

# Early echocardiographic evaluation following percutaneous implantation with the self-expanding CoreValve ReValving System aortic valve bioprosthesis

Peter-P.Th De Jaegere<sup>1\*</sup>, MD, PhD; Nicolo Piazza<sup>1</sup>, MD, FRCPC; Tjebbe W. Galema<sup>1</sup>, MD; Amber Otten<sup>1</sup>, MD; Osama I. Soliman<sup>1</sup>, MD; Bas M. Van Dalen<sup>1</sup>, MD; Marcel L. Geleijnse<sup>1</sup>, MD; Arie-Pieter Kappetein<sup>2</sup>, MD; Hector M. Garcia Garcia<sup>1</sup>, MD, MSc; Gerrit-Anne Van Es<sup>3</sup>, PhD; Patrick W. Serruys<sup>1</sup>, MD, PhD

1. Department of Cardiology, Thoraxcenter, Erasmus Medical Center, Rotterdam, The Netherlands; 2. Department of Cardiovascular Surgery, Thoraxcenter, Erasmus Medical Center, Rotterdam, The Netherlands; 3. Cardialysis, Corelab, Rotterdam, The Netherlands

Peter de Jaegere is a physician proctor for CoreValve. Gerrit-Anne van ES is an employee of Cardialysis BV, The Netherlands.

All other authors have no conflict of interest to declare.

## KEYWORDS

Aorta, valve, stenosis, catheters, echocardiography

## Abstract

**Aims:** Although safety and feasibility studies have been published, there are few reports dedicated to the echocardiographic evaluation of patients following percutaneous aortic valve replacement (PAVR). This report describes the early echocardiographic evaluation of patients undergoing PAVR with the CoreValve Revalving System.

**Methods and results:** The population consisted of 33 consecutive patients with aortic stenosis who underwent successful PAVR. Echocardiograms were performed pre-treatment (123±110 days prior), post-treatment (6±2 days) and post-discharge (80±64 days). Aortic valve function and left ventricular dimensions, systolic and diastolic function were assessed pre- and post-implantation. The mean age was 81±7 years and the mean Logistic Euroscore was 20±12. Following PAVR, the mean transaortic valve gradient decreased (46±16 mmHg pre-treatment vs. 12±7 mmHg post-treatment vs. 9±5 mmHg post-discharge,  $p<0.001$ ) and the mean effective orifice area increased (0.75±0.23 cm<sup>2</sup> pre-treatment vs. 1.97±0.85 cm<sup>2</sup> post-treatment vs. 1.72±0.45 cm<sup>2</sup> post-discharge,  $p<0.001$ ). There was no significant change in mean ejection fraction (41±12% pre-treatment vs. 46±15% post-treatment vs. 44±13% post-discharge,  $p=0.44$ ). Approximately two-thirds of patients had no change in diastolic function at follow-up.

**Conclusion:** Following implantation, there was a sustained decrease in aortic valve gradient and increase in aortic valve area. In addition, the mean ejection fraction did not change significantly and in the majority of patients, diastolic function was unchanged.

\* Corresponding author: Department of Cardiology, Thoraxcenter, Erasmus Medical Center, Rotterdam, The Netherlands

E-mail: p.dejaegere@erasmusmc.nl

## Introduction

Transcatheter aortic valve replacement has become regarded as a viable alternative to surgical aortic valve replacement in elderly patients with severe aortic stenosis who are considered too high a risk or denied surgery. Up until now, reports on transfemoral percutaneous aortic valve replacement have consisted mainly of safety and feasibility studies demonstrating a significant improvement in haemodynamic and clinical status<sup>1-8</sup>. Procedural-related and 30-day mortality rates after PAVR is reported to be 6% and 12%, respectively, while freedom from death, myocardial infarction or stroke at 30 days ranges from 74 - 86%<sup>3,6,7</sup>.

Echocardiography is the gold standard for the evaluation of pre- and post-surgical aortic valve replacement<sup>9</sup>. Following surgical aortic valve replacement, acute and long-term improvements in aortic valve gradients, regression in left ventricular mass and improvement in left ventricular diastolic function is reported<sup>11-21</sup>. There are, however, few reports dedicated on the echocardiographic evaluation post PAVR<sup>22</sup>.

In this study, we report on the early echocardiographic evaluation of patients undergoing PAVR with the CoreValve Revalving System (CRS) derived from an independent core laboratory (Cardialysis, Rotterdam, The Netherlands).

## Methods

### Patients

Thirty-three consecutive patients undergoing implantation with the CRS between November 2005 and December 2007 at the Thoraxcenter, Rotterdam were included in the analysis. Patients referred for the procedure were deemed either too high or prohibitive risk for surgical aortic valve replacement. Patients were treated in the framework of the CoreValve safety and feasibility protocols (COR 2005, COR 2006-02) and the CE post marketing surveillance registry.

The inclusion and exclusion criteria have been described elsewhere<sup>7</sup>. Briefly, patients were included if they had (1) severe native aortic valve stenosis with an area <1 cm<sup>2</sup> or <0.6 cm<sup>2</sup>/m<sup>2</sup> with or without aortic regurgitation; (2) aortic valve annulus diameter ≥ 20 mm and ≤ 27 mm and (3) sinotubular junction ≤ 43 mm measured by echocardiography.

Patients provided written consent after a consensus was achieved between a cardiologist and a cardiac surgeon that surgical aortic valve replacement was associated with either too high or prohibitive risk.

### Device description and procedure

Descriptions of the device and technical aspects of the procedure have been previously published<sup>23</sup>. The CoreValve aortic valve prosthesis consists of a self-expanding nitinol tri-level frame to which a trileaflet bioprosthetic porcine pericardial tissue valve is mounted and sutured.

The initial two procedures were performed under femoral-femoral circulatory support and a total of nine procedures were performed using the Tandem Heart (left atrial-to-femoral artery bypass system). The remaining 22 patients underwent a completely

percutaneous procedure with echo-assisted vascular access and percutaneous closure with a 10 Fr Prostar XL device<sup>23</sup>. Prior to the implantation of the prosthesis, percutaneous aortic balloon valvuloplasty was performed using rapid ventricular pacing. Device positioning and deployment was performed under fluoroscopic guidance only. Five patients (15%) were implanted with the 2<sup>nd</sup> generation 21 Fr catheter system and 28 patients (85%) were implanted with the 3<sup>rd</sup> generation 18 Fr catheter system. Twenty-six patients (79%) were implanted with the 26-mm inflow prosthesis and when it became later available, seven patients (21%) were implanted with the 29-mm inflow prosthesis. A total of nine patients had moderate-severe aortic regurgitation immediately after implantation of the prosthesis. As a result, five of these patients underwent re-dilatation with balloon valvuloplasty and the remaining four patients received a 2<sup>nd</sup> CRS during the index procedure.

### Echocardiographic assessment

A standard 2-D transthoracic echocardiogram (Philips Ie33 or Sonos 7500, Philips, Best, The Netherlands) was analysed pretreatment, post-treatment and early post-discharge. Two independent cardiologists reviewed the echocardiograms.

Echocardiographic studies were performed in a standard fashion. Quantification of left ventricular end-systolic and end-diastolic volumes and ejection fraction was performed using the biplane Simpson's method. Left ventricular end-systolic and end-diastolic dimensions (mm), interventricular septal and left posterior wall thickness (mm) and left atrial size (mm) measurements were obtained using the parasternal long axis view by either M-mode or 2-D echocardiography.

Diastolic function was assessed by pulsed-wave mitral inflow patterns and tissue Doppler imaging of the mitral annulus according to standard recommendations. Diastolic function was classified as normal, mild diastolic dysfunction (impaired relaxation), moderate diastolic dysfunction (pseudo-normal) and severe diastolic dysfunction<sup>24</sup>.

The diameters of the left ventricular outflow tract and aortic annulus were obtained by standard 2-D echocardiographic calliper measurement from the parasternal long axis view. Velocity-time integral (cm), peak aortic velocity (cm/sec), peak instantaneous gradient (mmHg) and mean transaortic gradient (mmHg) were measured. By using the modified Bernoulli equation ( $dP=4v^2$ ), a peak instantaneous valve gradient (mmHg) was derived from the continuous-wave Doppler velocity across the aortic valve. The effective orifice aortic valve area (cm<sup>2</sup>) was estimated by the continuity equation.

Colour-flow Doppler and continuous wave Doppler signal was used to quantitate aortic regurgitation<sup>9,10</sup>. Post-procedural aortic regurgitation was further classified as either central or paravalvular in origin. Mitral regurgitation was quantified using colour and continuous wave Doppler flow. Central valvular insufficiency was graded as none, mild, moderate or severe<sup>9,10</sup>.

Left ventricular mass index (g/m<sup>2</sup>) was estimated using the formula proposed by Devereux and colleagues:  $0.8 (1.04 ((LVIDD + PWTD + IVSTD)^3 - [LVIDD]^3)) + 0,6 g$  (where, LVIDD=left ventricular internal diameter, PWTD=posterior wall thickness at end-diastole, IVSTD=interventricular septal thickness at end-diastole)<sup>25</sup>.

## Statistical analysis

Categorical variables are presented as frequencies and continuous variables are presented as mean±standard deviation. A one-way ANOVA test was used for comparison between groups. A *p* value of < 0.05 was considered statistically significant.

## Results

The baseline patient characteristics are summarised in Table 1. Successful valve implantation was achieved in all patients. One patient died six days after PAVR as a consequence of procedural-related cardiac tamponade. Another patient died at 51 days related to sepsis. The completeness of the echocardiographic follow-up is shown in Figure 1. Patients with missing echocardiograms were all alive. The reason for missing echocardiograms included one of the following: (1) poor acoustic windows rendering the echocardiogram not suitable for interpretation; (2) follow-up care was reassigned to the referring hospital; or (3) the patient had not reached the follow-up time point.

**Table 1. Baseline characteristics (n=33).**

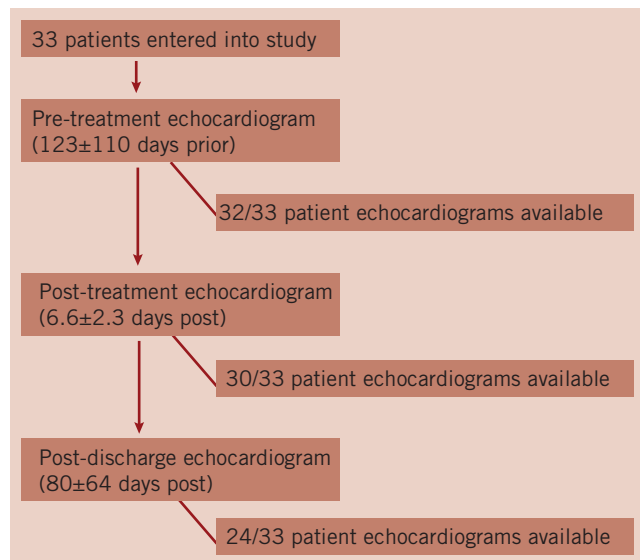
Age (yrs), mean±SD	81±7
Male, n (%)	17 (52%)
AMI, n (%)	7 (21%)
PCI, n (%)	7 (21%)
CABG, n (%)	10 (30%)
COPD, n (%)	6 (20%)
Diabetes, n (%)	8 (24%)
GFR (ml/min), mean (SD)	55 (21)
Logistic EuroScore, mean (SD)	20.3±12

AMI: acute myocardial infarction; CABG: coronary artery bypass; COPD: chronic obstructive pulmonary disease; GFR: glomerular filtration rate; PCI: percutaneous coronary intervention

**Table 2. Mean echocardiographic indices before and after percutaneous aortic valve replacement.**

Measurement	Pre-treatment (n=32)	Post-treatment (n=30)	Follow-up (n=24)	<i>p</i> value
Left atrium and ventricle				
LV end-diastolic dimension (mm)	51±8	49±9	49±7	0.61
LV end-systolic dimension (mm)	37±13	37±11	38±10	0.95
Septal thickness (mm)	14±3	13±2	13±2	0.2
LV posterior wall thickness (mm)	12±2	12±2	11±2	0.36
Left atrial dimension (mm)	48±9	45±9	44±9	0.32
LV mass indexed (g/m <sup>2</sup> )	151±44	148±44	128±37	0.14
LV ejection fraction - Biplane (%)	41±12	46±15	44±13	0.44
Valvular function				
Peak AV gradient (mmHg)	77±28	20±12	18±11	<0.001
Mean AV gradient (mmHg)	46±16	12±7	9±5	<0.001
Aortic annular dimension (mm)	22±3	N/A	N/A	N/A
LV outflow tract dimension (mm)	21±2	N/A	N/A	N/A
AVA (cm <sup>2</sup> )	0.75±0.023	1.97±0.85	1.72±0.45	<0.001
AVA - indexed (cm <sup>2</sup> /m <sup>2</sup> )	0.43±0.014	1.11±0.55	0.98±.25	<0.001
Aortic regurgitation grade (1-4)	1.8	1.7	1.8	0.81
Mitral regurgitation grade (1-4)	2.1	2.1	2.1	0.91

LV: left ventricle; AV: aortic valve; AVA: aortic valve area



**Figure 1. Flow diagram depicting time of follow-up and number of available echocardiograms at each time period.**

## Pre-treatment echocardiographic evaluation

Pretreatment echocardiographic analysis was available in 32 out of the 33 patients (Figure 1). The echo study was performed at a mean of 123±110 days prior to PAVR. The findings are summarised in Table 2.

The left ventricular end-diastolic and end-systolic dimensions were above the normal range (> 56 mm and > 40 mm, respectively) in 34% and 39% of patients, respectively. The septal thickness and left ventricular posterior wall thickness was above the normal range (> 12 mm) in 68% and 65% of patients, respectively. No patient had an interventricular septum-to-posterior wall thickness ratio greater than 1.3, therefore excluding asymmetric hypertrophic cardiomyopathy.

All patients had a pre-treatment aortic valve area index  $\leq 0.6 \text{ cm}^2/\text{m}^2$  (mean $\pm$ SD,  $0.43\pm 0.14 \text{ cm}^2/\text{m}^2$ ). Five patients had an ejection fraction  $< 30\%$ .

The frequencies of aortic and mitral regurgitation are depicted in figure 2 and 3, respectively. Classification of diastolic function was feasible in 27/32 patients and is summarised in Figure 4. The mean left ventricular mass index was increased in 84% of patients (normal range, male 49-115  $\text{g}/\text{m}^2$  and female 43-95  $\text{g}/\text{m}^2$ ).

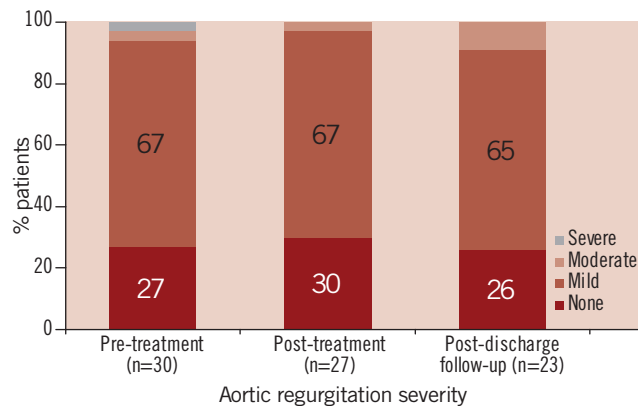


Figure 2. Severity of aortic regurgitation during serial echocardiographic evaluation (n=number of patient echocardiograms with sufficient information for assessment of aortic regurgitation).

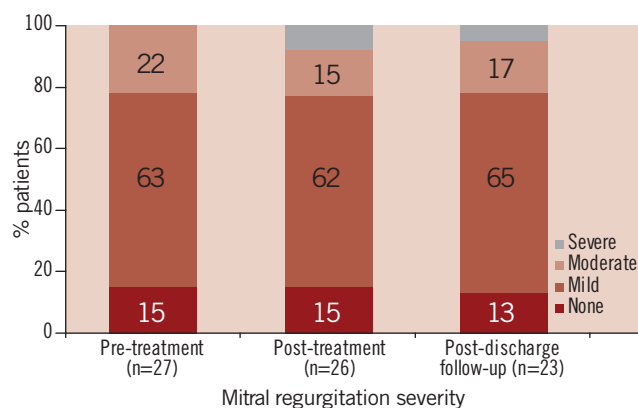


Figure 3. Severity of mitral regurgitation during serial echocardiographic evaluation (n=number of patient echocardiograms with sufficient information for assessment of mitral regurgitation).

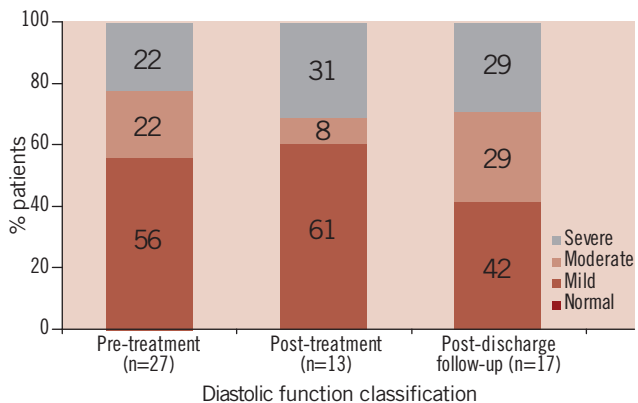


Figure 4. Classification of diastolic function during serial echocardiographic evaluation (n=number of patient echocardiograms with sufficient information for assessment of diastolic function).

## Post-treatment echocardiographic evaluation

Echocardiographic evaluation was performed at a mean of  $6\pm 2$  days following PAVR and was available in 30 out of the 33 patients (Table 2).

There was an immediate reduction in mean transaortic valve gradient from  $46\pm 16$  to  $12\pm 7$  mmHg and an increase in the estimated mean effective aortic orifice area from  $0.75\pm 0.23$  to  $1.97\pm 0.85 \text{ cm}^2$  (Figure 6 and 7).

The severity of aortic regurgitation post-treatment is shown in Table 2 and Figure 2. Figure 5 provides individual patient information regarding the change in aortic regurgitation. After PAVR, 23% of the patients had an improvement in the degree of aortic regurgitation 19% had a worsening. Aortic regurgitation was paravalvular in all patients except two in whom there was concomitant central aortic regurgitation. All patients who required either a re-dilatation with balloon valvuloplasty (n=5) or a valve-in-valve implantation (n=4) had mild paravalvular aortic regurgitation post-treatment.

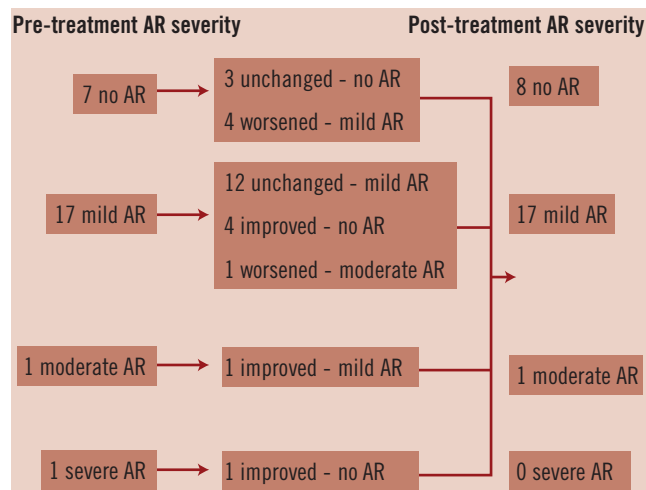


Figure 5. Descriptive change in aortic regurgitation grade from baseline to discharge (n=26 patients with both pre-treatment and post-treatment aortic regurgitation severity available for comparison).

The severity of mitral regurgitation post-treatment is shown in Table 2. It remained unchanged in 65%, improved in 12%, and worsened in 23% of the patients (Figure 3). In those patients who worsened, two had mild, two had moderate and one had severe mitral regurgitation.

After implantation of the prosthesis, the mean ejection fraction remained unchanged (Table 2).

Classification of diastolic function was possible in 13 patients (Figure 4). Of the 12 patients with pre- and post-treatment evaluation of diastolic function, there was no change in diastolic function in eight patients (66%) (Figure 8).

## Post-discharge echocardiographic evaluation

A post-discharge echocardiogram was available in 24/33 patients (73%) and was performed at a mean of  $80\pm 64$  days post PAVR (Table 2).

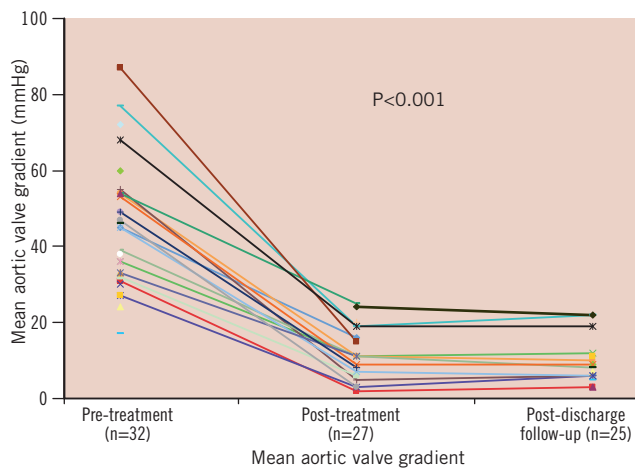


Figure 6. Mean aortic valve gradient (mmHg) during serial echocardiographic evaluation ( $n$ =number of patient echocardiograms with sufficient information for assessment of mean aortic valve gradient).

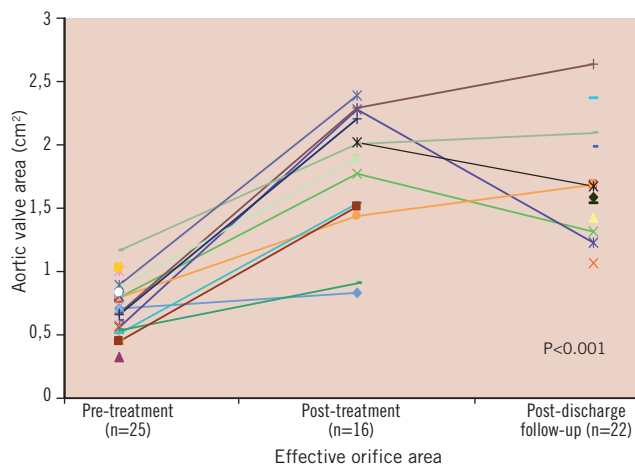


Figure 7. Effective orifice valve area ( $\text{cm}^2$ ) during serial echocardiographic evaluation ( $n$ =number of patient echocardiograms with sufficient information for assessment of effective orifice valve area).

The reduction in the mean aortic valve gradient was maintained at follow-up ( $9\pm 5$  mmHg) (Figure 6) as well as the increase in the mean effective aortic orifice area ( $1.72\pm 0.45$   $\text{cm}^2$ ) (Figure 7).

The mean aortic regurgitation grade did not change at post-discharge follow-up (Table 2). On an individual basis, aortic regurgitation did not change in 37% of the patients, improved by 1 grade in 37% and worsened by 1 grade in another 36%. No patient had severe aortic regurgitation (Figure 2). In all patients, aortic regurgitation was paravalvular.

The degree of mitral regurgitation during follow-up did not change in 44% of the patients, it improved in 33% improved and worsened in 23%.

There was no significant change in ejection fraction (Table 2).

Classification of diastolic function at follow-up is shown in Figure 4. In the majority of patients (69%), diastolic function did not change compared to baseline classification (Figure 8).

There was no statistically significant change in left ventricular mass index at a mean of  $80\pm 64$  days following PAVR ( $151\pm 44$   $\text{g}/\text{m}^2$  pre-treatment vs.  $128\pm 37$   $\text{g}/\text{m}^2$  post-discharge,  $p=0.14$ ).

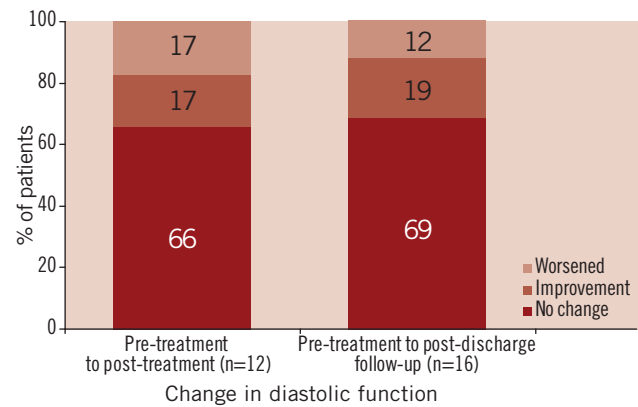


Figure 8. Intra-individual change in diastolic function during serial echocardiographic evaluation from pre-treatment to post-treatment and from pre-treatment to post-discharge follow-up ( $n$ =number of patient echocardiograms with sufficient information for assessment and comparison of diastolic function).

## Discussion

The results of this study demonstrate a significant decrease in aortic valve gradient and increase in valve area post PAVR. Yet, mild paravalvular aortic regurgitation after PAVR was present in the majority of patients (65%). There was no significant change in mean left ventricular ejection fraction. In addition, we found no significant change in diastolic function early after PAVR.

## Transaortic valve gradient and effective orifice aortic valve area

The changes in gradient and valve area observed in this study are consistent with the findings of others. Grube et al reported a reduction in the mean aortic gradient from 43.7 mmHg to 9.0 mmHg ( $p<0.001$ ) following the implantation of the CRS in 76 patients<sup>7</sup>. Webb et al found a reduction in the mean aortic gradient from  $46\pm 17$  mmHg to  $11\pm 5$  mmHg following the implantation of a balloon-expandable stent valve and an increase in AVA from  $0.6\pm 0.2$   $\text{cm}^2$  to  $1.7\pm 0.4$   $\text{cm}^2$ .<sup>6</sup> Cribier et al demonstrated a reduction in the mean aortic gradient from  $37\pm 13$  mmHg to  $9\pm 2$  mmHg and an increase in the mean AVA from  $0.60\pm 0.09$   $\text{cm}^2$  to  $1.7\pm 0.11$   $\text{cm}^2$ .<sup>5</sup> These results compare favourably to surgical aortic valve replacement whereby, following replacement with a bioprosthetic Carpentier-Edwards valve, the echocardiography-derived mean gradient is reported to be  $14\pm 6$  mmHg and AVA  $1.8$   $\text{cm}^2$ .<sup>26</sup>

## Paravalvular aortic regurgitation

In this study, mild paravalvular aortic regurgitation after PAVR was found in 67% of the patients. Although some had moderate to severe regurgitation immediately following PAVR, this was corrected by re-dilatation or the implantation of a second valve. These patients for whom further re-intervention was required had mild paravalvular aortic regurgitation on post-treatment follow-up. Importantly, no patient had severe regurgitation. In the study by Grube et al, re-dilatation was performed in 21 out of the 86 patients and a second valve was implanted in two patients<sup>8</sup>. Aortic regurgitation after PAVR is nearly always paravalvular and may be explained

by inadequate sizing of the valve due to inadequate annulus size measurement before PAVR or the lack of sufficient ranges of frame sizes, insufficient expansion of the frame due to the presence of calcium in the aortic root and leaflets, malpositioning of the valve (in most cases, too low), and leaflet malcoaptation which leads to central regurgitation. It must be recognised that there is currently no systematic or recommended method to adequately grade or characterise paravalvular aortic regurgitation. In addition, the results could have been different were transesophageal echocardiography had been used.

In 20-30% of patients, paravalvular aortic regurgitation improved or worsened during the short period of follow-up. The changes, however, were minimal. These observations are in parallel with those of others<sup>2-4</sup>. The changes in regurgitation may be related to the variability in repeat echo-Doppler studies and analyses, variable changes in the left ventricular outflow tract geometry after PAVR, recoil and/or further expansion of the nitinol frame. Aortic regurgitation may also be caused by dysfunction of the frame due to corrosion of the nitinol, leading to strut fracture<sup>27</sup>. This is unlikely in the present series due to the short follow-up.

### Left ventricular ejection fraction

We found no change in the left ventricular ejection fraction. These observations, in addition to being limited by the small sample size, may also be due to concomitant coronary artery disease and scar formation from old myocardial infarctions. In addition, no patient had worsening of ejection fraction of clinical relevance. Webb et al and Cribier et al found a significant increase in ejection fraction post-treatment from 53±15% to 57±13% and from 45±18% to 53±14%, respectively<sup>4,6</sup>. Not surprisingly, the improvement in left ventricular function in these studies was predominantly seen in patients who had moderate to severe left ventricular dysfunction at baseline. Similar observations have been observed after surgical aortic valve replacement<sup>10</sup>.

### Diastolic function

The majority of patients (69%) had no change in diastolic function and no patient had normalisation of diastolic function. The small sample size precludes firm conclusions. Diastolic dysfunction, however, is a determinant of poor cardiovascular outcome<sup>28</sup>. In surgical series, changes in diastolic function have been documented within two weeks following surgical aortic valve replacement<sup>29</sup>. These early changes have been attributed to a reduction in left atrial pressure but other factors, such as medication, volume status and loading conditions may also play a role. The longer-term improvements in diastolic function are likely an expression of the regression in left ventricular hypertrophy<sup>29</sup>.

### Limitations and conclusion

The present study is limited by the small sample size and short duration of follow-up. In addition, echocardiographic follow-up was not complete and the quality of certain frames precluded analysis of certain variables. Furthermore, potential differences in systolic or diastolic function could possibly exist between the patient cohorts treated with or without circulatory support, but the small sample

size would prohibit any reasonable conclusions. Short and long-term prospective echocardiographic studies are required. These studies may help to determine predictors of procedural and clinical outcomes. They may also evoke recommendations for improvements in the technology itself.

### References

1. Cribier A, Eltchaninoff H, Tron C, Bauer F, Agatiello C, Sebah L, Bash A, Nusimovici D, Litzler PY, Bessou JP, Leon M. Early experience with percutaneous transcatheter implantation of heart valve prosthesis for the treatment of end-stage inoperable patients with calcific aortic stenosis. *J Am Coll Cardiol* 2004; 43:698-703.
2. Grube E, Laborde JC, Gerckens U, Felderhoff T, Sauren B, Buellesfeld L, Mueller R, Menichelli M, Schmidt T, Zickmann B, Iversen S, Stone G. Percutaneous implantation of the CoreValve self-expanding valve prosthesis in high-risk patients with aortic valve disease -The Seigburg FIM study. *Circulation* 2006; 114:1616-24.
3. Webb J, Chandavimol M, Thompson C, Ricci D, Carere R, Munt B, Buller C, Pasupati S, Lichtenstein S. Percutaneous aortic valve implantation retrograde from the femoral artery. *Circulation* 2006; 113:842-50.
4. Cribier A, Eltchaninoff H, Tron C, Bauer F, Agatiello C, Nercolini D, Tapiero S, Litzler PV, Bessou JP, Babaliaros V. Treatment of calcific aortic stenosis with the percutaneous heart valve - midterm follow-up from the initial feasibility studies: The French Experience. *J Am Coll Cardiol* 2006; 47:1214-23.
5. Berry C, Asgar A, Lamarche Y, Marcheix B, Couture P, Basmadjian A, Ducharme A, Laborde JC, Cartier R, Bonan R. Novel therapeutic aspects of percutaneous aortic valve replacement with the 21F CoreValve Revalving System. *Cath Cardiovasc Interv* 2007; 70:610-16.
6. Webb J, Pasupati S, Humphries K, Thompson C, Altwegg L, Moss R, Sinhal A, Carere R, Munt B, Ricci D, Ye J, Cheung A, Lichtenstein S. Percutaneous transarterial aortic valve replacement in selected high-risk patients with aortic stenosis. *Circulation* 2007; 116:755-63.
7. Grube E, Schuler G, Buellesfeld L, Gerckens U, Linke A, Wenaweser P, Sauren B, Mohr FW, Walther T, Zickmann B, Iversen S, Felderhoff T, Cartier R, Bonan R. Percutaneous aortic valve replacement for severe aortic stenosis in high-risk patients using the second- and current third-generation self-expanding CoreValve prosthesis: device success and 30-day clinical outcome. *J Am Coll Cardiol*. 2007; 50:69-76.
8. Marcheix B, Lamarche Y, Berry C, Asgar A, Laborde JC, Basmadjian A, Ducharme A, Denault A, Bonan R, Cartier R. Surgical aspects of endovascular retrograde implantation of the aortic CoreValve bioprosthesis in high-risk older patients with severe symptomatic aortic stenosis. *J Thorac Cardiovasc Surg* 2007; 134:1150-56.
9. Bonow RO, Carabello BA, Kanu C, de Leon AC Jr, Faxon DP, Freed MD, Gaasch WH, Lytle BW, Nishimura RA, O'Gara PT, O'Rourke RA, Otto CM, Shah PM, Shanewise JS, Smith SC Jr, Jacobs AK, Adams CD, Anderson JL, Antman EM, Faxon DP, Fuster V, Halperin JL, Hiratzka LF, Hunt SA, Lytle BW, Nishimura R, Page RL, Riegel B. American College of Cardiology/American Heart Association Task Force on Practice Guidelines; Society of Cardiovascular Anesthesiologists; Society for the Cardiovascular Angiography and Interventions; Society of Thoracic Surgeons, ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the ACC/AHA task force on practice guidelines. *Circulation* 2006; 114:84-231.
10. Vahanian A, Baumgartner H, Bax J, Butchart E, Dion R, Filippatos G, Flachskampf F, Hall R, Jung B, Kasprzak J, Nataf P, Tornos P, Torracca L,

Wenink A. Guidelines on the management of valvular heart disease. The Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology. *Eur Heart J* 2007; 28:230-268.

11. Sharma, U, Barenburg P, Saraswati P, Dassen W, Pinto Y, Maessen JG. Systematic review of the outcome of aortic valve replacement in patients with aortic stenosis. *Ann Thorac Surg* 2004; 78:90-5.

12. Lamb H, Beyerbacht H, de Roos A, van der Laarse A, Vliegen HW, Leujes F, Bax JJ, van der Wall EE. Left ventricular remodeling early after aortic valve replacement: differential effects on diastolic function in aortic valve stenosis and aortic regurgitation. *J Am Coll Cardiol* 2002; 40:2182-88.

13. Vauri M, Shapira MR, Adler Y, Porter A, Birnbaum Y, Vered Z, Sagie A. The effect of aortic valve replacement on left ventricular mass assessed by echocardiography. *Eur J Echocardiography* 2000; 1:116-21.

14. Krayenbuehl HP, Hess OM, Schneider J, Turina M. Left ventricular function and myocardial structure in aortic valve disease before and after surgery. *Herz* 1984; 9:270-80.

15. De Paulis R, Sommariva L, Colagrande L, De Matteis GM, Fratini S, Tomai F, Bassano C, Penta de Peppo A, Chiariello L. Regression of left ventricular hypertrophy after aortic valve replacement for aortic stenosis with different valve substitutes. *J Thorac Cardiovasc Surg* 1998; 116:590-98.

16. Pibarot P, Dumesnil JG, Leblanc MH, Cartier P, Metras J. Changes in left ventricular mass and function after aortic valve replacement: a comparison between stentless and stented bioprosthetic valves. *J Am Soc Echocardiogr* 1999; 12:981-87.

17. Waszyrowski T, Kasprzak J, Krzeminska-Pakula M, Drozd J, Dziatkowiak A, J. Zaslonka. Regression of left ventricular dilatation and hypertrophy after aortic valve replacement. *Int J Cardiol* 1996; 57:217-25.

18. Henry WL, Bonow RO, Borer JS, Kent KM, Ware JH, Redwood DR, Itscoitz SB, McIntosh CL, Morrow AG, Epstein SE. Evaluation of aortic valve replacement in patients with valvular aortic stenosis. *Circulation* 1980; 61:814-25.

19. Gelsomino S, Frassani R, Morocutti G, Nucifora R, Da Col P, Minen G. Time course of left ventricular remodeling after stentless aortic valve replacement. *Am Heart J* 2001; 142:556-62.

20. Monrad ES, Hess OM, Murakami T, Nonogi H, Corin WJ, Krayenbuehl HP. Time course of regression of left ventricular hypertrophy after aortic valve replacement. *Circulation* 1988; 77:1345-55.

21. Robiolio PA, Rigolin VA, Hearne SE, Baker WA, Kisslo KB, Pierce CH. Left ventricular performance improves late after aortic valve replacement in patients with aortic stenosis and reduced ejection fraction. *Am J Cardiol* 1995; 76; 612-15.

22. Moss RR, Ivens E, Pasupati S, Humphries K, Thompson CR, Munt B, Sinhal A, Webb JG. Role of echocardiography in percutaneous aortic valve implantation. *J Am Coll Cardiol Img* 2008; 1:15-24

23. de Jaegere P, van Dijk LC, Laborde JC, Sianos G, Ramos FJO, Lighthart J, Kappeteijn AP, van der Ent M, Serruys PW. True percutaneous implantation of the CoreValve aortic valve prosthesis by combined use of ultrasound guided vascular access, Prostar XL, and the Tandem Heart. *Eurointerv* 2007; 2:500-05.

24. Khouri SJ, Maly GT, Suh DD, Walsh TE. A practical approach to the echocardiographic evaluation of diastolic function. *J Am Soc Echocardiogr* 2004; 17:290-97.

25. Devereux RB, Alonso DR, Lutas EM., Gottlieb GJ, Campo E, Sachs I, Reichel N. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol* 1986; 57: 450-58.

26. Jamieson WR, Munro AI, Miyagishima RT, Allen P, Burr LH, Tyers GF. Carpentier-Edwards standard porcine bioprosthesis: clinical performance to seventeen years. *Ann Thorac Surg* 1995; 60:999-1006.

27. Heintz C, Riepe G, Birken L, Kaiser E, Chakfe N, Morlock M, Delling G, Imig H. Corroded nitinol wires in explanted aortic endografts: an important mechanism of failure? *J Endovasc Ther* 2001; 8:248-53.

28. Lund O, Flot C, Jensen FT, Emmertsen K, Nielsen T, Rasmussen BS, Hansen OK, Pilegarrd HK, Kristensen. Left ventricular systolic and diastolic function in aortic stenosis. *Eur Heart J* 1997; 18:1977-1987.

29. Ikonomidis I, Tsoukas A, Parthenakis F, Gournizakis A, Kassimatis A, Rallidis L, and Nihoyannopoulos P. Four-year follow-up of aortic valve replacement for isolated aortic stenosis: a link between reduction in pressure overload, regression of left ventricular hypertrophy, and diastolic function. *Heart* 2001; 86:309-16.