PACEMAKER DEPENDENCE AFTER TRANSCATHETER AORTIC VALVE IMPLANTATION

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ABSTRACT

Transcatheter aortic valve implantation (TAVI) has emerged as an alternative for multimorbid patients not suitable for open heart surgery. The vicinity of the conduction system, especially the atrioventricular node and His bundle to the non-coronary and right coronary aortic cusp, predisposes these patients to conduction abnormalities. However, due to the shape of both available transcatheter aortic valves (CoreValve and Edwards SAPIEN valve) these rates are different. To date, there is no clear information about the true rate of atrioventricular block, the significance of left bundle branch block as well as the transient or permanent nature of these conduction disorders. Due to this, the rate of subsequent pacemaker implantation exceeds up to 50%, which itself may be associated with worse clinical outcomes. Thus, there is a need for further data from large-scale series with a glance to the true rate of clinically relevant conduction disorders.

INTRODUCTION

Calcific aortic stenosis is the most frequent expression of valvular heart disease in the Western world. Population-based observational studies have revealed that 1-2% of patients over 65 years have moderate to severe aortic stenosis.[1] Increased valve cusp thickness due to fibrosis and lipid accumulation, but without left ventricular outflow tract obstruction, is known as aortic valve sclerosis (Figure 1). It is a progressive disease that starts with initial changes in the cell biology of the valve leaflets, which develop into atherosclerotic-like lesions and aortic sclerosis, and eventually lead to calcification of the valve, causing left ventricular outflow tract obstruction. Even mild aortic stenosis is associated with adverse outcomes, with a 50% increased risk of cardiovascular death.[2] There are no known therapies that slow disease progression. Thus, current guidelines consider aortic valve replacement as a class I indication for symptomatic patients,[3,4] facing, however, the fact that one-third of patients are considered to have an unacceptably high risk for open surgery.[5] Current treatment options for those patients include medical treatment and percutaneous
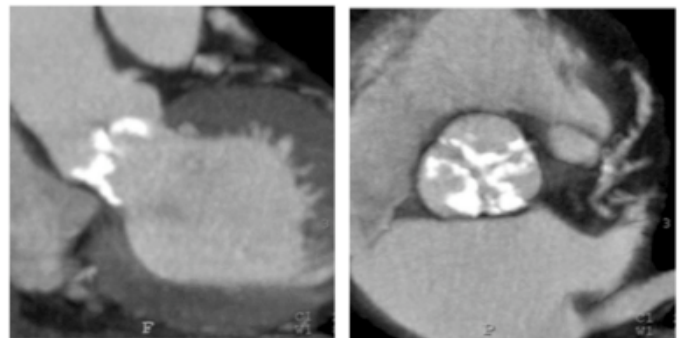
Figure 1. Calcific aortic valve stenosis in long-axis and short axis. The non-coronary cusp calcification extends to subvalvular regions, where the conduction system is located within the triangle of Koch.

balloon aortic valvuloplasty, although neither has been shown to reduce long-term mortality of medically treated patients with symptomatic aortic stenosis, with a 1 and 5-year survival rate of 60% and 32% respectively, and only minor short-term benefits were reported after balloon aortic valvuloplasty.[6-8] The search for a less invasive treatment option for patients with severe aortic stenosis was pioneered by Andersen et al.,[9] subsequently, the feasibility

Figure 2. Surface electrocardiogram of a patient before (A) and 2 days (B) after implantation of a CoreValve (A).

of percutaneous prosthetic valve delivery was demonstrated by others[10-13] and in 2000 Bonhoeffer et al.[14] described the first successful implantation of a catheter-based stent valve in a pulmonary conduit. Transcatheter aortic valve implantation (TAVI) has recently been developed to minimise surgical risk in high-risk patients with severe symptomatic aortic stenosis who are refused for conventional open aortic valve replacement. The anatomical proximity of the conduction system to the aortic annulus atrioventricular block, with subsequent pacemaker requirement, was described in 6% of cases after surgical aortic valve replacement, but varies after TAVI between 5.7% and 42.5%, while new left bundle branch block occurs in up to 50-70%[15-19] (Figure 2). Better prediction of pacemaker requirement would be of considerable benefit in patients undergoing TAVI with respect to potential needs and duration of postoperative monitoring.

ANATOMICAL CONSIDERATION

The aortic valve is normally composed of three cusps or leaflets. The individual cusps are attached to the aortic wall in a semilunar fashion, ascending to the commissures and descending to the basal attachment of each cusp to the aortic wall. The valvar leaflets and their supporting sinuses, which together make up the root, are related to all four cardiac chambers. Within the right atrium, the atrioventricular node is located within the triangle of Koch. This important triangle is demarcated

by the tendon of Todaro, the attachment of the septal leaflet of the tricuspid valve, and the orifice of the coronary sinus. The apex of this triangle is occupied by the atrioventricular component of the membraneous septum. The atrioventricular node is located just inferior to the apex of the triangle adjacent to the membranous septum, and therefore, the atrioventricular node is in close proximity to the subaortic region and membranous septum of the left ventricular outflow tract. Thus, pathologies involving the aortic valve can lead to complete heart block or intraventricular conduction abnormalities. The atrioventricular node continues as the bundle of His, piercing the membraneous septum and penetrating into the left through the central fibrous body. The branching bundle is intimately related to the base of the interleaflet triangle that separates the non-coronary and right coronary leaflets of the aortic valve.

CONDUCTION DISORDERS

Aortic valve disease has been associated with cardiac conduction system disease, as aortic stenosis and insufficiency have been associated with both prolonged atrioventricular conduction times and higher degrees of atrioventricular block.[20-22] Due to the vicinity of the aortic valve and atrioventricular node, as well as His bundle, complete atrioventricular block was reported in 5.7%, new left bundle branch block occurred in 18% with an association to complete atrioventricular block,
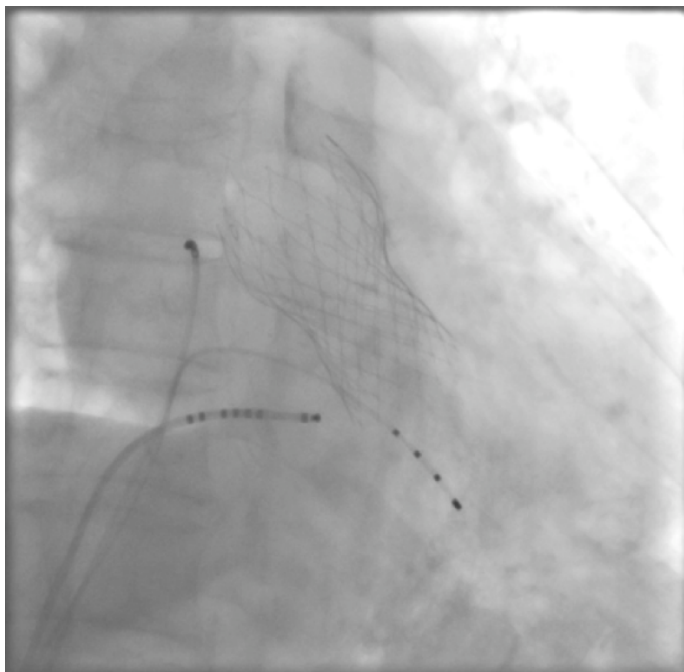
Figure 3. Setting of intracardiac measurements with a lead in right atrium, a lead in right ventricle and a lead in His bundle to measure intracardiac conduction.

syncope, and sudden cardiac arrest at long-term after open surgery.[23,24] Such conduction disturbances are presumed to result from surgical trauma to the cardiac conduction tissue during debridement of the calcified annulus.[23,24] Risk factors for complete atrioventricular block after surgical aortic valve replacement include previous aortic regurgitation, myocardial infarction, pulmonary hypertension, and postoperative electrolyte imbalance,[24,25] while among electrocardiographic criteria right bundle branch block was the strongest predictor for pacemaker requirement.[24,25] Several investigations report changes in surface electrocardiogram after TAVI.[15,26-31] The incidence of permanent pacemaker implantation after TAVI with the CoreValve system has been reported in 20% to 42.5%, and that of a new left bundle branch block in 50% to 70% [5,26-31]. Nevertheless, with the balloon-expandable, shorter Edwards SAPIEN prosthesis, which is placed in the aortic annulus without direct impact on left ventricular outflow tract, the incidence of atrioventricular conduction block requiring a pacemaker was reported between 0% to 6%, and new onset left bundle branch block of 3.3%.[32,33]

Differences to surgical aortic valve replacement might be due to the different techniques. In surgical approach the valve is replaced by another. Thus,

the amount of conduction damage is predictable because the local trauma is nearly the same in all patients. However, in TAVI the amount of local damage is dependent of local calcification, the height of implantation in left ventricular outflow tract, the extent of trauma during index procedure (balloon valvuloplasty, balloon-to-aortic annulus relation, post-TAVI dilatation) and from further aortic annulus geometry. Degenerative calcification of the aortic and mitral annulus is probably a diffuse process, in which the cardiac conduction system is often involved and making it vulnerable to injury when exposed to mechanical compression by the nitinol frame of the CoreValve, which seems to completely expand within the first 7-10 days.[31] Jilaihawi et al. reported first that pacemaker requirement after TAVI correlates to left axis deviation at baseline, left bundle branch block, baseline thickness of the native non-coronary cusp and to diastolic interventricular septal dimension >17 mm.[30] Similarly, Piazza et al. revealed no prosthesis-related left bundle branch block when the proximal end of the valve frame was positioned <6.7 mm from the lower edge of the non-coronary cusp.[27] In the study by Marcheix et al. 30% of patients required pacemaker implantation due to persistent atrioventricular block,[34] whereas Zahn et al. reported a permanent pacemaker rate of 42.5% in the German Transcatheter Aortic Valve Intervention-Registry.[16] Different rates of pacemaker implantation might be due to different indications for pacing (e.g. complete atrioventricular block, new left bundle branch, prolonged atrioventricular conduction). However, to date there is no evidence about the occurrence of left bundle branch block. Additionally, there is no information about the true long-term occurrence of relevant conduction disturbances and the permanent or transient nature of conduction disorders. A comparison of hard endpoints like high-grade atrioventricular block would be more convincing. Other reasons for different pacemaker implantation rates might be the learning curve with high implantation techniques resulting in less compromise of the compact atrioventricular node.

Akin et al.[35,36] was the first to describe intracardiac conduction abnormalities for better discrimination of new electrocardiographic changes on surface electrocardiogram, and to predict critical conduction delays (Figure 3, 4). The evolution to complete atrioventricular block and to left bundle branch block took place over an observation period of 7 days. Similarly, PQ interval and QRS duration, as well as AH and HV intervals prolonged. In the series of Akin et al, complete atrioventricular block was seen
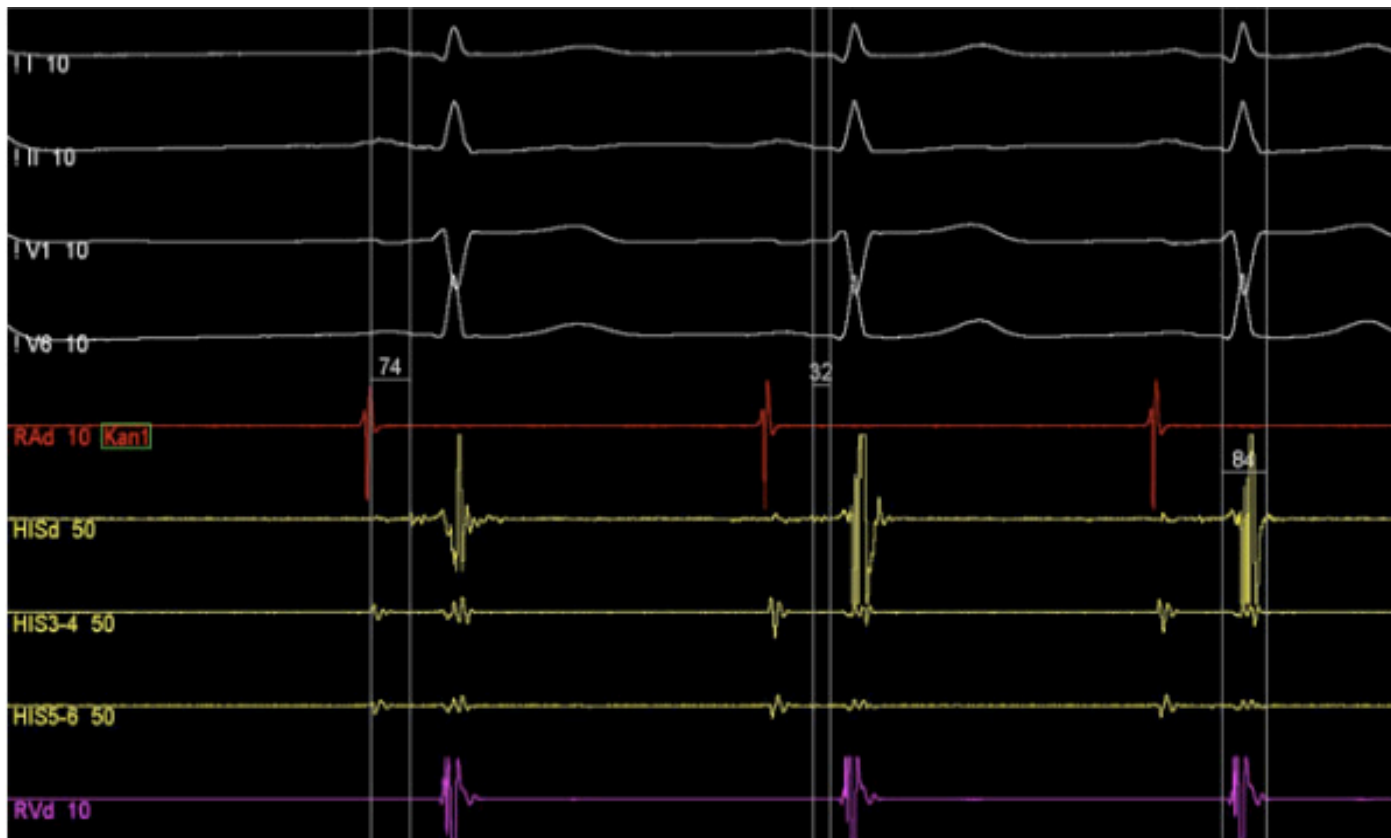
Figure 4. Intracardiac traces in a patient with normal AH and HV conduction.

in 13.3%, while 8.9% suffered from type II second-degree atrioventricular block; thus, 22.2% of patients developed an indication for permanent pacemaker implantation corroborating previous findings.[27-29,37-41] Their intracardiac measurements revealed that occurrence of first-degree atrioventricular block were predominantly due to prolongation of HV interval, which might be prognostically relevant.[42] Scheinman et al. have shown that patients with an HV interval greater than 100 msec are a at high risk to develop complete atrioventricular block.[42] Therefore, the possibility of progression of left bundle branch block to complete atrioventricular block should always be considered, and may explain the liberal use of pacemakers for conduction disorders observed in our series of TAVI patients. This liberal approach may be debatable, but in elderly patients with several comorbidities, preventive pacemaker insertion is justified by guideline recommendation.[43] Piazza et al. showed that some of the initial conduction delay after TAVI was partially reversible at 1 month follow-up and presumably related to inflammation and oedema around the conduction pathways;[27,31] Akin et al. could not identify a single case of conduction recovery.

The multivariate analysis of Akin et al. revealed that only PQ duration >200 msec, a left bundle branch block and QRS duration >120 msec immediately (within 60 minutes) after CoreValve implantation, seem to predict critical atrioventricular conduction delay. Other baseline clinical and electrocardiographic parameters had no impact. The occurrence of above electrocardiographic findings soon after TAVI may reflect the extent of trauma from the procedure. Interestingly, the exact determination of both the amount of valve calcification and the height of implantation turned out to be non-reproducible although both parameters have been claimed to impact on conduction physiology.[27,30] For example, the Edwards SAPIEN valve, shorter and less likely to extend into the left ventricular outflow tract, is obviously associated with a lower rate of complete AV block (0-6%).[33,44]

As demonstrated by Akin et al., we believe that, regardless of favourable anatomy, only the extent of trauma predict the occurrence of critical conduction delay after TAVI. However, to diminish trauma to the conduction system by TAVI using the CoreValve revalving system may reduce the risk of conduction abnormalities. Such strategies may include limiting the depth of the valve within the left ventricular outflow tract and keeping the number of pre and

post-valve implantation balloon valvuloplasties to a minimum. Additionally, operators should deploy the device only a few millimetres below the annulus and avoid impacting the septum. A modified implantation technique, however, may also require technical modifications to avoid malalignment of valves.

REFERENCES

1. Nkomo VT, Gardin JM, Skelton TN, et al. Burden of valvular heart disease: a population-based study. Lancet. 2006;368:1005-11.

2. Lloyd-Jones D, Adams R, Carnethon M, et al. Heart disease and stroke statistics-2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation. 2009;119:480-6.

3. Vahanian A, Baumgartner H, Bax J, et al. Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology; ESC Committee for Practice Guidelines. Guidelines on the management on valvular heart disease of the European Society of Cardiology. Eur Heart J. 2007;28:230-68.

4. Bonow RO, Carabello BA, Chatterjee K, et al. American College of Cardiology/American Heart Association Task Force on Practice Guidelines ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines: Developed in collaboration with the Society of Cardiovascular Anaesthesiologists: Endorsed by the Society for Cardiovascular Angiography and interventions and society of thoracic surgeons. Circulation. 2008;52:e1-142.

5. Lung 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.

6. Varadarajan P, Kapoor N, Bansal RC, et al. Clinical profile and natural history of 453 nonsurgically managed patients with severe aortic stenosis. Ann Thorac Surg. 2006;82:2111-5.

7. Shareghi S, Rasouli L, Shavelle DM, et al. Current results of balloon aortic valvuloplasty in high-risk patients. J Invasive Cardiol. 2007;19:1-5.

8. Sack S, Kahlert P, Khandanpour S, et al. Revival of an old method with new techniques: balloon aortic valvuloplasty of the calcified aortic stenosis in the elderly. Clin Res Cardiol. 2008;97:288-97.

9. Andersen HR, Knudsen LL, Hasenkam JM. Transluminal implantation of artificial heart valves. Description of a new expandable aortic valve and initial results with implantation by catheter technique in closed chest pigs. Eur Heart J. 1992;13:704-8.

10. Boudjemline Y, Agnoletti G, Bonnet D, et al. Percutaneous pulmonary valve replacement in a large right ventricular outflow tract: an experimental study. J Am Coll Cardiol. 2004;43:1082-7.

11. Cribrier A, Eltchaninoff H, Bash A, et al. Trans-catheter implantation of balloon-expandable prosthetic heart valves. Early results in an animal model. Circulation. 2001;104(Suppl 2):I552.

12. Lutter G, Kuklinski D, Berg G, et al. Percutaneous aortic valve replacement: an experimental study. I. Studies on implantation. J Thorac Cardiovasc Surg. 2002;123:768-76.

13. Sochman J, Peregrin JH, Pavcnik D, et al. Percutaneous transcatheter aortic disc valve prosthesis implantation: a feasibility study. Cardiovasc Intervent Radiol. 2000;23:384-8.

14. Bonhoeffer P, Boudjemline Y, Saliba Z, et al. Percutaneous replacement of pulmonary valve in a right-ventricle to pulmonary-artery prosthetic conduit with valve dysfunction. Lancet. 2000;356:1403-5.

15. Kodali SK, Williams MR, Smith CR, et al. PARTNER Trial Investigators: Two-year outcomes after transcatheter or surgical aortic valve replacement. N Engl J Med. 2012;366:1686-95.

16. Zahn R, Gerckens U, Grube E, et al. German Transcatheter Aortic Valve Interventions-Registry Investigators: Transcatheter aortic valve implantation: first results from a multi-centre real-world registry. Eur Heart J. 2011;32:198-204.

17. 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.

18. Webb JG, Chandavimol M, Thompson CR, et al. Percutaneous aortic valve implantation retrograde from the femoral artery. Circulation. 2006;112:842-50.

19. Bates MG, Matthews IG, Fazal IA, et al. Postoperative permanent pacemaker implantation in patients undergoing trans-catheter aortic valve implantation: what is the incidence and are there any predicting factors? Interact Cardiovasc Thorac Surg. 2001;12:243-53.

20. Yater WM, Cornell VH. Heart block due to calcareous lesions of the bundle of His. Ann Int Med. 1935;8:777.

21. Ablaza SG, Blanco G, Maranhao V, et al Calcific aortic valvular disease associated with complete heart block. Case reports of successful correction. Dis Chest. 1968;54:457-60.

22. Schwartz LS, Goldfischer J, Sprague GJ, et al. Syncope and sudden death in aortic stenosis. Am J Cardiol. 1969;23:647-58.

23. El-Khally Z, Thibault B, Stainloae C, et al. Prognostic significance of newly acquired bundle branch block after aortic valve replacement. Am J Cardiol. 2004;94:1008-11.

24. Marchenese K, Schenk EA. Atrioventricular conduction system lesion following cardiac valve replacement. Circulation. 1972;45-46(Suppl. II):II-188.

25. Copeland JG, Griepp RB, Stinson EB, et al. Long-term follow-up after isolated aortic valve replacement. J Thorac Cardiovasc Surg. 1977;74:875-89.

26. Erkapic D, De Rosa S, Kelava A, Lehmann R, Fichtlscherer S, Hohnloser SH. Risk for permanent pacemaker after transcatheter aortic valve implantation: a comprehensive analysis of the literature. J Cardiovasc Electrophysiol. 2012;23:391-7.

27. 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. JACC Cardiovasc Interv 2008;1:310-6.

28. 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.

29. Calvi V, Puzzangara E, Pruiti GP, et al. Early conduction disorders following percutaneous aortic valve replacement.

Pacing Clin Electrophysiol. 2009;32 (Suppl 1):S126-30.

30. 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.

31. Piazza N, Nuis RJ, Tzikas A, et al. Persistent conduction abnormalities and requirements for pacemaking six months after transcatheter aortic valve implantation. EuroIntervention. 2010;6:475-84.

32. Sinhal A, Altwegg L, Pasupati S, Humphries KH, et al. Atrioventricular block after transcatheter balloon expandable aortic valve implantation. J Am Coll Cardiol Intv. 2008;1:305-9.

33. Walther T, Falk V, Kempfert J, et al. Transapical minimally invasive aortic valve implantation; the initial 50 patients. Eur J Cardiothorac Surg. 2008;33:983-8.

34. Marcheix B, Lamarche Y, Berry C, et al. Surgical aspects of endovascular retrograde implantation of the aortic CoreValve bioprosthesis in high-risk older patients with severe symptomatic aortic stenosis. J Thorac Cardiovasc Surg. 2007;134:1150-6.

35. Akin I, Kische S, Schneider H, et al. Surface and intracardiac ECG for discriminating conduction disorders after CoreValve implantation. Clin Res Cardiol.

2012;5:357-64.

36. Akin I, Kische S, Paranskaya L, et al. Predictive factors for pacemaker requirement after transcatheter aortic valve implantation. BMC Cardiovasc Disord. 2012;Oct 4;12:87.

37. Fraccaro C, Buja G, Tarantini G, et al. Incidence, predictors, and outcome of conduction disorders after transcatheter self-expandable aortic valve implantation. Am J Cardiol. 2011;107:747-54.

38. Roten L, Wenaweser P, Delacretaz E, et al. Incidence and predictors of atrioventricular conduction impairment after transcatheter aortic valve implantation. Am J Cardiol. 2010;106:1473-80.

39. Ferreira ND, Caeiro D, Adão L, 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.

40. Khawaja MZ, Rajani R, Cook A, et al. Permanent pacemaker insertion after CoreValve transcatheter aortic valve implantation: incidence and contribution factors (the UK CoreValve Collaborative). Circulation. 2011;123:951-60.

41. Buellesfeld L, Stortecky S, Heg D, et al. Impact of permanent pacemaker implantation on clinical outcome among patients undergoing transcatheter aortic valve implantation. J Am Coll Cardiol.

2012;60:493-501.

42. Scheinman MM, Peters RW, Modin G, et al. Prognostic value of infranodal conduction time in patients with chronic bundle branch block. Circulation. 1977;56:240-4.

43. Epstein AE, DiMarco JP, Ellenbogen KA, et al. American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices); American Association for Thoracic Surgery; Society of Thoracic Surgeons ACC/AHA/HRS 2008 Guidelines dor Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the Association for Thoracic Surgery and Society of Thoracic Surgeons. J Am Coll Cardiol. 2008;51:e1-62.

44. Erkapic D, Kim WK, Weber M, et al. Electrocardiographic and further predictors for permanent pacemaker requirement after transcatheter aortic valve implantation. Europace. 2010;12:1188-90.