Europace (2015) **17**, 1136–1140
doi:10.1093/europace/euu362

Ventricular arrhythmias in aortic valve stenosis before and after transcatheter aortic valve implantation

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Received 30 June 2014; accepted after revision 17 November 2014; online publish-ahead-of-print 20 May 2015

Aims Transcatheter aortic valve implantation (TAVI) is a therapeutic treatment for patients with severe aortic stenosis (AS) at high surgical risk. Although the procedure is associated with a reduction in total mortality, there are no data regarding changing in the incidence of premature ventricular contractions (PVCs) and ventricular arrhythmias (VAs) after TAVI. The aim of this study was to assess the incidence of VAs before and after TAVI.

Methods and results We enrolled 237 patients who underwent TAVI at our centre. Ninety-one patients were excluded for the following reasons: presence of prior permanent pacemaker (PPM) ($n = 20$), new PPM implant after TAVI ($n = 48$), death during the follow-up period ($n = 16$), and lost at follow-up ($n = 7$). Finally, 146 patients were included in our analysis. The presence of VAs was evaluated in all patients recording a 24 h Holter monitoring before the procedure and after 1 and 12 months. Ventricular arrhythmias were classified according to a modified Lown grading system. Before the procedure, isolates PVCs (grade 1–2 of Lown grading system) were present in 34.9% of patients ($n = 51$). Complex PVCs (grade 3–4a–4b of Lown grading system) were present in 48.6% of the population (multifocal PVCs in 32 patients, 21.9%; pairs in 25 patients, 17.1%; ventricular tachycardia in 14 patients, 9.6%). One month after the procedure, we observed statistically significant incidence decrease of arrhythmias of grade 3 (from 21.9 to 17.1%) and grade 4 (pairs from 17.1 to 12.3%; ventricular tachycardia from 9.6 to 4.8%). After 12 months, there was a further significant reduction in the frequency and severity of PVCs. In particular, 45.8% of patients had isolates PVCs (<30 in all given hours of monitoring in 45 patients, 30.8%; higher than 30 in any hour of monitoring in 22 patients, 15%) while the frequency of complex arrhythmias was reduced to 16.4% (multifocal PVCs in 13 patients, 9%; couplets 8 patients, 5.5% and ventricular tachycardia in 3 patients, 2.0%). The difference was statistically significant ($P < 0.01$).

Conclusion This study indicates that VAs are common in patients with AS. We observed a significant decrease in the incidence and severity of PVCs since the first month after TAVI. Furthermore, after 1 year follow-up there was a further and significant reduction in the frequency of complex PVCs. This may be related to the benefits determined by valve replacement on left ventricular function.

Keywords Transcatheter aortic valve implantation • Ventricular arrhythmias • Lown grading system

Introduction

Several studies showed that malignant ventricular arrhythmias (VAs), documented by Holter monitoring, are frequent in patients with aortic stenosis (AS).[1–3] Furthermore, sudden death is observed among symptomatic patients, not suitable for surgery, with an incidence ranging from 8 to 34%.[4] Ventricular arrhythmia has been suggested to play an important role in these fatal outcomes. Transcatheter aortic valve implantation (TAVI) is a relatively new, less invasive treatment for severe, symptomatic AS and is advocated

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What's new?

- Currently we are not aware of other studies that have dealt with the variability of ventricular arrhythmias after transcatheter aortic valve implantation (TAVI).
- Our study shows that patients undergoing TAVI have an improvement of arrhythmic burden.
- It might be interesting evaluate the prognostic value of arrhythmic class in TAVI patients.

as an alternative to conventional surgical aortic valve replacement (SAVR) in patients who are not suitable for surgery due to the perceived high risk of operative mortality.[5–8] Although TAVI improves clinical and hemodynamic conditions, in the majority of these patients, sudden cardiac death remains an adverse outcome with an incidence of 17% in the peri-operative period.[9] To our knowledge, there are no reports in the literature investigating the variability of VAs after TAVI and their potential role in this adverse event. The aim of our study was to assess the severity and the frequency of VAs before and after TAVI.

Methods

Patients population

From June 2007 to November 2012, 237 consecutive patients underwent TAVI and completed 1 year follow-up at Ferrarotto Hospital (Catania, Italy). All patients underwent implantation of the third-generation percutaneous self-expanding CoreValve® prosthesis (CoreValve, Inc.) or the Edwards SAPIEN XT™ prosthesis (ES, Edwards Lifescience LLC).

Exclusion criteria for this retrospective analysis were presence of a preoperative permanent pacemaker (PPM) or PPM implantation during the follow-up, death during the procedure, death within the first year after the procedure, unavailability of baseline or 12 months follow-up data.

The study was conducted according to the Declaration of Helsinki. The local hospital ethics committee approved the protocol and a written informed consent was obtained from all patients or their closest relatives.

Study endpoint

The aim of this study was to analyse the incidence and severity of VAs before TAVI and after 1 and 12 months of follow-up and the factors influencing the postoperative variability of these arrhythmias during the follow-up period.

Procedure and post-procedural management

The technique of TAVI has been described extensively elsewhere.[10–12] In brief, a retrograde transfemoral technique was utilized. In all patients, a temporary pacemaker was placed in the right ventricle by femoral transvenous access. Burst rapid pacing at 180–220 beats/min was used to reduce cardiac motion and transvalvular flow during balloon dilatation. After balloon valvuloplasty, the release system was positioned across the native valve and with the aid of repeated contrast injections and under fluoroscopy guidance, deployed in a stepwise manner. Once the final release was obtained, final aortography was performed in order to assess the implant effectiveness.

The presence of VAs was evaluated in all patients recording a 24 h Holter monitoring before the procedure during hospital stay and after 1 and 12 months as outpatient. Ventricular arrhythmias were classified according to a modified version of the Lown grading system.[13] Ventricular arrhythmias were classified as follow: absence of premature ventricular contractions (PVCs), grade 0; occasional and isolated PVCs, <30 in all given hours of monitoring, grade 1; isolated and frequent PVCs, >30 in any hour of monitoring, grade 2; multifocal PVCs, grade 3; presence of couplets, grade 4a; ventricular tachycardia (VT) defined as three or more PVCs in succession with a frequency of over 100/min, grade 4b.

Severe arrhythmias were defined as grade Lown more than second.

Statistical analysis

This was an observational, retrospective, single-centre study. Continuous variables are reported as mean $\pm$ standard deviation and compared using independent sample Student's *t*-test. Categorical variables are reported as frequencies and compared using χ^2 test. A Friedman's test was used for non-parametric data when more than two related groups differ and a Cochran's Q for the analyses between non-parametric data in two related groups. A Cox multivariate analysis was performed including all the variables that were significant ($P < 0.20$) in the univariate analysis and those deemed clinically important. A P value of < 0.05 was considered statistically significant. All statistical tests were performed using SPSS for Windows 13.0 (SPSS).

Results

Clinical data

According to the criteria mentioned above, we excluded from the analysis a total of 91 patients on the basis of one or more of the following: presence of a preoperative PPM ($n = 20$) or PPM implanted during the follow-up period ($n = 48$); death during the procedure ($n = 6$); death within the first year after the procedure ($n = 10$); unavailability of baseline or 12 months follow-up data ($n = 7$). A total of 146 consecutive patients were finally analysed. Baseline clinical characteristics of these patients are summarized in *Table 1*. Overall, mean age was 80.53 $\pm$ 5.5 years and 92 patients (62.6%) were women. Before the procedure 125 (85%) patients were in sinus rhythm and 21 patients (15%) were in atrial fibrillation. A total of 58 patients (39.5%) were on beta-blockers and 15% of them were on antiarrhythmic drugs at baseline.

In our series, the average ejection fraction was 51.78 $\pm$ 11% (range 20–68), although among all treated patients there were also patients with left ventricular systolic dysfunction.

The high rate of patients with prior balloon aortic valvulopasty (52.4%) reflects our institutional practice in the early period of the TAVI programme.[14]

Incidence and severity of ventricular arrhythmias

Patients were reclassified, according to a modified version of Lown grading system, based on the arrhythmic data recorded during a 24 h Holter monitoring performed before TAVI. Twenty-four patients had no PVCs (grade 0); 48 patients had occasionally PVCs (grade 1); only 3 patients had >30 PVCs per hour (grade 2). Multiform PVCs occurred in 32 patients (grade 3); couplets of PVCs in 25 patients (grade 4a); and VT in 14 patients (grade 4b) (*Table 2*). All the episodes of VT were non-sustained. Patients with severe arrhythmias at baseline were 71 (48.6%).

Table 1 Baseline clinical characteristics

	Overall ($n = 146$)
Age (years), mean ± SD	80.53 ± 5.5
Female, *n* (%)	92 (62.6%)
Hypertension, *n* (%)	125 (85.0%)
Smoke, *n* (%)	46 (31.3%)
Diabetes, *n* (%)	39 (26.5%)
Dyslipidemia, *n* (%)	77 (52.4%)
Prior miocardial infarction, *n* (%)	38 (25.9%)
Coronary artery disease, *n* (%)	25 (31.7%)
Prior percutaneous coronary interventions, *n* (%)	46 (31.3%)
Prior cardiac surgery, *n* (%)	20 (13.6%)
Prior aortic valvuloplasty, *n* (%)	77 (52.4%)
Left ventricular ejection fraction, mean ± SD	51.78 ± 11
Mean pressure gradient, mean ± SD	54.9 ± 13
Aortic valve area (cm^2), mean ± SD	0.56 ± 0.2
Annulus, mean ± SD	20.9 ± 2.5
CoreValve prosthesis, *n* (%)	126 (86.4%)
Edwards prosthesis, *n* (%)	20 (13.6%)
Sinus rhythm, *n* (%)	125 (85%)
Atrial fibrillation, *n* (%)	21 (15%)
Beta-blockers, *n* (%)	58 (39.5%)
ACE inhibitors, *n* (%)	88 (59.9%)
Amiodarone, *n* (%)	14 (9.5%)
Flecainide, *n* (%)	2 (1.4%)
Propafenone, *n* (%)	1 (0.7%)
Sotalol, *n* (%)	1 (0.7%)
Digoxin, *n* (%)	4 (2.7%)

ACE, angiotensin-converting-enzyme.

Variability of ventricular arrhythmias after transcatheter aortic valve implantation

There were no significant differences in antiarrhythmic drugs before and after the procedure (P = NS). The distribution of patients throughout the study period according to the modified Lown grade classification is shown in *Table 2*. Overall, the serial comparison at baseline, 1 and 12 months showed an increase of patients in grade 0 and a reduction of patients in grade 3, 4a, and 4b. These modifications were highly statistically significant among all groups ($P < 0.01$), and within all single groups except for grade 1. In particular, patients in grade 0 increased from 24 (16.4%) at the baseline to 35 (24%) after 1 month and to 55 (38%) after 12 months ($P < 0.01$). Patients in grade 1 were 48 at baseline (32.9%) and after 1 month and decreased to 45 (30.9%) after 12 months (P = NS). Patients in grade 2 increased from 3 (2.1%) at the baseline to 13 (9%) after 1 month and to 22 (15%) after 12 months ($P < 0.05$). Those in grade 3 decreased from 32 (21.9%) to 25 (17.1%) at first month and to 13 (9%) at 1 year ($P < 0.05$). Patients in grade 4a, 25 (17.1%) at the baseline, became 18 (12.3%) after 1 month and then 8 (5.5%) after 12 months ($P < 0.05$). Finally those in class 4b were 14 (9.6%) before TAVI, became 7 (4.8%) after 1 month and 3 (2.1%) after 12 months ($P < 0.01$).

Table 2 Influence of aortic valve replacement on the frequency of ventricular arrhythmias (24 h Holter monitoring)

	Baseline	1 month	12 months	*P* value
Grade 0, *n* (%)	24 (16.4%)	35 (24%)	55 (38%)	<0.01
Grade 1, *n* (%)	48 (32.9%)	48 (32.9%)	45 (30.9%)	NS
Grade 2, *n* (%)	3 (2.1%)	13 (9%)	22 (15%)	<0.05
Grade 3, *n* (%)	32 (21.9%)	25 (17.1%)	13 (9%)	<0.05
Grade 4a, *n* (%)	25 (17.1%)	18 (12.3%)	8 (5.5%)	<0.05
Grade 4b, *n* (%)	14 (9.6%)	7 (4.8%)	3 (2.1%)	<0.01

Grade 0, no premature ventricular contractions (PVCs); grade 1, isolated and occasional PVCs, less than 30/h; grade 2, isolated and frequent PVCs, more than 30/h; grade 3, multiform PVCs; grade 4a, presence of couplets; grade 4b, ventricular tachycardia, defined as three or more PVCs in succession with a frequency of over 100 beat/min; NS, not significant.

Patients with severe arrhythmias decreased from 71 (48.6%) at baseline to 50 (34.2%) at first month and to 24 (16.4%) at the 1 year follow-up ($P < 0.01$).

Our echocardiographic data show an ejection fraction improvement from 51.78 ± 11% at baseline to 53.2 ± 8% after 12 months (P = NS).

Influence of baseline data on the variability of ventricular arrhythmias

In order to identify baseline variables that could be predictor of arrhythmic improvement after TAVI, patients were divided into two different groups: patients who showed a reduction of at least one Lown grade after 12 months were defined as group 1; patients who showed deterioration or did not show changes in Lown grade were defined as group 2. The univariate analysis was performed between preoperative parameters of groups 1 and 2 (*Table 3*). Group 1 enrolled patients with a lower end-systolic diameter, in grade Lown >2 and in sinus rhythm at baseline. The left bundle branch block after TAVI did not seem relevant on this analysis. A Cox multivariate analysis was performed including all the variables, which were significant at $P < 0.20$ in the univariate analysis. At multivariate Cox regression analysis, Lown class >2 resulted the only independent predictor of reduction in frequencies and severity of VAs after 12 months (*Table 4*).

Discussion

Our study evaluated the arrhythmic profile before and after procedure of patients who underwent TAVI, and showed a significant decrease in incidence of severe arrhythmias during long-term follow-up. Moreover, a significant decrease of arrhythmic events was observed in patients with a high baseline profile risk (Lown class >2). There are no data in the literature regarding arrhythmic class variation of patients underwent TAVI.

It is well known the relationship between VAs and AS, corroborating the hypothesis that there is a strong correlation between frequency and complexity of VAs and the risk of sudden death in these patients.

Sorgato *et al.* suggested that the frequency and complexity of VAs is major in patients with higher systolic ventricular overload and

reduced systolic function; the risk of VAs is also correlated to left ventricular hypertrophy.[3] Framingham Heart Study showed that hypertensive patients with left ventricular hypertrophy have an increased risk of premature cardiovascular and sudden death.[15]

Previous studies carried out in patients underwent SAVR, investigate the variation of arrhythmia before and after SAVR and showed a relationship between complex VAs and left ventricular performance.[2,16]

The results of Michel *et al.*[2], in surgical population, showed a decrease of PVCs in long follow-up period. This might be explained with the remodelling process involving the contractile and non-contractile elements of the left ventricle. In particular, Villari *et al.*[17] observed a reduction of 43% in left ventricular mass during the first two years after surgical valve replacement; this early remodelling of the left ventricle was mainly due to a decrease in muscle fibres diameters.

Moreover, in this group of patients was recorded an increase of PVCs early after surgery but these data are not correlate with preoperative or operative data.[17] A recent study of Eckart *et al.*[18] showed a possible mechanism behind the development of sustained VAs after corrective valve surgery. The appearance of VT after valve replacement was observed in a small group of patients, and an electrophysiological study was performed. The VT has been attributed to a scar reentry, to bundle branch reentry or to an outbreak of origin. The scars are often, but not always, located in proximity to valve annulus. Patients with scar reentry were treated with ablation with good success rate.

To our knowledge there are only two reports dealt with VAs in patients underwent TAVI.

Beinart *et al.*[19] published a case of VT in a 90-year-old patient who underwent Edwards prosthesis implantation using the transapical approach. Several days after discharge the patient experienced occasional palpitations; 24 h Holter monitoring showed frequent episodes of wide complex tachycardia. The 12 lead electrocardiogram (ECG) showed a VT with left bundle branch block morphology and inferior axis. The suspect VT origin was the aorto-mitral continuity area. The trigger of this arrhythmia, for the authors, could be a local tissue injury and oedema near the site of valve implantation. Given that the suspected mechanism was likely transient, patient was treated conservatively with beta-blockers, with resolution of symptoms and no more evidence of arrhythmia.

A second case, published by Dvir *et al.*[20], describes the onset of a sustained VT in a 90-year-old patient, eight days after the TAVI procedure. The patient with severe AS, chronic renal failure, and permanent atrial fibrillation underwent implantation of CoreValve prosthesis through transfemoral approach. Eight days after valve implantation, the patient complained palpitations and subsequently lost consciousness. The ECG revealed a monomorphic VT suggesting the origin from the left ventricular outflow tract. The

Table 4 Cox multivariate analysis

	Hazard ratio	CI lower	CI upper	*P* value
Lown pre >2	3.86	1.88	7.93	0.01
LVESD	0.96	0.91	1.02	0.10
Sinus rhythm	2.51	0.83	7.59	0.17

CI, confidence interval; LVESD, left ventricular end systolic diameter.

Table 3 Patients with class variation after 12 months

	Group 1 (*n* = 79, 54.1%)	Group 2 (*n* = 67, 45.9%)	*P* value
Age (years), mean ± SD	80.66 ± 5.2	80.33 ± 5.9	0.7
Female, *n* (%)	51 (64.6)	41 (61.2)	0.7
Smoke, *n* (%)	29 (36.7)	16 (23.9)	0.1
Diabetes, *n* (%)	19 (24.0)	20 (30.0)	0.5
Prior myocardial infarction, *n* (%)	22 (27.8)	15 (22.4)	0.6
Coronary artery disease, *n* (%)[a]	16 (22.8)	8 (12.0)	0.2
Prior percutaneous coronary interventions, *n* (%)	25 (31.6)	20 (29.9)	0.9
Prior cardiac surgery, *n* (%)	9 (11.4)	10 (14.9)	0.6
Prior aortic valvuloplasty, *n* (%)	40 (50.6)	37 (55.2)	0.6
Lown pre >2	48 (60.8)	21 (31.3)	**0.01**
Left ventricular ejection fraction (%)	51.32 ± 11.4	52.21 ± 10.6	0.6
Mean pressure gradient, mean ± SD	59.3 ± 18.7	58.3 ± 15.9	0.72
LVESD, mean ± SD	29.49 ± 6.1	32.09 ± 7.6	**0.03**
LVEDD, mean ± SD	46.09 ± 5.7	46.69 ± 5.9	0.5
Core valve prosthesis, *n* (%)	69 (87.3)	57 (85.1)	0.8
Edwards prosthesis, *n* (%)	10 (12.7)	10 (15.0)	0.8
Sinus rhythm, *n* (%)	71 (89.9)	53 (79.1)	0.1
Atrial fibrillation, *n* (%)	8 (10.1)	14 (20.9)	0.1
Post TAVI left bundle branch block, *n* (%)	31 (39.2)	34 (50.7)	0.2

LVESD, left ventricular end systolic diameter; LVEDD, left ventricular end diastolic diameter.
[a]Stenosis >50% major vessels.

adverse event was not associated with ischaemia or electrolyte disturbances and it was not drug related. The probable cause was the irritation of the myocardium caused by the prosthetic valve.

Our study highlights, from the early stages after TAVI, a significant reduction in severity and frequency of ventricular ectopic activity. After 12 months of follow-up, there is evident reduction of severe VAs (Lown class 3, 4a, 4b) compared with baseline. This result can be explained considering the ventricular remodelling after TAVI.

Another point is the timing of Lown class variation; after TAVI the arrhythmic benefit is more precocious than surgery, where the benefit has been shown only in the long-term follow-up. Probably this early benefit is secondary to a less invasive procedure: TAVI is associated with a lower perioperative catecholaminergic stress and a lower risk of *de novo* scars creation, themselves possible arrhythmic substrate.

Moreover, the arrhythmic improvement does not seem related to post-procedure therapeutic changes and is not compromised by new onset of bundle branch blocks.

In our study, after 12 months, a higher arrhythmic benefit was seen in patients in sinus rhythm and with lower left ventricular diameter. These results could be an indirect expression of a less advanced stage of cardiomyopathy.

In addition, Lown class >2 resulted the only independent predictor of reduction in frequencies and severity of VAs after 12 months. The arrhythmic class, at baseline, does not seem to affect the clinical improvements post-procedure.

Study limitations

This study has some limitations: it is a retrospective study conducted in a highly selected population. In order to complete follow-up of 12 months, we excluded patients died during this period, patients with PPM before and after the procedure and patients who failed to complete the follow-up maybe due to often re-hospitalizations.

The acute effect of after-load relief after TAVI would have been better evaluated by performing Holter monitoring in the close post-intervention period (i.e. 48 h to 1 week), which was not investigated for practical reasons in a proportion of patients and is therefore unavailable for the present analysis.

Moreover, the short duration of 24 h Holter monitoring can be inadequate to detect changes in the arrhythmic burden. Newer Holter monitors are now available with up to 2 weeks of recording capability, but these new devices are currently not available at our institute.

Another limitation regards the use of the Lown classification, which has not robustly correlated with adverse prognostic outcomes in patients with or without VAs. Moreover, information on the total VA burden is missing. However, the Lown classification was selected for the purpose of this study as a standardized tool to allow for an easy and broadly accepted descriptive and quantitative analysis of VAs following the TAVI procedure.

Currently, we are not aware of other studies that have dealt with the variability of VAs after TAVI and this underlines the need for multicenter and randomized studies with surgical population.

Conclusions

Our study shows that patients undergoing TAVI have an improvement of the arrhythmic burden, and this could lead to an improvement of symptoms too. The improvement can be noticed from the first follow-up after the procedure. Summarizing, greater is the arrhythmic class before TAVI greater seems to be the benefit derived. It might be interesting evaluate the prognostic value of arrhythmic class in TAVI patients.

Conflict of interest: none declared.

References

1. Martinez-Useros C, Tornos P, Montoyo J, Permanyer Miralda G, Alijarde M, García del Castillo H *et al.* Ventricular arrhythmias in aortic valve disease: a further marker of impaired left ventricular fuction. *Int J Cardiol* 1992;**34**:49–56.
2. Michel PL, Mandagout O, Vhanian A, Cormier B, Iung B, Luxereau P *et al.* Ventricular arrhythmias in aortic valve disease before and after aortic valve replacement. *Acta Cardiol* 1992;**2**:145–56.
3. Sorgato A, Faggiano P, Aurigemma GP, Rusconi C, Gaasch WH. Ventricular arrhythmias in adult aortic stenosis: prevalence, mechanisms and clinical relevance. *Chest* 1998;**113**:482–91. Reviews.
4. Matthews AW, Barritt DW, Keen GE, Belsey RH. Preoperative mortality in aortic stenosis. *Br Heart J* 1974;**36**:101–3.
5. Leon MB, Smith CR, Mack M, Miller DC, Moses JW, Svensson LG *et al.* Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. *N Engl J Med* 2010;**363**:1597–607.
6. Cribier A, Eltchaninoff H, Tron C, Bauer F, Gerber L. Percutaneous implantation of aortic valve prosthesis in patients with calcific aortic stenosis: technical advances, clinical results and future strategies. *J Invasive Cardiol* 2006;**19**;S88–96.
7. The Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). Guidelines on the management of valvular heart disease. *Eur Heart J* 2012;**33**:2451–96.
8. Grube E, Schuler G, Buellesfeld L, Gerckens U, Linke A, Wenaweser P *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 outcome. *J Am Coll Cardiol* 2007;**50**:69–76.
9. Moreno R, Calvo L, Salinas P, Dobarro D, Santiago JV, Sanchez-Recalde A *et al.* Causes of Peri-Operative Mortality After Transcatheter Aortic Valve Implantation: A Pooled Analysis of 12 Studies and 1,223 Patients. *J Invasive Cardiol* 2011;**23**:180–4.
10. Calvi V, Puzzangara E, Pruiti GP, Conti S, Di Grazia A, Ussia GP *et al.* Early conduction disorders following percutaneous aortic valve replacement. *Pacing Clin Electrophysiol* 2009;**32**:S126–30.
11. Ferreira ND, Caeiro D, Adao L, Oliveira M, Gonçalves H, Ribeiro J *et al.* Incidence and predictors of permanent pacemaker requirement after transcatheter aortic valve implantation with self-expanding bioprosthesis. *Pacing Clin Electrophysiol* 2010;**33**:1364–72.
12. Tamburino C, Capodanno D, Mulè M, Scarabelli M, Cammalleri V, Barbanti M *et al.* Procedural success and 30-day clinical outcomes after percutaneous aortic valve replacement using current third-generation self-expanding CoreValve prosthesis. *J Invasive Cardiol* 2009;**21**:93–8.
13. Ryan M, Lown B, Horn H. Comparison of ventricular ectopic beats during 24-hour monitoring and exercise testing in patients with coronary heart disease. *N Engl J Med* 1975;**292**:224–9.
14. Ussia GP, Capodanno D, Barbanti M, Scarabelli M, Imme S, Cammalleri V *et al.* Balloon aortic valvuloplasty for severe aortic stenosis as a bridge to high-risk transcatheter aortic valve implantation. *J Invasive Cardiol* 2010;**22**:161–6.
15. Levy D, Garrison GJ, Savage DD, Kannel WB, Castelli WP. Prognostic implication of echocardiographically determined left ventricular mass in the Framingham heart study. *N Engl J Med* 1190;**322**:1561–66.
16. Von Olshausen K, Amann E, Hofmann M, Schwarz F, Mehmel HC, Kübler W. Ventricular arrhythmias before and late after aortic valve replacement. *Am J Cardiol* 1984;**53**:142–46.
17. Villari B, Vassalli G, Monrad S, Chiariello M, Turina M, Hess OM. Normalization of diastolic dysfunction in aortic stenosis late after valve replacement. *Am J Cardiol* 1984;**53**:142–46.
18. Eckart RE, Hruczkowski TM, Tedrow U, Koplan BA, Epstein LM, Stevenson WG. Sustained Ventricular tachycardia associated with corrective valve surgery. *Circulation* 2007;**116**:2005–11.
19. Beinart R, Danik S, Palacios I, Barrett C, Inglessis I, Agnihotri A *et al.* Ventricular tachycardia following trans-apical aortic valve replacement. *Europace* 2012;**14**:450–2.
20. Dvir D, Haim M, Kornowski R. Left-ventricular outflow tract ventricular-tachycardia Event Following CoreValve Transcatheter Aortic-Valve Implantation. *Catheter Cardiovasc Interv* 2012;**79**:331–3.