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ECG Abnormalities After Transcatheter Aortic Valve Implantation

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Transcatheteraortic valve implantation, first introduced in 2002, has been established as an alternative modality for patients deemed not suitable for open-heart surgery. The anatomical vicinity of the atrioventricular node and the His bundle to the non-coronary and rightcoronary aortic cusps predisposes patients to conduction abnormalities in case of severe calcification or mechanical trauma during valve implantation. However, the two evaluated valves (CoreValve and Edwards SAPIEN valve) have different rates of these complications, mainly driven by their respective geometry.
Currently, there is ongoing evaluation of the true rate of conduction disorders and their clinical relevance or durability. The initial experience of fatal outcomes with conduction disorders such as complete atrioventricular block has increased the rate of subsequent pacemaker implantation up to 50%. However, prophylactic pacemaker implantation is associated with several possible complications. Thus, there is a need for further data from large-scale series taking into account the true rate of clinically relevant conduction disorders.

*Keywords:*Heart Valve Prosthesis Implantation; Atrioventricular Block; Bundle-Branch Block; Pacemaker; Artificial; Bundle of His

1. Introduction

Degenerative calcific aortic stenosis, which is an active inflammatory process, is the most frequent valvular heart disease in the Western world. It is estimated that 1-2% of patients aged over 65 years have moderate to severe aortic stenosis, whereas this rate increases to up to 12% in patients aged over 85 years (1). The preliminary stage, characterized by fibrosis, inflammation, and lipid accumulation, but without left ventricular outflow tract obstruction, is known as aortic valve sclerosis (Figure 1). It is a progressive disease, with a long-term asymptomatic phase, that starts with initial changes in the cell biology of the valve leaflets. It thereafter develops into atherosclerotic-like lesions and aortic sclerosis and eventually leads to the calcification of the valve, causing left ventricular outflow tract obstruction and symptoms like angina, syncope, and dyspnea. Even mild symptomatic 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. Even the use of Statins, which have a cholesterol effect and an anti-inflammatory effect, is not associated with better clinical outcomes than with placebo. Due to worse clinical outcomes, the current guidelines consider aortic valve replacement as a class I indication for symptomatic patients (3, 4). Nevertheless, one third of patients are considered to have an unacceptably high risk for open-heart surgery (5). The current treatment options for those patients include medical treatment and percutaneous balloon aortic valvuloplasty. However, neither has been shown to reduce long-term mortality with a one- and 5-year survival of 60% and 32%, respectively (6-8). A less invasive treatment option for patients with severe aortic stenosis was pioneered by Andersen et al. (9) and, subsequently, the feasibility of percutaneous prosthetic valve delivery was demonstrated by others (10-14). Transcatheter aortic valve implantation (TAVI) was developed one decade ago to minimize surgical risk in high-risk patients with severe symptomatic aortic stenosis refused for conventional open aortic valve replacement. With the anatomical proximity of the atrioventricular node to the aortic annulus, conduction disorders caused by calcification or mechanical trauma might result in atrioventricular block with subsequent pacemaker requirement. In literature, this is 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). A better understanding of the underlying conduction disorders and their dura-

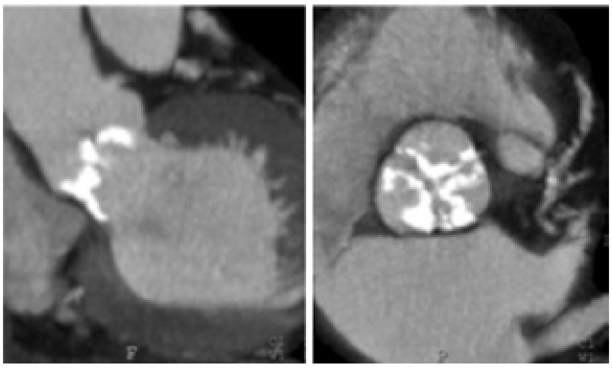
Implication for health policy/practice/research/medical education:
The transcatheter aortic valve implantation is an established tool for the treatment of multimorbid patients suffering from severe symptomatic aortic valve stenosis. However, it is much more associated with with pacemaker dependence as compared to classic open aortic valve surgery. Within this review we try to explain the differences between the interventional procedure and the open surgical procedure as well as the difference between different vlave types.

bility would help optimize ancillary therapy with the pacemaker and thus result in better clinical outcomes.

2. Anatomical Consideration

The aortic valve, which consists in the majority of cases of three cusps, is attached to the aortic wall. The valvular leaflets and their supporting sinuses, which together make up the root, are related to all four cardiac chambers. The atrioventricular node and the His bundle are located within the apex of the triangle of Koch, which itself is in the upper right atrium near the tricuspid valve and septum. The borders of this triangle are the tendon of Todaro, attachment of the septal leaflet of the tricuspid valve, and orifice of the coronary sinus. The atrioventricular node, which is near the apex of this triangle, is in close proximity to the subaortic region and the membranous septum of the left ventricular outflow tract. Thus, severe calcification, infectious diseases, and mechanical trauma to this region can induce conduction abnormalities like complete heart block. The atrioventricular node continues as the bundle of His, which is located in the membranous septum and branches into the left and right bundle.

Figure 1. Calcific Aortic Valve Stenosis in Long Axis and Short Axis



The non-coronary cusp calcification extends to the subvalvular regions, where the conduction system is located within the triangle of Koch.

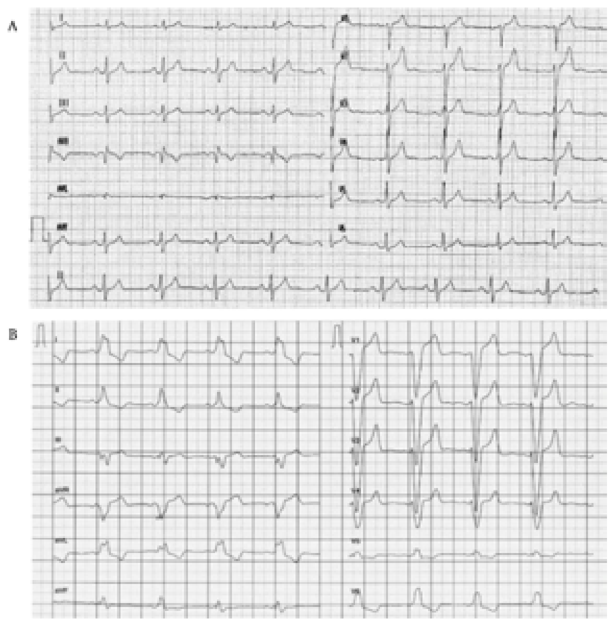


Figure 2. Surface Electrocardiograms of a Patient Before (A) and 2 Days (B) After the Implantation of a CoreValve (A)

3. Conduction Disorders

Aortic valve insufficiency as well as aortic valve stenosis has been associated with both prolonged atrioventricular conduction times and higher degrees of atrioventricular conduction disorders (20-22). The anatomical vicinity of the aortic valve and the atrioventricular node as well as the His bundle will lead to complete atrioventricular block in 5.7% and new left bundle branch block in 18% at long term after open-heart surgery (23, 24). Such complications are caused by surgical trauma to the cardiac conduction tissue during the preparation of the calcified annulus (23, 24). Predictive factors for complete atrioventricular block after conventional aortic valve replacement include previous aortic regurgitation, myocardial infarction, pulmonary hypertension, and postoperative electrolyte imbalance (24, 25). Among the electrocardiographic (ECG) criteria, right bundle branch block is the strongest predictor for pacemaker requirement (24, 25). To date, some investigations have reported descriptive changes in the surface ECG after TAVI (15, 26-31). In the published trials, the incidence of permanent pacemaker implantation after TAVI with the CoreValve system has been reported to be 20% to 42.5%, and that of a new left bundle branch block has been reported to range between 50% and 70% (5, 26-31). Nevertheless, with the balloon-expandable shorter Edwards SAPIEN prosthesis, which is placed in the aortic annulus without direct impact on the left ventricular outflow tract, the incidence of atrioventricular conduction block requiring pacemaker has been reported between 0% and 6% and the incidence of new-onset left bundle branch block has been reported to have a rate of 3.3% (32, 33).

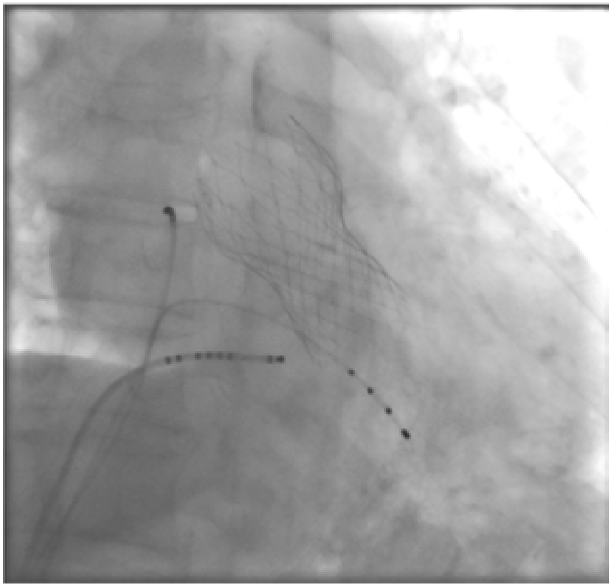
The discrepancies in the surgical technique might be explained by the amount of mechanical trauma. In the surgical approach, the amount of conduction damage is predictable because the local trauma is nearly the same in all patients. However, in TAVI, which displaces the anatomical structures, the amount of local damage is influenced by local calcification, height of implantation in the left ventricular outflow tract, and extent of trauma during the index procedure (i.e. balloon valvuloplasty, balloon-to-aortic annulus relation, and post-TAVI dilatation). Additionally, the calcification of the aortic and mitral annulus is probably not restricted to the valves and will impact on the membranous septum (31). In one study, the predictive factors for pacemaker requirement were determined by left axis deviation at baseline, left bundle branch block, baseline thickness of the native non-coronary cusp, and diastolic interventricular septal dimension > 17 mm (30). Similarly, another study revealed that if the proximal end of the valve frame was positioned < 6.7 mm from the lower edge of the non-coronary cusp, no prosthesis-related left bundle branch block would occur (27). Several groups have reported different rates of pacemaker implantation (15, 16), which might be due to different indications for pacing (e.g. complete atrioventricular block, new left bundle branch, and prolonged atrioventricular conduction). However, to date, there has been no evidence of the occurrence of left bundle branch block. Additionally, no information exists about the true long-term occurrence of relevant conduction disturbances and the permanent or transient nature of conduction disorders. Other reasons for the high pacemaker implantation rate might be the implantation technique. An implantation approach in which the valve would be implanted high with less compromise of the left ventricular outflow tracts and with this the compact AV ode would result in less pacemaker rates but would have the risk of valve dislocation.

Our group was the first to describe intracardiac conduction abnormalities for a better discrimination of new changes on the surface ECG (34) (Figures 3 and 4). The evolution of conduction disorders took place over a period of 7 days after implantation. In our series, complete atrioventricular block was seen in 13.3%, while 8.9% suffered from type II second-degree atrioventricular block; thus, 22.2% of the patients developed an indication for permanent pacemaker implantation corroborating previous findings (27-29, 35-39). Their intracardiac measurements revealed that the occurrence of first-degree atrioventricular block was predominantly due to the prolongation of HV interval, which might be prognostically relevant (40). However, Scheinman et al. showed that patients with an HV interval > 100 msec were at high risk to develop complete atrioventricular block (40). Therefore, the possibility of the progression of left bundle branch block and atrioventricular block grade I to complete atrioventricular block should always be considered and let us decide to a more liberal use of pacemakers for the 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 recommendations (41). In contrast to the other groups, we could not identify a recovery of conduction disorders (27, 31).

Our multivariate analysis revealed that only a PQ duration > 200 msec, left bundle branch block, and QRS duration > 120 msec immediately (within 60 minutes) after CoreValve implantation seemed to predict critical atrioventricular conduction delay without any impact of the other baseline parameters. This is plausible because the occurrence of the above predictive factors soon after TAVI may reflect the extent of trauma from the procedure. Interestingly, the exact determination 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). The importance of height was shown by the lower rates of complete atrioventricular block with the use of the Edwards SAPIEN valve (0-6%), which is shorter and less likely to extend into the left ventricular outflow tract (33, 42).

Thus, we believe that regardless of favorable anatomy, only the extent of trauma predicts the occurrence of critical conduction delay after TAVI. However, to diminish trauma to the conduction system by TAVI using the CoreValve Revalving System, several strategies may be helpful. 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 experienced in height implantation techniques will reduce the mechanical trauma to the apex of the Koch triangle.

Figure 3. Setting of Intracardiac Measurements with a Lead in the Right Atrium



A lead is placed in the right ventricle and a lead in the His bundle to measure intracardiac conduction.

Figure 4. Intracardiac Traces in a Patient with Normal AH and HV Conduction

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Authors' Contribution

Ibrahim Akin and Christoph A. Nienaber treated the patients and wrote the manuscript.

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