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ORIGINAL RESEARCH

STRUCTURAL

# Impact of Valve Frame Height on PCI Outcomes After TAVR

Carlo A. Pivato, MD, PhD,[a,b] Emanuele Di Angelantonio, MD, PhD,[c,d,e,f,g] Francesco Tartaglia, MD,[a,b] Gianluigi Condorelli, MD, PhD,[a,b] Nicole Fontana, MS,[c,h] Francesca Ieva, PhD,[c,h] Cosmo Godino, MD,[i] Masaaki Nakase, MD,[j] Karsten Hug, MD,[k] Tobias Rheude, MD,[k] Antonio J. Munoz-Garcia, MD, PhD,[l] Victor Alfonso Jimenez Diaz, MD,[m] Alfonso Ielasi, MD,[n] Marco Barbanti, MD,[o] Giuliano Costa, MD, PhD,[p] Angelo Anzuini, MD,[q] Giorgio Quadri, MD,[r] Diego Lopez-Otero, MD,[s] Philippe Garot, MD,[t] Ferdinando Varbella, MD,[u] Jorn Brouwer, MD, PhD,[v] Leon Gramss, MD,[a,b] Davide Cao, MD,[a,w] Stefano Figliozzi, MD,[a,b] Damiano Regazzoli, MD,[b] Luca Testa, MD,[x] Jorge Sanz Sanchez, MD,[y,z] Daijiro Tomii, MD,[j] Alaide Chieffo, MD,[aa,bb] Michael Joner, MD,[k,cc] Gennaro Sardella, MD,[dd] Enrico Cerrato, MD,[u,ee] Luis Nombela-Franco, MD,[ff] Thomas Pilgrim, MD,[j] Giulio Stefanini, MD, PhD[a,b]

## ABSTRACT

**BACKGROUND** Coronary access after transcatheter aortic valve replacement (TAVR) remains challenging, particularly with tall-framed valves (TFVs), raising concerns about long-term percutaneous coronary intervention (PCI).

**OBJECTIVES** The aim of this study was to evaluate the impact of bioprosthetic aortic valve type on long-term clinical outcomes in patients undergoing PCI following TAVR.

**METHODS** Data were derived from the multicenter REVIVAL-PCI registry, which included patients from 21 European centers who underwent PCI after TAVR between 2008 and 2023. Patients were classified according to valve frame height: TFVs or short-framed valves (SFVs). The primary endpoint was the 4-year incidence of major adverse cardiovascular events, defined as the composite of cardiovascular death, myocardial infarction, or stroke. Cumulative event rates were estimated using Kaplan-Meier method, and weighted Cox regression models using an entropy balance approach were used to adjust for imbalances in clinical and procedural confounders.

**RESULTS** The analysis included 441 patients, with 230 having undergone TAVR with SFVs (30.9% women) and 211 with TFVs (44.1% women). The median follow-up after PCI was 908 days (Q1-Q3: 322-1,728 days). The 4-year incidence of major adverse cardiovascular events was comparable between the SFV and TFV groups (38.1% [95% CI: 24.6%-43.9%] vs 31.9% [95% CI: 24.8%-41.0%]; HR: 1.04; 95% CI: 0.71-1.52; $P = 0.846$). Similar findings were observed after adjustment for potential confounders.

**CONCLUSIONS** In current practice, long-term outcomes after PCI in TAVR patients do not appear to be significantly different between those receiving SFVs and TFVs. Future investigations with newer generation valves and refined implantation techniques are needed to clarify these associations and optimize management strategies. (JACC Cardiovasc Interv. 2026;19:345–355) © 2026 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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**ABBREVIATIONS AND ACRONYMS**

**AS** = aortic stenosis

**CAD** = coronary artery disease

**CV** = cardiovascular

**MACE** = major adverse cardiovascular event(s)

**MI** = myocardial infarction

**PCI** = percutaneous coronary intervention

**SFV** = short-framed valve

**SMD** = standardized mean difference

**TAVR** = transcatheter aortic valve replacement

**TFV** = tall-framed valve

**TVR** = target vessel revascularization

The development of coronary artery disease (CAD) and aortic stenosis (AS) shares common pathogenic mechanisms.[1] Consequently, these conditions frequently coexist, with CAD found in up to 75% of patients with AS and associated with worse clinical outcomes following aortic valve interventions.[2-4]

Transcatheter aortic valve replacement (TAVR) has become the standard of care for symptomatic severe AS in patients across a broad spectrum of surgical risk. However, as TAVR is increasingly performed in younger individuals with longer life expectancies, there is growing concern regarding the feasibility of subsequent coronary revascularization. Unplanned percutaneous coronary intervention (PCI) following TAVR remains relatively infrequent. However, its incidence is increasing as these patients live longer and remain at risk for progressive CAD.[2,5-7] This underscores the importance of strategic valve selection at the time of TAVR to optimize future coronary access and ensure long-term procedural success.[4,8]

Although SFVs are associated with more favorable coronary reaccess, PCI remains feasible with tall-framed valves (TFVs), albeit with potentially greater complexity.[9-11] A recent study by Zendjebil et al[12] highlighted this distinction, reporting a higher long-term risk for death or heart failure rehospitalization in patients with TFVs after coronary events. However, this finding, driven only by heart failure hospitalizations, was derived from an unadjusted analysis. This raises the possibility that the finding was due to confounding rather than a true effect of valve type. Furthermore, the impact of valve design on other pivotal endpoints such as myocardial infarction (MI) and stroke remains unknown.

Given that valve selection is influenced by multiple clinical and anatomical factors, a robustly adjusted analysis is essential. Although prior work has focused on procedural feasibility of PCI following TAVR,[6] data on long-term clinical outcomes remain scarce. This study from the multicenter REVIVAL-PCI registry addresses these critical evidence gaps.

We hypothesized that valve frame height, by influencing coronary access, may affect long-term cardiovascular (CV) outcomes after PCI in TAVR patients. Therefore, we provide the first comprehensive, adjusted comparison of a broad range of CV outcomes in patients undergoing PCI after TAVR with either short-framed valves (SFVs) or TFVs.

## METHODS

**STUDY POPULATION.** The REVIVAL-PCI registry included consecutive patients undergoing PCI after TAVR for significant CAD from 2008 to 2023 at

From the [a]Department of Biomedical Sciences, Humanitas University, Pieve Emanuele, Milan, Italy; [b]IRCCS Humanitas Research Hospital, Rozzano, Milan, Italy; [c]HDS, Health Data Science Centre, Human Technopole, Milan, Italy; [d]British Heart Foundation Cardiovascular Epidemiology Unit, Department of Public Health and Primary Care, University of Cambridge, Cambridge, United Kingdom; [e]Victor Phillip Dahdaleh Heart and Lung Research Institute, University of Cambridge, Cambridge, United Kingdom; [f]British Heart Foundation Centre of Research Excellence, University of Cambridge, Cambridge, United Kingdom; [g]National Institute for Health and Care Research Blood and Transplant Research Unit in Donor Health and Behaviour, University of Cambridge, Cambridge, United Kingdom; [h]MOX, Department of Mathematics, Politecnico di Milano, Italy, Milan; [i]Cardiology Unit, Heart Valve Center, IRCCS San Raffaele Scientific Institute and Vita-Salute University, Milan, Italy; [j]Department of Cardiology, Inselspital, University of Bern, Bern, Switzerland; [k]Department of Cardiology, TUM University Hospital German Heart Center, Munich, Germany; [l]Department of Cardiology, University Hospital Virgen de la Victoria, Malaga, Spain; [m]Department of Cardiology, Hospital Álvaro Cunqueiro, University Hospital of Vigo, Vigo, Spain; [n]Cardiology Division, Istituto di Ricovero e Cura a Carattere Scientifico Ospedale Galeazzi Sant'Ambrogio, Milan, Italy; [o]Università degli Studi di Enna "Kore," Enna, Italy; [p]A.O.U. Policlinico "G. Rodolico - San Marco," Catania, Italy; [q]Ospedale Humanitas Mater Domini, Castellanza, Italy; [r]Interventional Cardiology, Mauriziano Hospital, Turin, Italy; [s]Cardiology Department, Complexo Hospitalario Universitario De Pontevedra, Pontevedra, Spain; [t]Institut Cardiovasculaire Paris Sud, Hôpital Jacques Cartier, Ramsay-Santé, Massy, France; [u]Interventional Cardiology Unit, San Luigi Gonzaga University Hospital, Orbassano, Italy; [v]Department of Cardiology, St. Antonius Hospital, Nieuwegein, the Netherlands; [w]Department of Cardiology, Humanitas Gavazzeni, Bergamo, Italy; [x]Department of Cardiology, Istituto di Ricovero e Cura a Carattere Scientifico Policlinico San Donato, Milan, Italy; [y]Hospital Universitari i Politecnic La Fe, Valencia, Spain; [z]Centro de Investigación Biomedica en Red, Madrid, Spain; [aa]Vita Salute San Raffaele University, Milan, Italy; [bb]Interventional Cardiology Unit Istituto di Ricovero e Cura a Carattere Scientifico San Raffaele Hospital, Milan, Italy; [cc]German Center for Cardiovascular Research, Partner Site Munich Heart Alliance, Munich, Germany; [dd]Policlinico Umberto I University, Rome, Italy; [ee]Rivoli Infermi Hospital, Rivoli (Turin), Italy; and the [ff]Cardiovascular Institute, Hospital Clinico San Carlos, IdISSC, Madrid, Spain.

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21 centers across Europe (Supplemental Table 1). The study was conducted following the ethical principles of the Declaration of Helsinki and was approved by the Institutional Review Board of each center.

Detailed information on baseline characteristics, PCI and TAVR procedures, antithrombotic regimen after PCI, and clinical outcomes was retrospectively collected by each participating center in a preformatted extraction sheet. For the present analysis, patients were classified according to the implanted valve type according to frame height: SFV vs TFV.

Both TAVR and PCI procedures were performed according to standard techniques, with device choice based on operator preference. Antithrombotic therapy and duration were left to the discretion of the treating physician. Clinical follow-up was conducted via either inpatient or outpatient visits or telephone interviews. All adverse events were site reported.

**ENDPOINTS.** The primary outcome of interest of this study was major adverse CV events (MACE), defined as the composite of CV death, MI, or stroke at 4-year follow-up. Secondary outcomes included the single components of the primary outcome, CV death, target vessel MI, periprocedural MI, target lesion revascularization, target vessel revascularization (TVR), target vessel failure (defined as a composite of TVR, MI, and CV death), definite or probable stent thrombosis, and CV hospitalization. Outcome definitions can be found in Supplemental Table 2.

**STATISTICAL ANALYSIS.** Continuous variables are reported as mean ± SD or median (Q1-Q3), while categorical variables are expressed as absolute numbers and percentages. Patients undergoing PCI with prior SFVs were compared with those with prior TFVs. Covariate balance between the 2 groups was assessed using the standardized mean difference (SMD), with an SMD $<0.1$ indicating good balance.

To control for confounding variables, we used entropy balancing, a powerful reweighting method designed to achieve superior covariate balance compared with traditional propensity score methods.[13] The selection of covariates for adjustment was prespecified on the basis of clinical expertise and a directed acyclic graph model to identify potential confounders while excluding known mediators (eg, procedural success) that lie on the causal pathway between valve type and outcome. We chose entropy balancing more than alternatives like inverse probability of treatment weighting because of its methodological advantages in this setting. Whereas inverse probability of treatment weighting relies on a predictive model for treatment assignment that may still leave residual imbalances, entropy balancing is an optimization algorithm that computed weights to make the covariate distributions nearly identical across the 2 valve groups. This is achieved by matching not just their means, but also their variance and skewness. This approach is particularly effective in studies with modest sample sizes and significant baseline imbalances, as seen in our cohort. Final weights were truncated at the 1st and 99th percentiles, a standard technique to improve the stability and precision of the estimates by preventing subjects with extreme weights from having undue influence.[14]

To assess the impact of both the baseline clinical profile and the PCI procedure itself, we computed 2 distinct weighted models, as outlined in Supplemental Figure 1. Our primary analysis (model 1) adjusted for a comprehensive set of clinical, demographic, and TAVR-related confounders that were present before the index PCI. For sensitivity analysis, we then included all variables from model 1 and additionally adjusted for a detailed set of procedural characteristics from index PCI (model 2). A complete list of the specific covariates included in each of these 2 adjustment models is provided in Supplemental Table 3. The pre- and postweighting balance diagnostics for these models are detailed in Supplemental Figures 2 and 3. The number of missing values for each variable in each model is reported in Supplemental Table 4.

We estimated 95% CIs using a robust variance estimator to account for potential correlations introduced by weighting. The association between valve type and outcomes was estimated using weighted Cox regression models. The Kaplan-Meier method was used to estimate both crude and weighted cumulative incidences, which were compared between groups using the log-rank test for the time-to-first event. The proportional hazards assumption was evaluated for the weighted Cox models using Schoenfeld residuals and corresponding global tests. No relevant violations of the assumption were found.

To explore for potential treatment effect modification, we performed formal tests for interaction within our primary adjusted Cox regression model (model 1). Subgroup analyses were conducted for the primary endpoint on the basis of clinical presentation (acute vs chronic coronary syndrome), sex, and median age.

To ensure the robustness of our findings with respect to country-level differences, we conducted a dedicated sensitivity analysis (Supplemental Table 5). To reduce statistical instability, we applied a 2-step filtering process: 1) exclusion of small centers contributing fewer than 10 patients; and 2)

**TABLE 1 Baseline Clinical Characteristics of the Population With No Adjustment and After Adjustment With Model 1**

| | Unadjusted | | | Adjusted | | |
|---|---|---|---|---|---|---|
| | SFV (n = 230) | TFV (n = 211) | SMD | SFV (n = 191.8) | TFV (n = 182.0) | SMD |
| Clinical characteristics | | | | | | |
| Age, y | 80.7 ± 5.8 | 81.1 ± 6.4 | 0.065 | 80.9 ± 5.4 | 80.9 ± 6.3 | 0.001 |
| Women | 71 (30.9) | 93 (44.1) | 0.275 | 73.0 (38.1) | 70.8 (38.9) | 0.017 |
| Logistic EuroSCORE | 12.6 ± 11.7 | 14.7 ± 11.6 | 0.177 | 14.1 ± 13.8 | 13.39 ± 11.23 | 0.058 |
| EuroSCORE II | 5.2 ± 6.7 | 5.8 ± 5.8 | 0.095 | 6.2 ± 8.3 | 5.50 ± 5.39 | 0.102 |
| BMI, kg/m$^2$ | 26.7 ± 4.7 | 27.5 ± 4.9 | 0.170 | 27.30 ± 4.8 | 27.22 ± 4.45 | 0.017 |
| Hypertension | 202 (87.8) | 193 (91.5) | 0.120 | 172.6 (90.0) | 163.7 (89.9) | 0.002 |
| Diabetes | 70 (30.4) | 70 (33.2) | 0.059 | 57.3 (29.9) | 56.3 (31.0) | 0.023 |
| Dyslipidemia | 162 (70.4) | 154 (73.0) | 0.057 | 138.4 (72.2) | 132.4 (72.8) | 0.013 |
| Previous PCI | 85 (37.0) | 105 (49.8) | 0.261 | 80.0 (41.7) | 75.6 (41.5) | 0.004 |
| Previous CABG | 25 (10.9) | 39 (18.5) | 0.216 | 28.0 (14.6) | 26.0 (14.3) | 0.009 |
| Previous PCI or CABG | 95 (41.3) | 118 (55.9) | 0.296 | 90.6 (47.2) | 82.5 (45.3) | 0.038 |
| Peripheral arterial disease | 42 (18.3) | 42 (19.9) | 0.040 | 35.7 (18.6) | 34.7 (19.0) | 0.011 |
| eGFR, mL/min/1.73 m$^2$ | 63.5 ± 22.3 | 60.2 ± 20.8 | 0.152 | 62.0 ± 22.5 | 62.1 ± 20.9 | 0.005 |
| LVEF,% | 53.6 ± 12.7 | 55.6 ± 11.0 | 0.165 | 54.8 ± 11.6 | 54.9 ± 11.0 | 0.014 |
| TAVR characteristics | | | | | | |
| Year of TAVR procedure | | | 0.263 | | | 0.022 |
| 2008-2012 | 33 (14.3) | 33 (15.7) | | 25.9 (13.5) | 24.1 (13.2) | |
| 2012-2017 | 102 (44.3) | 116 (55.2) | | 93.2 (48.6) | 90.5 (49.7) | |
| 2018-2023 | 95 (41.3) | 61 (29.0) | | 72.6 (37.9) | 67.4 (37.0) | |
| Transapical approach | 24 (10.4) | 12 (5.7) | 0.175 | 15.0 (7.8) | 14.0 (7.7) | 0.006 |
| Valve size, mm | 25.90 ± 2.11 | 27.19 ± 2.39 | 0.571 | 26.40 ± 2.00 | 26.39 ± 2.22 | 0.004 |
| Valve-in-valve | 2 (0.9) | 3 (1.4) | 0.052 | 1.0 (0.5) | 1.9 (1.0) | 0.056 |
| Postdilatation | 35 (15.2) | 90 (42.7) | 0.635 | 55.4 (28.9) | 55.4 (30.4) | 0.034 |

Values are mean ± SD or n (%). Variables included in the adjusted model are reported in Supplemental Table 3.

BMI = body mass index; CABG = coronary artery bypass grafting; eGFR = estimated glomerular filtration rate; EuroSCORE = European System for Cardiac Operative Risk Evaluation; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention; SFV = short-framed valve; SMD = standardized mean difference; TAVR = transcatheter aortic valve replacement; TFV = tall-framed valve.

exclusion of countries where, after this step, one valve type was no longer represented. In the resulting restricted cohort, we re-estimated the weighted Cox regression using the same covariates as in model 1, while additionally including country as a balancing variable in the entropy weighting procedure.

Moreover, to assess the potential for overestimation of risk by the Kaplan-Meier method, we performed 2 sensitivity analyses on model 1. First, we evaluated the adjusted outcomes at 1 year. Second, as an alternative to the primary Cox model, we used an adjusted Fine-Gray subdistribution hazard model to re-estimate the subdistribution HRs while accounting for competing risks. To account for the nature of the different endpoints, the competing risks were defined as follows: for the composite endpoint of MACE and the individual endpoint of CV death, non-CV death was treated as the competing risk; for all other nonfatal endpoints, all-cause death was treated as the competing risk.

*P* values <0.05 were considered to indicate statistical significance. All analyses were performed using R version 4.3.1 (R Foundation for Statistical Computing).

## RESULTS

**BASELINE CLINICAL AND PROCEDURAL CHARACTERISTICS.** Of the 464 patients in the REVIVAL-PCI registry, 441 patients were included in this analysis after excluding 23 recipients of mechanically expandable or transapical valves. A total of 230 patients received SFVs and 211 received TFVs across 21 centers. SFVs were almost entirely balloon expandable SAPIEN valves (226 [98.3%]; Edwards Lifesciences). Conversely, all TFVs were self-expanding, predominantly CoreValve or Evolut (141 [66.8%]; Medtronic), Symetis or ACURATE (54 [25.6%]; Boston Scientific), and Portico or Navitor devices (13 [6.2%]; Abbott Laboratories).

Tables 1 and 2 report clinical and procedural characteristics according to valve type before weighting and after weighting for clinical confounders.

Before weighting, SFV patients were less likely to be female (30.9% vs 44.1%, SMD = 0.275) and had lower rates of hypertension (87.8% vs 91.5%, SMD = 0.120) and prior PCI (37.0% vs 49.8%, SMD = 0.261) or CABG (10.9% vs 18.5%, SMD = 0.216) compared with TFV patients. Age was

**TABLE 2 PCI Characteristics of the Population With No Adjustment and After Adjustment With Model 1**

| | Unadjusted | | | Adjusted | | |
|---|---|---|---|---|---|---|
| | SFV (n = 230) | TFV (n = 211) | SMD | SFV (n = 191.8) | TFV (n = 182.0) | SMD |
| PCI characteristics | | | | | | |
| Days from TAVR to PCI | 89.5 (40.0-613.5) | 143.0 (40.0-396.2) | 0.088 | 87.6 (41.0-536.7) | 140.7 (39.1-402.6) | 0.011 |
| PCI planned at time of TAVR | 59 (25.7) | 57 (27.0) | 0.031 | 51.6 (26.9) | 50.1 (27.5) | 0.014 |
| Indication for PCI | | | 0.397 | | | 0.006 |
| CCS | 164 (72.6) | 113 (54.1) | | 127.3 (66.4) | 120.9 (66.4) | |
| Unstable angina | 13 (5.8) | 21 (10.0) | | 16.8 (8.8) | 15.9 (8.7) | |
| NSTEMI | 38 (16.8) | 54 (25.8) | | 36.3 (18.9) | 34.7 (19.0) | |
| STEMI | 9 (4.0) | 17 (8.1) | | 8.8 (4.6) | 8.2 (4.5) | |
| AHF or cardiac arrest | 2 (0.9) | 4 (1.9) | | 2.6 (1.4) | 2.4 (1.3) | |
| Radial access | 114 (52.3) | 84 (42.0) | 0.207 | 84.3 (45.7) | 69.6 (39.2) | 0.132 |
| Number of diseased vessels | 1.0 (1.0-2.0) | 1.0 (1.0-1.0) | 0.176 | 1.0 (1.0-1.0) | 1.0 (1.0-1.0) | 0.036 |
| ACC/AHA type 2B or C | 172 (90.1) | 130 (86.1) | 0.122 | 155.1 (89.8) | 121.5 (89.5) | 0.010 |
| Severe calcifications | 58 (25.2) | 51 (24.2) | 0.024 | 49.7 (25.9) | 44.0 (24.2) | 0.040 |
| CTO | 8 (3.5) | 3 (1.4) | 0.133 | 9.6 (5.0) | 3.3 (1.8) | 0.176 |
| Evidence of thrombus | 8 (3.7) | 9 (4.7) | 0.052 | 9.3 (5.0) | 7.7 (4.6) | 0.016 |
| Intravascular imaging | 14 (6.1) | 18 (8.5) | 0.094 | 6.6 (3.4) | 14.1 (7.7) | 0.189 |
| Bifurcation | 66 (28.7) | 43 (20.4) | 0.194 | 54.3 (28.3) | 43.0 (23.6) | 0.107 |
| ISR | 26 (11.3) | 30 (14.2) | 0.087 | 21.1 (11.0) | 26.9 (14.8) | 0.113 |
| Thrombus aspiration | 1 (0.5) | 1 (0.5) | 0.007 | 0.4 (0.2) | 1.0 (0.6) | 0.057 |
| Use of plaque modification device | 14 (6.6) | 5 (2.7) | 0.189 | 10.7 (5.9) | 2.4 (1.5) | 0.234 |
| Predilatation | 183 (86.7) | 154 (82.8) | 0.110 | 159.9 (87.1) | 143.3 (86.7) | 0.012 |
| PCI on LM | 33 (15.1) | 37 (18.6) | 0.094 | 25.1 (13.5) | 27.2 (15.5) | 0.058 |
| PCI on LAD | 105 (47.9) | 83 (41.7) | 0.126 | 87.0 (46.6) | 88.0 (50.1) | 0.070 |
| PCI with DES | 202 (93.5) | 180 (92.3) | 0.047 | 173.4 (93.7) | 156.1 (90.3) | 0.126 |
| Number of lesions per patients | 1.0 (1.0-2.0) | 1.0 (1.0-2.0) | 0.128 | 1.0 (1.0-2.0) | 1.0 (1.0-2.0) | 0.067 |
| Number of stents per lesion | 1.0 (1.0-2.0) | 1.0 (1.0-2.0) | 0.118 | 1.0 (1.0-2.0) | 1.0 (1.0-2.0) | 0.006 |
| Total stent length per lesion, mm | 40.4 ± 27.8 | 35.1 ± 24.4 | 0.203 | 38.5 ± 26.1 | 38.5 ± 26.6 | 0.001 |
| Minimum stent diameter, mm | 2.9 ± 0.5 | 3.0 ± 0.6 | 0.217 | 3.0 ± 0.6 | 3.0 ± 0.6 | 0.035 |
| Total number of stents per patient | 1.0 (1.0-2.0) | 1.0 (1.0-2.0) | 0.186 | 1.0 (1.0-2.0) | 1.0 (1.0-2.0) | 0.094 |
| Total stent length per patient, mm | 35.2 ± 29.6 | 27.9 ± 25.3 | 0.264 | 36.6 ± 29.9 | 30.7 ± 24.5 | 0.217 |
| Complex PCI | 76 (36.2) | 57 (30.0) | 0.132 | 67.0 (36.5) | 51.3 (31.2) | 0.113 |
| Successful PCI | 221 (98.2) | 197 (95.2) | 0.171 | 187.0 (97.6) | 171.2 (94.7) | 0.152 |
| Angiographic success | 215 (98.6) | 192 (99.0) | 0.032 | 185.0 (99.1) | 168.2 (99.0) | 0.013 |
| Complete revascularization | 158 (71.2) | 137 (67.8) | 0.073 | 130.6 (68.2) | 121.7 (67.4) | 0.018 |
| Coronary occlusion | 3 (1.3) | 1 (0.5) | 0.089 | 1.0 (0.5) | 0.7 (0.4) | 0.022 |
| Post-PCI antiplatelet therapy | | | | | | |
| OAC | 59 (26.7) | 47 (23.2) | 0.082 | 50.0 (26.1) | 46.7 (25.7) | 0.010 |
| Short DAPT (≤3 mo) | 59 (28.9) | 39 (20.3) | 0.201 | 48.3 (27.6) | 43.2 (25.7) | 0.044 |

Values are median (Q1-Q3), n (%), or mean ± SD. Variables included in the adjusted model are reported in Supplemental Table 3. CCS was defined as stable angina, silent ischemia, or left ventricular ejection fraction deterioration.

ACC = American College of Cardiology; AHA = American Heart Association; AHF = acute heart failure; CCS = chronic coronary syndrome; CTO = chronic total occlusion; DAPT = dual antiplatelet therapy; DES = drug-eluting stent(s); ISR = in-stent restenosis; LAD = left anterior descending coronary artery; LM = left main coronary artery; NSTEMI = non-ST-segment elevation myocardial infarction; OAC = oral anticoagulation; STEMI = ST-segment elevation myocardial infarction; other abbreviations as in Table 1.

similar in the 2 groups (81.1 ± 6.4 years vs 80.7 ± 5.8 years, SMD = 0.065), as well as the prevalence of diabetes (33.2% vs 30.4%, SMD = 0.059). In the SFV group, mean valve size was smaller (25.9 ± 2.1 mm vs 27.2 ± 2.4 mm, SMD = 0.571), and a transapical approach was more frequently used (10.4% vs 5.7%, SMD = 0.175) than in the TFV group.

PCI was performed 89.5 days (Q1-Q3: 40.0-613.5 days) after TAVR implantation in the SFV group and 143.0 days (Q1-Q3: 40.0-396.25 days) in the TFV group. Radial access was more commonly used in the SFV group than in the TFV group (52.3% vs 42.0%, SMD = 0.207). An acute coronary syndrome as index presentation was more common in the TFV cohort (43.9% vs 26.6% in the SFV cohort, SMD = 0.397). PCI was more frequently successful in the SFV group than in the TFV group (98.2% vs 95.2%, SMD = 0.171) despite the higher rate of complex lesions treated (36.2% vs 30.0%, SMD = 0.132).

**TABLE 3 4-Year Clinical Outcomes in the Unadjusted Population and After Adjustment With Model 1**

| | Unadjusted | | | | Adjusted for Clinical Variables | | | |
|---|---|---|---|---|---|---|---|---|
| | KM Incidence at 4 Years (95% CI) | | | | KM Incidence at 4 Years (95% CI) | | | |
| | SFV (n = 230) | TFV (n = 211) | HR (95% CI) | *P* Value | SFV (n = 191.8) | TFV (n = 182.0) | HR (95% CI) | *P* Value |
| MACE | 38.1 (24.6-43.9) | 31.9 (24.8-41.0) | 1.04 (0.71-1.52) | 0.846 | 40.4 (25.8-51.9) | 34.1 (22.6-44.0) | 1.13 (0.64-2.00) | 0.674 |
| Death | 39.3 (30.3-49.2) | 35.8 (28.8-45.4) | 1.09 (0.76-1.57) | 0.632 | 46.3 (31.5-57.9) | 38.5 (26.2-49.1) | 1.28 (0.78-2.01) | 0.336 |
| MI | 10.7 (1.9-11.8) | 13.7 (7.4-19.3) | 0.62 (0.32-1.20) | 0.156 | 6.1 (0.1-11.8) | 15.1 (6.2-23.3) | 0.43 (0.12-1.56) | 0.201 |
| Stroke | 11.4 (3.8-16.7) | 4.2 (1.1-8.5) | 2.03 (0.81-5.10) | 0.133 | 12.6 (2.9-21.5) | 6.1 (0.0-12.2) | 1.65 (0.41-6.75) | 0.482 |
| CV death | 26.5 (17.1-35.3) | 21.6 (15.8-30.8) | 1.22 (0.76-1.96) | 0.412 | 28.4 (15.5-39.4) | 19.5 (11.0-27.4) | 1.45 (0.76-2.78) | 0.258 |
| Stent thrombosis | 1.0 (0.0-2.7) | 2.1 (0.0-4.7) | 0.46 (0.08-2.50) | 0.366 | 2.8 (0.0-7.6) | 0.8 (0.0-1.6) | 3.62 (0.46-28.4) | 0.221 |
| Periprocedural MI | 7.7 (0.8-13.2) | 9.8 (4.1-16.8) | 0.80 (0.32-2.00) | 0.636 | 8.4 (0.4-15.8) | 11.2 (2.8-19.1) | 0.95 (0.27-3.28) | 0.936 |
| Target vessel MI | 4.8 (0.0-6.8) | 8.7 (3.9-14.5) | 0.55 (0.22-1.38) | 0.204 | 5.0 (0.0-10.6) | 8.5 (1.2-15.2) | 0.74 (0.16-3.39) | 0.699 |
| TLR | 8.0 (2.8-13.6) | 13.2 (8.2-20.6) | 0.56 (0.27-1.15) | 0.114 | 13.1 (3.9-21.4) | 13.9 (5.1-22.0) | 1.00 (0.39-2.55) | 0.997 |
| TVR | 12.0 (4.8-18.5) | 19.6 (14.1-28.9) | 0.54 (0.29-0.99) | 0.046 | 14.1 (4.9-22.5) | 18.7 (9.4-27.1) | 0.78 (0.33-1.84) | 0.577 |
| TVF | 35.1 (25.4-44.8) | 36.6 (31.0-48.0) | 0.90 (0.61-1.31) | 0.567 | 40.4 (26.2-52.0) | 34.8 (23.8-44.4) | 1.15 (0.67-1.95) | 0.607 |
| CV rehospitalization | 36.6 (22.0-42.0) | 31.9 (25.0-42.2) | 0.98 (0.66-1.47) | 0.931 | 37.0 (22.2-49.1) | 31.2 (19.6-41.5) | 1.05 (0.58-1.89) | 0.875 |

Variables included in the adjusted model are reported in Supplemental Table 3.

CV = cardiovascular; KM = Kaplan-Meier; MACE = major adverse cardiovascular event(s); MI = myocardial infarction; TLR = target lesion revascularization; TVF = target vessel failure; TVR = target vessel revascularization; other abbreviations as in Table 1.

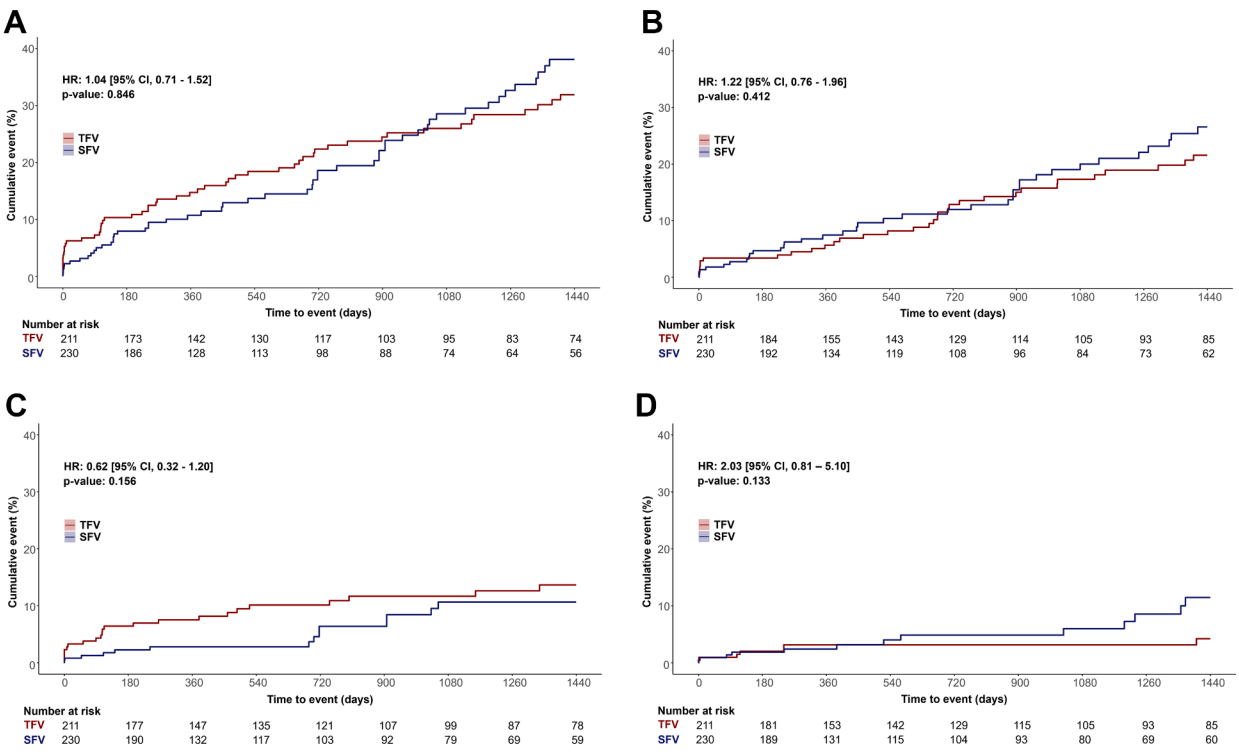
After adjustment for a comprehensive set of clinical, demographic, and TAVR-related confounders (model 1), balance was achieved for all included variables (Table 1, Supplemental Figure 2), though some procedural differences remained (Table 2).

**OUTCOMES BEFORE WEIGHTING.** The median follow-up duration after PCI was 908 days (Q1-Q3: 322-1,728 days), and 274 patients (62.1%) completed 4-year follow-up. Table 3 reports clinical outcomes according to valve type in the crude population and after weighting for clinical confounders. The 4-year crude incidence of MACE was 38.1% (95% CI: 24.6%-43.9%) in the SFV group vs 31.9% (95% CI: 24.8%-41.0%) in the TFV group (HR: 1.04; 95% CI: 0.71-1.52; $P = 0.846$) (Figure 1A). Similarly, we did not find significant differences in terms of all-cause mortality (39.3% [95% CI: 30.3%-49.2%] in the SFV group vs 35.8% [95% CI: 28.8%-45.4%] in the TFV group; HR: 1.09; 95% CI: 0.76-1.57; $P = 0.632$), CV death (26.5% [95% CI: 17.1%-35.3%] vs 21.6% [95% CI: 15.8%-30.8%]; HR: 1.22; 95% CI: 0.76-1.96; $P = 0.412$) (Figure 1B), MI (10.7% [95% CI: 1.9%-11.8%] vs 13.7% [95% CI: 7.4%-19.3%]; HR: 0.62; 95% CI: 0.32-1.20; $P = 0.156$) (Figure 1C), stroke (11.4% [95% CI: 3.8%-16.8%] vs 4.2% [95% CI: 1.1%-8.5%]; HR: 2.03; 95% CI: 0.81-5.10; $P = 0.133$) (Figure 1D), or any other secondary endpoint, except for TVR, whose incidence was lower in the SFV group than in the TFV group (12.0% [95% CI: 4.8%-18.5%] vs 19.6% [95% CI: 14.1%-28.9%]; HR: 0.54; 95% CI: 0.29-0.99; $P = 0.046$).

**OUTCOMES AFTER WEIGHTING (MODEL 1).** After weighting for clinical confounders, the 4-year adjusted incidence of MACE was 40.3% (95% CI: 25.8%-51.9%) in the SFV group vs 34.1% (95% CI: 22.6%-44.0%) in the TFV group (HR: 1.13; 95% CI: 0.64-2.00; $P = 0.674$) (Table 3, Figure 2A). Similarly, there were no statistically significant differences in the rates of all-cause mortality (46.3% [95% CI: 31.5%-57.9%] in the SFV group vs 38.5% [95% CI: 26.2%-49.1%] in the TFV group; HR: 1.28; 95% CI: 0.78-2.01; $P = 0.336$), CV death (28.4% [95% CI: 15.5%-39.4%] vs 19.5% [95% CI: 11.0%-27.4%]; HR: 1.45; 95% CI: 0.76-2.78; $P = 0.258$) (Figure 2B), MI (6.1% [95% CI: 0.1%-11.8%] vs 15.1% [95% CI: 6.2%-23.3%]; HR: 0.43; 95% CI: 0.12-1.56; $P = 0.201$) (Figure 2C), stroke (12.6% [95% CI: 2.9%-21.5%] vs 6.1% [95% CI: 0.0%-12.2%]; HR: 1.65; 95% CI: 0.41-6.75; $P = 0.482$) (Figure 2D), or any other secondary endpoint.

**SENSITIVITY ANALYSES.** The robustness of our primary findings from model 1 was confirmed across several sensitivity analyses. A country-level analysis, which excluded centers and countries with statistical instability, yielded an adjusted HR for MACE consistent with the main analysis (Supplemental Table 5). To address potential overestimation by the Kaplan-Meier method, both an analysis of adjusted outcomes at 1 year and a formal competing risk analysis confirmed the lack of significant differences between groups (Supplemental Tables 6 and 7). Formal tests for interaction showed that the effect of valve type

**FIGURE 1** Kaplan-Meier Estimates of 4-Year Clinical Outcomes in the Unadjusted Population




(A) Major adverse cardiovascular events. (B) Cardiovascular death. (C) Myocardial infarction. (D) Stroke. SFV = short-framed valve; TFV = tall-framed valve.

on 4-year MACE was consistent across prespecified subgroups of clinical presentation, sex, and age (*P* for interaction > 0.10 for all) (Supplemental Figure 4).

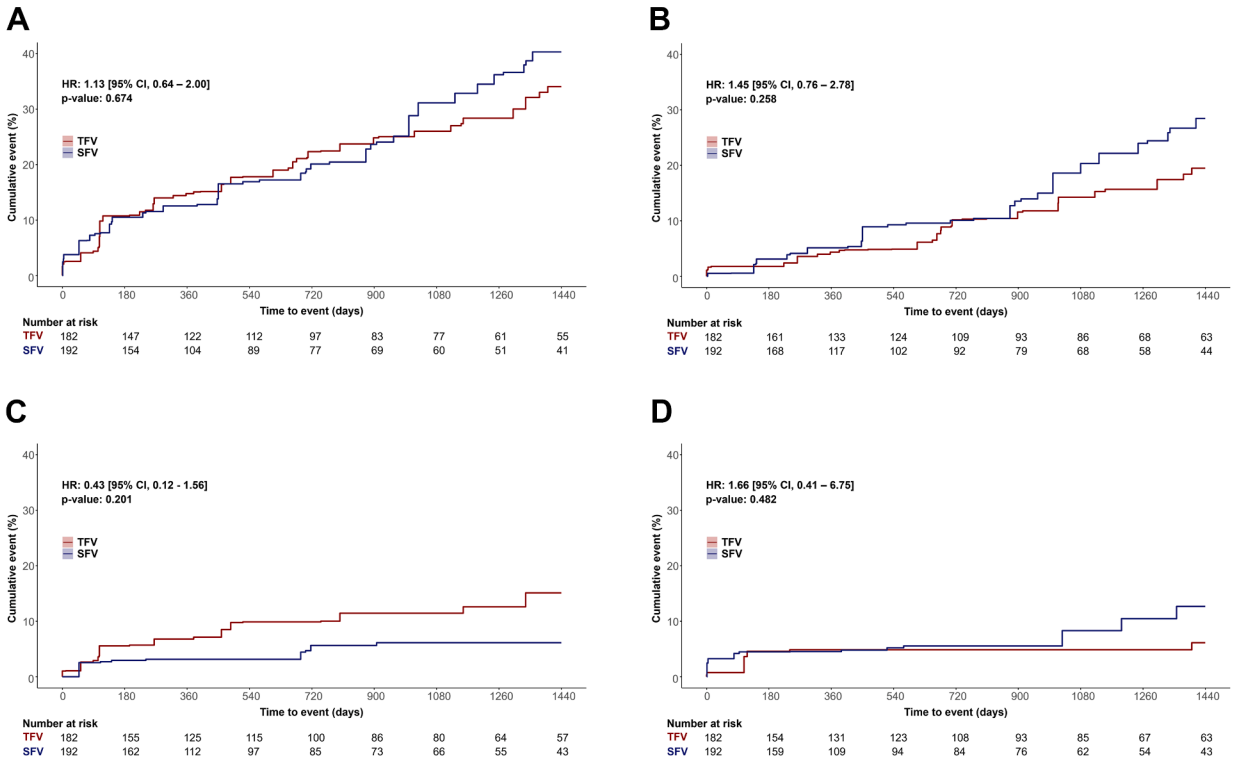
In the sensitivity analysis that additionally adjusted for procedural characteristics of the index PCI (model 2), all included baseline and procedural variables were well balanced between the 2 groups (Supplemental Tables 8 and 9, Supplemental Figure 3). Consistent with the findings from model 1, there were no significant differences in the rates of MACE or any other adverse events between the SFV and TFV groups (Supplemental Table 10, Supplemental Figure 5).

## DISCUSSION

This study represents the largest and most comprehensive analysis to date evaluating the impact of transcatheter heart valve frame height on long-term clinical outcomes in patients undergoing PCI after TAVR (**Central Illustration**). We leveraged a large, multicenter registry and applied robust statistical methods to account for confounding bias. Using this approach, we found no significant differences in 4-year MACE risk between the 2 cohorts. Rates of MI were higher in the TFV group, while strokes occurred more frequently in the SFV group. However, these differences did not reach statistical significance.

Valve selection in TAVR remains a matter of debate, particularly given its implications for future coronary access.[12] Suboptimal coronary engagement in patients with a TFV may increase procedural complexity and duration, necessitate greater contrast volume, and hinder equipment delivery.[6,9-12,15-18] This is reflected in our findings, as patients with TFVs demonstrated lower rates of drug-eluting stent implantation (90.3% vs 93.7%), overall PCI success (94.7% vs 97.6%), and radial access (39.2% vs 45.7%) compared with those with SFV, even after weighting for clinical confounders. These procedural hurdles may partially explain why prior studies revealed that patients with balloon-expandable SFVs were more likely to undergo PCI compared with self-expanding TFVs, despite similar rates of unplanned coronary angiography between the 2 groups.[12,15] Beyond procedural challenges, TFVs have also been associated with an elevated risk for delayed coronary obstruction, which may contribute to the numerically higher

**FIGURE 2** Kaplan-Meier Estimates of 4-Year Clinical Outcomes After Adjustment With Model 1




(A) Major adverse cardiovascular events. (B) Cardiovascular death. (C) Myocardial infarction. (D) Stroke. Variables included in the adjusted model are reported in Supplemental Table 3. Abbreviations as in **Figure 1**.

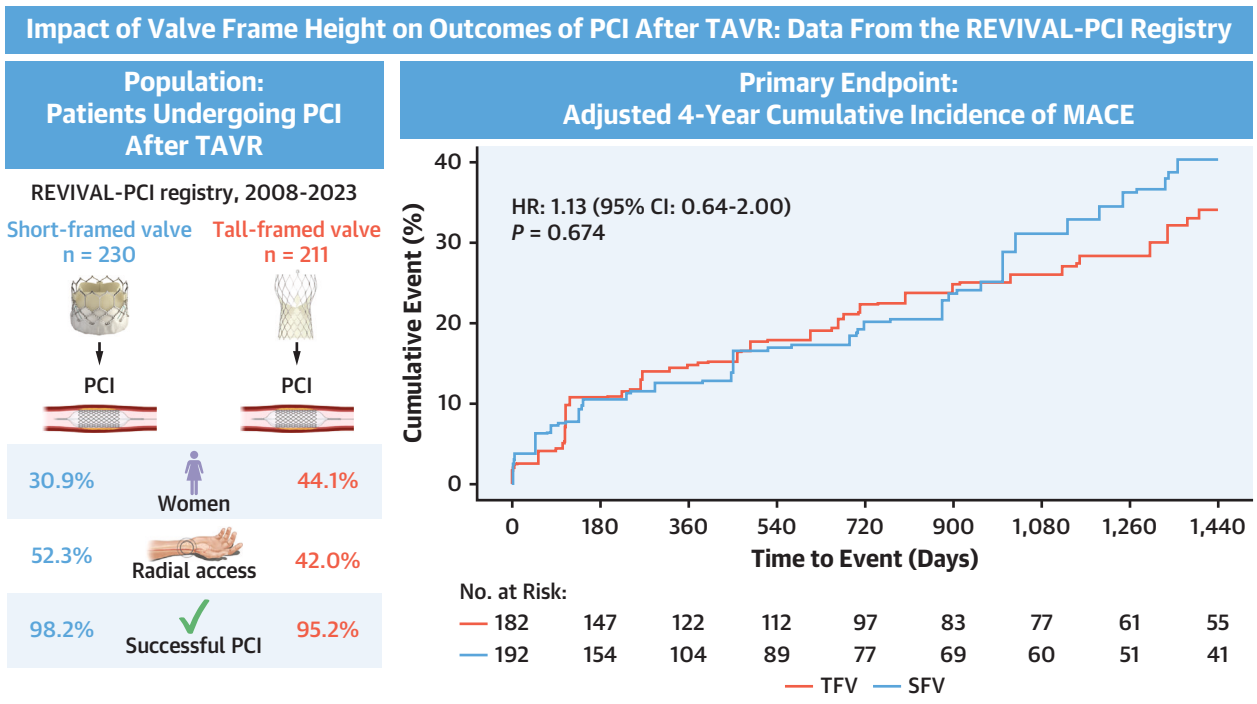
MI rates observed in this study.[19,20] This heightened risk is multifactorial, but the challenges in coronary reaccess with target vessel failure likely play a significant role.[21-23] Nevertheless, given the lack of statistical significance, these results should be regarded as hypothesis generating only.

Recent retrospective data have suggested that patients undergoing PCI after TAVR with balloon-expandable SFVs experience fewer rehospitalizations for heart failure compared with those with self-expanding TFVs, although no significant difference in all-cause death was detected.[12] Notably, that study also reported a numerically higher incidence of stroke in the SFV group compared with the TFV group (9.3% vs 8.4%), which aligns with our findings. However, the absence of adjustment for confounding variables in that analysis raises concerns about residual bias, underscoring the need for more rigorous evaluation of these associations.

A key strength of our registry is the availability of granular details, allowing a comprehensive adjustment for both baseline (ie, before PCI) and procedural (ie, at the time of PCI) confounding factors. Notably, the initially significant difference in unadjusted TVR rates (favoring SFVs with an HR of 0.54) became nonsignificant after adjustment for confounding variables. This change appears to be driven by 2 distinct mechanisms. First, the effect size was substantially attenuated after accounting for baseline clinical characteristics alone (HR increased to 0.78), indicating that much of the initial disparity was likely due to confounding by indication; that is, the TFV cohort presented with a higher intrinsic risk for repeat revascularization. Second, the adjustment process reduced statistical precision, widening the 95% CI (from 0.29-0.99 to 0.33-1.84). The effect was further nullified when procedural variables were included in model 2.

The issue of prosthesis-related coronary access limitations is expected to diminish with the development of new-generation TFVs featuring dedicated ostium-sparing designs. Additionally, advanced implantation techniques, including commissural and coronary alignment, leaflet modification strategies, and computed tomography-guided PCI, may further mitigate these challenges.[24-31]

**CENTRAL ILLUSTRATION** Study Population and Main Finding




**Pivato CA, et al. JACC Cardiovasc Interv. 2026;19(3):345-355.**

On the left, the study population of 441 patients undergoing percutaneous coronary intervention (PCI) after transcatheter aortic valve replacement (TAVR), stratified by valve frame height (short-framed valves [SFVs] vs tall-framed valves [TFVs]); on the right, the main result showing no significant difference in long-term major adverse cardiovascular event (MACE) risk between the 2 groups. HRs and Kaplan-Meier estimates are adjusted (model 1).

**STUDY LIMITATIONS.** To our knowledge, this is the first study to report a comprehensive set of clinical outcomes in patients undergoing PCI after TAVR according to valve frame height. Our analytical approach incorporated 2 separate adjustments: one accounting for baseline clinical confounders before PCI and another incorporating procedural characteristics of PCI itself, yielding consistent findings. Additionally, sensitivity analyses stratified by country demonstrated similar results, reducing the potential impact of regional practice variations on our conclusions.

Nevertheless, several limitations must be acknowledged. First, as an observational study, our ability to reduce potential bias from unmeasured confounders is inherently limited. Although a randomized trial is necessary to provide definitive evidence, conducting such a study would be highly challenging because of the relatively low incidence of PCI after TAVR.

Second, although participating centers were instructed to report all consecutive cases to minimize selection bias, the retrospective nature of the registry introduces the risk for information and reporting biases. However, the high procedural success rates observed in our cohort are highly consistent with those reported in large national and single center registries, which are less susceptible to selection bias. Reported success rates in these registries range from 92.5% to 99%, suggesting that a major reporting bias is unlikely in our study.[17,32]

Third, despite being the largest study to date, the relatively low incidence of PCI after TAVR limited our overall sample size.[6] This is directly reflected in the wide CIs observed for our adjusted HRs, indicating a lack of statistical precision. Consequently, our study had limited power to detect modest but potentially clinically relevant differences between the groups, and the risk for a type II error cannot be dismissed.

This limitation is particularly pronounced for our secondary endpoints, where the number of events was even smaller. Therefore, although our primary analysis revealed no statistically significant difference in MACE, our results should be interpreted with caution. They do not definitively rule out a benefit for one valve type over another but rather suggest that any difference, if one exists, is unlikely to be large.

Last, the prolonged data collection period, required by the low event rate, introduces potential temporal biases. However, adjustment for time-related factors mitigated concerns regarding evolving practice patterns.

## CONCLUSIONS

In this large, multicenter registry, we found that PCI following TAVR was highly feasible for recipients of both SFV and TFV, and critically, there were no significant differences in 4-year MACE risk. These findings provide reassurance that the potential PCI complexities associated with TFV do not translate into adverse long-term outcomes. Future investigations are warranted to determine if newer generation valves with access-preserving designs and refined implantation techniques can further improve outcomes for this growing patient population.

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**ADDRESS FOR CORRESPONDENCE:** Prof Giulio Stefanini, IRCCS Humanitas Research Hospital, Via Alessandro Manzoni, 56, 20089 Rozzano, Milan, Italy. E-mail: giulio.stefanini@hunimed.eu. X handle: @GGStefanini.

## PERSPECTIVES

**WHAT IS KNOWN?** As TAVR expands to patients with longer life expectancies, the incidence of PCI following TAVR is increasing. Preserving coronary reaccess is pivotal in the lifetime management of these patients, but data on the impact of valve design on clinical outcomes following PCI are lacking.

**WHAT IS NEW?** At 4-year follow-up, major clinical outcomes after PCI did not differ significantly between patients receiving SFVs and TFVs. This offers important reassurance on long-term outcomes in patients with TFVs, even though PCI in this setting may be more challenging. Accordingly, operators should anticipate procedural complexity and prioritize access-preserving techniques, such as commissural alignment, at the time of initial TAVR.

**WHAT IS NEXT?** Future studies should evaluate if newer generation TFVs with dedicated coronary access features can reduce procedural complexity. Furthermore, prospective data are needed to confirm if systematic strategies such as computed tomography–guided PCI planning and commissural alignment translates into improved procedural efficiency and long-term clinical outcomes for this growing patient population.

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**KEY WORDS** PCI, short-framed valve, tall-framed valve, TAVR

**APPENDIX** For supplemental tables and figures, please see the online version of this paper.