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**Right ventricular function following Surgical Aortic Valve Replacement and  
Transcatheter Aortic valve implantation: a Cardiovascular MR study.**

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## ABSTRACT

**Objective:** The response of the RV following treatment of aortic stenosis is poorly defined, reflecting the challenge of accurate RV assessment. Cardiovascular magnetic resonance (CMR) is the established reference for imaging of RV volumes, mass and function. We sought to define the impact of transcatheter aortic valve implantation (TAVI) and surgical aortic valve replacement (SAVR) upon RV function in patients treated for severe aortic stenosis using CMR.

**Methods:** A 1.5T CMR scan was performed preoperatively and 6 months postoperatively in 112 (56 TAVI, 56 SAVR;  $76\pm 8$  years) high-risk severe symptomatic aortic stenosis patients across two UK cardiothoracic centres.

**Results:** TAVI patients were older ( $80.4\pm 6.7$  vs.  $72.8\pm 7.2$  years,  $p<0.05$ ) with a higher STS score ( $2.13 \pm 0.73$  vs.  $5.54 \pm 3.41\%$ ,  $p< 0.001$ ). At 6 months, SAVR was associated with a significant increase in RV end systolic volume ( $33\pm 10$  vs.  $37\pm 10\text{ml/m}^2$ ,  $p=0.008$ ), and decrease in RV ejection fraction ( $58\pm 8$  vs.  $53\pm 8\%$ ,  $p=0.005$ ) and tricuspid annular plane systolic excursion ( $22\pm 5$  vs.  $14\pm 3\text{mm}$ ,  $p<0.001$ ). Only 4 (7%) SAVR patients had new RV late gadolinium hyper-enhancement with no new cases seen in the TAVI patients at 6 months. Longer surgical cross-clamp time was the only predictor of increased RV end systolic volume at 6 months. Post-TAVI, there was no observed change in RV volumes or function. Over a maximum 6.3 year follow-up, 18(32%) of TAVI patients and 1(1.7%) of SAVR patients had died ( $p=0.001$ ). On multivariable Cox analysis, the RV mass at 6m post-TAVI was independently associated with all-cause mortality (HR 1.359, 95% CI 1.108-1.666,  $p=0.003$ ).

**Conclusions:** SAVR results in a deterioration in RV systolic volumes and function associated with longer cross-clamp times and is not fully explained by suboptimal RV protection during cardiopulmonary bypass. TAVI had no adverse impact upon RV volumes or function.

**KEYWORDS:** aortic stenosis, transcatheter aortic valve implantation, aortic valve replacement, right ventricular function, cross-clamp time



## **ABBREVIATIONS**

AR	= aortic regurgitation
AVA	= aortic valve area
CABG	= coronary artery bypass grafting
CMR	= cardiovascular magnetic resonance
EuroSCORE	= European System for Cardiac Operative Risk Evaluation
LGE	= late gadolinium enhancement
LVEF	= left ventricular ejection fraction
MI	= myocardial infarction
MR	= mitral regurgitation
NYHA	= New York Heart Association
PCI	= percutaneous coronary intervention
RVEF	= right ventricular ejection fraction
RVESVI	= right ventricular end systolic volume index
RVEDVI	= right ventricular end diastolic volume index
SAVR	= surgical aortic valve replacement
STS	= Society of Thoracic Surgeons' risk model
TAVI	= transcatheter aortic valve implantation

## INTRODUCTION

Surgical aortic valve replacement (SAVR) is first-line therapy for symptomatic patients with severe aortic valve stenosis. Transcatheter aortic valve implantation (TAVI) has emerged as a clinical and cost-effective treatment for patients deemed inoperable or with too high predicted mortality(1). Reverse remodelling of the left ventricle observed following both TAVI and SAVR has been well documented(2). However, much less is understood about the response of the right ventricle (RV) in these settings.

RV dysfunction is thought to occur following cardiac surgery for both valvular(3) and coronary disease(4) and is an independent predictor of late survival and adverse clinical outcomes(5). The precise mechanism of this dysfunction remains to be elucidated; a number of theories have been proposed based on conflicting evidence. The EuroSCORE II and the STS models for calculating operative mortality of cardiac surgery do not incorporate preoperative RV dysfunction, despite its' association with a high mortality(6). This in part reflects the challenging nature of reliably evaluating RV performance(7) with its asymmetric and variable 3D geometry.

Cardiovascular magnetic resonance (CMR) is the established reference modality for imaging of both left and right ventricular volumes and function. CMR affords reproducible 3D volume acquisition, can image in any plane, has excellent blood-tissue contrast and can detect subtle wall motion abnormalities(8).

Studies directly comparing the impact of SAVR with TAVI upon RV function are limited(6, 9, 10) and have depended upon 2D transthoracic echocardiographic (TTE) parameters with relatively short follow-up. This study was designed specifically to determine the impact of SAVR and TAVI upon RV performance using CMR at 6 months. We hypothesised that SAVR, but not TAVI (which obviates the need of cardiopulmonary bypass and pericardiotomy), would be associated with decline in RV function. Furthermore, we sought to elucidate potential mechanisms, by defining the contribution of procedural factors and CMR derived parameters to any observed change in RV performance.

## **METHODS**

### Study population

This study prospectively recruited 167 patients with severe trileaflet degenerative AS (TTE valve area  $\leq 1.0\text{cm}^2$  or peak velocity  $>4\text{m/s}$ ) who were referred for either TAVI (n=101) or SAVR (n=66) at the University Hospitals of Leeds and Leicester, UK, between July 2008 and December 2013. Higher-risk (higher EuroSCORE) SAVR patients were recruited in preference to ensure baseline demographics were more comparable to the TAVI group. Exclusion criteria included any contraindication to CMR. The study was approved by a national ethics committee, complied with the Declaration of Helsinki and all patients provided written informed consent.

### Transcatheter Aortic Valve Implantation

TAVI was performed under general anaesthesia. Either an 18F CoreValve Revalving system (CVR, Medtronic, Minneapolis, Minnesota, USA) or an 18F or 20F Lotus™ Aortic Valve system (Boston Scientific Corporation, Natick, MA, USA) were deployed.

### Surgical Aortic Valve Replacement

SAVR was performed by standard midline sternotomy with cardiopulmonary bypass and mild hypothermia. Biological or mechanical prostheses of varying sizes were used according to surgical preference; concomitant coronary artery bypass grafting (CABG) was performed as indicated.

### CMR Protocol

For each individual patient, identical baseline preoperative and 6 month postoperative scans were performed on the same 1.5T MRI vendor platform (Intera, Phillips Healthcare, Best, Netherlands or Avanto, Siemens Medical Systems, Erlangen, Germany). Both sites used the identical CMR protocol as previously described(2).

### CMR Image Analysis

Image analysis was performed blinded off-line, using commercially available software (QMass 7.5 and QFlow 7.2, Medis Medical Imaging Systems, Leiden, The Netherlands – used for LV

and RV chamber quantification and valvular haemodynamics; CVI42, Circle Cardiovascular Imaging, Calgary, Alberta, Canada – used for assessment of LGE). Standard ventricular and valvular assessment was performed as previously described(2).

For patients in normal sinus rhythm, the left atrium emptying fraction was determined, defined as  $(LAVol_{max} - LAVol_{min}) \times 100 / LAVol_{max}$ . Similarly, the right atrium emptying fraction was determined, defined as  $(RAVol_{max} - RAVol_{min}) \times 100 / RAVol_{max}$ .

The tricuspid annular plane systolic excursion (TAPSE) was measured as the maximum apical displacement of the lateral tricuspid valve annulus from end-diastole to end-systole (Figures 1A and 1B). Delayed late gadolinium enhanced images were reviewed by two experienced observers for focal myocardial fibrosis and scarring (secondary to infarction) and then reported qualitatively, as either present or absent, and, for the LV, quantified using the full-width half-maximum technique.

#### Statistical Analysis

Based on published data, 45 patients per group were required to detect a 7ml change in RVEDV or 2% difference in EF between the two treatments (80% power and an  $\alpha$  error of 0.05)(8). Continuous variables are presented as mean $\pm$ SD. Normality was determined by the Shapiro–Wilk test. Frequencies are reported as number(%). The Student t-test and Wilcoxon signed rank test were used for continuous variables. Changes over time were assessed for differences between the treatment groups and clinical variables by two-way repeated measures ANOVA. Predictors of functional change were calculated by a stepwise multiple linear regression model with baseline measurements entered as covariates. Variables with a univariate  $p < 0.05$  were deemed significant. All statistical analyses were performed using the PASW software package (V.21.0 SPSS, IBM, Chicago, Illinois, USA), with a two-sided significance level of  $p < 0.05$  considered statistically significant. Intra-observer (12 data sets 6 months apart) and inter-observer (12 data sets) agreement was assessed and expressed as coefficient of variation.

## RESULTS

### Patient population

A total of 112 patients (56 TAVI and 56 SAVR) completed both preoperative and 6 month post-operative scans. Reasons for non-completion of the CMR protocol were varied (Figure 2). Baseline characteristics of the final study population are reported in Table 1. TAVI patients were older, with a higher STS score and greater frequency of coronary intervention. There was no difference in baseline pulmonary pressure, as estimated by echocardiography, between the two intervention groups ( $p=0.159$ ).

### Procedural data

For the TAVI group, 46(82%) patients received a Medtronic CoreValve and 10(18%) a Boston Scientific Lotus valve. The femoral artery was the route of access for 51(91%) patients. Three TAVIs were performed via the subclavian artery, one via the carotid artery and one via a direct aortic approach. Procedural success was 100% with an average catheterisation time of  $162\pm 53$ min, fluoroscopy time  $25\pm 7$ min and  $147\pm 50$ mls of contrast agent. One patient had concomitant PCI at the time of TAVI.

For the surgical group, seven patients received a mechanical prosthesis and the remaining 49(88%) a tissue bioprosthesis. Sixteen (29%) received concomitant CABG, of which 9 involved use of the left internal mammary artery. None of the surgical patients received a concomitant tricuspid or mitral valve annuloplasty ring and none underwent surgical closure of the pericardium. For the group as a whole, the average bypass time was  $104\pm 47$ min and cross clamp time  $76\pm 40$ min. The average length of stay in intensive care was  $3.1\pm 2.5$  days.

### Haemodynamics, valvular function and LV reverse remodelling

Baseline and follow-up CMR scan results are shown in Table 2. Comparable degrees of reduction in aortic valve gradient and LV reverse remodelling were seen following TAVI and SAVR.

A significant decline in the RA emptying fraction was seen following SAVR (baseline  $34.7\pm 8.7\%$  vs. 6m  $25.5\pm 9.7\%$ ,  $p<0.001$ ) and increase following TAVI (baseline  $31.6\pm 10.8\%$  vs. 6m  $35.7\pm 12\%$ ,  $p=0.009$ ). No change in LA emptying fraction was seen following SAVR

(baseline  $48.5 \pm 12.8\%$  vs. 6m  $48.7 \pm 9.1\%$ ,  $p=0.945$ ) but a significant improvement occurred following TAVI (baseline  $36.9 \pm 12.6\%$  vs. 6m  $43.4 \pm 10.4\%$ ,  $p=0.011$ ).

#### Impact of intervention upon Right Ventricular size and function

No difference existed between the groups' preoperative indexed measurements of right ventricular EDV ( $p=0.547$ ) or mass ( $p=0.462$ ). Although both groups had preserved RV systolic function, the baseline RV ejection fraction ( $p=0.001$ ) and TAPSE ( $p=0.026$ ) were significantly higher in the SAVR group. SAVR, but not TAVI, was associated with a statistically significant decrease in RV ejection fraction with a concomitant increase in indexed RVESV at 6 months. Similarly post-operative SAVR TAPSE values were significantly lower than the TAVI group ( $p<0.001$ ). The effect of intervention upon RV mass at 6 months was comparable between the two groups ( $p=0.259$ ).

#### Late Gadolinium Enhancement

LGE imaging was performed in all but three TAVI patients, in whom renal impairment was prohibitive. For the TAVI group, 26(49%) had mid-wall/patchy LV fibrosis and 8(15%) prior myocardial infarction prior to intervention. Only 3(6%) patients had RV hyper-enhancement at baseline with no new cases seen at 6 months. For the SAVR group, 18(32%) had midwall/patchy LV fibrosis and 7(13%) had evidence of previous myocardial infarction. Only two SAVR patients had RV fibrosis at baseline and 4 (7%) had new hyper-enhancement at 6 months. No change in total quantity of scar (% LV myocardium) was seen following SAVR (2.4 vs 2.3%,  $p=0.759$ ) or TAVI (3.1 vs 3.6%,  $p=0.795$ ). In the subgroup of SAVR patients without baseline LV scar ( $n=31(55\%)$ ), no significant change was seen in RVEF post-operatively ( $56.9 \pm 7.8\%$  vs.  $53.0 \pm 8.8\%$ ,  $p=0.071$ ).

#### Demographic and procedural risk factors associated with RV functional decline

Table 3 shows the results of univariate regression analyses of clinical and CMR variables associated with change in RV indices. Surgical cross clamp time statistically was the only factor significantly associated with an increase in RVESV index in the SAVR group at follow up.

#### Predictors of Mortality following Intervention

Over a maximum 6.3yrs follow up (median 2.8yrs); there were 19 deaths (all-cause mortality) out of the 112 patients that completed 6 month follow-up imaging. Of the 56 TAVI patients, 18 (32%) died compared to only 1 (1.7%) from the SAVR group ( $p=0.001$ ). For the TAVI group, bivariate regression analysis was performed to assess for an association between CMR measures of RV function (as listed in Table 2) and total mortality. The only independently associated measure with survival post-TAVI was RV mass at 6 months (Hazard ratio 1.359, 95% CI 1.108-1.666,  $p$  Value 0.003).

#### Measurement variability

Our variability in RV measurements was comparable to published values(8) (Table 4).

### **DISCUSSION**

This prospective multicentre study, designed specifically to use CMR for the assessment of RV function in patients with symptomatic severe aortic stenosis, has shown that SAVR resulted in deterioration in RV systolic volumes and function, which was associated with longer surgical cross-clamp times. In contrast, RV volumes and systolic function were unaltered following TAVI.

The prognostic importance of the right ventricle and its contribution to exercise capacity in a number of cardiac conditions is well recognised(11). Recently it has been demonstrated that RV dysfunction is independently associated with late survival after left heart valve surgery(12). There have been inconsistent findings from studies assessing RV function post-TAVI, in part due to the variety of echocardiographic definitions for systolic function being used(9, 10, 13). Following SAVR however, an early decline in RV ejection fraction appears ubiquitous(6). TAPSE has been the principal measurement studied in this context. However, TAPSE assessment maybe insensitive to global RV performance and is confounded by paradoxical interventricular septal motion, and particularly following SAVR, thoracic wall pericardial adhesions. Furthermore, TAPSE is an insensitive marker of RVEF unless it falls below 35%(14). Even 3D echo can systematically underestimate RV volumes(15), such that CMR is considered the reference investigation for RV morphological and functional assessment.

We have demonstrated using CMR that there is no change in RV volumes or ejection fraction at 6 months following TAVI. SAVR on the other hand is associated with a significant increase in RV end systolic volume, preserved end diastolic volume and overall reduction in ejection fraction. Consistent with this observation was a significant reduction in TAPSE.

CMR has previously been used to assess RV function in a comparison between off-pump and on-pump techniques for CABG(4). CABG was associated with a significant reduction in RV function 6 days post-operatively which normalised by 6 months. This was independent of surgical technique and thus not compounded by the use of a cross-clamp or cardiopulmonary bypass. The early decline was due to a decrease in the RV end diastolic volume, with the indexed RV end systolic volume remaining unchanged. Our surgical group was on average ten years older than the previously studied CABG cohort with a larger baseline RVESVi. Furthermore, there was no change in indexed LV volumes or mass seen in the CABG studies(4). This is very different to the reverse remodelling seen post-SAVR(2) and together suggests our SAVR cohort and the CABG group are not directly comparable.

Our study has uniquely combined CMR volumetric RV analysis with the measurement of TAPSE as part of a comprehensive assessment of systolic function. TAPSE measurement disregards RV dimensions and is less sensitive to subtle RV changes(14). This is an important limitation to relying on TAPSE alone to assess treatment response. Our observed combined reduction in both TAPSE and volumetric ejection fraction following SAVR, and not TAVI, implies SAVR confers a genuine functional decline in RV systolic function, and not merely a geometric change post-operatively, such as that described following mitral valve surgery(11). Our findings allow us to consider further the pathophysiology of RV deterioration observed following SAVR which remains poorly understood. In our study, LA emptying fraction did not change, mitral regurgitation decreased and LV ejection fraction improved 6 months following SAVR. These findings strongly suggest the pathophysiology of RV systolic decline post SAVR is independent of left heart function.

Our study indicates the increase in RVESV following SAVR is statistically significantly associated with longer aortic cross clamp times at surgery. This is a new, previously

undescribed observation. Prolonged cardiopulmonary bypass time is associated with increased mortality and morbidity(16). Longer cross-clamp times are associated with a greater risk of myocardial ischaemia(17) and raised biomarkers of myocardial damage(18).

Tissue characterisation is a pivotal and unique strength of CMR. However, the thin RV wall, susceptibility to artefact and close association with pericardial fat are all limitations to LGE assessment. Nonetheless, we detected new infarction in only 7% of patients. This is, to our knowledge, the first study to utilise LGE in the assessment of RV response to surgery. Our findings suggest the decline in RV function we observed following SAVR is not fully explicable by suboptimal RV protection during cardiopulmonary bypass. The lack of association with bypass grafting at the time of surgery is also consistent with a process unrelated to epicardial coronary disease.

It is noteworthy that RV dysfunction post operatively is an adverse prognostic marker(5) and in a small study, patients without LV LGE had no 30-day MACCE events and no deaths up to 2 years following SAVR(19). In our patients without baseline hyperenhancement, SAVR was not associated with a change in RV ejection fraction at 6 months. Further work is needed to investigate the potential role of CMR in risk stratifying patients that are potentially most susceptible to RV deterioration following aortic valve surgery.

Incision of the pericardium has been suggested as the principal factor responsible for RV deterioration post-cardiac surgery(20). A significant decline in RV systolic tissue Doppler velocity occurs within minutes and is sustained, possibly through alterations in pericardial constraint and subsequently RV geometry(21). Our findings of RV preservation following TAVI which obviates any pericardial insult, supports this hypothesis. Alternatively, a reduction in myocardial strain of the right atrium may confer a reduction in RV inlet long-axis systolic function post-SAVR (10). In experimental canine models, selective RA ischemic injury increases RV free wall dyskinesia (22). The significant decline in RA emptying fraction following SAVR, and not TAVI, is likely a sequela to traumatic surgical venous cannulation and may contribute to the RV systolic decline observed at 6 months.

Previous studies demonstrating an early decline in RV function following cardiac surgery have implicated an increase in pulmonary vascular resistance(4). It is conceivable that such an increase in afterload could mediate RV dysfunction through end systolic cavity dilatation. The use of mechanical ventilation, anaesthesia and pro-inflammatory cytokines have all been implicated(23). However, pulmonary vascular resistance is thought to normalize soon after surgery and thus unlikely to fully explain our findings at 6 months(24). Furthermore, cross-clamping of the thoracic aorta significantly increases mean pulmonary arterial and pulmonary capillary wedge pressures(25). Canine models suggest this is mediated through blood volume redistribution and increased afterload(26). Such an afterload mismatch may contribute to the increased RVESV observed at 6 months and underscore the influence of aortic cross-clamp time at surgery.

The finding that RV mass at 6 months following TAVI is independently associated with all-cause mortality is novel and most likely a reflection of worse outcome in those with right-sided pressure overload from more significant underlying pulmonary disease, despite the correction of aortic stenosis. Further work in a larger population is needed to clarify the prognostic value of CMR RV mass quantification in this context.

### **Study Limitations**

Our study is not randomised and baseline differences in demographics between our study groups are unavoidable due to current TAVI implantation guidelines.

The higher mortality and pacing rates in the TAVI group may have a confounding effect, potentially excluding patients with worse cardiac function from the analysis. However, there was statistically no difference in the STS score between the included 56 TAVI patients and those that withdrew/died (n=18) ( $5.54\pm 3.4\%$  vs.  $5.28\pm 3.82\%$ ,  $p=0.791$ ). Furthermore, RV function at baseline as assessed by CMR, was also equivalent between these two groups.

Our study did not include patients undergoing trans-apical TAVI or sutureless AVR. We have not assessed the impact of tricuspid regurgitation (TR) quantified by CMR upon the changes in RV function seen. Based on qualitative echocardiography (grading TR as none, mild,

moderate or severe), no significant change in degree of tricuspid regurgitation was seen following TAVI (average interval of 5 months,  $p=0.144$ ) nor SAVR (average interval of 6 months,  $p=0.819$ ). We can infer from this that deterioration in RV systolic function is not likely to have been related to post-operative tricuspid regurgitation. Furthermore, RV dysfunction, and not significant TR, seems independently associated with survival late following a left heart valve operation(12).

## **CONCLUSION**

SAVR, but not TAVI, resulted in RV dysfunction that was associated with longer aortic cross clamp times. Further work is needed to determine whether reduction in cross clamp times can preserve RV function following SAVR, and whether TAVI may be the preferable intervention in patients with pre-existing RV dysfunction. Assessment of both left and right ventricular function by CMR may be clinically important when making treatment decisions for high-risk patients with severe aortic stenosis.

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## **COMPETING INTERESTS**

DB is a proctor for the Medtronic CoreValve and Boston Scientific Lotus valve.

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Table 1. Patient characteristics and baseline echocardiographic data

<b>Characteristics</b>	<b>SAVR (n=56)</b>	<b>TAVI (n=56)</b>	<b>p Value*</b>
<b>Age</b>	72.8 ± 7.2	80.4 ± 6.6	< 0.001
<b>Male gender, n (%)</b>	38 (72%)	32 (57%)	0.12
<b>EuroSCORE II (%)</b>	1.51 ± 0.91	5.84 ± 5.10	< 0.001
<b>STS Mortality (%)</b>	2.13 ± 0.73	5.54 ± 3.41	< 0.001
<b>BMI (kgm<sup>-2</sup>)</b>	27.6 ± 4.71	27.6 ± 3.81	0.98
<b>Previous MI, n (%)</b>	7 (13)	11 (20)	0.31
<b>Previous PCI, n (%)</b>	2 (4)	14 (25)	0.001
<b>Previous CABG, n (%)</b>	0 (0)	16 (29)	< 0.001
<b>Stroke/TIA, n (%)</b>	8 (14)	10 (18)	0.61
<b>Peripheral vascular disease, n (%)</b>	2 (4)	13 (23)	0.002
<b>Diabetes Mellitus, n (%)</b>	11 (21)	11 (20)	0.89
<b>Hyperlipidaemia, n (%)</b>	32 (60)	35 (63)	0.82
<b>COPD, n (%)</b>	4 (8)	13 (23)	< 0.001
<b>Atrial Fibrillation, n (%)</b>	4 (8)	14 (25)	0.018
<b>eGFR (ml/min/1.73m<sup>2</sup>)</b>	72.7 ± 13.5	63.7 ± 18.9	0.01
<b>AVA (cm<sup>2</sup>)</b>	0.82 ± 0.4	0.60 ± 0.2	0.002
<b>Mean aortic valve PG (mmHg)</b>	46 ± 13	52 ± 18	0.07
<b>Pulmonary Hypertension**, n (%)</b>	8 (14)	16 (29)	0.05
<b>Moderate (31-55 mmHg), n (%)</b>	6 (11)	10 (18)	
<b>Severe (&gt;55 mmHg), n (%)</b>	2 (3)	6 (11)	
<b>ValvuloArterial Impedance (Z<sub>va</sub>)</b>	3.86 ± 1.0	3.76 ± 1.4	0.70

Values are mean±SD or n (%). \*p Value for comparison between TAVI and SAVR groups.

Z<sub>va</sub>, valvuloarterial impedance (systolic arterial pressure + mean transvalvular gradient / stroke volume index). \*\*Pulmonary hypertension defined as estimated pulmonary artery systolic pressure by transthoracic echocardiography to be >35mmHg. BMI: body mass index, COPD: chronic obstructive pulmonary disease, eGFR: estimated glomerular filtration rate.

Table 2. Preoperative baseline measurements and postoperative changes in the two procedural groups.

	SAVR		TAVI		p Value <sup>†</sup>
	Baseline	6 months	Baseline	6 months	
<b>Haemodynamics</b>					
<b>Heart Rate (bpm)</b>	64±11	65±12	66±11	67±14	0.950
<b>Systolic BP (mmHg)</b>	131±22	132±20	127±27	134±25	0.316
<b>Diastolic BP (mmHg)</b>	73±10	71±11	64±10	65±10	0.257
<b>Valves</b>					
<b>AV peak PG (mmHg)<sup>††</sup></b>	56±19	29±13 <sup>***</sup>	53±15	23±11 <sup>***</sup>	0.485
<b>AR fraction (%)</b>	19±17	10±10 <sup>**</sup>	17±12	8±7 <sup>***</sup>	0.932
<b>MR fraction (%)</b>	13±14	6±9 <sup>**</sup>	26±17	16±19 <sup>**</sup>	0.445
<b>Left Ventricle</b>					
<b>EDVI (ml/m<sup>2</sup>)</b>	90±26	74±13 <sup>***</sup>	96±23	87±20 <sup>**</sup>	0.09
<b>ESVI (ml/m<sup>2</sup>)</b>	43±22	31±9 <sup>***</sup>	48±24	42±17 <sup>**</sup>	0.06
<b>LVEF (%)</b>	54±11	58±8 <sup>**</sup>	52±13	53±11	0.098
<b>Mass Index (g/m<sup>2</sup>)</b>	77±24	61±16 <sup>***</sup>	80±20	67±18 <sup>***</sup>	0.694
<b>LVM/LVEDV (g/ml)</b>	0.88±0.2	0.85±0.2	0.86±0.2	0.79±0.2 <sup>**</sup>	0.224
<b>LGE (%LV)</b>	2.4±3.0	2.3±3.8	3.1±3.1	3.6±4.2	0.466
<b>Right Ventricle</b>					
<b>EDVI (ml/m<sup>2</sup>)</b>	78±17	78±16	80±18	77±19	0.334
<b>ESVI (ml/m<sup>2</sup>)</b>	33±10	37±10 <sup>**</sup>	40±15	38±14	0.005
<b>RVEF (%)</b>	58±8	53±9 <sup>**</sup>	52±10	52±10	0.013
<b>Mass Index (g/m<sup>2</sup>)</b>	15±4	15±4	15±5	13±4 <sup>**</sup>	0.259
<b>RVM/RVEDV (g/ml)</b>	0.21±0.07	0.20±0.07	0.20±0.08	0.18±0.06 <sup>**</sup>	0.499
<b>TAPSE (mm)</b>	22±5	14±3 <sup>***</sup>	19±6	19±7	0.001

Paired t test vs baseline: \*p<0.05, \*\*p<0.01, \*\*\*p<0.001. † Independent samples t-test to compare degree of change seen following SAVR with that seen following TAVI. †† Derived from MRI assessment.

Table 3. Univariate regression analysis of clinical and CMR variables for the identification of factors associated with change in RV volume/function indices

Variables	Univariate analysis			
	B Coefficient±SE	R <sup>2</sup>	95% CI	p Value
<b>RVESVI (ml/m<sup>2</sup>)</b>				
<b>Concomitant CABG</b>	-3.86±2.87	0.035	-9.6 to 1.88	0.185
<b>Bypass time</b>	0.05±0.03	0.066	-0.01 to 0.11	0.059
<b>Cross Clamp time</b>	0.07±0.03	0.088	0.01 to 0.13	0.028
<b>Mechanical SAVR</b>	5.93±3.63	0.048	-1.33 to 13.19	0.108
<b>Pulmonary Hypertension</b>	-3.31±3.28	0.019	-9.87 to 3.25	0.318
<b>COPD</b>	2.80±4.73	0.006	-6.66 to 12.26	0.556
<b>RVEF (%)</b>				
<b>Concomitant CABG</b>	5.83±3.39	0.056	-0.95 to 12.61	0.091
<b>Bypass time</b>	-0.03±0.03	0.014	-0.09 to 0.03	0.387
<b>Cross Clamp time</b>	-0.05±0.04	0.028	-0.13 to 0.04	0.221
<b>Mechanical SAVR</b>	-5.08 ± 4.36	0.025	2.94 to -13.8	0.249
<b>Pulmonary Hypertension</b>	-4.52±3.55	0.030	-11.62 to 2.58	0.209
<b>COPD</b>	-4.08±5.75	0.010	-15.58 to 7.42	0.481
<b>TAPSE (mm)</b>				
<b>Concomitant CABG</b>	0.61±1.74	0.003	-2.87 to 4.09	0.729
<b>Bypass time</b>	0.01±0.02	0.007	-0.03 to 0.05	0.610
<b>Cross Clamp time</b>	0.01±0.02	0.009	-0.03 to 0.05	0.560
<b>Mechanical SAVR</b>	-3.28±2.25	0.054	-7.78 to 1.17	0.154
<b>Pulmonary Hypertension</b>	0.23±2.37	0.000	-4.51 to 4.97	0.924
<b>COPD</b>	2.20±2.81	0.017	-3.42 to 7.82	0.438

Each parameter had a separate regression analysis performed.

Table 4. Observer variability of right ventricular quantification

<b>RV parameter</b>	<b>Intra-observer variability</b>	<b>Inter-observer variability</b>
<b>RV End Diastolic Volume</b>	3.9%	4.7%
<b>RV End Systolic Volume</b>	4.8%	8.8%
<b>RV Mass</b>	7.4%	8.8%
<b>TAPSE</b>	4.3%	8.9%

## Figure Titles and Legends

### Figure 1.

Title: Method for calculation of TAPSE

Legend: TAPSE was measured as the maximum apical displacement of the lateral tricuspid valve annulus from end-diastole to end-systole.

TAPSE, tricuspid annulus systolic plane excursion

### Figure 2.

Title: Study profile

Legend: A flow diagram demonstrating patient recruitment with reasons for non-completion of study protocol.

TAVI, transcatheter aortic valve implantation.

SAVR, surgical aortic valve replacement