Left ventricular remodelling pattern and its relation to clinical outcomes in patients with severe aortic stenosis treated with transcatheter aortic valve implantation

Bartosz Rymuza1, Karol Zbroński1, Piotr Scisło1, Radosław Wilimski2, Janusz Kochman1, Agata Ćwiek1, Krzysztof J. Filipiak1, Grzegorz Opolski1, Zenon Huczek1

1First Department of Cardiology, Medical University of Warsaw, Warsaw, Poland
2Department of Cardiac Surgery, Medical University of Warsaw, Warsaw, Poland

Adv Interv Cardiol 2017; 13, 4 (50): 288–294
DOI: https://doi.org/10.5114/aic.2017.71609

Abstract

Introduction: Left ventricular hypertrophy (LVH) is a common compensating process in the pressure overload mechanism of aortic stenosis (AS).

Aim: To identify a group of patients with a LVH pattern which may alter periprocedural and 1-year outcomes after transcatheter aortic valve implantation (TAVI).

Material and methods: Echocardiographic examinations of 226 patients with severe AS treated with TAVI between March 2010 and February 2016 were retrospectively analysed and correlated with echocardiographic parameters and clinical outcomes in the study group. Ultimately 208 patients were enrolled in the study. Based on left ventricular mass index (LVMI) and relative wall thickness (RWT) patients were divided into three categories: concentric remodelling (CR), concentric hypertrophy (CH) and eccentric hypertrophy (EH). Most of the patients with severe AS referred for TAVI were found to have CH (n = 150, 72.8%), then EH (n = 33, 16%) and CR (n = 16, 7.8%).

Results: There were no significant differences between groups in terms of periprocedural outcomes or complications. After a mean observation time of 561.8 ±239.0 days, the observed all-cause mortality rate was 19.9%. After multivariable adjustment, CR remained associated with a higher risk of mortality (HR = 4.31; 95% CI: 1.607–11.538; p = 0.004).

Conclusions: Left ventricular hypertrophy is common in patients with severe AS prior to TAVI. The LVH pattern does not affect TAVI-related complications. In patients with severe AS referred for TAVI, CR seems to be the least favourable geometry of LVH, increasing the risk of 1-year all-cause death.

Key words: mortality, left ventricular hypertrophy, left ventricular remodelling, transcatheter aortic valve implantation, severe aortic stenosis, concentric remodelling.

Introduction

Aortic stenosis (AS) is the most common cardiovascular disease besides hypertension and coronary artery disease in the adult European population [1]. During the long asymptomatic phase of AS the walls of the left ventricle (LV) are subjected to increasing pressure overload which causes gradual thickening of the muscle. Along with growing left ventricular mass (LVM) and occurrence of interstitial fibrosis, the development of diastolic and systolic dysfunction begins, slowly leading to the symptomatic phase, heart failure and death. The risk of an adverse outcome may be diminished by relieving afterload by valve replacement therapy, but the high mortality risk may persist in patients with severe left ventricular hypertrophy (LVH) [2]. The pathomechanism of this relation may be explained by slower regression of LVM after surgical aortic valve replacement (SAVR) and transcatheter aortic valve implantation (TAVI) [3–7].

Geometric changes of LV dimensions in AS are heterogeneous and fall into three categories: concentric remodelling (CR), concentric hypertrophy (CH) and eccentric hypertrophy. They differ between one another in terms of left ventricular diastolic diameter (LVDD), intraventricular septum diastolic diameter (IVSd) and posterior wall thickness (PWT), which contribute to calculating LVM. Given the known effect of LV geometry on TAVI outcomes
we hypothesized that different LVH patterns may affect peri-procedural outcomes as well as 1-year prognosis. Some forms of hypertrophy are recognized as predictors of long-term mortality in patients with AS, and preserved ejection fraction (EF) [15], but this relation has not been confirmed in the high-risk TAVI population.

Aim

The aim of the study was to analyse the distribution of different models of LVH in the studied group, to assess the possible link between the abovementioned geometries on peri-procedural outcomes, and finally to test whether any of the patterns has an effect on 1-year mortality.

Material and methods

Study design and population

The study was designed as a retrospective, single-centre, observational study with 1-year follow-up of events. Pre- and post-procedural echocardiographic examinations of 226 consecutive patients with severe AS, referred by a local Heart Team’s decision for TAVI between March 2010 and March 2016, were analysed. After substracting data of patients whose examinations were of reduced quality, and those with valve-in-valve procedures, ultimately 208 patients were enrolled in the current study. In each patient relative wall thickness (RWT) and left ventricular mass index (LVMI) were calculated, and according to the results, patients were classified into four categories: concentric hypertrophy, concentric remodelling, eccentric remodelling or normal geometry. The recorded echocardiograms of patients were examined, and accurate measurement of post-procedural values of depth of implantation and parameters describing paravalvular leak (PVL) were obtained. The 1-year follow-up echocardiograms were analysed to determine changes in LVM. Information regarding baseline characteristics and peri-procedural proceedings was collected as well as follow-up data concerning outcomes and events. The study was approved by the bioethical commission of the Medical University of Warsaw.

Echocardiography

Two-dimensional Doppler transthoracic echocardiography was performed. The images were obtained in parasternal long- and short-axis views and also two-and four-chamber views. Continuous wave Doppler was used to estimate transvalvular gradients using the Bernoulli equation. Ventricular diameters and posterior and septal wall thickness were measured in two dimensions in the parasagittal view according to guideline recommendations [16]. Each included examination was assessed and besides standard parameters, post-procedural frame borders of the implanted valves were analysed as well as PVL location, volume and number. All measurements were obtained by a single, trained echocardiographer who evaluates TAVI patients on a daily basis.

Study definitions and endpoints

RWT was calculated as RWT = (2 × PWTD ÷ LVDD) and LVMI as LVMI = 0.8 × ((LVDD + PWTD + IVS)2 – LVDD2) + 0.6 and indexed to body surface area (BSA) [17].

Patients with RWT ≤ 0.42 were divided into two categories. Those with LVMI above the cut-off values of 95 g/m² for women and 115 g/m² for men were included in the eccentric hypertrophy group and the rest were considered as normal. The group with RWT ≥ 0.42 was also divided according to LVMI into concentric hypertrophy (LVMI ≥ 95 g/m² for women and 115 g/m² for men) and concentric remodelling [16]. The left ventricular end diastolic volume was assessed using the Teichholz formula. All clinical endpoints were defined by VARC 2 criteria [17].

Statistical analysis

In order to identify the group with the least favourable left ventricle (LV) geometry the 4 groups of patients were compared using the one-way ANOVA test with Tukey’s post hoc test when appropriate. The Shapiro-Wilk test was used to confirm or reject normal distribution of each continuous variable. Categorical variables, expressed as counts and percentages, and continuous variables are expressed as means ± SD. Data concerning the number of post-procedural events and complication rate were compared using the χ² test or Fisher’s exact test, as appropriate. All probability values reported are 2-sided and a value < 0.05 was considered to be significant. Kaplan-Meier curves and log-rank tests of the time-to-event data were used to assess the effect of LV remodelling patterns on all-cause mortality. Afterwards Cox proportional hazard analysis was performed to find possible predictors of endpoints. The proportional-hazards assumption was checked using Schoenfeld residuals. The baseline variables which differed between the predictor and the rest of the group with a p-value < 0.10 were entered in the multivariable Cox model to find independent predictors of 1-year mortality. Data were processed using the SPSS software, version 22 (IBM SPSS Statistics, New York, US) and MedCalc, version 13 (MedCalc Software, Ostend, Belgium).

Results

Differences between remodelling pattern groups

In the study population most of the patients with severe AS referred for TAVI were found to have CH
Table I. Group characteristics with electrocardiographic and echocardiographic findings

| Parameter | Concentric remodelling | Eccentric hypertrophy | Concentric hypertrophy | Normal geometry | P-value* |
|-----------|------------------------|-----------------------|------------------------|----------------|----------|
| Baseline: |                        |                       |                        |                |          |
| Age [years] | 80.6 ±9.1 | 78.0 ±6.7 | 79.7 ±7.5 | 79.1 ±9.5 | NS |
| Female sex, n (%) | 10 (62.5) | 11 (33.3) | 82 (54.7) | 4 (44.4) | 0.023 |
| BMI [kg/m²] | 30.4 ±6.2 | 27.2 ±4.1 | 26.6 ±4.4 | 25.8 ±3.9 | NS |
| BSA (Du Bois) [m²] | 1.9 ±0.2 | 1.8 ±0.1 | 1.8 ±0.2 | 1.8 ±0.2 | NS |
| EuroSCORE I Logistic (%) | 11.1 ±5.8 | 22.0 ±13.3 | 16.7 ±11.8 | 23.5 ±21.2 | 0.044 |
| EuroSCORE II (%) | 3.6 ±2.9 | 6.3 ±5.0 | 4.1 ±3.1 | 5.0 ±3.5 | 0.001 |
| STS (%) | 4 (25.0) | 14 (42.4) | 57 (38.0) | 0 (0.0) | NS |
| Hypertension, n (%) | 15 (93.8) | 21 (63.6) | 108 (72.0) | 5 (55.6) | NS |
| Diabetes, n (%) | 4 (25.0) | 12 (36.4) | 56 (37.3) | 4 (44.4) | NS |
| eGFR < 30 ml/min, n (%) | 2 (12.5) | 5 (15.2) | 13 (8.7) | 1 (11.1) | NS |
| AF , n (%) | 3 (18.8) | 12 (36.4) | 56 (37.3) | 4 (44.4) | NS |
| COPD, n (%) | 2 (12.5) | 7 (21.2) | 26 (17.3) | 1 (11.1) | NS |
| NYHA ≥ III, n (%) | 5 (31.3) | 25 (75.8) | 68 (45.3) | 4 (44.4) | 0.001 |
| CCS ≥ 3, n (%) | 3 (18.8) | 10 (30.3) | 23 (15.3) | 2 (22.2) | NS |
| Myocardial infarction, n (%) | 2 (12.5) | 11 (33.3) | 42 (28.0) | 4 (44.4) | NS |
| PCI, n (%) | 6 (37.5) | 10 (30.3) | 53 (35.3) | 5 (55.6) | NS |
| CABG, n (%) | 0 (0) | 6 (18.2) | 13 (8.7) | 1 (11.1) | NS |
| Stroke/TIA, n (%) | 2 (12.5) | 3 (9.1) | 23 (15.3) | 0 (0.0) | NS |
| Permanent pacemaker, n (%) | 1 (6.3) | 5 (15.2) | 26 (17.3) | 1 (11.1) | NS |
| PAD, n (%) | 2 (12.5) | 11 (33.3) | 22 (14.7) | 0 (0.0) | 0.006 |

Electrocardiography:

| Parameter | Concentric remodelling | Eccentric hypertrophy | Concentric hypertrophy | Normal geometry | P-value* |
|-----------|------------------------|-----------------------|------------------------|----------------|----------|
| QRS [ms] | 104.1 ±25.1 | 107.4 ±43.5 | 102.5 ±34.6 | 90.8 ±61.4 | NS |
| Any AVB, n (%) | 1 (6.3) | 2 (6.1) | 10 (6.7) | 2 (22.2) | NS |
| RBBB, n (%) | 1 (6.3) | 4 (12.1) | 13 (8.7) | 1 (11.1) | NS |
| LBBB, n (%) | 1 (6.3) | 11 (33.3) | 20 (13.3) | 2 (22.2) | 0.004 |

Echocardiography:

| Parameter | Concentric remodelling | Eccentric hypertrophy | Concentric hypertrophy | Normal geometry | P-value* |
|-----------|------------------------|-----------------------|------------------------|----------------|----------|
| Moderate/severe MR, n (%) | 0 (0) | 12 (36.4) | 19 (12.7) | 0 (0.0) | 0.001 |
| RV [mm] | 28.8 ±4.0 | 32.1 ±4.0 | 28.8 ±4.1 | 28.7 ±6.7 | 0.001 |
| IVSd [mm] | 12.8 ±1.8 | 12.1 ±2.4 | 14.5 ±2.1 | 9.4 ±1.9 | 0.001 |
| LVDD [mm] | 38.0 ±10.3 | 60.2 ±5.4 | 47.4 ±1.6 | 51.8 ±7.5 | 0.001 |
| PWTd [mm] | 11.3 ±1.6 | 10.2 ±1.3 | 13.2 ±1.9 | 8.7 ±1.2 | 0.001 |
| LA [mm] | 39.1 ±5.9 | 49.4 ±5.8 | 43.0 ±6.5 | 42.0 ±6.2 | 0.001 |
| EF [%] | 61.2 ±4.8 | 36.3 ±15.0 | 53.0 ±15.0 | 54.9 ±16.5 | 0.001 |
| RWT [mm] | 0.8 ±1.0 | 0.3 ±0.1 | 0.6 ±0.1 | 0.3 ±0.1 | 0.001 |
| LVMI [g] | 160.3 ±30.9 | 287.5 ±54.2 | 268.6 ±71.7 | 167.2 ±31.5 | 0.017 |
| LVMI [g/m²] | 84.6 ±26.7 | 158.7 ±27.9 | 151.7 ±40.5 | 92.5 ±8.2 | 0.044 |
| LVEDV [ml] | 68.2 ±28.8 | 183.2 ±37.1 | 107.0 ±32.5 | 131.6 ±47.0 | 0.001 |
| LVEDVI [ml/m²] | 36.0 ±15.1 | 100.9 ±18.0 | 60.4 ±18.4 | 73.3 ±25.8 | 0.001 |
| BAV, n (%) | 1 (6.3) | 4 (12.1) | 15 (10.0) | 1 (11.1) | NS |
| Aortic annulus [mm] | 22.5 ±3.0 | 23.9 ±5.6 | 22.8 ±3.3 | 25.1 ±2.4 | NS |
| LVOT minimal diameter [mm] | 19.2 ±3.4 | 21.5 ±3.1 | 19.4 ±4.1 | 21.0 ±3.2 | 0.006 |
| AVA [cm²] | 0.7 ±0.2 | 0.9 ±0.8 | 0.7 ±0.3 | 1.2 ±1.4 | NS |
| AVAI [cm²/m²] | 0.4 ±0.1 | 0.5 ±0.5 | 0.4 ±0.1 | 0.5 ±0.1 | 0.024 |
| Vmax [m/s] | 4.2 ±0.5 | 3.8 ±0.7 | 4.6 ±0.7 | 3.6 ±1.1 | 0.001 |
| PG mean [mm Hg] | 51.4 ±22.1 | 36.6 ±14.4 | 51.5 ±18.5 | 36.0 ±15.5 | 0.001 |
| PG max [mm Hg] | 83.6 ±58.2 | 59.8 ±18.4 | 80.1 ±30.1 | 64.7 ±25.3 | 0.001 |

PAD – peripheral artery disease, AVB – atrioventricular block, RWT – relative wall thickness, LVM – left ventricular mass, LVMI – left ventricular mass index, LVEDV – left ventricular end-diastolic volume, LVEDVI – left ventricular end-diastolic volume index, BAV – bicuspid aortic valve, AVA – aortic valve area, AVAI – aortic valve area index, NS – non-significant. *p-value determined by one-way ANOVA.
Table II. Procedural data and pre-discharge echocardiographic findings

| Parameter | Concentric remodelling | Eccentric hypertrophy | Concentric hypertrophy | Normal geometry | P-value* |
|-----------|------------------------|-----------------------|------------------------|-----------------|----------|
| Procedural data: | | | | | |
| Time of the procedure [min] | 208.5 ±18.9 | 216.2 ±67.9 | 209.5 ±47.3 | 200.6 ±34.5 | NS |
| Contrast volume [ml] | 212.4 ±31.2 | 199.5 ±48.0 | 206.0 ±65.1 | 231.7 ±54.5 | NS |
| Time of fluoroscopy [min] | 29.6 ±8.1 | 28.9 ±9.0 | 30.7 ±11.7 | 31.1 ±8.6 | NS |
| Radiation dose [mGy] | 967.9 ±726.9 | 1208.2 ±626.0 | 1215.2 ±731.0 | 1338.6 ±524.3 | NS |
| Cover index [mm] | 16.4 ±11.9 | 12.6 ±12.6 | 15.5 ±10.2 | 9.9 ±7.6 | NS |
| TF, n (%) | 12 (75.0) | 22 (66.7) | 121 (80.7) | 8 (88.9) | NS |
| CV, n (%) | 8 (50.0) | 14 (42.4) | 66 (44.0) | 1 (11.1) | NS |
| ES, n (%) | 3 (18.8) | 5 (15.2) | 23 (15.3) | 1 (11.1) | NS |
| XT, n (%) | 3 (18.8) | 2 (6.1) | 26 (17.3) | 0 (0.0) | NS |
| Lotus, n (%) | 0 (0) | 2 (6.1) | 8 (5.3) | 1 (11.1) | NS |
| EV, n (%) | 0 (0) | 5 (15.2) | 9 (6.0) | 3 (33.3) | NS |
| Predilatation, n (%) | 13 (81.3) | 18 (54.5) | 111 (74.0) | 3 (33.3) | NS |
| Postdilatation, n (%) | 3 (18.8) | 6 (18.2) | 34 (22.7) | 3 (33.3) | NS |
| Echo at discharge: | | | | | |
| Any central regurgitation, n (%) | 3 (18.8) | 3 (9.1) | 11 (7.3) | 2 (22.2) | NS |
| Mean PVL grade | 1.5 ±1.0 | 1.6 ±1.0 | 1.4 ±1.0 | 1.3 ±1.0 | NS |
| Valve frame border (mitral side) [mm] | 3.7 ±2.8 | 7.2 ±2.9 | 6.5 ±2.8 | 8.6 ±3.3 | NS |
| Valve frame border (IVS side) [mm] | 7.9 ±2.9 | 7.1 ±2.8 | 6.4 ±2.8 | 7.1 ±2.2 | NS |
| Number of PVLs | 1.3 ±1.3 | 1.7 ±0.9 | 1.7 ±1.3 | 1.5 ±1.0 | NS |
| AVA [cm²] | 1.7 ±0.3 | 1.7 ±0.3 | 1.7 ±0.2 | 1.8 ±0.3 | NS |
| AVAI [cm²/m²] | 1.3 ±0.6 | 1.5 ±0.8 | 1.2 ±0.7 | 1.2 ±0.4 | NS |
| Vmax [m/s] | 1.9 ±0.5 | 2.0 ±0.3 | 2.2 ±0.5 | 2.0 ±0.4 | NS |
| PG mean [mm Hg] | 10.3 ±6.2 | 8.3 ±3.2 | 10.9 ±6.0 | 9.4 ±4.6 | NS |
| PG max [mm Hg] | 15.1 ±9.6 | 16.4 ±6.1 | 21.1 ±14.5 | 17.1 ±6.8 | NS |

TF – transfemoral access, CV – CoreValve, ES – Edwards Sapien, XT – Edwards Sapien XT, EV – Evolut R, PVL – paravalvular regurgitation, IVS – intraventricular septum, AVA – aortic valve area, AVAI – aortic valve area index, Vmax – peak aortic jet velocity, PG – pressure gradient.

Table III. Postprocedural outcomes defined by VARC-2 criteria

| Parameter | Concentric remodelling | Eccentric hypertrophy | Concentric hypertrophy | Normal geometry | Total | P-value |
|-----------|------------------------|-----------------------|------------------------|-----------------|-------|---------|
| Stroke/TIA | 0 (0%) | 2 (6.1%) | 6 (4.0%) | 0 (0.0%) | 8 (3.8%) | 0.08 |
| Severe PPM | 1 (7.1%) | 1 (3.2%) | 5 (3.3%) | 0 (0.0%) | 7 (3.4%) | 0.81 |
| PM implantation | 2 (14.3%) | 5 (16.1%) | 22 (15.8%) | 0 (0.0%) | 29 (15.2%) | 0.08 |
| PG mean > 20 mm Hg | 3 (21.4%) | 0 (0.0%) | 8 (5.8%) | 0 (0.0%) | 11 (5.8%) | 0.19 |
| Moderate/severe PVL | 2 (14.3%) | 9 (29.0%) | 28 (20.1%) | 1 (14.3%) | 40 (20.9%) | 0.18 |
| 30-day mortality | 2 (12.5%) | 2 (6.1%) | 11 (7.3%) | 2 (22.2%) | 17 (8.2%) | 0.12 |

PPM – patient-prosthesis mismatch, PM – permanent pacemaker.
Table IV. Cox proportional hazard analysis. Factors included in univariate analysis were significantly different between concentric remodelling group and others at $p < 0.1$

| Parameter       | Univariate | Multivariate |
|-----------------|------------|--------------|
|                 | $P$-value  | Hazard (HR)  | 95% confidence intervals | $P$-value  | Hazard (HR)  | 95% confidence intervals |
|                 |            |              | Lower | Upper |            |              | Lower | Upper |
| Weight [kg]     | 0.24       | 1.23         | 0.87  | 1.75  |            |              | Lower | Upper |
| BMI [kg/m²]     | 0.20       | 0.73         | 0.45  | 1.18  |            |              | Lower | Upper |
| Hypertension, n (%) | 0.86   | 1.09         | 0.41  | 2.92  |            |              | Lower | Upper |
| Severe/moderate MR | 0.40   | 1.77         | 1.03  | 3.04  |            |              | Lower | Upper |
| IVSd [mm]       | 0.15       | 0.57         | 0.26  | 1.24  |            |              | Lower | Upper |
| LVDD [mm]       | 0.21       | 0.48         | 0.15  | 1.53  |            |              | Lower | Upper |
| PWDTd [mm]      | 0.99       | 0.99         | 0.08  | 11.87 |            |              | Lower | Upper |
| Ao [mm]         | 0.27       | 1.05         | 0.96  | 1.14  |            |              | Lower | Upper |
| LA [mm]         | 0.69       | 0.99         | 0.91  | 1.06  |            |              | Lower | Upper |
| EF (%)          | 0.73       | 1.01         | 0.98  | 1.04  |            |              | Lower | Upper |
| Concentric remodelling | 0.05 | 4.68         | 0.99  | 21.9  | 0.01       | 4.31         | 1.61  | 11.54 |
| LVM             | 0.25       | 1.04         | 0.97  | 1.11  |            |              | Lower | Upper |
| LVMI            | 0.83       | 0.99         | 0.88  | 1.11  |            |              | Lower | Upper |
| LVEDV [ml]      | 0.05       | 1.04         | 0.86  | 1.26  | 0.01       | 1.01         | 1.00  | 1.02  |
| LVEDVI [ml/m²]  | 0.12       | 1.05         | 0.82  | 1.35  |            |              | Lower | Upper |
| IVS maximal diameter [mm] | 0.16 | 0.92         | 0.81  | 1.03  |            |              | Lower | Upper |
| Valve frame border (IVS side) [mm] | 0.99 | 0.99         | 0.88  | 1.14  |            |              | Lower | Upper |

BMI – body mass index, MR – mitral regurgitation, IVSd – interventricular septum diastolic diameter, LVDD – left ventricular diastolic diameter, PWDTd – posterior wall diastolic thickness, Ao – aorta, LA – left atrium, EF – ejection fraction, LVM – left ventricular mass, LVMI – left ventricular mass index, LVEDV – left ventricular end diastolic volume, LVEDVI – left ventricular end diastolic volume index.
Periprocedural outcomes

In terms of periprocedural proceedings there was a significant difference in pre-dilation number, with CR being the most frequent recipients (81.3%) (Table II). Analysing postprocedural outcomes there were no statistically significant between-group differences, with non-significantly higher rates of pacemaker implantation and stroke in EH patients (Table III).

One-year follow-up results

In 1-year observation the overall, all-cause mortality rate was 19.7%. In the Kaplan-Meier survival plot there were no statistically significant differences in terms of 1-year mortality (log rank 0.11; Figure 1). In the Cox proportional hazard model the presence of CR was a significant predictor of 1-year mortality. After including confounding factors from baseline variables which differed between the predictor and rest of the group with a p-value < 0.10, CR and LVEDV were found to be independent predictors of 1-year mortality (Table IV).

Discussion

In the present study we recognized patients with CR as potentially being at increased risk of mortality after TAVI. The main findings of this observational study were: (1) the most frequent LVH pattern in patients with severe AS was CH; (2) LVH patterns did not have a significant impact on periprocedural complications; (3) CR and LVEDV are independent predictors of 1-year mortality after TAVI.

In previous studies of the effect of remodelling patterns on mortality in patients with AS, the group which was at highest risk was the CH group [7]. In our study we did not find an association between having CH and increased risk of death, but these studies were performed on different AS populations, with ours being an older group just prior to TAVI.

To the best of our knowledge this was the first study to compare periprocedural outcomes after TAVI in different LVH pattern groups. What may be surprising is that the group with the thickest IVS and smallest LVOT diameter (CR) did not have an increase in complications known to be related to those parameters i.e. pacemaker implantations (PM) and moderate/severe paravalvular leak. Although this relation may be mostly due to the relatively small sample size, this may also contribute to the high mean cover index in the CR group (16.4 ±11.9 mm).

In the present study we found a non-significantly higher PM rate in EH patients. Considering the highest percentages of right bundle branch block (RBBB) and LBBB in this group, this result supports earlier reports regarding bundle branch blocks as an important factors affecting the PM rate. Also in the EH group there was a non-significantly higher stroke rate. Whether this is just an aleatory finding or is in some way in concordance...
with the low EF and its potentially thrombogenic effect needs to be confirmed in a larger, randomized trials.

While trying to explain the effect of CR on 1-year mortality, we analysed the differences between pre-procedural and 1-year echocardiograms of the remaining group (Table V). The crucial difference between CR and other groups was that it was the only group which showed a significant LVEDV and LVEDVi increase, driven by a significant increase in LVDD. This also explains the role of LVEDV on mortality found in proportional hazard analysis. Based on these results we can assume that it is not the absolute value of LVEDV that drives the mortality, but the increase of this factor.

The biggest limitation of the current study is that it was a retrospective analysis undertaken on a relatively small group of patients. There was also a lack of mid-term analysis which could enhance the insight into echocardiographic parameter changes of those patients who were still alive. Information regarding patients’ baseline medications as well as biomarker values was unavailable in this study.

Conclusions
Most of the patients with severe symptomatic AS referred for TAVI already have one type of LV remodelling (95.7% of the studied group). The LV remodelling patterns have no influence on the occurrence of periprocedural complications. Concentric remodelling and increase in LVEDV are independent predictors of 1-year mortality after TAVI.

Conflict of interest
The authors declare no conflict of interest.

References
1. Jung B, Baron G, Butchart EG, et al. A prospective survey of patients with valvular heart disease in Europe: The Euro Heart Survey on Valvular Heart Disease. Eur Heart J 2003; 24: 1231-43.
2. Beach JM, Mihaljevic T, Rajeswaran J, et al. Ventricular hypertrophy and left atrial dilatation persist and are associated with reduced survival after valve replacement for aortic stenosis. J Thorac Cardiovasc Surg 2014; 147: 362-9.e8.
3. Lindman BR, Stewart WJ, Pibarot P, et al. Early regression of severe left ventricular hypertrophy after transcatheter aortic valve replacement is associated with decreased hospitalizations. JACC Cardiovasc Interv 2014; 7: 662-73.
4. Une D, Mesana L, Chan V, et al. Clinical impact of changes in left ventricular function after aortic valve replacement: analysis from 3112 patients. Circulation 2015; 132: 741-7.
5. Douglas PS, Hahn RT, Pibarot P, et al. Hemodynamic outcomes of transcatheter aortic valve replacement and medical management in severe, inoperable aortic stenosis: a longitudinal echocardiographic study of cohort B of the PARTNER trial. J Am Soc Echocardiogr 2015; 28: 210-7.e1-9.
6. Orsinelli DA, Aurigemma GP, Battista S, et al. Left ventricular hypertrophy and mortality after aortic valve replacement for aortic stenosis. A high risk subgroup identified by preoperative relative wall thickness. J Am Coll Cardiol 1993; 22: 1679-83.
7. Sato K, Kumar A, Jones BM, et al. Reversibility of cardiac function predicts outcome after transcatheter aortic valve replacement in patients with severe aortic stenosis. J Am Heart Assoc 2017; 6: pii: e005798.
8. 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.
9. 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.
10. Wong DT, Bertaso AG, Liew GY, et al. Relationship of aortic annular eccentricity and paravalvular regurgitation post transcatheter aortic valve implantation with CoreValve. J Invasive Cardiol 2013; 25: 190-5.
11. Giannini F, Montorfano M, Romano V, et al. Valve embolization with a second-generation fully-retrievable and repositionable transcatheter aortic valve. Int J Cardiol 2016; 223: 867-9.
12. Stoklosa R, Szymański P, Dąbrowski M, et al. The impact of transcatheter aortic valve implantation on left ventricular performance and wall thickness – single-centre experience. Postep Kardiol Inter 2015; 11: 37-43.
13. Bagiński M, Kiec-Zawadzki P, Dziewierz A, et al. Early- and mid-term outcomes after transcatheter aortic valve implantation. Data from a single-center registry. Adv Interv Cardiol 2016; 12: 122-7.
14. Bochenek T, Kusz B, Mizia M, et al. Echocardiographic evaluation of myocardial strain in patients after transcatheter aortic valve implantation. Postep Kardiol Inter 2015; 11: 95-9.
15. Capoulade R, Clavel MA, Le Ven F, et al. Impact of left ventricular remodelling patterns on outcomes in patients with aortic stenosis. Eur Heart J Cardiovasc Imaging 2017 Jan 6. pii: jew288.
16. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging 2015; 16: 233-70.
17. Kappetein AP, Head SJ, Généreux P, et al. Updated standardized endpoint definitions for transcatheter aortic valve implantation: the Valve Academic Research Consortium-2 consensus document. EurUroIntervention 2012; 8: 782-95.