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ORIGINAL PAPER

Left and right ventricular longitudinal systolic function following aortic valve replacement in the PARTNER 2 trial and registry

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Aims

Evaluation of left and right ventricular (RV) longitudinal systolic function may enhance risk stratification following aortic valve replacement (AVR). The study objective was to evaluate the changes in left and RV longitudinal systolic function and RV–pulmonary artery (RV–PA) coupling from baseline to 30 days and 1 year after AVR.

Methods and results

Left ventricular (LV) longitudinal strain (LS), tricuspid annulus plane systolic excursion (TAPSE), and RV–PA coupling were evaluated in patients from the PARTNER 2A surgical AVR (SAVR) arm ($n = 985$) and from the PARTNER 2 SAPIEN 3 registry ($n = 719$). TAPSE and RV–PA coupling decreased significantly following SAVR, but remained stable following TAVR. Lower LV LS, TAPSE, or RV–PA coupling at baseline was associated with increased risk of the composite of death, hospitalization, and stroke at 5 years [adjusted hazard ratios (HRs) for LV LS < 15%: 1.24, 95% confidence interval (CI) 1.05–1.45, $P = 0.001$; TAPSE < 14 mm: 1.44, 95% CI 1.21–1.73, $P < 0.001$; RV–PA coupling < 0.55 mm/mmHg: 1.32, 95% CI 1.07–1.63, $P = 0.011$]. Reduced TAPSE at baseline was the most powerful predictor of the composite endpoint at 5 years. Patients with LV ejection fraction <50% at baseline had increased risk of the primary endpoint with SAVR (HR: 1.34, 95% CI 1.08–1.68, $P = 0.009$) but not with TAVR (HR: 1.12, 95% CI 0.88–1.42). Lower RV–PA coupling at 30 days showed the strongest association with cardiac mortality.

Conclusion

SAVR but not TAVR was associated with a marked deterioration in RV longitudinal systolic function and RV–PA coupling. Lower TAPSE and RV–PA coupling at 30 days were associated with inferior clinical outcomes at 5 years. In patients with LVEF < 50%, TAVR was associated with superior 5-year outcomes.

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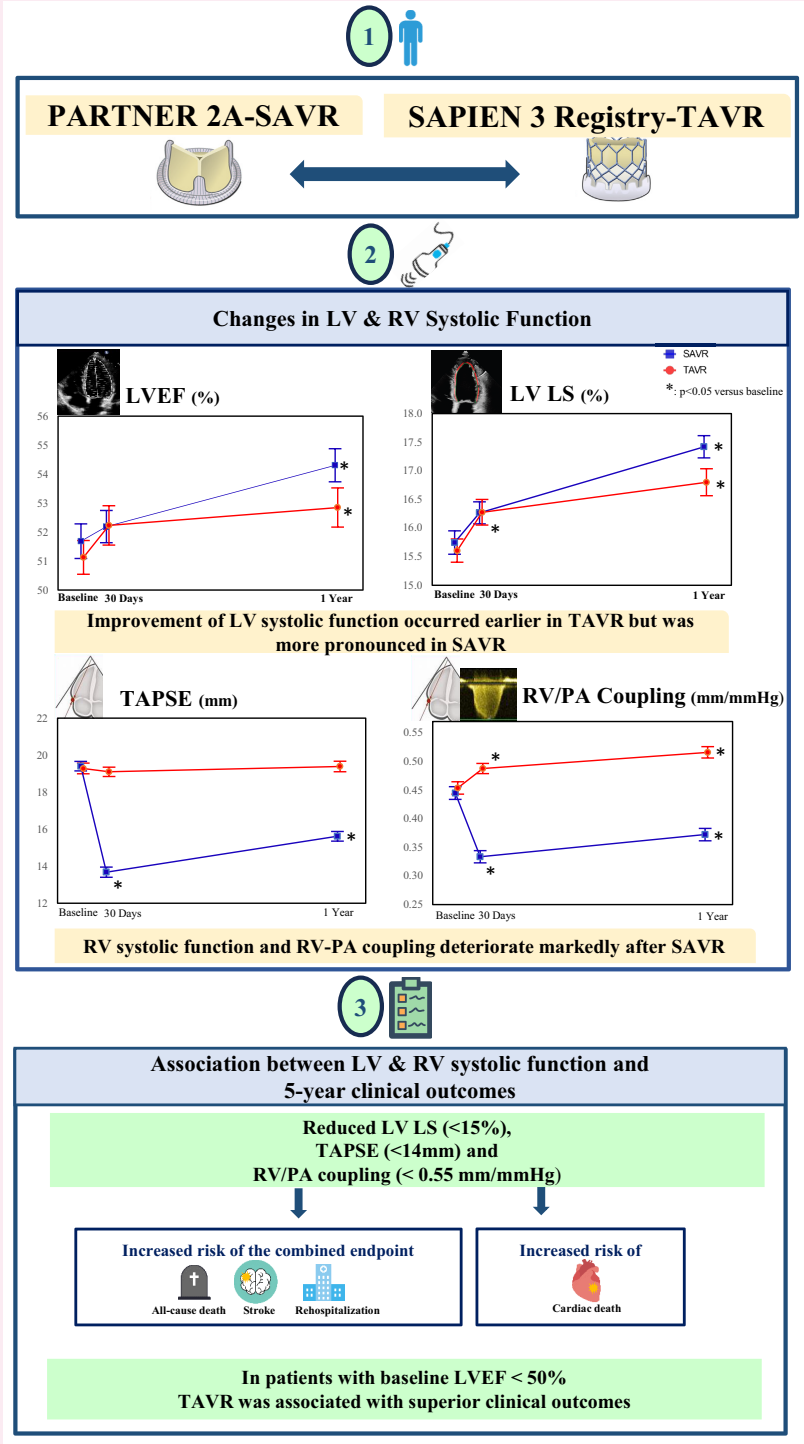
Graphical Abstract



Keywords aortic valve replacement • TAVR • SAVR • longitudinal systolic function • RV–PA coupling

Introduction

Current guidelines recommend aortic valve replacement (AVR) in patients with symptomatic severe aortic stenosis (AS) or asymptomatic severe AS with left ventricular (LV) systolic dysfunction defined by a LV ejection fraction (LVEF) < 50%.[1,2] However, LVEF underestimates the extent of myocardial systolic dysfunction in the presence of concentric hypertrophy, such as is often the case in patients with severe AS.[3] Hence, LVEF lacks sensitivity to identify subclinical LV systolic dysfunction prior to AVR and to demonstrate an improvement in LV systolic function following AVR. LV longitudinal strain (LS) has been shown to be superior to LVEF for early detection

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and quantification of intrinsic myocardial systolic dysfunction in patients with AS.[4–7] There are scarce data regarding the changes in LV LS after AVR and its association with clinical outcomes following surgical (SAVR) or transcatheter AVR (TAVR).

Right ventricular (RV) dysfunction occurs in up to 30% of patients with severe AS and is associated with worse prognosis following AVR.[8–10] In previous analyses of the PARTNER 2 and 3 trials, we reported that worsening of RV function assessed by a multiparameter integrative approach including visual assessment, RV fractional area change, and tricuspid annulus plane systolic excursion (TAPSE) was more common following SAVR vs. TAVR and was associated with worse prognosis.[11,12] In a recent analysis of PARTNER 3, we also reported that baseline RV–pulmonary artery (RV–PA) uncoupling, documented by a TAPSE/systolic pulmonary arterial pressure ratio < 0.55 mm/mmHg, was associated with adverse clinical outcomes at 2 years post AVR.[13] However, there is no large and echocardiography core lab–adjudicated analysis and comparison of the evolution and clinical impact of both LV and RV systolic function in patients undergoing TAVR or SAVR. Hence, the objectives of the present analysis of the PARTNER 2A trial and registry were (i) to determine and compare the changes in LV and RV longitudinal systolic function from baseline to 30 days and 1 year after SAVR or TAVR in patients with severe AS and intermediate surgical risk and (ii) to assess the association between parameters of LV and RV longitudinal systolic function and RV–PA coupling measured at baseline and 30 days with 5-year clinical outcomes.

Methods

Study design and population

The populations from the PARTNER (Placement of Aortic Transcatheter Valves, NCT01314313) 2A randomized trial[14,15] and the PARTNER 2 SAPIEN 3 intermediate-risk observational study (NCT0322128)[16] were considered in the present analysis (*Figure 1*). These two prospective, multicentre studies enrolled patients with symptomatic severe AS who were considered to be at intermediate surgical risk. In the PARTNER 2A trial, patients were randomly assigned to receive either SAVR or TAVR using the SAPIEN XT device (Edwards Lifesciences, Irvine, CA). In the SAPIEN 3 single-arm study, all patients underwent TAVR with the SAPIEN 3 valve. The PARTNER 2A trial and SAPIEN 3 registry had similar inclusion and exclusion criteria.[14,15] The institutional review boards of each participating site approved both studies, and written informed consent was provided by all patients. For the present study, we included the surgical cohort of the PARTNER 2A randomized trial ($n = 985$) and the PARTNER 2 SAPIEN 3 intermediate-risk registry ($n = 719$) with echocardiographic data available at baseline and 30 days and 1-year follow-up.

Doppler echocardiographic data

In both the PARTNER 2A trial and SAPIEN 3 registry, a transthoracic echocardiogram was performed at baseline prior to the procedure, at 30 days, and annually thereafter. All baseline and follow-up transthoracic echocardiograms were analysed by a Core Lab Consortium composed of four core labs: Cardiovascular Imaging C5 Research Corelab, Cleveland Clinic (Cleveland, OH), Québec Heart & Lung Institute, Laval University (Québec, Canada), Cardiovascular Research Foundation (New York City, NY), and MedStar Health Research Institute (Washington, DC). The processes for image analysis and quality assurance have previously been described.[17] Parameters of LV (i.e. LS) and RV (i.e. TAPSE) longitudinal systolic function were analysed with the TomTec Cardiac Performance Analysis platform by the same core lab (Québec Heart & Lung Institute).

LVEF was measured by the biplane Simpson method. LV LS was measured on the apical four-chamber view using the speckle-tracking method and expressed in absolute values. The RV free wall LS was performed, but the feasibility of this measurement was too low in our cohort (<50%), so these data were not reported in the present analysis. TAPSE was measured using the 2D method as previously described.[12] RV–PA coupling was assessed by calculating the ratio between TAPSE to the systolic PA pressure estimated by Doppler echocardiography from the peak tricuspid regurgitation systolic velocity.

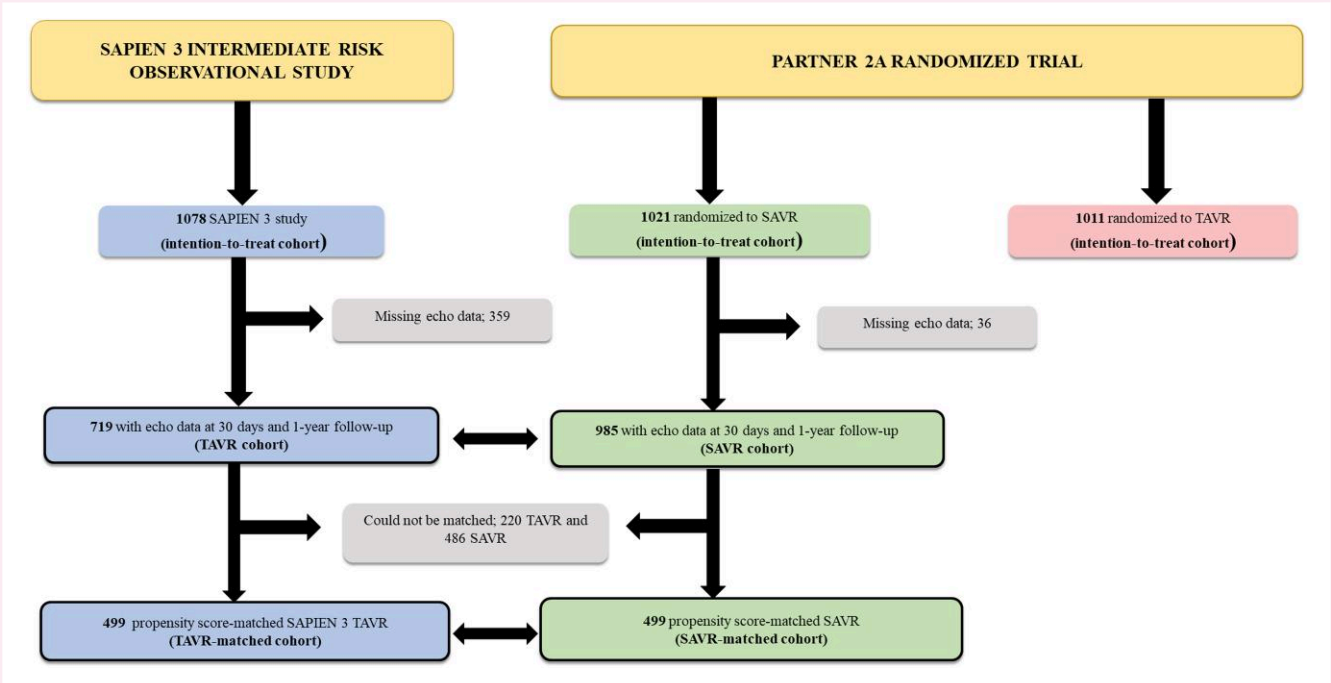

Figure 1 Study flowchart. SAVR, surgical aortic valve replacement; TAVR, transcatheter aortic valve replacement.

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Study endpoints

The echocardiographic endpoints were the changes in LVEF, LV LS, TAPSE, and RV–PA coupling from baseline to 30 days and 1 year post AVR. The primary clinical endpoint was the composite of all-cause death, hospitalization, and stroke at 5-year follow-up. The secondary endpoint was cardiac death at 5-year follow-up. Clinical outcomes were reported as defined by Valve Academic Research Consortium-2 definitions.[18]

Statistical analyses

Continuous variables are presented as mean ± SD and were compared using Student's *t*-test. Categorical variables are presented as proportions and were compared using the χ^2 or Fisher exact tests. Cox regression analyses were performed to compare 5-year rates for the primary and secondary endpoints according to LVEF < or ≥50%, LV LS < or ≥15%, TAPSE < or ≥14 mm, RV–PA coupling < or ≥0.55 mm/mmHg, and the treatment received (TAVR vs. SAVR). These cut-off values were selected according to previous studies that proposed and validated these values for risk stratification in patients with AS undergoing SAVR or TAVR.[4,13,19–21] Hazard ratios (HRs) with their 95% confidence intervals (CIs) were reported. Kaplan–Meier estimates and log-rank test were used to compare and graphically display outcomes between groups. Multivariable analysis was performed with the Cox proportional hazard model including the Society of Thoracic Surgeons (STS) score, baseline stroke volume index < 35 mL/m^2, mean aortic gradient < 20 mmHg, and ≥moderate mitral regurgitation for adjustment. For the analyses of the association of echocardiographic parameters of LV and RV longitudinal systolic function measured at 30 days vs. clinical outcomes, the analyses were landmarked at 30 days.

Moreover, a propensity score (PS) matching analysis was done to adjust for differences in baseline characteristics and potential confounders that may lead to biased estimates of the TAVR vs. SAVR comparison. A PS was calculated for each patient to estimate the propensity towards being included in a specific treatment group (TAVR vs. SAVR). This was done by logistic regression including 27 baseline covariates (see Supplementary data online, *Table S1*). Missing baseline data were imputed using the Markov chain Monte Carlo method prior to modelling. Based on their PSs, each TAVR patient was matched to a SAVR patient (1:1) to create two balanced cohorts, using a greedy matching strategy with calliper of width equal to 0.2 of the SD of the logit of the PS. All analyses were performed first in the whole cohorts and then in the PS-matched cohorts.

A subgroup analysis comparing patients with values above and below the thresholds established for LV and RV function with the four parameters evaluated (LVEF, LV LS, TAPSE, and RV–PA coupling) was performed, and *P*-values for interaction were calculated for the following covariates: age (≥75 years vs. <75 years), gender, baseline mean gradient (≥40 mmHg vs. <40 mmHg), and type of AVR (TAVR vs. SAVR). A $P < 0.05$ was considered significant for all statistical tests. All analyses were performed using SAS software, version 9.4 (SAS Institute, Cary, NC, USA).

Results

Overall, the study cohort was composed of 1704 patients including 985 patients from the SAVR arm of the PARTNER 2A randomized trial and 719 from the PARTNER 2 SAPIEN 3 intermediate-risk registry (*Figure 1*). Baseline clinical, echocardiographic, and procedural characteristics are displayed in Supplementary data online, *Table S2*. SAVR recipients were more frequently females (45.1% vs. 39.9%, $P = 0.03$), exhibited slightly higher STS score (5.8% vs. 5.3%, $P < 0.001$), and AS severity (aortic peak velocity 438.6 ± 57.2 cm/s vs. 428.5 ± 55.2 cm/s, $P < 0.001$) compared to TAVR recipients. They also had higher LVEF, LV LS, and lower RV–PA coupling ratio at baseline (see Supplementary data online, *Table S2* and *Figure S1*). After PS matching, a total of 499 pairs of patients were obtained, and the baseline characteristics of the matched cohorts are displayed in *Table 1*.

Changes in LV and RV function from baseline to 30 days and 1 year post AVR

In the PS-matched cohorts, LVEF improved from baseline to 1 year in both TAVR and SAVR groups. However, LV LS improved more rapidly in the TAVR group, resulting in significantly higher values at 30 days and 1-year follow-up compared to baseline, whereas the improvement in LV LS was only seen at 1-year follow-up in the SAVR group (*Figure 2A* and *B* and *Table 2*). LVEF and LV LS were not significantly different between TAVR vs. SAVR at 30 days. SAVR recipients showed significantly higher LV LS at 1-year follow-up compared to TAVR ones.

RV longitudinal systolic function as assessed by TAPSE remained stable from baseline to 30 days and 1 year after TAVR, whereas it decreased significantly after SAVR from baseline to 30 days and recovered, only in part, from 30 days to 1 year (*Figure 2C* and *D* and *Table 2*). RV–PA coupling improved from baseline to 30 days and 1 year after TAVR, whereas it decreased significantly after SAVR. TAPSE and RV–PA coupling ratio were significantly better in TAVR at 30 days and 1-year follow-up compared to SAVR. The results for the unmatched cohorts are shown in Supplementary data online, *Figure S1* and *Table S3*.

In the PS-matched cohorts, the percentages of patients with LV systolic dysfunction defined as LVEF < 50% and of abnormal LV LS (<15%) decreased from baseline to 1 year in both the TAVR and SAVR groups. The percentages of patients with abnormal LV LS were similar (41.4% vs. 40.4% at baseline; 33.0% vs. 35.7% at 30 days; and 24.7% vs. 28.6% at 1 year) in SAVR vs. TAVR (*Figure 3*). The percentages of patients with reduced TAPSE (<14 mm; 18.4% vs. 18.5%, $P = 0.98$) and reduced RV–PA coupling (76.7% vs. 72.8%, $P = 0.29$) were similar in SAVR vs. TAVR at baseline. These percentages of reduced TAPSE and/or RV–PA coupling increased markedly from baseline to 30 days (18.4–50.5%, $P < 0.001$ for TAPSE; 76.7–95.2%, $P < 0.001$ for RV–PA coupling) after SAVR, whereas they remained stable after TAVR (*Figure 3*). The rates of reduced TAPSE were significantly higher after SAVR vs. TAVR at both 30 days (50.5% vs. 16.7%, $P < 0.001$) and 1-year follow-up (36.4% vs. 14.0%, $P < 0.001$). The percentages of patients with LV and/or RV dysfunction for the unmatched cohorts are shown in Supplementary data online, *Figure S2*.

Association between parameters of LV and RV longitudinal systolic function and clinical outcomes

After adjustment for STS score, baseline stroke volume index < 35 mL/m^2, mean aortic gradient < 20 mmHg, and ≥moderate mitral regurgitation, the presence of LV or RV systolic dysfunction at baseline were all associated with increased risk of the primary clinical endpoint [adjusted HRs for LVEF <50%: 1.23 (95% CI 1.04–1.44), $P = 0.013$; LV LS <15%: 1.24 (95% CI 1.05–1.45), $P = 0.001$; TAPSE <14 mm: 1.44 (95% CI 1.21–1.73), $P < 0.001$; RV–PA coupling <0.55 mm/mmHg: 1.32 (95% CI 1.07–1.63), $P = 0.011$] and of cardiac mortality [adjusted HRs for LVEF <50%: 1.28 (95% CI 1.00–1.63), $P = 0.005$; LV LS <15%: 1.44 (95% CI 1.13–1.83), $P = 0.003$; TAPSE <14 mm: 1.55 (95% CI 1.19–2.03), $P = 0.001$; RV–PA coupling <0.55 mm/mmHg: 1.87 (95% CI 1.31–2.65), $P < 0.001$] at 5 years (*Table 3*). TAPSE < 14 mm at baseline was the strongest predictor of the primary clinical endpoint in the whole cohort, as well as in both TAVR and SAVR groups (*Table 3*). Only parameters of RV dysfunction were associated with increased risk of cardiac death in both SAVR and TAVR groups, with RV–PA coupling < 0.55 mm/mmHg being the strongest predictor of cardiac death in all groups (*Table 3*).

The presence of LV systolic dysfunction (LVEF < 50%, LV LS <15%) or TAPSE <14 mm at 30 days were associated with increased risk of the composite endpoint at 5 years [adjusted HR for LVEF < 50%: 1.20 (95%

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Table 1 Baseline, procedural, and 30-day data in the TAVR and SAVR PS-matched groups

	All ($n = 998$)	TAVR ($n = 499$)	SAVR ($n = 499$)	*P*-value
Baseline demographic and clinical data				
Age, years	81.8 ± 6.49	81.9 ± 6.35	81.6 ± 6.64	0.530
Female sex	432 (43.29)	213 (42.69)	219 (43.89)	0.700
Body surface area, m^2	1.89 ± 0.24	1.89 ± 0.22	1.89 ± 0.25	0.930
Body mass index, kg/m^2	28.60 ± 5.99	28.50 ± 5.76	28.60 ± 6.22	0.850
Previous or current smoker	504 (50.50)	254 (50.90)	250 (50.10)	0.800
Hypertension	933 (93.49)	466 (93.39)	467 (93.59)	0.900
Dyslipidaemia	813 (81.46)	405 (81.16)	408 (81.76)	0.810
Diabetes mellitus	335 (33.57)	171 (34.27)	164 (32.87)	0.640
Peripheral arterial disease	320 (32.06)	153 (30.66)	167 (33.47)	0.340
Chronic kidney disease	64 (6.41)	35 (7.01)	29 (5.81)	0.440
Prior atrial fibrillation	346 (34.67)	184 (36.87)	162 (32.46)	0.140
Previous or current cancer	318 (31.86)	153 (30.66)	165 (33.07)	0.410
Current or previous COPD	279 (28.07)	142 (28.51)	137 (27.62)	0.750
Prior stroke or TIA	174 (17.43)	86 (17.23)	88 (17.64)	0.870
Congestive heart failure	844 (84.57)	436 (87.37)	408 (81.76)	0.010
Coronary artery disease	759 (76.05)	382 (76.55)	377 (75.55)	0.710
Prior myocardial infraction	161 (16.13)	78 (15.63)	83 (16.63)	0.670
Prior PCI	280 (28.06)	139 (27.86)	141 (28.26)	0.890
Prior CABG	265 (26.55)	130 (26.05)	135 (27.05)	0.720
Permanent pacemaker	123 (12.32)	61 (12.22)	62 (12.42)	0.920
STS score	5.40 ± 1.45 (997)	5.50 ± 1.28	5.40 ± 1.60 (498)	0.770
NYHA class III or IV	748 (74.95)	373 (74.75)	375 (75.15)	0.880
Baseline Doppler echocardiographic data				
Peak aortic velocity (cm/s)	432.38 ± 55.77 (979)	430.65 ± 51.42 (490)	434.11 ± 59.82 (489)	0.330
Aortic valve mean gradient (mmHg)	45.38 ± 12.57 (979)	45.16 ± 11.39 (490)	45.59 ± 13.65 (489)	0.590
Aortic valve area (cm^2)	0.69 ± 0.18 (932)	0.70 ± 0.17 (475)	0.69 ± 0.18 (457)	0.180
Aortic regurgitation ≥ moderate	65/969 (6.71)	29/488 (5.94)	36/481 (7.48)	0.340
Mitral regurgitation ≥ moderate	86/953 (9.02)	39/484 (8.06)	47/469 (10.02)	0.290
Tricuspid regurgitation ≥ moderate	77/912 (8.44)	28/472 (5.93)	49/440 (11.14)	0.005
LV stroke volume (mL)	71.72 ± 18.04 (934)	71.61 ± 17.45 (476)	71.84 ± 18.64 (458)	0.850
LV ejection fraction, %	51.39 ± 11.56 (767)	51.13 ± 11.97 (403)	51.69 ± 11.09 (364)	0.500
LV longitudinal strain, %	15.67 ± 4.01 (768)	15.61 ± 4.12 (403)	15.75 ± 3.89 (365)	0.630
TAPSE, mm	19.32 ± 5.52 (824)	19.28 ± 5.34 (433)	19.41 ± 5.72 (391)	0.750
PASP, mmHg	45.47 ± 11.96 (652)	45.39 ± 12.700 (347)	45.57 ± 11.08 (305)	0.840
RV–PA coupling	0.45 ± 0.19 (569)	0.45 ± 0.20 (316)	0.44 ± 0.17 (253)	0.570
Procedural data				
Transfemoral access	N/A	437 (87.58)	N/A	N/A
Pre-dilatation	N/A	126/493 (25.56)	N/A	N/A
Post-dilatation	N/A	55 (11.02)	N/A	N/A
Concomitant PCI	N/A	3 (0.60)	N/A	N/A
Concomitant CABG	N/A	N/A	65/470 (13.83)	N/A
30-day Doppler echocardiographic data				
Peak aortic velocity (cm/s)	226.43 ± 44.22 (856)	226.54 ± 43.39 (461)	226.31 ± 45.22 (395)	0.940
Aortic valve mean gradient (mmHg)	11.26 ± 5.06 (856)	11.49 ± 4.98 (461)	10.98 ± 5.13 (395)	0.130
Aortic valve area (cm^2)	1.61 ± 0.44 (809)	1.70 ± 0.423 (438)	1.50 ± 0.427 (371)	<0.001
Aortic regurgitation ≥ moderate	19/851 (2.23)	16/465 (3.44)	3/386 (0.78)	0.009

Continued

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Table 1 Continued

	All ($n = 998$)	TAVR ($n = 499$)	SAVR ($n = 499$)	*P*-value
Severe PPM at 30 days	139/805 (17.27)	41/438 (9.36)	98/367 (26.70)	<0.001
Mitral regurgitation ≥ moderate	79/832 (9.50)	31/462 (6.71)	48/370 (12.97)	0.002
Tricuspid regurgitation ≥ moderate	79/829 (9.53)	26/463 (5.62)	53/366 (14.48)	<0.001
LV stroke volume (mL)	71.20 ± 19.83 (814)	77.07 ± 18.86 (440)	64.29 ± 18.71 (374)	<0.001
LV ejection fraction, %	52.21 ± 11.04 (659)	52.23 ± 10.89 (383)	52.19 ± 11.27 (276)	0.960
LV longitudinal strain, %	16.27 ± 3.75 (660)	16.27 ± 3.78 (384)	16.27 ± 3.72 (276)	0.980
TAPSE, mm	16.89 ± 5.68 (672)	19.10 ± 5.54 (395)	13.68 ± 4.22 (277)	<0.001
PASP, mmHg	41.21 ± 9.86 (636)	40.51 ± 10.32 (358)	42.10 ± 9.19 (278)	0.040
RV–PA coupling, mm/mmHg	0.43 ± 0.18 (519)	0.49 ± 0.19 (312)	0.33 ± 0.13 (207)	<0.001

Values are expressed as mean ± SD or absolute number (%). Values between parentheses are number of patients with data available for continuous variables (shown only if missing data) or percentage of patients for binary variables. In case data are missing for a given variable, we added the denominator corresponding to the number of patients with data available.
CABG, coronary artery bypass graft; COPD, chronic obstructive pulmonary disease; LV, left ventricle; N/A, not available; NYHA, New York Heart Association; PA, pulmonary artery; PASP, pulmonary artery systolic pressure; PCI, percutaneous coronary intervention; PPM, patient–prosthesis mismatch; RV, right ventricle; SAVR, surgical aortic valve replacement; STS, Society of Thoracic Surgeons; TAPSE, tricuspid annular plane systolic excursion; TAVR, transcatheter aortic valve replacement; TIA, transient ischaemic attack.

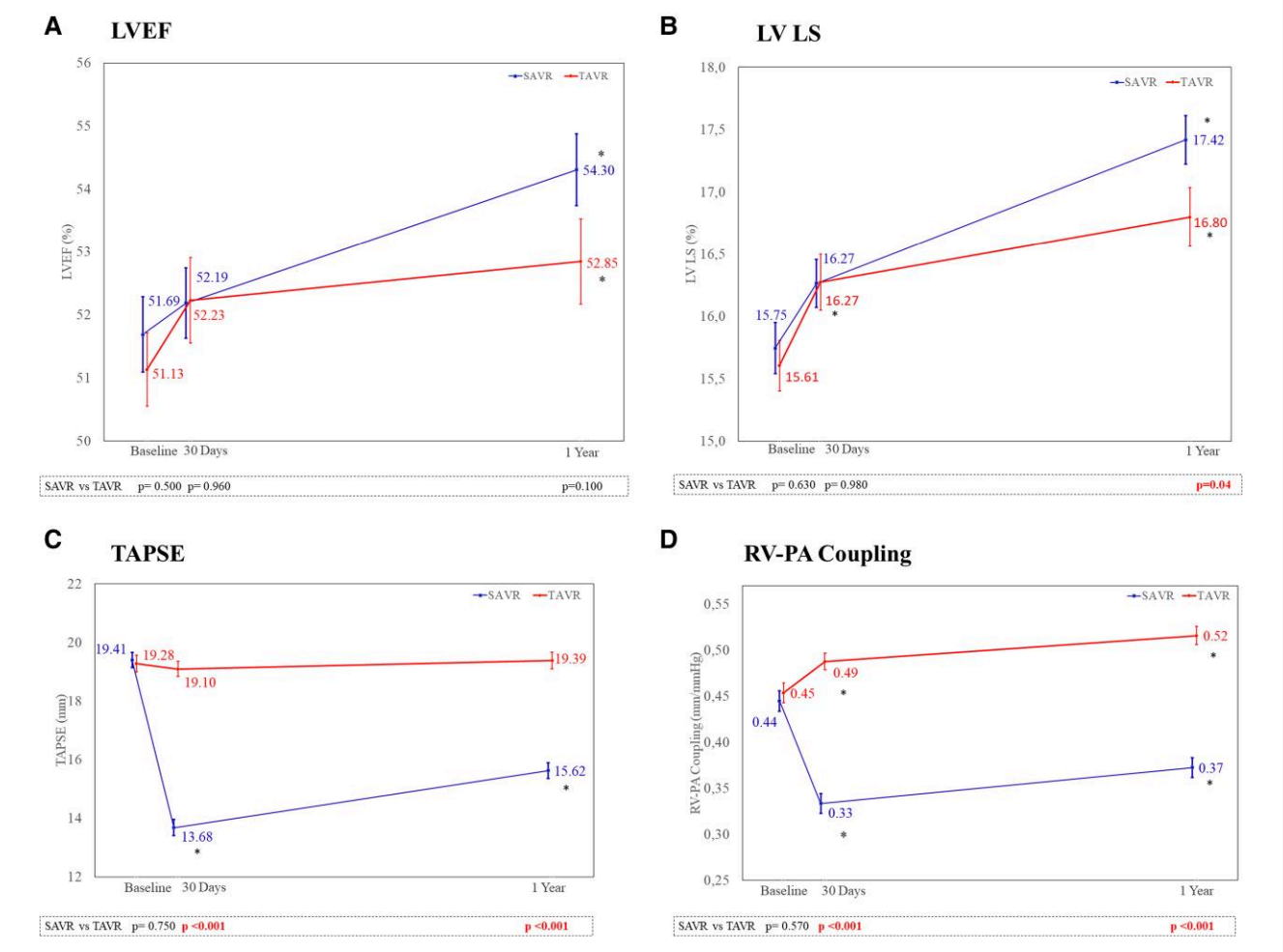

Figure 2 Comparison of echocardiographic parameters of LV and RV systolic function between TAVR and SAVR for the PS-matched cohorts. Line charts showing pre- and post-procedure data of LVEF (*A*), LV LS (*B*), TAPSE (*C*), and RV–PA coupling (*D*) in TAVR vs. SAVR. LS, longitudinal strain; LV, left ventricle; LVEF, left ventricular ejection fraction; RV–PA coupling, right ventricle–pulmonary artery coupling; SAVR, surgical aortic valve replacement; TAPSE, tricuspid annular plane systolic excursion; TAVR, transcatheter aortic valve replacement. *$P < 0.05$ vs. baseline.

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Table 2 Comparison of echocardiographic parameters of LV and RV systolic function over time (baseline, 30 days, and 1 year) for PS-matched cohorts

		Baseline	30 days	1 year	*P*-value (baseline vs. 30 days)	*P*-value (baseline vs. 1 year)	*P*-value (30 days vs. 1 year)
TAVR	LVEF	51.13 ± 11.97	52.23 ± 10.89	52.85 ± 10.35	0.190	0.090	0.760
	LV LS	15.61 ± 4.12	16.27 ± 3.78	16.80 ± 3.54	**<0.001**	**<0.001**	**0.040**
	TAPSE	19.28 ± 5.32	19.10 ± 5.54	19.39 ± 4.53	0.160	0.900	0.610
	RV–PA coupling	0.45 ± 0.20	0.49 ± 0.19	0.52 ± 0.18	**0.040**	**0.003**	0.580
SAVR	LVEF	51.69 ± 11.09	52.19 ± 11.27	54.30 ± 10.32	0.470	**0.005**	0.29
	LV LS	15.75 ± 3.89	16.27 ± 3.72	17.42 ± 3.60	0.08	**<0.001**	**<0.001**
	TAPSE	19.41 ± 5.72	13.68 ± 4.22	15.62 ± 4.41	**<0.001**	**<0.001**	**<0.001**
	RV–PA coupling	0.44 ± 0.17	0.33 ± 0.13	0.37 ± 0.13	**<0.001**	**<0.001**	**0.010**

P-values in bold indicate significant association.
LS, longitudinal strain; LV, left ventricle; LVEF, left ventricular ejection fraction; RV–PA coupling, right ventricle–pulmonary artery coupling; SAVR, surgical aortic valve replacement; TAPSE, tricuspid annular plane systolic excursion; TAVR, transcatheter aortic valve replacement.

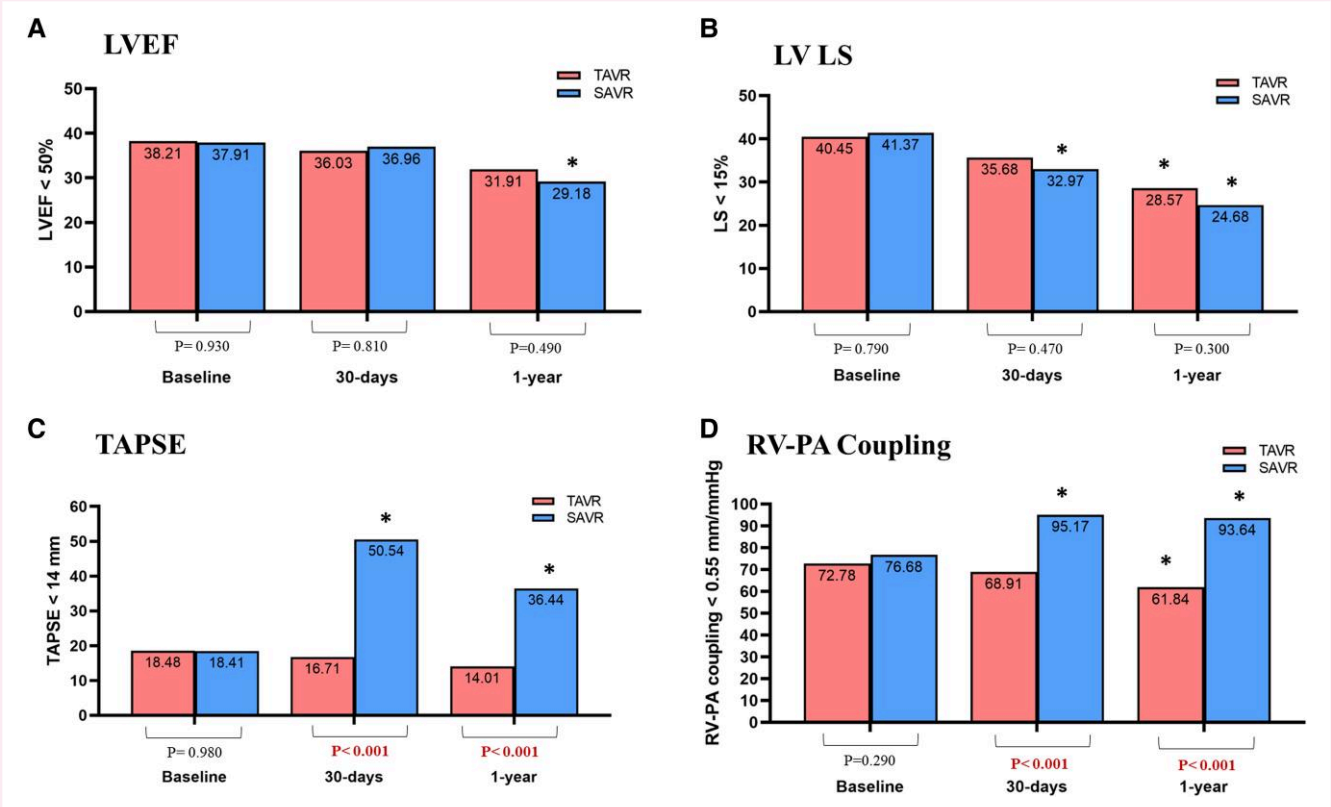

Figure 3 Proportion of patients with abnormal echocardiographic parameters of LV and RV systolic dysfunction in TAVR vs. SAVR groups at baseline, 30 days, and 1 year for the PS-matched cohorts. Bar chart showing pre- and post-procedure percentages of patients with (*A*) LVEF <50%, (*B*) LV LS < 15%, (*C*) TAPSE <14 mm, and (*D*) RV–PA coupling < 0.55 mm/mmHg in TAVR vs. SAVR patients. *$P < 0.05$ vs. baseline. Abbreviations as in *Figure 2*.

CI 1.00–1.44), $P = 0.049$; LS <15%: 1.29 (95% CI 1.08–1.56), $P = 0.006$; TAPSE <14 mm: 1.21 (95% CI 1.00–1.45), $P = 0.044$; *Table 4*]. The presence of LV systolic dysfunction (LVEF <50%, LV LS <15%) or RV–PA coupling < 0.55 mm/mmHg at 30 days were associated with increased risk of cardiac mortality at 5 years [adjusted HR for LVEF < 50%: 1.43 (95% CI 1.08–1.90), $P = 0.013$; LS <15%: 1.47 (95% CI 1.10–1.95), $P = 0.008$; RV–PA coupling <0.55 mm/mmHg: 1.71 (95% CI 1.08–2.72), $P = 0.023$; *Table 4*]. Reduced RV–PA coupling at 30 days was the strongest predictor of cardiac mortality.

Patients with impaired LV or RV longitudinal systolic function (i.e. LVEF < 50%, LV LS < 15%, TAPSE < 14 mm, and/or RV–PA coupling ratio < 0.55 mm/mmHg) undergoing SAVR had increased risk of clinical

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Table 3 Association between baseline LVEF < 50%, LV LS < 50%, TAPSE < 14 mm, and RV coupling < 0.55 mm/mmHg and clinical outcomes at 5 years for the PS-matched cohorts

5-year clinical outcomes	Echo variable at baseline	Adjusted hazard ratio (95% CI)[a] *P*-value		
		TAVR	SAVR	Whole cohort
Combined endpoint of death, hospitalization, and stroke	LVEF < 50%	1.12 (0.88, 1.42), 0.364	1.34 (1.08, 1.68), **0.009**	1.23 (1.04, 1.44), **0.013**
	LV LS < 15%	1.19 (0.94, 1.51), 0.149	1.29 (1.03, 1.60), **0.023**	1.24 (1.05, 1.45), **0.001**
	TAPSE < 14mm	1.49 (1.15, 1.93), **0.003**	1.43 (1.11, 1.83), **0.006**	1.44 (1.21, 1.73), **<0.001**
	RV–PA coupling < 0.55 mm/mmHg	1.29 (0.97, 1.73), 0.079	1.34 (0.97, 1.86), 0.076	1.32 (1.07, 1.63), **0.011**
Cardiac death	LVEF < 50%	1.14 (0.79, 1.65), 0.483	1.36 (0–98, 1.89), 0.064	1.28 (1.00, 1.63), **0.050**
	LV LS < 15%	1.44 (1.00, 2.07), 0.051	1.40 (1.01, 1.94), **0.041**	1.44 (1.13, 1.83), **0.003**
	TAPSE < 14mm	1.50 (1.01, 2.21), **0.043**	1.37 (0–89, 2.09), **0.018**	1.55 (1.19, 2.03), **0.001**
	RV–PA coupling < 0.55 mm/mmHg	1.61 (1.01, 2.58), **0.047**	2.03 (1.17, 3.51), **0.012**	1.87 (1.31, 2.65), **<0.001**

P-values in bold indicate significant association.
CI, confidence interval; others as in *Table 2*.
[a]Hazard ratios have been adjusted by Society of Thoracic Surgeons score, baseline stroke volume index < 35 mL/m^2, mean gradient < 20 mmHg, and ≥moderate mitral regurgitation. For HR, the values below every specified threshold are considered as the reference.

Table 4 Association between 30 days LVEF < 50%, LV LS < 50%, TAPSE < 14 mm, and RV coupling < 0.55 mm/mmHg and clinical outcomes at 5 years for the PS-matched cohorts

5-year clinical outcomes	Echo variable at 30 days	Adjusted hazard ratio (95% CI)[a] *P*-value		
		TAVR	SAVR	Whole cohort
Combined endpoint of death, hospitalization, and stroke	LVEF < 50%	1.16 (0.90, 1.49), 0.245	1.26 (0.97, 1.65), 0.089	1.20 (1.00, 1.44), **0.049**
	LV LS < 15%	1.22 (0.94, 1.57), 0.132	1.41 (1.08, 1.84), **0.012**	1.29 (1.08, 1.56), **0.006**
	TAPSE < 14mm	1.24 (0.91, 1.68), 0.176	1.29 (0.99, 1.70), 0.062	1.21 (1.00, 1.45), **0.044**
	RV–PA coupling < 0.55 mm/mmHg	1.26 (0.93, 1.60), 0.132	1.54 (0.74, 3–76), 0.339	1.19 (0.91, 1.55), 0.211
Cardiac death	LVEF < 50%	1.37 (0.92, 2.04), 0.124	1.42 (0–95, 2.35), 0.091	1.43 (1.08, 1.90), **0.013**
	LV LS < 15%	1.19 (0.79, 1.79), 0.413	1.72 (1.15, 2.59), **0.009**	1.47 (1.10, 1–95), **0.008**
	TAPSE < 14mm	1.46 (0.91, 2.33) 0.117	1.40 (0.95, 2.06), 0.150	1.30 (0–97, 1.73), 0.080
	RV–PA coupling < 0.55 mm/mmHg	2.06 (1.23, 3.48), **0.007**	1.14 (0.36, 3.62), 0.827	1.71 (1.08, 2.72), **0.023**

P-values in bold indicate significant association.
Abbreviations as in *Tables 2* and *3*.
[a]Hazard ratios have been adjusted by Society of Thoracic Surgeons score, baseline stroke volume index < 35 mL/m^2, mean gradient < 20 mmHg, and ≥ moderate mitral regurgitation. For HR, the values below every specified threshold are considered as the reference.

outcomes in the short term (as early as the first year), whereas those undergoing TAVR also had increased risk of adverse outcomes but with a more delayed impact: i.e. beyond 2–3 years (*Figure 4*). The PS-matched analyses provided similar results (see Supplementary data online, *Results* and *Figure S3*). The results for cardiac mortality are shown in Supplementary data online, *Figures S4* and *S5*, for the unmatched and PS-matched cohorts, respectively.

In the subgroup analyses (see Supplementary data online, *Figure S6*), TAVR appeared to be associated with a lower rate of the primary outcome at 5 years compared to SAVR in patients with LVEF < 50% at baseline (see Supplementary data online, *Figure S6A*).

Discussion

This study constitutes, by far, the largest and most comprehensive echocardiography core lab analysis of the changes over time and the clinical impact of both LV and RV longitudinal systolic function in patients undergoing TAVR or SAVR. Furthermore, this is the first study to report LV LS data in a TAVR vs. SAVR randomized trial and registry. The main findings of study could be summarized as follows: (i) LV LS improved to a slightly larger extent from baseline to 1 year in SAVR vs. TAVR patients, although the improvement occurs earlier in TAVR; (ii) TAPSE remained unchanged and RV–PA coupling improved following TAVR but deteriorated significantly following SAVR; (iii) lower LV LS, TAPSE, or RV–PA coupling at baseline were associated with increased risk of all-cause death, stroke, and heart failure hospitalization at 5 years in the whole cohort; (iv) reduced TAPSE (<14 mm) at baseline appears to be a powerful predictor of adverse clinical outcomes in the whole cohort as well as in the TAVR and SAVR cohorts, separately; (v) reduced RV–PA coupling (<0.55 mm/mmHg) at baseline and 30 days showed the strongest association with cardiac death at 5 years; and (vi) TAVR is associated with better clinical outcomes than SAVR in the subset of patients with LVEF <50%.

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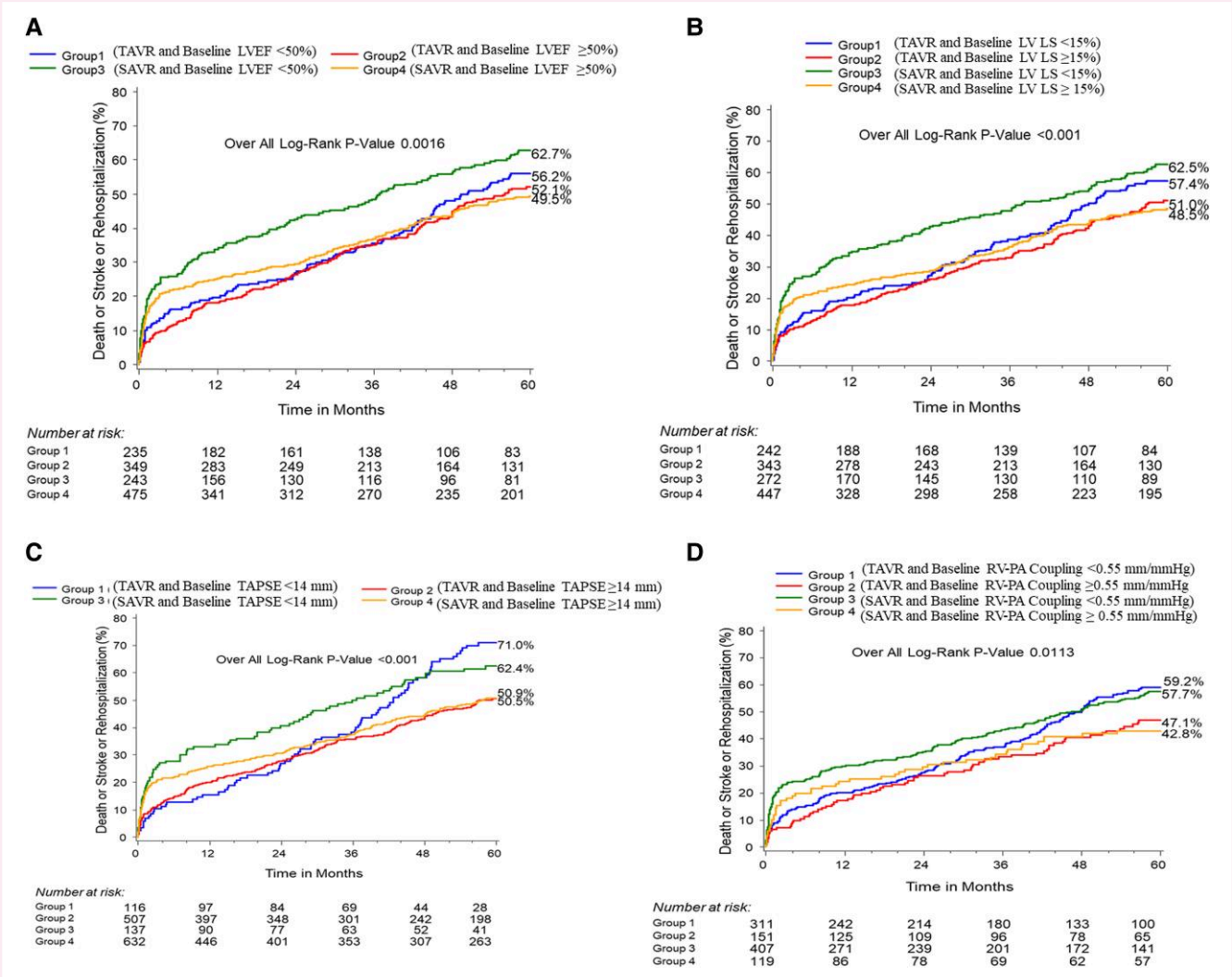

Figure 4 Incidence of clinical outcomes according to presence of LV or RV systolic dysfunction and type of AVR: TAVR vs. SAVR in the unmatched cohorts. Kaplan–Meier curves for the combined endpoint (all-cause death, rehospitalization, or stroke) at 5-year follow-up according to the treatment (TAVR vs. SAVR) group and the presence of LVEF < or ≥50% (*A*), LV LS < or ≥15% (*B*), TAPSE < or ≥14 mm (*C*), and RV–PA coupling < or ≥0.55 mm/mmHg (*D*). AVR, aortic valve replacement. Other abbreviations as in *Figure 2*.

LVEF is a powerful marker of clinical outcomes in both asymptomatic and symptomatic patients with severe AS. AVR is recommended in asymptomatic patients with depressed LVEF (i.e. <50%) to avoid irreversible LV systolic dysfunction leading to cardiac events.[1,2] In symptomatic patients with an indication of aortic valve intervention, LVEF may be used to stratify the risk of adverse outcomes following AVR and potentially to determine the optimal type of AVR (TAVR vs. SAVR). In the present study, TAVR was associated with better clinical outcomes compared to SAVR in the subset of patients with LVEF < 50%. In asymptomatic patients with severe AS, LV LS appears to provide incremental prognostic value compared to LVEF.[4] This may be explained by the fact that in patients with LV concentric hypertrophy such in AS, LV LS is an earlier and more sensitive marker of LV subclinical dysfunction. In the present study, LV LS at 30 days was superior to LVEF to predict subsequent clinical outcomes following SAVR.

There are few data on the comparison of the evolution of LV systolic function according to the type of AVR: TAVR vs. SAVR. In the present study, LV systolic function (i.e. LVEF and LV LS) improved similarly after both SAVR and TAVR, but this improvement occurred earlier in the TAVR group. Several factors may be associated with improvement in LV systolic function after AVR, including the reduction in the LV pressure overload, imposed by the severe AS, the reduction in LV myocardial ischaemia, the improvement in haemodynamic conditions, and the regression of LV hypertrophy and fibrosis. Myocardial injury caused by ischaemia–reperfusion and inflammation during cardiopulmonary bypass, aortic cross-clamping, and immediate postoperative period may explain the delayed recovery of LV systolic function in patients undergoing SAVR. In this study, lower LVEF or LV LS at 30 days were associated with 5-year clinical outcomes, underscoring the importance to prevent perioperative myocardial injury during AVR and particularly during SAVR.

Although the rates of ≥moderate residual aortic regurgitation were low in both groups, they were higher in TAVR patients. It cannot be excluded that this fact may have contributed to the better LVEF and LV LS parameters observed in the SAVR group at follow-up.

The echocardiographic parameters of RV systolic function may be helpful to predict the risk of adverse events not only in asymptomatic patients with severe AS[22] but also in those undergoing left-sided surgery including AVR.[23,24] In the PARTNER 3 trial including low surgical

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risk patients with severe AS undergoing AVR, we previously reported a reduction in TAPSE in the SAVR arm but not in the TAVR arm, which was associated with increased risk of mortality, stroke, and hospitalization at follow-up.[12] Recently, we also reported, in a sub-analysis of the PARTNER 3 trial, that RV–PA uncoupling was associated with adverse outcomes.[13] In the present study, we investigated all these markers of RV function to determine their evolution and clinical impact after AVR. The post-procedural changes in RV systolic function and of RV–PA coupling ratio also differed markedly according to the type of AVR. RV function was preserved after TAVR, whereas SAVR was associated with a postoperative deterioration in RV function that persisted at 1-year follow-up. Furthermore, a lower TAPSE and RV–PA coupling were independent predictors of adverse outcomes at 5 years following AVR. The factors that may explain the deterioration in RV longitudinal systolic function and RV–PA coupling after SAVR may include the following: (i) the uncoupling between the RV and the pericardial sac due to the pericardiectomy; (ii) incomplete RV myocardial protection during cardiopulmonary bypass; (iii) transient pulmonary hypertension during the peri-operative period; (iv) gaseous emboli during cardiopulmonary bypass[25–28]; and (v) higher rates of postoperative mitral regurgitation in the SAVR vs. TAVR group. We have recently proposed a classification to determine the extent of extra-valvular cardiac damage associated with AS and enhance risk stratification prior or after AVR.[22,29,30] We have also published that among patients with severe AS in the PARTNER 2 and 3 trials, the extent of cardiac damage is associated with health status before and after AVR.[31] In light of the results of the present study, including LV LS < 15% in the criteria for Stage 1 (LV damage) and TAPSE < 14 mm and RV–PA coupling ratio < 0.55 mm/mmHg in Stage 4 (RV damage) could potentially further improve the prognostic value of this staging scheme, but this remains to be validated. Moreover, TAVR should be systematically discussed as an alternative to SAVR in patients with altered LVEF (<50%, Stage 1), severe pulmonary hypertension (i.e. systolic pressure > 60 mmHg, Stage 3), and/or RV damage (Stage 4). In particular, the results of the present study provide support to the selection of TAVR rather than SAVR in patients with LVEF < 50%, TAPSE < 14 mm, and/or RV–PA coupling ratio < 0.55 mm/mmHg.

Study limitations and strengths

Patients were not randomized to SAVR vs. TAVR with the SAPIEN 3 valve, and patients with SAVR had higher LVEF, LV LS, and RV–PA coupling at baseline compared to those with TAVR. Selection biases could have occurred because this analysis has been performed only in the subset of patients with available echocardiographic data at baseline, 30 days, and 1 year. Although subgroup analyses [i.e. patients with or without patient–prosthesis mismatch (PPM), post-procedural significant mitral or tricuspid regurgitation] might be of interest, the lack of statistical power prevents us to perform further subanalyses. The echocardiographic parameters of LV and RV longitudinal systolic function analysed in the present study were measured by the same echocardiographic core laboratory. The lack of proper image quality in the three-chamber and two-chamber apical views limited our analyses for the LV strain to four-chamber data. The measurement of RV free wall LS was attempted, but feasibility was poor (<50%). In the subset of patients in whom the measurement of RV free wall strain was feasible, the patterns of changes in RV free wall strain in the SAVR vs. TAVR groups were similar to those of TAPSE (see Supplementary data online, *Results* and *Figure S7*). In the present study, TAPSE was used to assess RV longitudinal systolic function. Some studies suggested that TAPSE may overestimate the degree of RV systolic dysfunction following pericardiectomy. Several studies[32] reported that TAPSE or RV free wall strain decrease after SAVR, whereas the RV ejection fraction or the fractional area change do not change following surgery. A multiparametric approach including parameters that take into account not only the longitudinal RV systolic function but also the radial systolic component (e.g. fractional area change and RV ejection fraction by 3D) may, nonetheless, have provided a more comprehensive assessment of the global RV systolic function post SAVR. Nevertheless, it is crucial to highlight that TAPSE remains widely accessible and extensively used in clinical practice, with established prognostic significance.

Conclusion

In patients with severe AS and intermediate surgical risk, LV systolic function improved from baseline to 1 year to a slightly larger extent in SAVR vs. TAVR, but the improvement occurred earlier in TAVR. SAVR was associated with a marked deterioration in RV longitudinal systolic function and RV–PA coupling ratio, which persisted at 1 year, whereas parameters of RV function remained stable or improved following TAVR. Reduced TAPSE and RV–PA coupling at baseline or 30 days were the most powerful independent predictors of clinical outcomes at 5 years. In patients with baseline LVEF <50%, SAVR was associated with worse clinical outcomes at 5 years compared to TAVR.

Supplementary data

Supplementary data are available at *European Heart Journal - Cardiovascular Imaging* online.

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Conflict of interest: J.T. is consultant for Abbott, Philips Healthcare, and General Electric. R.T.H. reports speaker fees from Abbott Structural, Baylis Medical, Edwards Lifesciences, Medtronic, and Philips Healthcare; she has institutional consulting contracts for which she receives no direct compensation with Abbott Structural, Edwards Lifesciences, Medtronic, and Novartis; she is Chief Scientific Officer for the Echocardiography Core Laboratory at the Cardiovascular Research Foundation for multiple industry-sponsored tricuspid valve trials, for which she receives no direct industry compensation. M.-A.C. has research grant with Medtronic and core laboratory contract with Edwards Lifesciences without direct compensation. W.J. is consultant for Boston Scientific and BridgeBio. F.M.A. and N.J.W. have institutional research grants as directors of an academic core lab from Edwards, Abbott, Medtronic, Boston Scientific, Biotronik, Corcyn, and Foldax. They have no personal disclosures. N.A. received speakers fees from Abbott Vascular, GE Healthcare, and Philips ultrasound and research grants from Alnylam and Pfizer. H.C.H. reports institutional research funding from Abbott, Boston Scientific, Edwards Lifesciences, Highlife, Medtronic, and WL Gore; consulting fees from Edwards Lifesciences, Medtronic, Wells Fargo, and WL Gore; and equity in Holistick Medical and Micro Interventional Devices. M.J.M. has served as a Co-Pi for clinical trials for Abbott and Edwards Lifesciences and as Study Chair for a trial for Medtronic. All roles were uncompensated. V.T. is consultant or researcher for Abbott Vascular, Boston Scientific, CryoLife, Edwards Lifesciences, Medtronic, and Shockwave. The other authors have nothing to disclose.

Data availability

The data, analytic methods, and study materials of the PARTNER 2 trial and SAPIEN 3 registry will not be made available to other researchers for purposes of reproducing the results or replicating the procedure.

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