

Case Reports

Transvenous Valve-in-Valve Replacement Preserving the Function of a Transvalvular Defibrillator Lead

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Although feasibility and efficacy of percutaneous tricuspid valve-in-valve implantation have been established, a transtricuspid pacing or defibrillator lead might preclude this technique: lead damage can cause lead dysfunction resulting in inappropriate or inefficient pacing or shocks. In these cases, lead removal is thought to be the only option. We describe a patient who presented with rapid clinical deterioration due to tricuspid valve stenosis early after implantation of an internal defibrillator with a transvalvular shock-lead. A transvenous valve-in-valve implantation of the tricuspid valve was performed after protecting the defibrillator-lead with a custom-made covered stent. We describe the technical issues, the clinical outcome, and the evolution of lead function after implantation. © 2014 Wiley Periodicals, Inc.

Key words: tricuspid valve; percutaneous intervention; internal cardiac defibrillator; lead protection

INTRODUCTION

In patients at high risk for cardiac surgery, feasibility and efficacy of percutaneous tricuspid valve-in-valve implantation have been established [1–3]. However, patients with a transtricuspid pacing or defibrillator lead challenge the interventional cardiologist with a dilemma: the valved stent can damage the lead causing dysfunction, inadequate sensing or pacing, which in turn may result in delivering inappropriate or inefficient shocks [4,5].

We describe a patient who presented with symptomatic tricuspid valve stenosis after implantation of a transvalvular shock-lead. A transvenous valve-in-valve implantation of the tricuspid valve was performed after protecting the defibrillator lead with a covered stent.

CASE REPORT

A 42-year-old male presented for percutaneous revaluation of the tricuspid valve. He was born with an arterial trunc, underwent pulmonary artery banding in infancy, and was repaired at the age of five. Later, multiple re-interventions were needed. At the age of 29 years, right ventricular outflow tract reconstruction with a Carpentier-Edwards size 27 mm bioprosthesis, as well as implantation of a tricuspid valve Carpentier-Edwards size

33 mm bioprosthesis was performed. Symptomatic runs of ventricular tachycardia at the age of 40 years were treated with an internal defibrillator (ICD) Medtronic Virtuoso® VR connected to a 9 Fr transvalvular shock lead (Medtronic 6947).

In the years following ICD implantation, tricuspid valve stenosis developed with rapid clinical deterioration in 2 years. He presented now in NYHA functional class IV with severe right heart failure and anasarca edema.

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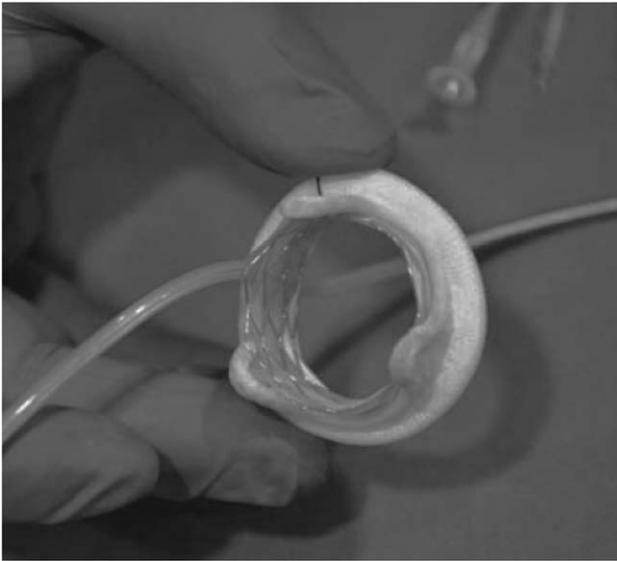


Fig. 1. Bench testing of percutaneous valve-in-valve replacement of the tricuspid valve. A 39 mm 8 Zig Covered CP stent™ (NuMED®, NY, USA) was positioned into the 33 mm Carpentier Edwards bioprosthesis. The 9 Fr defibrillator lead is protected as it passes between the CE ring and the newly positioned CCP stent.

The patient was refused for surgery by a leading center for cardiovascular surgery because of the high operative risk after five previous sternotomies, the last one being complicated by bleeding and difficult chest closure. Therefore, a transvenous valve-in-valve implantation in the tricuspid position seemed a better alternative. The patient declined percutaneous removal of the defibrillator lead with replacement by an epicardial-subcutaneous lead system. As valved stents could damage complex electrical leads, we chose to protect the defibrillator lead by means of a custom-made Expanded polytetrafluoroethylene (ePTFE) covered stent before implantation of the valved stent.

BENCH TESTING AND CLINICAL PROCEDURE

Before the procedure was performed, we did bench testing of the implantation technique used. A 39 mm 8 Zig Covered CP stent™ (NuMED®, NY) was mounted on a 28 mm balloon (NuMED®) to protect the 9 Fr ICD wire crossing a 33 mm Carpentier Edwards bioprosthesis (Fig. 1). Next an Edwards SAPIEN XT 29 mm valve was implanted in the covered stent. It should be noted that the 8 Zig stent foreshortens significantly when it is dilated beyond 23 mm (70% at 26 mm). Furthermore, the ePTFE membrane may tear when dilated beyond 24 mm. Therefore, when performing the clinical intervention, we chose a 10 Zig CCP stent (custom made, no CE mark; NuMED®) which can be dilated up to 36 mm with significantly less foreshortening (20% at 26 mm) while the ePTFE membrane remains intact.

The patient was anesthetized with endotracheal intubation and artificial ventilation. Veno-arterial extracorporeal membrane oxygenation (ECMO) with femoral access was started as a safety measure at 4 l/min to give hemodynamic support during the procedure: we felt that because of the poor clinical status, the patient would not tolerate the hemodynamic consequences of leaving the tricuspid valve temporary stented open during the procedure. Percutaneous access was obtained by inserting a 9–24 Fr sheath in the right jugular vein using a dilator kit.

First, right heart catheterization during minimal flow ECMO confirmed severe tricuspid valve stenosis with a mean transtricuspid gradient of 20 mm Hg at 80 bpm heart rate, despite low output (Fig. 2).

After balloon interrogation of the inner diameter (25 mm), the defibrillator lead was protected with a custom made 39 mm 10 Zig Covered CP stent™ (NuMED®) mounted on a 32 mm Balloon-in-Balloon catheter (NuMED®) (Fig. 3A). Next, a 26 mm Edwards SAPIEN valve (9000 TFX 26) was deployed in the tricuspid position (Fig. 3B)

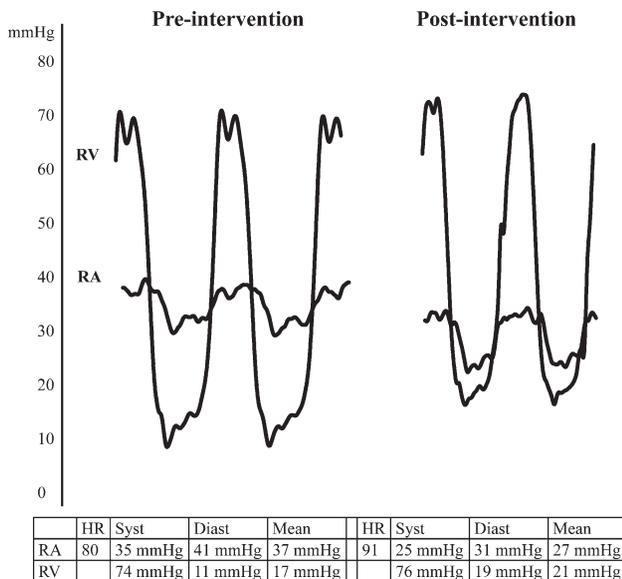


Fig. 2. Hemodynamic evaluation of the tricuspid valve stenosis pre- and post-intervention. RA indicates right atrium; RV = right ventricle.

Doppler echocardiography revealed severe tricuspid valve stenosis with a mean transvalvular gradient of 20 mm Hg at 75 bpm heart rate. Right ventricular function was severely impaired. Moderate dysfunction of the left ventricle (ejection fraction = 45%) was present. Signs of increased right atrial pressure with severe dilatation of the right atrium and a dilated inferior vena cava without respiratory variation were noted.

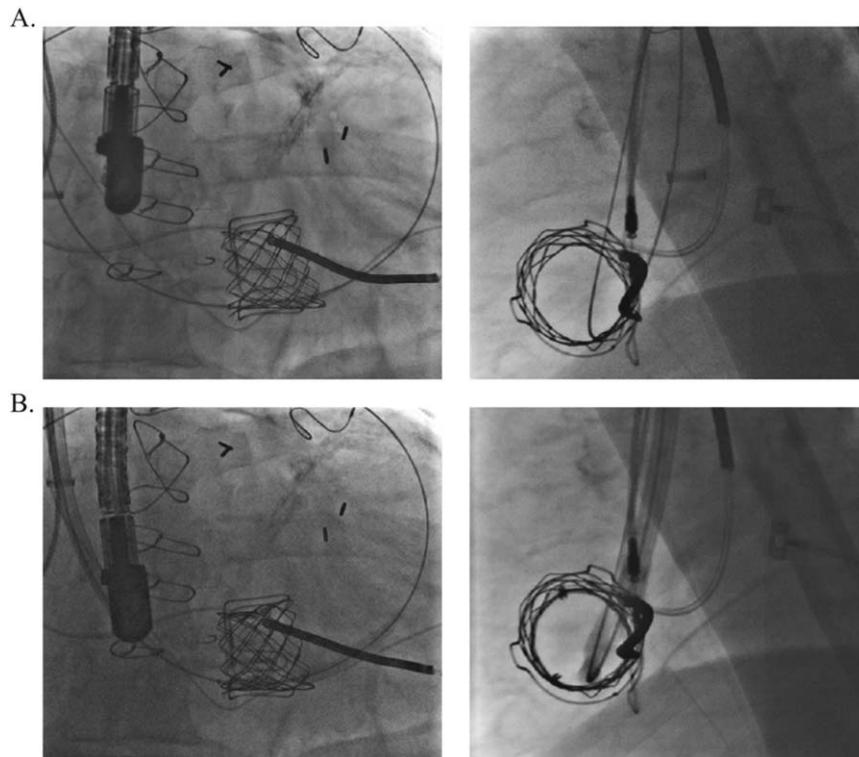


Fig. 3. Angiographic images during intervention showing (A) status after deployment of the covered CP stent excluding the defibrillator lead outside the annulus and (B) status after deployment of the Edwards SAPIEN valve.

TABLE I. Evolution of Lead-Impedances Pre- and Post-Intervention in the Patient

Date	Pacing impedance	RV defib impedance	SVC defib impedance	RV pacing threshold
Baseline	480 Ω	49 Ω	61 Ω	0.5V at 0.4 msec
Post-procedure (% compared to baseline)	392 Ω (82%)	25 Ω (51%)	33 Ω (54%)	1.0 V at 0.4 msec
Day 2 (% compared to baseline)	368 Ω (75%)	25 Ω (51%)	33 Ω (54%)	1.0 V at 0.4 msec
13 months (% compared to baseline)	336 Ω (70%)	28 Ω (57%)	37 Ω (60%)	1.0 V at 0.4 msec

RESULT AND FOLLOW-UP

Valvular function improved dramatically with an invasive transtricuspid gradient of 6 mm Hg at 91 bpm heart rate post-procedure and only minimal valvular regurgitation (Fig. 2). Tricuspid valve function was preserved at 13-months follow-up without echocardiographic evidence of restenosis or valvular regurgitation.

Before and after the procedure, ICD lead impedances and function were evaluated (Table I). Immediately after the procedure, lead impedances lowered. In the follow-up, no further decline was noted.

During a 13-months follow-up, the patient had two appropriate ICD interventions due to ventricular tachycardia, reconverted by anti-tachypacing. He had ventricular pacing for 20% of the time. No inappropriate

shocks were fired. Impedance as well as the pacing thresholds remained stable (Table I).

Because of the decrease in impedance, the effect of lead crushing was tested ex vivo. Shock leads were tested in a saline solution to reproduce the osmolality of the blood and a sponge placed in the saline solution to mimic human tissue contact. Impedance measurements of a Medtronic 6947 defibrillator lead connected to a Medtronic Maximo® VR 7232 ICD was measured at baseline. Next, the lead was crushed at 15 atm between a 29 mm Sapien stented valve within a covered CP stent, and the ring of a 33 mm Carpentier Edwards bioprosthesis. The results of impedance testing can be found in Table II.

A Medtronic 6947 lead that had been crushed between the stents and the valvular ring for 17 months was connected to the same device. Impedance testing

TABLE II. Bench Testing of Lead-Impedances in Tested Conditions

Measured impedance	New lead			Lead 17 months placed between stent and homograft ring	
	Free (Ω)	Balloon inflated to 15-atmosphere (Ω)	Placed between stent and homograft ring (Ω)	17 months in place (Ω)	After release (Ω)
RV pacing	1,984	1,904	1,744	576	576
RV Defib	26	26	25	20	20
SVC Defib	31	31	31	26	23

was performed and again after release of the pressure by the stents (Table II).

Superior vena cava (SVC) and right ventricular (RV) impedances were rather low in the test conditions. However, no significant changes could be observed in SVC impedance and RV shock impedance during either active crushing of the lead and after deflation of the balloon. Release of the lead after 17 months did not alter the impedance.

DISCUSSION

Stenosis of a tricuspid bioprosthetic valve is common after 13 years although accelerated dysfunction after lead implantation is sometimes reported [6,7]. When a transvalvular lead is present and a new valve needs to be implanted, several approaches can be considered.

A surgical tricuspid valve intervention allows the lead to be extracted and replaced by a transvenous extra-annular or epicardial-subcutaneous lead system; however, because of previous multiple complicated sternotomies the risk in this patient was deemed excessively high. A percutaneous approach offers two options. First, lead extraction with subsequent epicardial sensing-pacing and subcutaneous shock lead placement could be followed by percutaneous reevaluation of the tricuspid valve. However, percutaneous lead extraction is difficult and is associated with significant morbidity and mortality [8]. Additional risk factors for lead extraction are severe fibrosis and the use of a screw-in lead, which were both present in this patient [8]. Furthermore, the patient refused percutaneous lead extraction and the significant cardiomegaly raised doubt about epicardial sensing and pacing and adequate subcutaneous shock function. Lastly, a new percutaneous valved stent can sandwich the lead between the old bioprosthesis and the new, valved stent.

The Edwards Sapien valve has a stiff stent with sharp edges, designed to anchor well into the calcified aortic valve ring. When placed in the tricuspid position, it could cause lead malfunction either by damaging the insulation causing a short circuit, dysfunction of sensing or (sub-) total lead fracture with failure of

pacing and defibrillator therapy. Therefore, we chose to protect the lead with a covered CP stentTM. This stent consists of relatively thick (0.013") rounded wire that is little abrasive to the surrounding tissue. To further reduce the local stress as well as to reduce a possible paravalvular leak after stent and valve placement, we chose an ePTFE covered stent. The Carpentier-Edwards 33 mm bioprosthesis has a profile height of 25 mm and the Edwards Sapien valve has a frame height of 16.1 mm. Therefore, we implanted a 10 Zig covered stent in this patient, which can be dilated up to 36 mm with its ePTFE membrane remaining intact. Only minimal foreshortening occurs when it is dilated less than 30 mm. After stent placement, the 26 mm Edwards Sapien valve could be easily deployed in appropriate position.

No complications occurred; rapid improvement of venous congestion, right heart failure, and exercise tolerance were noted. Lead impedances immediately after procedure lowered, probably due to local stress on the lead; the lead-resistance remained stable during follow-up. At 13 months, pacing function was preserved and correct detection of ventricular tachycardia with appropriate delivery of anti-tachypacing was noted. The patient did not receive inappropriate shocks. As the evolution is unpredictable, the patient was instructed to have a magnet available to reset the device into VOO when inappropriate shocks would be fired.

A decrease in lead impedance is most often caused by damage of the insulation, either by mechanical disruption or by degradation and metal ion oxidation [4]. The leads have a multilumen design with a silicone insulation of each conductor individually to withstand mechanical stress and lead degradation. Although bench testing could not identify changes in impedances by actively crushing the lead, or after long-term presence of the lead sandwiched between the valvular ring and the stents, changes in SVC and RV impedances could be seen in our clinical case immediately post-intervention. Disruption of the insulation could have occurred; however, if the cardiac motion would cause mechanical disruption of the lead, further decline in lead impedances should have occurred in follow-up. Furthermore, as the lead was already implanted for

2 years, silicone stiffening and ion diffusion into the lead insulation could cause a low-resistance circuit between conductors after crushing. We could not reproduce these conditions in our *ex vivo* experiment. Lastly, an altered orientation of the lead closer to the RV free wall or changes in volume-status after the procedure could have influenced lead impedances [9,10]. It should be noted that, although impedances lowered, values remained on the safe side.

CONCLUSION

This case shows an alternative approach when confronted with patients with a transvalvular lead and the indication for percutaneous tricuspid valve replacement. Exclusion of the lead outside the valvular ring is feasible and efficient.

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