Transcatheter Aortic Valve Implantation for Failed Surgical Aortic Bioprostheses Using a Self-Expanding Device (from the Prospective VIVA Post Market Study)

Ran Kornowski, MD[a,*], Bernard Chevalier, MD[b], Jean-Philippe Verhoye, MD[c], David Holzhey, MD[d], Axel Harnath, MD[e], Ulrich Schäfer, MD[f], Emmanuel Teiger, MD, PhD[g], Thibaut Manigold, MD[h], Thomas Modine, MD[i], Geraud Souteyrand, MD[j], Didier Champagnac, MD[k], Jae K. Oh, MD[l], Shuzhen Li, PhD[m], and Didier Tchétché, MD[n], on behalf of the VIVA Investigators

Patients with symptomatic aortic stenosis are often treated with a surgical valve replacement. Surgical bioprosthetic valves degenerate over time and therefore may necessitate a redo surgery. This analysis reports the 2-year clinical outcomes of the Valve-in-Valve study, which evaluated transcatheter aortic valve implantation using the CoreValve and Evolut R devices in patients with degenerated surgical aortic bioprostheses at high risk for surgery. The prospective Valve-in-Valve study enrolled 202 eligible patients with failing surgical aortic bioprostheses due to stenosis, regurgitation, or a combination of both. The Evolut R bioprosthesis was used in 90.5% of valve-in-valve transcatheter aortic valve implantation cases. Two-year all-cause and cardiovascular mortality rates were 16.5% and 11.1%, respectively. Other clinical events included stroke (7.9%), disabling stroke (1.7%), and new pacemaker implantation (10.1%). The 2-year all-cause mortality rate was significantly higher in patients with discharge mean gradients ≥20 mmHg vs. those with lower mean gradients (21.0% vs 7.6%, p = 0.025). Discharge mean gradients ≥20 mm Hg were associated with smaller surgical bioprostheses (OR, 7.2 [95% CI 2.3 to 22.1]. In patients with failing surgical aortic bioprostheses, valve-in-valve treatment using a supra-annular self-expanding bioprosthesis provides significant functional improvements with acceptable rates of complications, especially if a postprocedural mean gradient of <20 mmHg can be achieved. **(Am J Cardiol 2021;144:118−124)**

In elderly patients who present with symptomatic, severe aortic stenosis or regurgitation, the need for valve replacement is required.[1,2] The surgical options include mechanical or bioprosthetic valve implants, and the decision is based primarily on the patient's age.[3] Over time the bioprosthetic valves degenerate and become less effective, which results in the need for replacement of the failed bioprostheses.[4−6] For many patients who are at high risk for surgery due to age or comorbidities, reoperation is not feasible. Implantation of a transcatheter bioprosthesis in a failed surgical valve offers a less invasive option for these patients and has shown good results.[7−12] In the postmarket multicenter Valve-in-Valve (VIVA) trial, eligible symptomatic patients with degenerated aortic bioprosthesis who underwent elective treatment with a self-expanding transcatheter aortic valve were evaluated. One-year results showed improved valve hemodynamics and low mortality rates, and confirmed the safety and efficacy of a valve-in-valve (ViV) using the CoreValve or Evolut R.[9] The aim of this study was to evaluate the 2-year clinical outcomes.

Methods

VIVA is a prospective, observational, single-arm, postmarket multicenter study that assessed the safety and effectiveness of ViV transcatheter aortic valve implantation (TAVI) using a CoreValve or Evolut R bioprosthesis (Medtronic, Minneapolis, Minnesota). Details of the inclusion and exclusion criteria for selection of patients and procedures of the trial have been previously published.[9] The present analysis evaluated the final 2-year clinical outcomes of the patients from the VIVA trial. This study complied with

[a]Department of Cardiology, Rabin Medical Center, Petah Tikva, Israel; [b]Ramsay Générale de Santé, Institut Cardio-vasculaire Paris-Sud, Massy, France; [c]Department of Cardiovascular Surgery, CHU Rennes, Rennes, France; [d]Department of Cardiac Surgery, Leipzig Heart Institute, Leipzig, Germany; [e]Department of Cardiology, Sana-Herzzentrum Cottbus, Cottbus, Germany; [f]Department of Cardiology, Angiology and Intensive Care Medicine, Marienkrankenhaus Hamburg, Hamburg, Germany; [g]Interventional Cardiology Unit, CHU Mondor, Créteil, France; [h]Cardiology Service, CHU de Nantes, Nantes, France; [i]Department of Cardiovascular Surgery, CHU Lille, Lille, France; [j]Department of Cardiology, CHU Clermont-Ferrand, Université Clermont Auvergne, Clermont-Ferrand, France; [k]Department of Cardiology, Medipole Hospital Privé, Villeurbane, France; [l]Department of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota; [m]Coronary and Structural Heart Clinical Department, Medtronic, Minneapolis, Minnesota; and [n]Groupe CardioVasculaire Interventionnel, Clinique Pasteur, Toulouse, France. Manuscript received September 10, 2020; revised manuscript received and accepted December 15, 2020.

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*Corresponding author: Tel: 009-723-937-6441.

E-mail address: rkornowski@clalit.org.il (R. Kornowski).

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the Declaration of Helsinki, and the research protocol was approved by each site's ethics committee according to individual national requirements. The Cardiovascular European Research Center (Massy, France) was responsible for independent site management, monitoring, and clinical events committee adjudication.

The primary safety end point for this study was cardiovascular death 30 days post-procedure. Secondary end points were defined according to Valve Academic Research Consortium-2 (VARC II) criteria[13]; clinical outcomes included peri-procedural myocardial infarction, major and minor access site complications, major bleeding, stroke, acute kidney injury stage II/III, and new pacemaker implantation. Echocardiograms were completed at screening, discharge, and 1-year post-procedure. Two-year echo data were not collected in this study. Echocardiograms were analyzed by an independent core laboratory (Mayo Clinic, Rochester, Minnesota).[14]

For the present analysis, expected patient-prothesis mismatch (PPM) was determined for each patient based on instruction for use and valve hemodynamic performance of preexisting surgical valve size and the patient's body surface area. PPM was defined using the VARC II definitions for patients with body mass index (BMI) <30 kg/m^2 (severe PPM: effective orifice area index (EOAi) ≤0.65 cm^2/m^2; moderate PPM : 0.65cm^2/m^2 ≤ EOAi ≤ 0.85 cm^2/m^2; and no PPM: EOAi >0.85 cm^2/m^2) and for patients with BMI ≥30 kg/m^2 (severe PPM: EOAi <0.60 cm^2/m^2; moderate PPM: 0.60 cm^2/m^2 ≤ EOAi ≤ 0.70 cm^2/m^2; and no PPM : EOAi >0.70 cm^2/m^2). Valve fracturing prior to a ViV procedure was uncommon practice during patient accrual for this trial and was not captured in this study.

The analytic cohort comprised all patients undergoing attempted ViV. Categorical variables are presented as the number of subjects (%) and were compared using the chi-square test, while continuous variables are presented as mean ± SD or median (interquartile range) and were compared using independent samples *t*-tests or analysis-of-variance F test (for 3 or more group comparisons). Patients were stratified by discharge mean gradient ≥20 mm Hg vs <20 mm Hg, true inner aortic diameter (≤20 mm vs >20 mm), and failure mode (stenotic, regurgitant, or combined). Clinical outcomes at 2 years are reported as Kaplan-Meier estimates in time-to-event analyses, and subgroups were compared using the log-rank test. The Kaplan-Meier estimate of all-cause mortality stratified by discharge mean gradient was landmarked at day 14 to exclude patients who died or were censored before discharge. No statistical techniques were used to impute missing data. Subjects with missing data were not included in the corresponding portion of the analysis. The number of subjects included in each analysis is reported. For predictors of 2-year mortality, a complete list of variables included in the univariable analysis are provided in Supplemental Table 1. Predictors of 2-year mortality were analyzed after discharge (Supplementary Table 1) and after 1 year (Supplemental Table 2). In the multivariable Cox proportional hazard model, candidate variables were selected from univariable predictors with a p value ≤0.20. The final stepwise method with thresholds for entry and exit required a p value of 0.15. All testing used a 2-sided alpha level of 0.05. Statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, North Carolina).

Results

From November 2014 to June 2016 a total of 202 patients were enrolled and underwent attempted implant (Figure 1). Patient demographics are shown in Table 1. Patients in this trial had a mean age of 79.9 ± 7.2 years. The mean Society of Thoracic Surgeons score was 6.6 ± 5.1%, and the mean European System for Cardiac Operative Risk Evaluation score (EuroSCORE) was 25.0 ± 14.3%. Most patients were in New York Heart Association (NYHA) functional class III or IV (71%). The comorbidities of hypertension (84%) and chronic obstructive pulmonary disease (21%) were among the most pronounced in the trial patients.[9]

As previously described, 17 deaths occurred post-procedure through 12 months.[9] There were an additional 15 deaths between 12 months and 2 years, resulting in an all-cause mortality rate of 16.5% with cardiovascular mortality at 11.1% (Figure 2). Two-year mortality was not significantly different when stratified by true inner diameter (≤20 mm vs >20 mm, p = 0.866) (Figure 2). There was no relationship found between expected PPM and 2-year mortality (Supplementary Figure 1). Two-year mortality was significantly associated with discharge mean gradients ≥20 mm Hg compared with mean gradients <20 mm Hg (21.0% vs 7.6%, p = 0.025) (Figure 2). Discharge mean

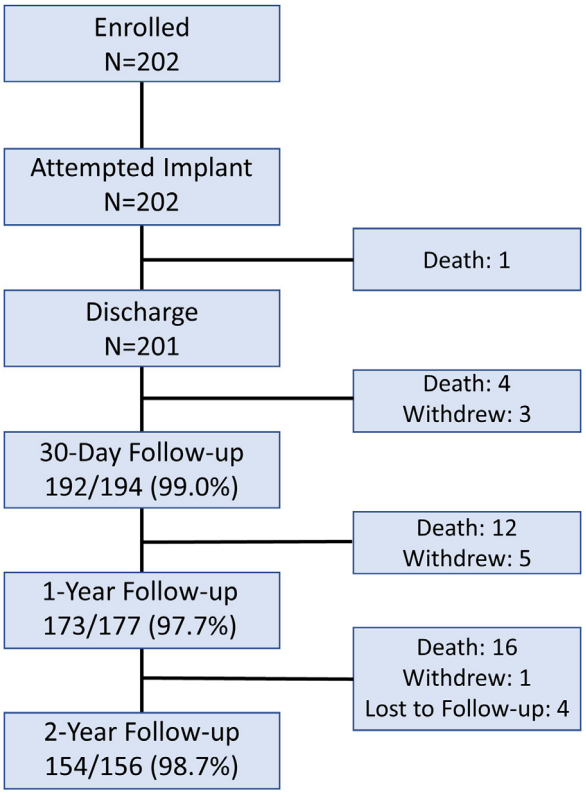

Figure 1. Patient flow diagram.

Table 1
Baseline characteristics according to failure mode of surgical valve

Characteristic	All (N=202)	Bioprostheses Failure Mode			p Value
		Stenosis (N=114)	Regurgitation (N=46)	Combined (N=42)	
Age (years)	79.9 ± 7.2	79.4 ± 7.1	80.1 ± 8.6	81.1 ± 5.6	0.446
BSA (m^2)	1.8 ± 0.2	1.8 ± 0.2	1.8 ± 0.2	1.8 ± 0.2	0.780
Men	96 (48%)	51 (45%)	27 (59%)	18 (43%)	0.221
STS-PROM (%)	6.6 ± 5.1	6.4 ± 4.6	6.1 ± 4.9	7.6 ± 6.5	0.335
Diabetes mellitus	53 (26%)	36 (32%)	7 (15%)	10 (24%)	0.096
History of hypertension	167/200 (84%)	94 (83%)	39/44 (89%)	34 (81%)	0.568
Peripheral vascular disease	28 (14%)	15 (13%)	6 (13%)	7 (17%)	0.840
Previous stroke	10 (5%)	10 (9%)	0	0	0.017
Previous transient ischemic attack	5 (3%)	2 (2%)	2 (4%)	1 (2%)	0.602
Chronic lung disease/COPD	42 (21%)	26 (23%)	5 (11%)	11 (26%)	0.152
Percutaneous coronary intervention	54 (27%)	36 (32%)	11 (24%)	7 (17%)	0.155
Balloon valvuloplasty	12 (6%)	9 (8%)	0	3 (7%)	0.123
Previous myocardial infarction	22 (11%)	15 (13%)	3 (7%)	4 (10%)	0.531
NYHA classification					0.536
I	7/198 (3%)	7/113 (6%)	0/45	0/40	
II	51/198 (26%)	26/113 (23%)	11/45 (24%)	14/40 (35%)	
III	108/198 (55%)	64/113 (57%)	25/45 (56%)	19/40 (47%)	
IV	32/198 (16%)	16/113 (14%)	9/45 (20%)	7/40 (18%)	
Surgical valve age (years)	9.3 ± 4.4	8.9 ± 4.4	9.7 ± 3.5	9.9 ± 5.0	0.339
Failed bioprosthetic surgical valve					0.035
Stented	188 (93%)	108 (95%)	39 (85%)	41 (98%)	
Stentless	14 (7%)	6 (5%)	7 (15%)	1 (2%)	
Homograft	0	0	0	0	
Bioprosthesis labeled size (mm)	22.7 ± 2.1	22.6 ± 2.0	23.2 ± 2.2	22.5 ± 2.0	0.172
≤21	84/201 (42%)	48 (42%)	16 (35%)	20 (48%)	0.151
>21 and <25	65/201 (32%)	39 (35%)	12 (26%)	14 (33%)	
≥25	52/201 (26%)	26 (23%)	18 (39%)	8 (19%)	
Calcified aorta					0.065
None	71/159 (45%)	34/85 (40%)	22/41 (54%)	15/33 (46%)	
Mild	56/159 (35%)	28/85 (33%)	17/41 (41%)	11/33 (33%)	
Moderate	25/159 (16%)	16/85 (19%)	2/41 (5%)	7/33 (21%)	
Severe	7/159 (4%)	7/85 (8%)	0/41	0/33	
Bioprosthesis valve internal diameter (mm)	20.9 ± 2.7	21.0 ± 2.7	21.0 ± 3.0	20.4 ± 2.3	0.425
<20	70/171 (41%)	40/96 (42%)	15/40 (37%)	15/35 (43%)	0.719
≥20 and <23	60/171 (35%)	32/96 (33%)	14/40 (35%)	14/35 (40%)	
≥23	41/171 (24%)	24/96 (25%)	11/40 (28%)	6/35 (17%)	

Values are mean ± SD, n (%). Denominators are presented if different from column headers. BSA = body surface area, COPD = chronic obstructive pulmonary disease, NYHA = New York Heart Association, STS-PROM = Society of Thoracic Surgeons Predicted Risk of Mortality.

gradients of ≥20 mm Hg were shown to be associated with female sex and smaller size of a patient's first bioprosthesis (Table 2). Similarly, smaller transcatheter valve sizes were associated with discharge mean gradients ≥20 mm Hg (Table 2).

Two-year all-cause mortality rates were similar across the 3 surgical aortic valve (SAV) failure modes (p = 0.257), although the rate was numerically higher in patients with a stenotic SAV (20.4%) compared with regurgitation (11.2%) or a combined failure mode (12.2%) (Figure 2). However, cardiovascular death at 2 years was significantly different across the 3 SAV failure modes of stenosis (15.9%), regurgitation (5.0%), or a combination (4.9%, p = 0.050) (Table 3).

The 2-year stroke rate was 7.9%, which increased by 2 patients from the 1-year follow-up; both were disabling strokes (1.7%). At 2 years there were 2 additional prosthetic valve endocarditis cases (1.8%) and 1 additional prosthetic valve thrombosis case (1.6%) reported. The pacemaker rate remained unchanged at 10.1% at 2 years (Table 3). Two-year NYHA functional class remained predominately class I or II with no class IV reported (91.0% in class I/II and 9.0% in class III, Figure 3).

Multivariable analysis of mortality showed that a discharge mean gradient of ≥20 mmHg, low BMI , and NYHA functional classification IV were indicators of increased risk for all-cause mortality between 14 days and 2 years (Figure 4). The expected PPM was not shown to be related to mortality during this time period. Univariable analysis of mortality between 1 and 2 years shows only age as a predictor for 2-year mortality. A full list of variables from the univariable model at discharge and 1 year can be found in Supplementary Tables 1 and 2.

Discussion

This analysis of patients who underwent a TAVI ViV procedure using a supra-annular, self-expanding CoreValve or

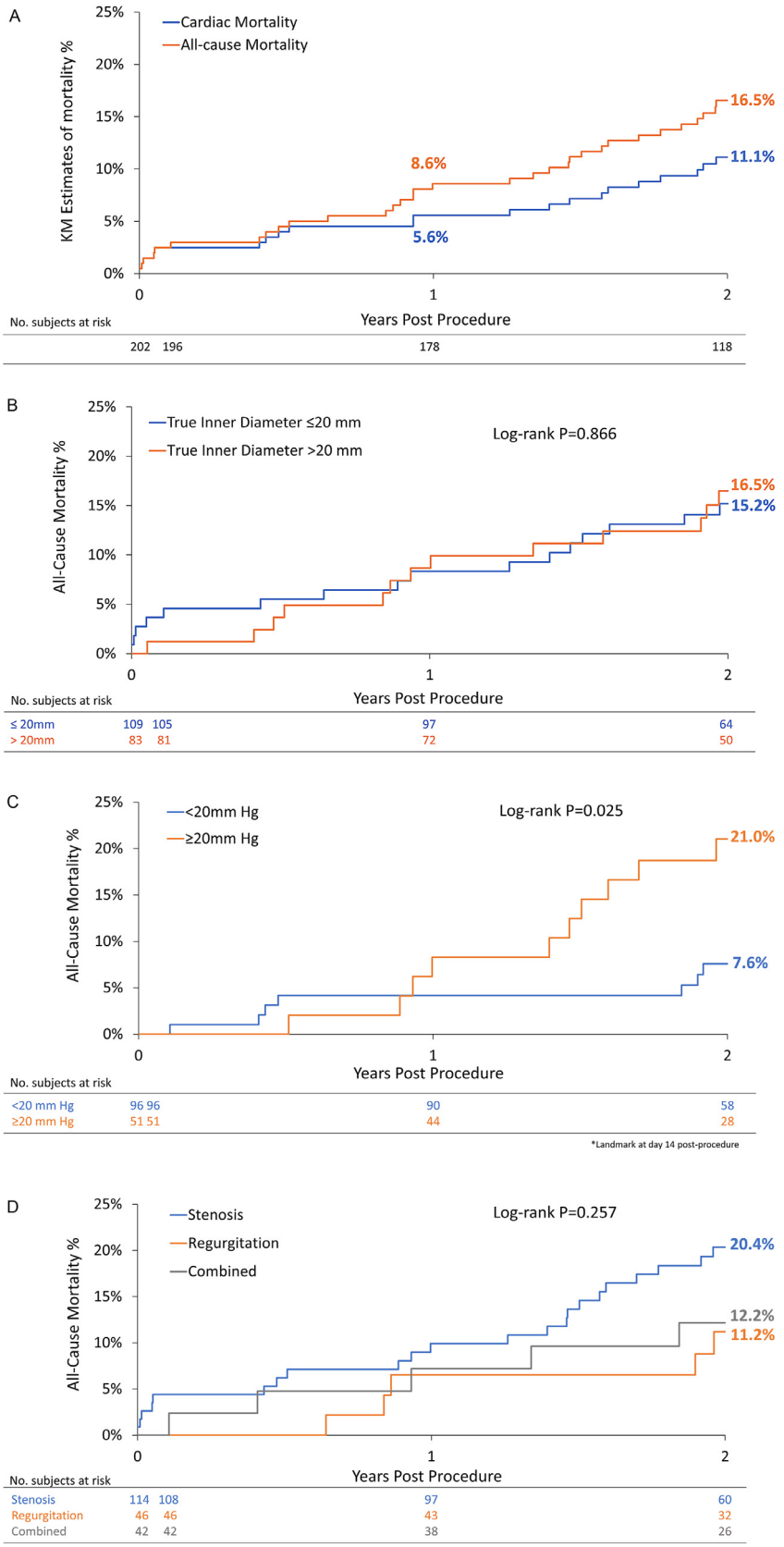

Figure 2. Clinical outcomes after valve-in-valve transcatheter aortic valve implantation (TAVI). *(A)* Kaplan-Meier all-cause mortality and cardiovascular mortality time to event curves through 2 years. *(B)* Kaplan-Meier estimates for all-cause mortality through 2 years stratified by true inner diameter (≤20 vs >20 mm). *(C)* Kaplan-Meier estimates of all-cause mortality through 2 years stratified by discharge mean gradient (<20 mm Hg vs ≥20 mm Hg). Data for discharge mean gradient were landmarked at day 14. *(D)* Kaplan-Meier estimates of all-cause mortality through 2 years stratified by surgical valve failure mode.

Table 2
Characteristics associated with discharge mean gradient

	Discharge Mean Gradient <20 mm Hg (N=100)	Discharge Mean Gradient ≥20 mm Hg (N=51)	p Value
Baseline characteristic			
Men	51 (51%)	17 (33%)	0.039
STS score (%)	6.5 ± 4.9	7.2 ± 6.4	0.489
SAV age (years)	9.8 ± 4.5	8.4 ± 3.5	0.041
Size of first bioprosthesis (mm)	23.2 ± 2.0	21.7 ± 1.7	<0.001
≤21	28/99 (28%)	32 (63%)	
>21 and <25	41/99 (41%)	14 (27%)	
≥25	30/99 (30%)	5 (10%)	
Post-procedure characteristics			
CoreValve (mm)			0.033
23	1/9 (11%)	3/4 (75%)	
26	6/9 (67%)	1/4 (25%)	
29	2/9 (22%)	0/4	
31	0/9	0/4	
Evolut R (mm)			<0.001
23	51/91 (56%)	41/47 (87%)	
26	29/91 (32%)	4/47 (9%)	
29	11/91 (12%)	2/47 (4%)	

Values are mean ± SD or n (%). Denominators are presented if different from column headers. SAV = surgical aortic valve, STS = Society of Thoracic Surgeons.

Evolut R bioprosthesis confirms the safety and sustainable effectiveness of these devices for treatment of surgical valve failure through 2 years and provides further insights on the importance of improved hemodynamics after TAVI. This report highlights several key findings: 1, Low 2-year rates of all-cause and cardiovascular mortality after ViV treatment were observed; 2, tThere was no association between surgical valve size or failure mode and risk for mortality through 2 years; and 3, discharge mean gradients ≥20 mm Hg were associated with a greater risk of all-cause mortality between 14 days and 2 years compared with a discharge mean gradient <20 mm Hg.

The present analysis showed that patients with higher discharge mean gradients were more commonly female and their failed surgical valve was predominantly ≤21 mm. Small failed surgical valves require implantation of small transcatheter replacement valves, resulting in a greater likelihood of larger than desired mean gradients across the aortic valve area.[8,10,15] Mean gradients ≥20 mm Hg after a ViV TAVI have been found to be associated with increased mortality[11] and were more common in patients that had small failed surgical valves due to small aortic valve area.[8] In this study 63% of patients who had a discharge mean gradient ≥20 mm Hg also had a first bioprosthesis ≤21 mm, highlighting the association between small original valve size and increased mean gradients and consistent with observations by Deeb et al, who also noted the importance of surgical valve size on post-procedure hemodynamics where patients with small surgical valves were more likely to have mean gradients ≥20 mm Hg at discharge and 1 month.[10]

Previous studies have also shown an association between SAV failure mode and increased mean gradients, where SAV failure due to stenosis was shown to be associated with increased mean gradients after a TAVI procedure.[10] In this study the association of SAV failure mode and all-cause mortality was not significant. However, it is worth

Table 3
Two-year clinical outcomes

Variable	All (N=202)	Bioprostheses Failure Mode			p Value
		Stenosis (N=114)	Regurgitation (N=46)	Combined (N=42)	
Outcome					
All-cause mortality	32 (16.5%)	22 (20.4%)	5 (11.2%)	5 (12.2%)	0.257
Cardiovascular	21 (11.1%)	17 (15.9%)	2 (5.0%)	2 (4.9%)	0.050
Non-cardiovascular	11 (6.1%)	5 (5.4%)	3 (6.5%)	3 (7.6%)	0.830
Peri-procedural myocardial infarction	1 (0.5%)	0	0	1 (2.4%)	0.149
Access-site complication	14 (7.0%)	9 (7.9%)	2 (4.3%)	3 (7.1%)	0.719
Major access site complication	3 (1.5%)	2 (1.8%)	0	1 (2.4%)	0.614
VARCII bleeding	39 (20.0%)	21 (19.6%)	12 (26.1%)	6 (14.4%)	0.406
Life-threatening bleeding	2 (1.2%)	1 (1.3%)	1 (2.2%)	0	0.640
Major bleeding	21 (10.8%)	11 (10.1%)	6 (13.0%)	4 (9.9%)	0.831
Acute kidney injury	3 (1.7%)	3 (3.1%)	0	0	0.282
Stage II or III	2 (1.2%)	2 (2.2%)	0	0	0.420
Prosthetic valve endocarditis	3 (1.8%)	2 (2.1%)	1 (2.4%)	0	0.660
Prosthetic valve thrombosis	3 (1.6%)	1 (0.9%)	1 (2.2%)	1 (2.6%)	0.751
Coronary artery obstruction requiring intervention	5 (2.5%)	2 (1.8%)	2 (4.3%)	1 (2.4%)	0.641
All stroke	15 (7.9%)	12 (11.4%)	2 (4.4%)	1 (2.6%)	0.131
Disabling stroke	3 (1.7%)	3 (3.2%)	0	0	0.280
New pacemaker implantation*	20 (10.1%)	14 (12.7%)	2 (4.3%)	4 (9.5%)	0.303
New pacemaker implantation†	20 (12.0%)	14 (15.8%)	2 (5.0%)	4 (10.5%)	0.229

Values are number of patients with events (%), depicted as Kaplan-Meier event rates.
* Includes patients with baseline pacemaker.
† Excludes patients with baseline pacemaker. VARC-II = Valve Academic Research Consortium-2.

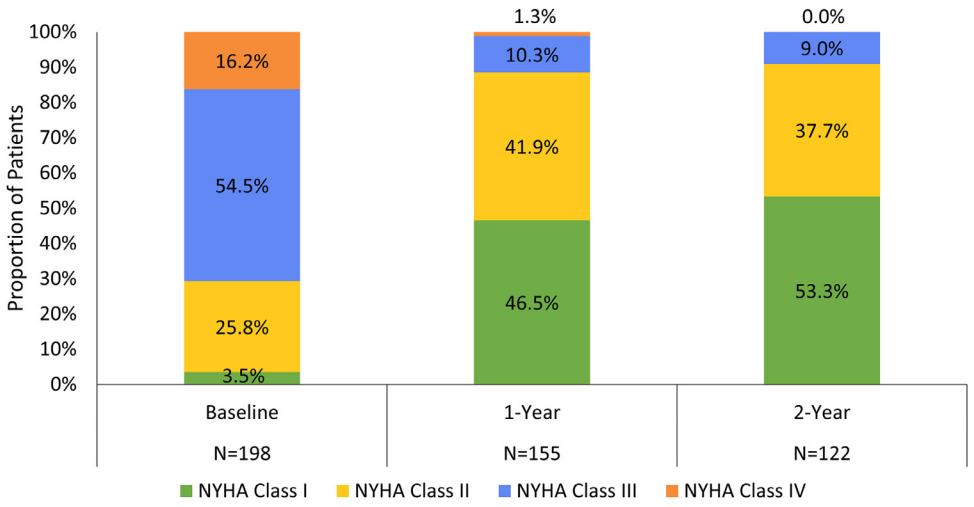

Figure 3. New York Heart Association (NYHA) classification at baseline, 1 year, and 2 years.

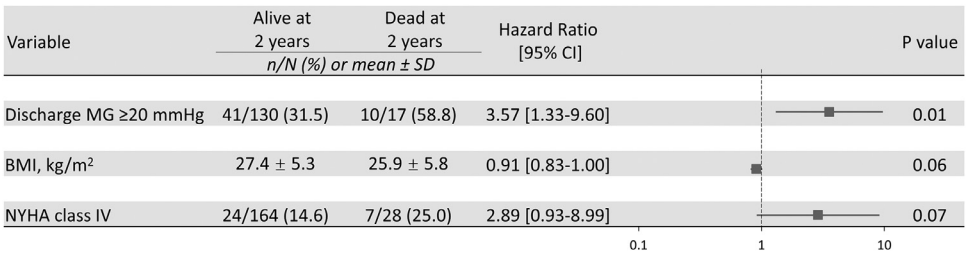

Figure 4. Multivariable predictors of 2-year mortality. Discharge mean gradient (MG) ≥20 mmHg, low body mass index (BMI), and New York Heart Association (NYHA) class IV are significant multivariable predictors of 2-year mortality. Data for discharge mean gradient were landmarked at day 14. A full list of variables included in the univariable model are provided in Supplemental Table 1.

noting that of the 32 patients with all-cause mortality events, 22 of them had a surgical valve fail due to stenosis. Similar findings were reported in the 1-year paper for the VIVA study, where mortality was numerically higher in patients with SAV failure mode of stenosis.[9] Tuzcu et al found that mean gradients in patients with stenotic original bioprostheses were higher after a ViV TAVI compared with patients with regurgitation or combined SAV failure mode.[15] Considering our findings of higher mortality in patients with higher mean gradient post-procedurally, this could explain our findings of higher cardiovascular mortality according to the SAV failure mode.[16]

In this study patients with discharge mean gradients ≥20 mm Hg had a significantly higher rate of all-cause mortality at 2 years, which was associated with female sex and smaller failed surgical valves. This emphasizes that the valve size used in patients for both surgical and replacement valve procedures impacts long-term outcomes in patients and confirms observations that the largest valve size possible should be chosen to optimize outcomes in patients with symptomatic severe aortic stenosis.[10] Contemporary practice often includes ring fracturing, which was not common practice at the time of the VIVA study and was not performed.

The NYHA class at discharge was shown to be a predictor of mortality among patients who underwent a ViV TAVI, where patients in NYHA class IV had a higher rate of mortality than those in a lower class. Patients who received a ViV TAVI had major improvements in NYHA class at 1 year,[9] and these improvements were maintained through 2 years, demonstrating the long-term benefits and functional improvement patients can receive from a ViV TAVI.[17]

The 2-year clinical outcomes of patients with a failing surgical aortic valve bioprosthesis demonstrate that ViV TAVI using the self-expanding CoreValve and Evolut R devices was safe and clinically effective. Nonetheless, the 2-year all-cause mortality and cardiovascular mortality were significantly higher in patients with a discharge mean gradient ≥20 mm Hg, primarily among those who received smaller implanted valves, calling for an effort to minimize the post-procedural valvular gradients. Thus, the catheter-based ViV TAVI procedure using the supra-annular self-expanding devices is a viable treatment option for patients suffering from degenerated bioprosthetic surgical valves.

There are several limitations to the VIVA study. At the time this study was conducted there were no standardized best practices to guide performance of the ViV procedures. Therefore, procedural details such as use of post-dilation and target implant depth were left to the treating physician. Additionally, implant depth was not collected in this study, which prevents analysis of implant depth-related outcomes. The experimental technique of bioprosthetic ring fracture, aimed to diminish post-procedural gradients, was not engaged in the VIVA protocol; thus, its impact on post-procedural valve

gradients or overall outcomes has not been assessed in the current study. Finally, echocardiograms were not collected at the 2-year follow-up, limiting any analysis of hemodynamic measurements out to 2 years.

Author's Contributions

Ran Kornowski: conceptualization, methodology, investigation, writing-original draft preparation, resources. Jean-Philippe Verhoye, Didier Tchétché, Bernard Chevalier, David Holzhey, Axel Harnath, Ulrich Schäfer, Emmanuel Teiger, Thibaut Manigold, Thomas Modine, Geraud Souteyrand, Didier Champagnac: methodology, investigation, writing-review and editing, resources. Jae K. Oh: methodology, formal analysis, writing−review and editing. Shuzhen Li: formal analysis, validation, methodology writing-review and editing.

Disclosures and conflicts of interest

Dr. Kornowski is a clinical investigator and proctor for Medtronic. Dr. Tchétché is a consultant for Medtronic. Dr. Holzhey is a proctor and advisor for Medtronic; proctor for Edwards Lifesciences, and Boston Scientific. Dr. Harnath received payments for clinical study involvement and traveling compensation from, and is a proctor and consultant for, Medtronic. Dr. Schäfer is a proctor and speaker for, and has received travel support and grant support from, Medtronic. Dr. Teiger is a consultant and proctor for Medtronic. Dr. Modine is a consultant, proctor, and an advisory board member for Medtronic. Dr. Souteyrand is a proctor for Medtronic. Dr. Champagnac is a proctor for Abbott (Mitraclip). Dr. Oh is a consultant to Medtronic with grants paid to his institution. Shuzhen Li is an employee and shareholder of Medtronic. Dr. Chevalier is a proctor for Medtronic. Dr. Verhoye and Dr. Manigold have nothing to declare.

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Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.amjcard.2020.12.047.

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