(lengthening of QT interval correct with Bazett formula with amiodarone and sotalol and significant heart rate bradycardia with beta-blockers therapy). The patient was admitted to our centre for catheter ablation of arrhythmic substrate. Access to the cardiac chambers was attempted through the left femoral vein and artery. A four-pole catheter (Dynamic XT steerable BARD Electrophysiology, Lowell, MA, USA) was placed in the coronary sinus, a four-pole catheter (Supreme, St Jude Medical, St Paul, MN, USA) was placed in right ventricular apex, and an irrigated 4 mm tip ablation catheter (Cool Path Duo, St Jude Medical) was used for geometrical reconstruction of the right and left ventricles. The electroanatomical mapping and ablation was obtained by a non-fluoroscopic navigation system (Ensite NavX, St Jude Medical) without use of fluoroscopy. During the map creation aortic valvular plane and coronary ostia are localized and the entrance into the ventricle is confirmed not only by the anatomical map but also by the endocardiac potential.

Mapping of the left ventricular outflow tract with a retrograde approach showed the earliest activation under the left coronary cusp (Figure 1E and F). Ablation at the site terminated the VT and returned it non-inducible. The procedural time was 156 min. After 3 months the ECG Holter showed only 85 PVBs vs. 32 900 PVBs, 516 couplets, 23 NSVT, and 1 SVT registered before ablation. Some studies showed feasibility, safety, and efficacy of using three-dimensional mapping systems as the primary guide for catheter visualization and radiofrequency catheter ablations for supraventricular and ventricular tachyarrhythmias, with a significant reduction of fluoroscopy use and then a concomitant reduction of possible X-ray side effects. One other study showed the feasibility of procedures without use of fluoroscopy. Considering the well-known cumulative inherent risk of radiation, a significant reduction of fluoroscopy exposure time represents a clear-cut benefit both for the patient as well as for the physician. In our patient VT ablation was achieved without complication and without fluoroscopy guide despite the intrinsic complexity in the anatomy of the heart. Catheter entanglement or catheter knot is a potential problem with non-fluoroscopic navigation. If a change in resistance to catheter manipulation is found, at which point the catheters could be visualized under fluoroscopy and if there is entanglement, appropriate manoeuvres to disentangle them could be performed.

Conflict of interest: none declared.

References

CASE REPORT

Ventricular tachycardia following trans-apical aortic valve replacement

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Transcatheter aortic valve replacement (TAVR) is a relatively new procedure for high-risk patients with severe aortic stenosis. We report a case of a new left ventricular outflow tract ventricular tachycardia following TAVR.

Case summary

This is a case of a 90-year-old woman with a history of highly symptomatic severe aortic stenosis with a calculated aortic valve (AV) area of 0.5 cm² and left ventricular ejection fraction of 72%. The patient was deemed to be at high risk for death or major morbidity with AV replacement surgery (Logistic EuroScore 15.8%) and was therefore enrolled in the PARTNER (Placement of AoRTic TraNs catheterER) valve trial. She underwent successful transcatheter aortic valve replacement (TAVR) via a trans-apical approach utilizing a 26 mm Sapien valve (Edwards Lifesciences, CA, USA). The left ventricular outflow tract measured 23 mm (Figure 2A) on the intraprocedural transoesophageal echocardiography. Follow-up echocardiograms showed a well-seated valve with a mild paravalvular leak and preserved left ventricular ejection fraction.

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Several days after discharge the patient reported occasional palpitations. A 24 h Holter monitor showed frequent episodes of wide complex tachycardia at an average heart rate of 112 bpm with AV dissociation, consistent with ventricular tachycardia (VT) (Figure 1A). Subsequent 12-lead electrocardiogram recording revealed VT with right bundle branch block/inferior axis morphology, and qR pattern at lead V1 (Figure 1B). These findings are consistent with VT originating from the aorto-mitral continuity (AMC) region. The patient’s beta-blocker dose was increased with subsequent resolution of her symptoms and no further evidence of arrhythmia.

Transcatheter aortic valve replacement is a relatively new procedure for high-risk patients with severe aortic stenosis. New conduction abnormalities following TAVR have been reported in up to 67% of the patients.1 In this report we describe the case of a VT following TAVR. The most commonly observed VT following TAVR involves reentry around the apical access site. Based on VT morphology in this case, however, this arrhythmia likely originated from the region at the AMC. Ventricular arrhythmias originating from the AMC have been reported in patients with structural heart disease, including those who had undergone prior AV replacements.2 Electrophysiology study (EPS) in this cohort of patients revealed scar in the majority of these patients serving as a potential substrate for reentry. In contrast, we suspect that the mechanism in our patient was more likely secondary to local tissue injury and oedema near the site of the aortic valve replacement early following placement of the valve. Of note, VT due to local tissue injury has been described in the experimental setting with the mechanism felt to be due to abnormal automaticity.3

Given that the suspected mechanism was likely transient, we elected to treat our patient conservatively with beta-blockers rather than proceeding to an EPS. To our knowledge this is the first report of VT related to TAVR, which originates from the peri-AMC region.
We did not perform an EPS and characterize the properties of the tissue responsible for her arrhythmia. We cannot definitively rule out that a small area of permanent tissue damage occurred at the time of valve deployment. However, she has been arrhythmia-free following the initiation of beta blockade.

**Conflict of interest:** none declared.

**References**


**Figure 2** (A) Intraprocedural transoesophageal echocardiography prior to valve deployment. The left ventricular outflow tract measures 23 mm. (B) Intraprocedural transoesophageal echocardiography following valve deployment demonstrating a well-seated Sapien valve in the aortic position.

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**CASE REPORT**

**Arrhythmias in heart transplant recipients**

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The suture between the recipient and donor atrium in a heart transplant patient usually gives complete electric isolation. In this case report, we describe two transplant patients with an atrial tachycardia in the recipient atrium. In the first patient there was no conduction to the donor atrium, whereas the second patient had a breakthrough with 2-to-1 conduction.

**Case**

A 78-year-old male patient, who underwent orthotopic heart transplantation 12 years before because of dilated cardiomyopathy, was admitted for his yearly transplant check-up. The routine Holter revealed an atrial arrhythmia with slightly irregular ventricular rate ([Figure 1](#)). Another heart transplant patient, a 59-year-old female, was admitted because of paroxysmal palpitations, 5 years after transplantation. An atrial tachycardia with a 2-to-1 conduction block was diagnosed and confirmed by electrophysiological investigation ([Figure 2](#)).

**Discussion**

During an orthotopic heart transplantation, a part of the posterior right and left donor atrium is attached to the recipient atrium. The suture between both atria usually leads to complete electric isolation. In the first patient, the recipient atrium developed tachycardia but without breakthrough to the donor heart. When watching carefully, a (donor) P-wave can be seen in front of each QRS complex ([Figure 1](#); black arrowheads). In the second patient, an atrial arrhythmia originating from the recipient atrium was recorded, conducting in a 2-to-1...