

# Impact of a New Conduction Defect After Transcatheter Aortic Valve Implantation on Left Ventricular Function

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**Objectives** This study sought to evaluate the impact of new conduction defects after transcatheter aortic valve implantation (TAVI) on the evolution of left ventricular (LV) function during 1-year follow-up.

**Background** New left bundle branch block (LBBB) or need for permanent pacing due to atrioventricular (AV) block are frequent after TAVI.

**Methods** A total of 90 consecutive patients treated with TAVI and who had 12-month echocardiographic follow-up were included in the study. In 39 patients, a new conduction defect (new LBBB or need for permanent pacemaker activity) persisted 1 month after TAVI. In 51 patients, no persistent new conduction defect was observed. Two-dimensional echocardiography using parasternal short-axis, apical 4-chamber, and apical 2-chamber views was performed before TAVI and at 1-year follow-up to determine LV volumes and ejection fraction based on Simpson's rule. Speckle-tracking echocardiography was applied using standard LV short-axis images to assess the effect of new conduction defects on time-to-peak radial strain of different LV segments as a parameter of LV dyssynchrony.

**Results** New conduction defects resulted in marked heterogeneity in time-to-peak strain between the 6 analyzed short-axis segments. During 1-year follow-up after TAVI, there was a significant increase in left ventricular ejection fraction (LVEF) in patients without new LBBB ( $53 \pm 11\%$  pre TAVI to  $59 \pm 10\%$  at follow-up;  $p < 0.001$ ), whereas there was no change in LVEF in patients with a new conduction defect ( $52 \pm 11\%$  pre TAVI to  $51 \pm 12\%$  at follow-up,  $p = 0.740$ ). Change in LV end-systolic volume was also significantly different between patient groups ( $-1.0 \pm 14.2$  vs.  $-11.2 \pm 15.7$  ml,  $p = 0.042$ ). New conduction defect and LVEF at baseline were independent predictors of reduced LVEF at 12-month follow-up after TAVI.

**Conclusions** LVEF improves after TAVI for treatment of severe aortic stenosis in patients without new conduction defects. In patients with a new conduction defect after TAVI, there is no improvement in LVEF at follow-up. (J Am Coll Cardiol Intv 2012;5:1257–63) © 2012 by the American College of Cardiology Foundation

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Transcatheter aortic valve implantation (TAVI) has become a therapeutic option for high-risk or nonoperable patients with severe symptomatic aortic valve stenosis (1–6). A major limitation of TAVI is the frequent occurrence of new conduction defects and a subsequent need for pacemaker implantation (7–11). Pressure induced by the procedure and by the valve implant on the atrioventricular (AV) bundle located close to the aortic valve has been discussed as the reason for new conduction defects. The frequency of new left bundle branch blocks (LBBB) and the need for pacemaker implantation is known to be higher with the self-expanding CoreValve prosthesis (Medtronic, Minneapolis, Minnesota) than with the balloon-expandable Edwards SAPIEN valve (Edwards Lifesciences, Irvine, California). LBBB is known to be associated with left ventricular (LV) dyssynchrony, ventricular remodeling, and impaired function (12). Implantation of a permanent right ventricular pacemaker (PPM) is also known to result in impairment of LV function (13–15). The cause of the deleterious effect of PPM has been shown to be LV dyssynchrony (15). Although LV dyssynchrony is the consequence of a new LBBB or a PPM after TAVI, the impact of a new conduction defect after TAVI on the development of LV function during 1-year follow-up has not been evaluated.

### Abbreviations and Acronyms

**AV** = atrioventricular

**LBBB** = left bundle branch block

**LV** = left ventricle/ventricular

**LVEF** = left ventricular ejection fraction

**PPM** = permanent pacemaker

**TAVI** = transcatheter aortic valve implantation

TAVI induces LV dyssynchrony and has a negative impact on LV function.

The current study: 1) used speckle-tracking echocardiography to determine the impact of a new conduction defect after TAVI on heterogeneity of LV contraction; 2) analyzed the impact of a new conduction defect after TAVI on LV volumes and ejection fraction at 12-month follow-up; and 3) determined predictors of failed recovery of LV function at 12-month follow-up after TAVI.

### Methods

**Study design and patient population.** We prospectively evaluated 125 consecutive patients (39 male, mean age  $81 \pm 7$  years) with severe symptomatic calcified native valve stenosis in whom either a CoreValve or Edwards SAPIEN Revalving system was implanted between January 2009 and July 2010 and in whom 1-year follow-up echocardiography was aimed for. Twenty-seven patients died during 1-year follow-up. Another 8 patients refused to return for

follow-up echocardiography. In 90 patients, echocardiography was performed before TAVI, at 1 month, and at 1-year follow-up. These patients formed the study group. Patients were referred for percutaneous aortic valve implantation after a senior cardiologist and senior cardiac surgeon reached consensus that surgical replacement would be associated with either high or prohibitive risk. The inclusion and exclusion criteria of patients for implantation of the catheter-based revalving systems have been described elsewhere (9). In patients with significant femoral or iliac artery disease, transapical TAVI was the preferred procedure, whereas transfemoral TAVI was preferred in very old patients and in patients with pulmonary disease. Patients had to have a valve area  $<1 \text{ cm}^2$ , and aortic valve annulus diameter measuring  $\geq 18$  and  $\leq 27$  mm. Presence of a bicuspid aortic valve as defined by echocardiographic screening was an exclusion criteria for TAVI. This study was approved by the ethical committee of the University Aachen, and all patients gave informed consent.

**Description of the device and the procedure.** In 52 patients (58%), the CoreValve Revalving system was used. In 38 patients (42%), the Edwards SAPIEN valve was implanted. Details of the devices, and the technical aspects of the procedure, have been previously published (1–4). The CoreValve Revalving system consists of a self-expanding nitinol trilevel frame to which a trileaflet bioprosthetic porcine pericardial tissue valve is secured. In this study, prosthesis sizes of 26 and 29 mm have been used. The Edwards SAPIEN valve is a balloon-expandable, bovine pericardium prosthesis. In this study, prosthesis sizes of 23 and 26 mm have been used. All patients treated by apical access TAVI obtained an Edwards SAPIEN valve, whereas all patients treated by femoral access TAVI obtained a CoreValve Revalving system device. Selection of the device size was based on measurements of the aortic root anatomy and aortic valve measurements, in particular the aortic valve annulus diameter, obtained by transesophageal echocardiography, calipered angiography, and multislice computed tomography. Selection of prosthesis size intended slight oversizing of the prosthesis diameter relative to the annulus diameter to securely fix the prosthesis within the native valve and annulus, and minimize the potential for paravalvular leaks between the prosthetic valve and native annulus.

Balloon valvotomy was performed before implantation. Positioning and deployment of the device was performed under fluoroscopic guidance using the CoreValve prosthesis and with combined fluoroscopic and transesophageal echocardiography guidance for Edwards SAPIEN implantation. **Echocardiography.** Pre-interventional patient screening included transthoracic as well as transesophageal echocardiography to confirm diagnosis, define aortic valve morphology, and measure the aortic valve annular diameter.

Follow-up echocardiography was performed at 1 month and at 12 months. Echocardiography at baseline and at

12-month follow-up was used to evaluate LV end-diastolic and end-systolic volumes based on apical 4- and 2-chamber views and application of the Simpson's method. The LVEF was calculated based on LV volume measurements. Echocardiography at baseline and at 1-month follow-up was performed to evaluate myocardial deformation.

**Myocardial deformation analysis.** Speckle-tracking analysis was performed on LV short-axis images at the papillary muscle level to determine radial strain (17). Off-line analysis of radial strain was performed on digitally stored images (EchoPAC, GE Vingmed, Horton, Norway) by 2 independent observers blinded to the clinical and other echocardiographic information. The speckle-tracking software tracks natural acoustic markers frame to frame on grayscale images of the myocardium (frame rate varied from 40 to 80 frames/s), as previously described (17). Myocardial strain is determined from temporal differences in the mutual distance of neighboring speckles. Radial strain is calculated as change in length/initial length of the speckle pattern over the cardiac cycle, with myocardial thinning represented as negative strain and myocardial thickening as positive strain. The traced endocardium is automatically divided into 6 standard segments: septal, anteroseptal, anterior, lateral, posterior, and inferior (17). For all 6 segments, time-strain curves were constructed. Time from QRS onset to peak radial strain was obtained for each curve. The location of the earliest- and latest-activated segments and the heterogeneity in time-to-peak radial strain for the 6 segments was determined. The time difference between the earliest- and latest-activated segment was calculated. An interval  $\geq 130$  ms for the absolute difference in time-to-peak radial strain for the septal or anteroseptal wall versus the posterior or lateral wall was defined as LV dyssynchrony (17). On short-axis images of 20 randomly selected patients, intra- and interobserver variability in the analysis of time-to-peak radial strain were determined. The intraobserver variability for determining the time-to-peak radial strain of all 6 segments was  $12 \pm 9$  ms, and the interobserver variability  $15 \pm 11$  ms.

**Electrocardiography and pacemaker requirements.** Electrocardiographic tracings obtained before and after valve implantation, along with 1-month follow-up recordings, were interpreted in the core laboratory at the University Aachen. Serial electrocardiographic recordings were performed within the first 5 days after the TAVI procedure. The recordings were analyzed for rhythm, heart rate (beats/min), PQ and QRS intervals (in milliseconds), and presence of second- or third-degree AV block. The requirements after the procedure for a temporary pacemaker or PPM were documented. A PPM was implanted based on PQ and QRS interval measurements and serial changes within the first days after the procedure. A deliberate approach towards PPM implantation was applied. Registration of intermittent second- or third-degree AV block was an indication for

PPM implantation. A QRS interval  $> 130$  ms together with a PQ interval  $> 250$  ms was also used as an indication for PPM implantation unless serial studies indicated a reduction of intervals immediately after TAVI.

On the basis of the 1-month follow-up ECG, patients were divided into those without any new electrocardiographic conduction defects and those with a new conduction defect. Documentation of either a new LBBB or the new need for PPM action due to total AV block determined the presence of a new conduction defect.

**Statistical analysis.** Statistical analysis was performed using the SPSS software version 17.0 (IBM, Armonk, New York). Categorical data were presented as frequencies and compared with the Pearson chi-square test. Continuous data were presented as mean  $\pm$  SD and compared with the Student *t* test or analysis of variance as appropriate. Logistic regression analysis was performed to identify univariate and multivariate predictors for a reduction of left ventricular ejection fraction (LVEF) at 12-month follow-up (LVEF follow-up minus LVEF before TAVI  $< 0\%$ ). Included variables were age, sex, aortic valve area before TAVI, LVEF at baseline, number of significantly diseased coronary arteries on angiography, severity of aortic regurgitation after TAVI, type of implanted TAVI device (either the self-expanding CoreValve or the balloon-expandable Edwards SAPIEN), and a new conduction defect after TAVI. Univariate predictors with  $p < 0.20$  were included in the multivariate analysis. In addition, an analysis of covariance was performed to determine in a multiple linear regression model factors with impact on change in LVEF during 12-month follow-up.  $p < 0.05$  was considered statistically significant.

## Results

In 90 patients, baseline, 1-month, and 12-month follow-up echocardiography was obtained. Fifteen of these patients had atrial fibrillation (17%) at baseline, and 12 patients (13%) had baseline conduction defects. In 7 patients, there was an LBBB, and 5 patients had PPM action due to AV block.

**Frequency of new conduction deficit.** Thirty-nine patients (43%) had a new conduction defect after TAVI, and in 51 patients (57%), no new conduction defect was documented. Thirty-one patients (35%) had a new LBBB. Twelve of these 31 patients with new LBBB had a new PPM implanted but only intermittent pacemaker action. An additional 8 patients (9%) had new need for PPM activation due to complete AV block after TAVI. In 3 of the 51 patients considered to have no new conduction defect, a new PPM was implanted due to documentation of intermittent AV block immediately after TAVI. However, at 1-month follow-up, pacemaker analysis documented only sporadic pacemaker action.

| Table 1. Patient Baseline and Procedural Characteristics |                                     |  |         |
|--|-------------------------------------|--|---------|
|  | New Conduction Disturbance (n = 39) | No New Conduction Disturbance (n = 51) | p Value |
| Age, yrs   | 80 ± 6                              | 79 ± 6                                 | 0.422   |
| Male   | 14 (36)                             | 20 (40)                                | 0.387   |
| Log EuroSCORE  | 21 ± 13                             | 19 ± 12                                | 0.324   |
| NYHA functional class                                    | 3.1 ± 0.6                           | 3.0 ± 0.6                              | 0.535   |
| CAD  | 25 (65)                             | 34 (67)                                | 0.848   |
| PAH  | 6 (16)                              | 9 (18)                                 | 0.778   |
| Mitral regurgitation ≥grade 2                            | 11 (27)                             | 13 (26)                                | 0.921   |
| GFR <60 ml/min   | 12 (29)                             | 22 (43)                                | 0.172   |
| TAVI type  |                                     |  |         |
| Edwards SAPIEN   | 7                                   | 31                                     | <0.001  |
| CoreValve  | 32                                  | 20                                     |         |

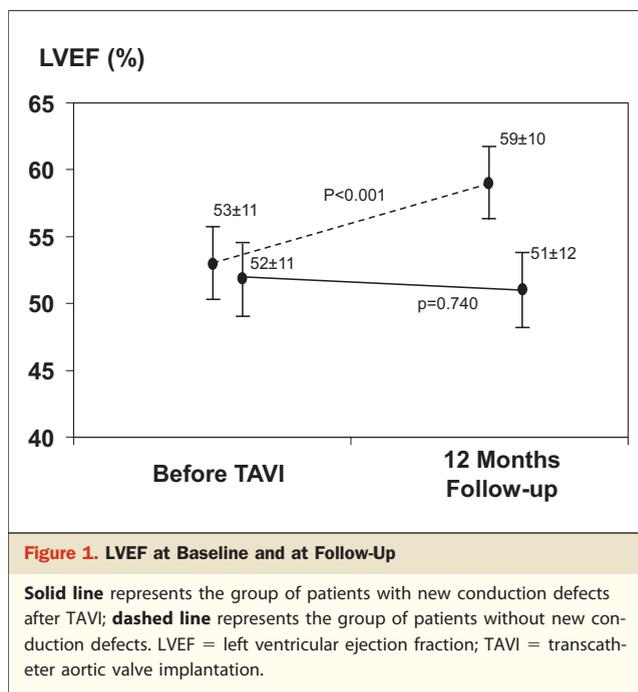
Values are n, n (%), or mean ± SD.  
CAD = coronary artery disease; GFR = glomerular filtration rate; NYHA = New York Heart Association; PAH = pulmonary artery hypertension (mean pressure >25 mm Hg); TAVI = transcatheter aortic valve implantation.

Baseline and procedural characteristics of patients with new conduction defects as compared with patients without new conduction defects are given in Table 1. In 32 of the 52 CoreValve patients (62%) and in 7 of the 38 Edwards SAPIEN patients (18%), a new conduction defect was observed after the procedure. Thus, the frequency of new conduction defects was significantly higher after CoreValve implantation compared with Edwards SAPIEN implantation ( $p < 0.001$ ).

**LV volumes and LVEF.** At baseline before TAVI, there were no differences in LV end-diastolic and end-systolic volumes as well as LVEF between patients with new and those without new conduction defects after TAVI (Table 2). At 12-month follow-up, LV end-systolic volume had decreased for the total study population from  $65 \pm 31$  to  $60 \pm 31$  ml ( $p = 0.039$ ), whereas end-diastolic volume was unchanged ( $136 \pm 51$  vs.  $135 \pm 50$  ml). Patients without new conduction defects after TAVI had a significantly

| Table 2. LV Volumes and LVEF Before TAVI and at 12-Month Follow-Up Related to New Conduction Disturbance |                                     |  |         |
|--|-------------------------------------|--|---------|
|  | New Conduction Disturbance (n = 39) | No New Conduction Disturbance (n = 51) | p Value |
| ESV before TAVI, ml  | 64 ± 31                             | 65 ± 30                                | 0.771   |
| Δ ESV at follow-up, ml   | -1.0 ± 14.2                         | -11.2 ± 15.7                           | 0.042   |
| EDV before TAVI, ml  | 136 ± 46                            | 134 ± 54                               | 0.843   |
| Δ EDV at follow-up, ml   | -3.3 ± 25.2                         | -7.5 ± 27.2                            | 0.374   |
| LVEF before TAVI, %  | 52 ± 11                             | 53 ± 11                                | 0.854   |
| LVEF at follow-up, %   | 51 ± 12                             | 59 ± 10                                | 0.052   |
| Δ LVEF at follow-up, %   | -0.5 ± 9.4                          | 5.8 ± 7.9                              | 0.002   |

Values are mean ± SD or % ± SD.  
EDV = end-diastolic volume; ESV = end-systolic volume; LV = left ventricular; LVEF = left ventricular ejection fraction; TAVI = transcatheter aortic valve implantation.



smaller end-systolic volume at 12-month follow-up compared with patients with new conduction defects (Table 2). Changes in end-diastolic LV volumes were less different between patients with and without new conduction defects. The LVEF changed at 12-month follow-up after TAVI from  $54 \pm 11\%$  to  $57 \pm 11\%$  for the total study group. In patients without new conduction defects, there was an increase in LVEF of  $5.8 \pm 7.9\%$  ( $p < 0.001$  for LVEF at follow-up vs. baseline), whereas there was almost no change at 12-month follow-up in the group with new conduction defects ( $-0.5 \pm 9.4\%$ ,  $p = 0.740$  between baseline and follow-up) (Fig. 1). Eight patients (16%) of the no new conduction defect group demonstrated impairment of LVEF at follow-up, whereas 19 patients (49%) in the new conduction defect group demonstrated impairment of LVEF at follow-up. Thus, there was a significant difference between both groups in the change of LVEF at follow-up ( $p = 0.002$ ). Considering only CoreValve patients, LVEF increased by  $1.7 \pm 10.4\%$ , whereas LVEF increased by  $4.4 \pm 6.8\%$  in Edwards SAPIEN patients ( $p = 0.230$ ).

**Myocardial deformation analysis.** At baseline, there was a high level of homogeneity in the time-to-peak radial strain between the 6 analyzed segments of a short-axis image. At baseline, there was also no difference between patients with and without new conduction defects after TAVI in the homogeneity of LV contraction expressed as the time difference between earliest and latest segment reaching the peak radial strain (Table 3). One month after TAVI, the time difference between the earliest and latest peak radial strain reached in the 6 segments of 1 short-axis image had increased significantly from  $43 \pm 39$  to  $142 \pm 52$  ms ( $p <$

**Table 3. Analysis of LV Contraction Synchrony Using Echocardiographic Deformation Imaging**

|   | New Conduction Disturbance (n = 39) | No New Conduction Disturbance (n = 51) | p Value |
|---|-------------------------------------|--|---------|
| Pre-TAVI Δ time-to-peak systolic radial strain, ms          | 36 ± 24                             | 29 ± 22                                | 0.101   |
| 1-month post-TAVI Δ time-to-peak systolic radial strain, ms | 139 ± 35                            | 38 ± 34                                | <0.001  |

Values are mean ± SD. Δ Time-to-peak systolic radial strain relates to the time difference to peak systolic strain between the 6 analyzed segments of the short axis image.  
 Abbreviations as in Table 2.

0.001) in the new conduction defect group. By contrast, there was no significant change in the no new conduction defect group. At 1 month after TAVI, the time difference between earliest and latest segment reaching the peak radial strain was found to be significantly different between the groups with and without new conduction defects (Table 3). Considering the definition for LV dyssynchrony based on absolute difference in time-to-peak radial strain for the septal or anteroseptal wall versus the posterior or lateral wall, 34 patients (87%) were found to have LV dyssynchrony in the new conduction defect group at 1-month follow-up after TAVI and 5 patients (10%) in the no new conduction defect group.

**Predictors of impaired LV function after TAVI.** In a univariate logistic regression analysis induction of a new conduction defect, LVEF at baseline and the time difference to peak radial strain 1 month after TAVI were univariate predictors of a reduction in LV function at 12-month follow-up (Table 4). In a multivariate logistic regression analysis, new conduction defect (odds ratio: 2.001, 95% confidence interval: 1.271 to 3.045,  $p = 0.003$ ) and LVEF at baseline (odds ratio: 1.010, 95% confidence interval: 1.001 to 1.019,  $p = 0.021$ ) remained independent predictors of a reduction in LV function at 12-month follow-up. Age, sex, aortic valve area before TAVI, prosthesis diameter, and aortic regurgitation were not independent predictors of impaired LVEF at follow-up. Considering a covariance analysis, only LVEF at baseline (regression coefficient  $b: -0.271$ ,  $p < 0.001$ ) as a covariate and new conduction defect (adjusted mean difference: 5.595,  $p < 0.001$ ) had influence on change in LVEF at 12-month follow-up.

## Discussion

The main findings of this study are: 1) TAVI is frequently associated with new conduction defects; 2) patients with new conduction defects demonstrate dyssynchrony in LV function 1 month after TAVI; 3) although there is improvement in LVEF after TAVI for severe aortic stenosis in patients without new conduction defects, patients with new conduction defects have a significantly worse development

in LV function at 12-month follow-up; and 4) development of a new conduction defect is a predictor of absent improvement in LV function at 12-month follow-up.

**Conduction defects after TAVI.** New conduction defects develop in as many as one-third of patients undergoing surgical replacement of the aortic valve, with LBBB being the most common abnormality (18–20). After TAVI, new conduction defects have been described in an even significantly higher number of patients, with new LBBB being the predominant new conduction defect (7–11,21–24). The anatomic neighborhood of the aortic valve to the atrioventricular bundle; degeneration of the conduction system; trauma by guidewires, catheters, and pre-implantation balloon valvuloplasty; and direct constant pressure of the implanted valve on the left bundle branch at the base of the interleaflet triangle between the right and noncoronary triangle are the potential causes (25). The frequency of new LBBB has been reported to be higher after implantation of the CoreValve prosthesis as compared with the Edwards SAPIEN prosthesis (24). This has been explained by the larger size of the CoreValve prosthesis and, in particular, the greater depth of implantation within the LV outflow track. An implantation depth into the LV outflow track >6 mm has been associated with a significantly increased risk of LBBB (7,24). In a study on 30 patients treated with a CoreValve prosthesis, Calvi et al. (10) observed a frequency of early conduction disorders after TAVI in 68% of patients. LBBB was the most common conduction abnormality, with an incidence of 45.8%. In a study on 40 consecutive patients in whom a CoreValve device was implanted, the frequency of LBBB increased from 15% before treatment to 55% after valve implantation (7). In a recent study on 270 patients, the incidence of LBBB was reported to be 13% before TAVI and 61% after TAVI using the CoreValve prosthesis (25). New-onset LBBB has been reported with lower frequency after implantation of Edwards SAPIEN valve. Gutiérrez et al. (11) reported an increase in the frequency of LBBB from 9% at baseline to 27% after Edwards SAPIEN implantation. Need for PPM implantation after TAVI has been reported in up to 39.3% of patients in a registry of 697 patients (26). Significantly higher rates of pacemaker requirement have been reported after CoreValve implantation

**Table 4. Univariate Predictors of Reduced LVEF at 12-Month Follow-Up After TAVI**

|   | OR    | 95% CI      | p Value |
|---|-------|-------------|---------|
| New LBBB or new PPM   | 1.551 | 1.294–1.844 | <0.001  |
| 1-month post-TAVI Δ time-to-peak systolic radial strain (per additional ms) | 1.003 | 1.001–1.005 | 0.001   |
| LVEF before TAVI (per additional %)   | 1.012 | 1.003–1.022 | 0.007   |

LVEF at 12-month follow-up minus LVEF at baseline <0%.  
 CI = confidence interval; LBBB = left bundle branch block; OR = odds ratio; PPM = permanent pacemaker; other abbreviations as in Table 2.

compared with after Edwards SAPIEN implant. Khawaja et al. (25) reported in a study of 270 patients a PPM rate of 33.3%, and Piazza et al. (7) reported in a smaller study a rate of 40% after CoreValve implantation. By contrast, need for a new PPM has been reported to be as low as 3.4% and 3.8%, respectively, in both arms of the PARTNER (Placement of Aortic Transcatheter Valve) trial involving a total of 527 TAVI patients with an Edwards SAPIEN implant (4,27).

**LV function after TAVI.** LV function is known to improve after aortic valve replacement for severe aortic stenosis. Furthermore, better post-procedural recovery of LVEF has been shown to be associated with better improvement in functional status and better late survival (28). Inconsistent results have been reported after TAVI. Some studies have also documented improvements in LVEF after TAVI (29,30). In a study comparing the development of LV function between patients undergoing transcatheter and surgical prosthetic valve implantation, TAVI patients had even better recovery of LVEF at 1 year (28). However, in this study as well as others demonstrating improvement in LVEF after TAVI, the Edwards SAPIEN valve was used for the procedure. By contrast, studies reporting on the development of LVEF after implantation of the CoreValve prosthesis did not demonstrate significant improvement (31). The different rates of conduction defects, and in particular LBBB, may be the likely explanation for the different development of LVEF after TAVI using Edwards SAPIEN versus CoreValve implants.

This study evaluated the important link between new conduction defects and subsequent development of LV function. New conduction defects resulted immediately in LV dyssynchrony. At 12-month follow-up, patients with new conduction defects had significantly worse development of the LV function than patients without new conduction defects. The impact of PPM action on ventricular synchrony and development of LV function has been demonstrated in several trials (13–15). Tops et al. (15) demonstrated that patients with new LV dyssynchrony with PPM action had a decrease in LVEF from 48% at baseline to 39% at 1-year follow-up. Previous studies have demonstrated that the cumulative percent right ventricular pacing increased the risk of heart failure and risk of death. Although in previous studies on right ventricular pacing, the implantation of the device induced only deleterious effects, TAVI results in a potentially positive impact on LV function due to afterload reduction and, simultaneously, the deleterious effect of LV dyssynchrony due to new conduction defects. The induction of a new conduction defect and its impact on LV function may be of limited importance in the very old, high-risk patients receiving TAVI. However, in the moderate surgical risk patient of lower age, increasingly treated with TAVI, the effect of new conduction

defects may be of greater importance for subsequent patient outcome.

**Study limitations.** This is a single-center experience. Further studies with higher patient numbers will be necessary to further assess the development of LV volumes and function after TAVI. Future studies need to assess the impact of new conduction defects on patient functional class and life quality. The indication for pacemaker implantation did not follow strict rules after TAVI. Thus, some patients may have received “prophylactic” pacing in the presence of LBBB in combination with prolonged PQ interval. However, only patients with PPM action at 1-month follow-up were included in the group with new conduction defects.

This study did not demonstrate a significant difference in the change of LV function at follow-up between patients treated with a CoreValve prosthesis and those treated with an Edwards SAPIEN prosthesis although there was a significantly higher rate of new conduction defects after implantation of the CoreValve prosthesis. A randomized study with higher patient numbers may be required to evaluate a potential difference in LV function at follow-up between both valve types. This study did not find the access site to be a predictor of impairment in LV function at follow-up. However, the apical access TAVI may result in minor apical scar formation and thus impairment of LV function at follow-up.

## Conclusions

TAVI is frequently associated with new conduction defects. New conduction defects result in LV dyssynchrony and subsequently impaired development of LV function at 12-month follow-up.

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## REFERENCES

1. Grube E, Schuler G, Buellesfeld L, et al. 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.
2. Webb JG, Altwegg L, Boone RH, et al. Transcatheter aortic valve implantation: impact on clinical and valve-related outcomes. *Circulation* 2009;119:3009–16.
3. Cribier A, Eltchaninoff H, Tron C, et al. Treatment of calcific aortic stenosis with the percutaneous heart valve: mid-term follow-up from the initial feasibility studies: the French experience. *J Am Coll Cardiol* 2006;47:1214–23.
4. Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. *N Engl J Med* 2010;363:1597–607.
5. Himbert D, Descoutures F, Al-Attar N, et al. Results of transfemoral or transapical aortic valve implantation following a uniform assessment

- in high-risk patients with aortic stenosis. *J Am Coll Cardiol* 2009;54:303-11.
6. Rodés-Cabau J, Webb JG, Cheung A, et al. Transcatheter aortic valve implantation for the treatment of severe symptomatic aortic stenosis in patients at very high or prohibitive surgical risk: acute and late outcomes of the multicenter Canadian experience. *J Am Coll Cardiol* 2010;55:1080-90.
  7. Piazza N, Onuma Y, Jesserun E, et al. Early and persistent intraventricular conduction abnormalities and requirements for pacemaking after percutaneous replacement of the aortic valve. *J Am Coll Cardiol Intv* 2008;1:310-6.
  8. Sinhal A, Altwegg L, Pasupati S, et al. Atrioventricular block after transcatheter balloon expandable aortic valve implantation. *J Am Coll Cardiol Intv* 2008;1:305-9.
  9. Vahanian A, Alferi O, Al-Attar N, et al., European Association of Cardio-Thoracic Surgery; European Society of Cardiology; European Association of Percutaneous Cardiovascular Interventions. Transcatheter valve implantation for patients with aortic stenosis: a position statement from the European Association of Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). *Eur Heart J* 2008;29:1463-70.
  10. Calvi V, Puzangara E, Pruiti GP, et al. Early conduction disorders following percutaneous aortic valve replacement. *Pacing Clin Electrophysiol* 2009;32 Suppl 1:S126-30.
  11. Gutiérrez M, Rodés-Cabau J, Bagur R, et al. Electrocardiographic changes and clinical outcomes after transapical aortic valve implantation. *Am Heart J* 2009;158:302-8.
  12. Vernooy K, Verbeek XA, Peschar M, et al. Left bundle branch block induces ventricular remodeling and functional septal hypoperfusion. *Eur Heart J* 2005;26:91-8.
  13. Steinberg JS, Fischer A, Wang P, et al. The clinical implications of cumulative right ventricular pacing in the Multicenter Automatic Defibrillator Trial II. *J Cardiovasc Electrophysiol* 2005;16:359-65.
  14. Vernooy K, Dijkman B, Cheriex EC, Prinzen FW, Crijns HJ. Ventricular remodeling during long-term right ventricular pacing following His bundle ablation. *Am J Cardiol* 2006;97:1223-7.
  15. Tops LF, Suffoletto MS, Bleeker GB, et al. Speckle-tracking radial strain reveals left ventricular dyssynchrony in patients with permanent right ventricular pacing. *J Am Coll Cardiol* 2007;50:1180-8.
  16. Amundsen BH, Helle-Valle T, Edvardsen T, et al. Noninvasive myocardial strain measurement by speckle tracking echocardiography: validation against sonomicrometry and tagged magnetic resonance imaging. *J Am Coll Cardiol* 2006;47:789-93.
  17. Suffoletto MS, Dohi K, Cannesson M, Saba S, Gorcsan J. Novel speckle-tracking radial strain from routine black-and-white echocardiographic images to quantify dyssynchrony and predict response to cardiac resynchronization therapy. *Circulation* 2006;113:960-8.
  18. Koplan BA, Stevenson WG, Epstein LM, Aranki SF, Maisel WH. Development and validation of a simple risk score to predict the need for permanent pacing after cardiac valve surgery. *J Am Coll Cardiol* 2003;41:795-801.
  19. Dawkins S, Hobson AR, Kalra PR, Tang ATM, Monro JL, Dawkins KD. Permanent pacemaker implantation after isolated aortic valve replacement: incidence, indications, and predictors. *Ann Thorac Surg* 2008;85:108-12.
  20. El-Khally Z, Thibault B, Staniloae C, et al. Prognostic significance of newly acquired bundle branch block after aortic valve replacement. *Am J Cardiol* 2004;94:1008-11.
  21. Moreno R, Dobarro D, López de Sá E, et al. Cause of complete atrioventricular block after percutaneous aortic valve implantation: insights from a necropsy study. *Circulation* 2009;120:e29-30.
  22. Baan J Jr., Yong ZY, Koch KT, et al. Factors associated with cardiac conduction disorders and permanent pacemaker implantation after percutaneous aortic valve implantation with the CoreValve prosthesis. *Am Heart J* 2010;159:497-503.
  23. Jilaihawi H, Chin D, Vasa-Nicotera M, et al. Predictors for permanent pacemaker requirement after transcatheter aortic valve implantation with the CoreValve bioprosthesis. *Am Heart J* 2009;157:860-6.
  24. Aktug Ö, Dohmen G, Brehmer K, et al. Incidence and predictors of left bundle branch block after transcatheter aortic valve implantation. *Int J Cardiol* 2012;160:26-30.
  25. Khawaja MZ, Rajani R, Cook A, et al. Permanent pacemaker insertion after CoreValve transcatheter aortic valve implantation: incidence and contributing factors (the UK CoreValve Collaborative). *Circulation* 2011;123:951-60.
  26. Zahn R, Gerckens U, Grube E, et al. Transcatheter aortic valve implantation: first results from a multi-centre real-world registry. *Eur Heart J* 2011;32:198-204.
  27. Smith CR, Leon MB, Mack MJ, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. *N Engl J Med* 2011;364:2187-98.
  28. Clavel MA, Webb JG, Rodés-Cabau J, et al. Comparison between transcatheter and surgical prosthetic valve implantation in patients with severe aortic stenosis and reduced left ventricular ejection fraction. *Circulation* 2010;122:1928-36.
  29. Webb JG, Pasupati S, Humphries K, et al. Percutaneous transarterial aortic valve replacement in selected high-risk patients with aortic stenosis. *Circulation* 2007;116:755-63.
  30. Bauer F, Eltchaninoff H, Tron C, et al. Acute improvement in global and regional left ventricular systolic function after percutaneous heart valve implantation in patients with symptomatic aortic stenosis. *Circulation* 2004;110:1473-6.
  31. Tzikas A, Geleijnse ML, Van Mieghem NM, et al. Left ventricular mass regression one year after transcatheter aortic valve implantation. *Ann Thorac Surg* 2011;91:685-91.
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- Key Words:** aortic stenosis ■ conduction defect ■ dyssynchrony ■ left bundle branch block ■ valve.