immediate post-TTVR inflow gradient was associated with greater risk of both leaflet thrombosis and TV reintervention. Although these findings should be considered preliminary, they suggest that implanters should strive for maximal gradient relief during the implant, and perhaps consider post-dilution of the implanted TTVR even for relatively minor residual gradients.

Only 2 of the 8 patients with a clinically evident thrombus, and 1 of the 5 with acute or early valve thrombosis, were on anticoagulation at the time, less than patients who did not develop a thrombus, although this finding should be interpreted with caution due to the small number of events. In addition, there have been anecdotal cases in which rising TV inflow gradients were reversed with initiation of anticoagulation. Thus, whereas frank thrombosis was uncommon in this series, the importance of thrombus prevention after TTVR may not yet be fully appreciated, and it is worth considering whether conditions after TTVR merit utilization of anticoagulant or dual antiplatelet therapy rather than a single antiplatelet
agent. Over one-half of the patients in this series were discharged on some form of anticoagulant therapy, with or without an antiplatelet medication, whereas almost all of the others were on antiplatelet therapy alone. We did not collect data on changes in or compliance with these therapies, and the absolute number of clinically detected thrombotic episodes was small, precluding analysis that might provide insight into risks and benefits associated with different approaches.

**TV FUNCTION.** As we reported previously, TV function remained stable in the majority of patients who underwent TTVR. At most recent follow-up, which was beyond 3 years in a substantial number of patients, the mean Doppler inflow gradient was typically in the 4 to 5 mm Hg range, and TR was mild or less. This is similar to gradients observed across newly implanted surgical bioprosthetic valves in the TV position (35), but as we discussed in a prior report, the magnitude of gradient across a TTVR that should be considered significant has not been determined (6). As discussed in the preceding text, the hemodynamic implications of subtle leaflet thrombus were not investigated in this study. Subclinical thrombosis has emerged as an important concern after transcatheter aortic valve replacement (15), and insofar as the valves employed for TTVR are often transcatheter aortic valves used off-label, it is reasonable to hypothesize that this may be a relevant concern with TTVR as well. Accordingly, it will important to examine this issue in depth, particularly because small increases in the TV inflow gradient can translate into significant symptoms and sequelae.

**METHODOLOGICAL CONSIDERATIONS AND STUDY LIMITATIONS.** In addition to the issues addressed in the preceding text, there were several important limitations to this study. Data were collected as part of a voluntary unsponsored retrospective registry with no core labs, no auditing of data, and no standardized criteria for anticoagulation/antiplatelet therapy or reintervention. Similarly, assessment of TV function by echocardiography depends on a number of factors, which were not standardized or analyzed in this study, and further studies will be necessary to characterize post-TTVR hemodynamics more rigorously. Details of prior endocarditis episodes, including the organism, duration before TTVR, involvement of the native or prosthetic TV, and number of episodes were unknown for this study, so we cannot make inferences about risks related to specific circumstances. Incomplete follow-up was also an important limitation: in most surviving patients, follow-up was available for <3 years, limiting our ability to assess freedom from valve-related events over time. The disparity of loss to follow-up among patients with a prior history of endocarditis may confound assessment of this as a potential risk factor for post-TTVR adverse outcomes. Thus, these findings should be considered preliminary, and more robust estimates will require longer evaluation in a larger cohort of patients. There were few patients with underlying functional TR related to left-heart disease or valve-in-ring TTVR procedures, so future studies will be required to provide insight into these important cohorts.

**CONCLUSIONS**

TTVR was hemodynamically and clinically beneficial in patients of various ages and underlying disease states. Adverse valve-related outcomes were relatively uncommon in this high-risk cohort, and valve function remained excellent in the vast majority of patients followed beyond 3 years post-TTVR. Although a history of endocarditis and prosthetic material in the heart are both risk factors for subsequent endocarditis, patients with prior endocarditis were not at higher risk for adverse valve-related outcomes after TTVR. Endocarditis after TTVR was uncommon, and occurred with similar frequency in patients with both valves. Leaflet thrombosis was also an uncommon but important adverse outcome. Further study is necessary to determine the appropriate level of thromboprophylactic therapy after TTVR.

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**PERSPECTIVES**

**COMPETENCY IN PATIENT CARE AND PROCEDURAL SKILLS:** Early experience suggests that TTVR can be associated with favorable mid-term clinical outcomes in patients with bioprosthetic tricuspid stenosis and/or regurgitation.

**TRANSLATIONAL OUTLOOK:** Potential relationships between hemodynamic factors and valve leaflet thrombosis after TTVR require further study to guide antithrombotic therapy.


**KEY WORDS** endocarditis, percutaneous valve, stenosis, thrombus, transcatheter valve implantation

[APPENDIX For a list of additional centers and investigators participating in the VIVID registry as well as a supplemental table and videos, please see the online version of this paper.]