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Ventricular Tachycardia in Repaired Tetralogy of Fallot

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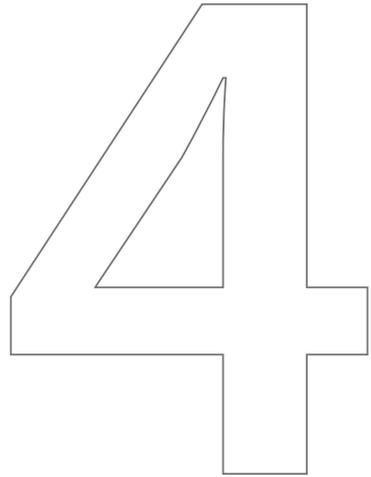


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Left-Sided Ablation of Ventricular Tachycardia in Adults with Repaired Tetralogy of Fallot: a Case Series

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ABSTRACT

Background

Radiofrequency catheter ablation (RFCA) of ventricular tachycardia (VT) in repaired Tetralogy of Fallot (rTOF) focuses on isthmuses in the right ventricle but may be hampered by hypertrophied myocardium or prosthetic material. These patients may benefit from ablation at the left side of the ventricular septum.

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Methods and Results

Records from 28 consecutive rTOF patients from two centres, who underwent VT ablation, were reviewed. Ablation targeted anatomical isthmuses containing VT reentry circuits identified by 3D substrate, pace and/or entrainment mapping. A left-sided approach was considered beneficial if (1) right-sided RFCA failed, (2) part of the circuit was mapped to the left side and (3) left-sided RFCA resulted in isthmus transection and prevention of VT induction. In 4/28 patients (52 ± 13 years, 75% male), inducible for 1.5 (quartiles, 1.0 – 2.0) VTs (335 ± 58 msec), left-sided RFCA was performed. In 3 patients RFCA at aortic sites terminated VT related to a septal isthmus and prevented re-induction. In 1 patient, with prior biventricular ICD, diastolic activity was recorded at the left side of the septum in proximity to the His-bundle. RFCA prevented VT re-induction with anticipated complete AV-block. The left-sided approach resulted in complete procedural success (transection of anatomical isthmus and non-inducibility) and freedom of VT recurrence during follow-up (20 ± 15 months) in all patients. Right-sided RFCA failure was likely due to septal hypertrophy in 2, the overlying pulmonary homograft in 1 and overlying VSD patch in 1.

Conclusion

Left-sided RFCA for VTs dependent on septal anatomical isthmuses improves ablation outcome in rTOF.

INTRODUCTION

The population of adults with repaired Tetralogy of Fallot (rTOF) is increasing, as improved surgical techniques are extending survival.¹ Ventricular arrhythmias (VA) are an important reason for late morbidity and mortality.^{1,2} The majority of VA in rTOF are monomorphic VTs.³ The potential re-entry paths causing these VTs are now well described and contain anatomical isthmuses bordered by unexcitable tissue from the tricuspid or pulmonic valve annuli, surgical scars and patch material.⁴ Radiofrequency catheter ablation (RFCA) of the anatomical isthmus causing VT can be highly effective for preventing recurrent VT.⁴ However, achieving conduction block across an isthmus can be difficult due to the hypertrophic myocardium or protection of portions of the isthmus under prosthetic patches or valve homografts. This is particularly true for isthmuses in the outlet septum where the VSD patch is typically located. The aim of this study was to assess the feasibility and utility of ablation from the left ventricle or aorta when ablation from the right ventricle fails. We demonstrate for the first time, to our knowledge, successful ablation of VTs involving the outflow septum using a combined right and left ventricular/aortic root ablation approach.

METHODS

Population

The procedural data of twenty-eight consecutive patients with rTOF, who underwent RFCA of VT in two centres between 2001 and 2013, were reviewed. Patients in whom a left-sided approach was performed were included in the current series. A left-sided approach was considered beneficial if (1) RFCA from the RV failed to prevent VT induction and/or isthmus transection and/or VT recurrence, (2) parts of the VT re-entry circuit isthmus were mapped to the left ventricle (LV) or aortic root and (3) RFCA applied from the left side resulted in isthmus transection and prevented VT induction. The medical records of the patients were reviewed for the surgical history, documentation of spontaneous VT, previous VT ablation and failed drugs. Imaging studies were reviewed for left and right ventricular function at the time of referral. The RV systolic function was semi-quantitatively analysed.^{5,6} The LV ejection fraction was derived by Simpson's method. All patients were treated according to the routine clinical protocol and provided informed consent.

Procedure

The systematic approach for ablation of macro-reentry VT in rTOF has been previously described in detail.⁴ Briefly, the (re-)induction protocol existed of two or three drive cycle lengths (600, 500 and 400 msec) with up to three extra stimuli, burst pacing and isoproterenol from ≥ 2 RV sites (apex and RV outflow tract (RVOT)). A RV bipolar electroanatomical voltage map (EAM) was created using a 3D mapping system (Carto XP/3, Thermocool catheter, Biosense Webster, Inc; Diamond Bar, Calif). At low voltage sites (bipolar recording < 1.5 mV), high output pacing (10 mA at 2-ms pulse width) was performed to identify areas of electrically unexcitable scar/tissue (EUS).⁷ EUS (i.e. VSD-patch, pulmonary homograft, and

previous RV incisions), the pulmonary valve (PV) and the tricuspid annulus (TA) were defined as boundaries of the four previously described anatomical isthmuses in rTOF (for details see figure 1). Two of these anatomical isthmuses (isthmus 3 and 4) involve the septum. The relationship between the critical reentry circuit isthmus of each VT and the anatomical isthmuses was confirmed by concealed entrainment, diastolic activity with VT termination during RF delivery for tolerated VT and/or pace mapping (PM) at sites where QRS morphology matched VT morphology ($\geq 11/12$).⁸ The anatomical isthmus containing the critical VT reentry site was transected with RF current using an open saline-irrigated catheter (power 35-50 W, maximum temperature 45°C) until high output (at least 10 mA, 2 ms pulse width) pacing fails to capture along the ablation line. Block was further supported by the presence of double potentials and/or a change in activation sequence during SR or RV-pacing after ablation. More recently differential pacing was performed to confirm bidirectional conduction block.⁹ After transection of the anatomical isthmus programmed stimulation was repeated.

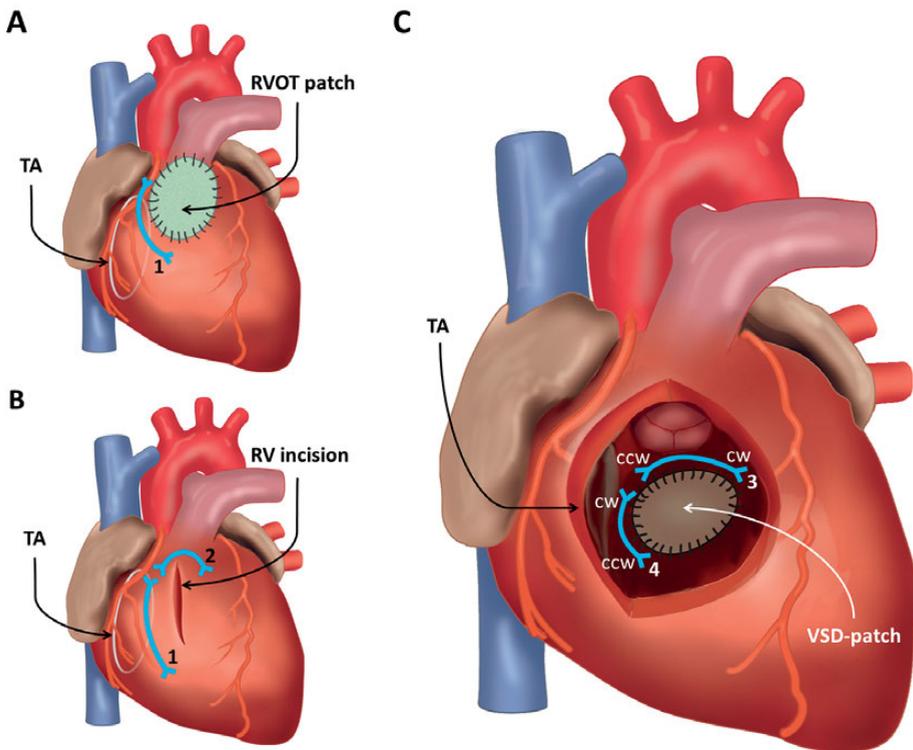


Figure 1. The previously described four anatomical isthmuses for VT in rTOF.⁴ Anatomical isthmus 1 is located between the TA and a RV incision/RVOT patch (panel A, B) and anatomical isthmus 2 is located between a RV incision and PV (panel B). Anatomical isthmus 3, located between the PV and VSD-patch and anatomical isthmus 4, located between the VSD-patch and TA (panel C) are bordering on the septum. Ccw indicates counter clockwise; cw, clockwise; PV, pulmonary valve; rTOF, repaired Tetralogy of Fallot; RV, right ventricle; RVOT, RV outflow tract; TA, tricuspid annulus; VSD, ventricular septal defect; VT, ventricular tachycardia.

Left-sided approach

If VT could not be abolished from the RV and the anatomical isthmus containing reentry circuit sites was in or bordering on the septum, the aorta and LV were mapped using a retrograde approach. The described criteria were applied to confirm that parts of the reentry circuit were located within the same anatomical isthmus but approachable from the left ventricle or aorta. Before RF delivery at aortic sites coronary angiography or aortography was performed to identify the coronary ostia. In the aorta power was limited to a maximal of 35 Watts (figure 2). After RFCA, the induction protocol was repeated.

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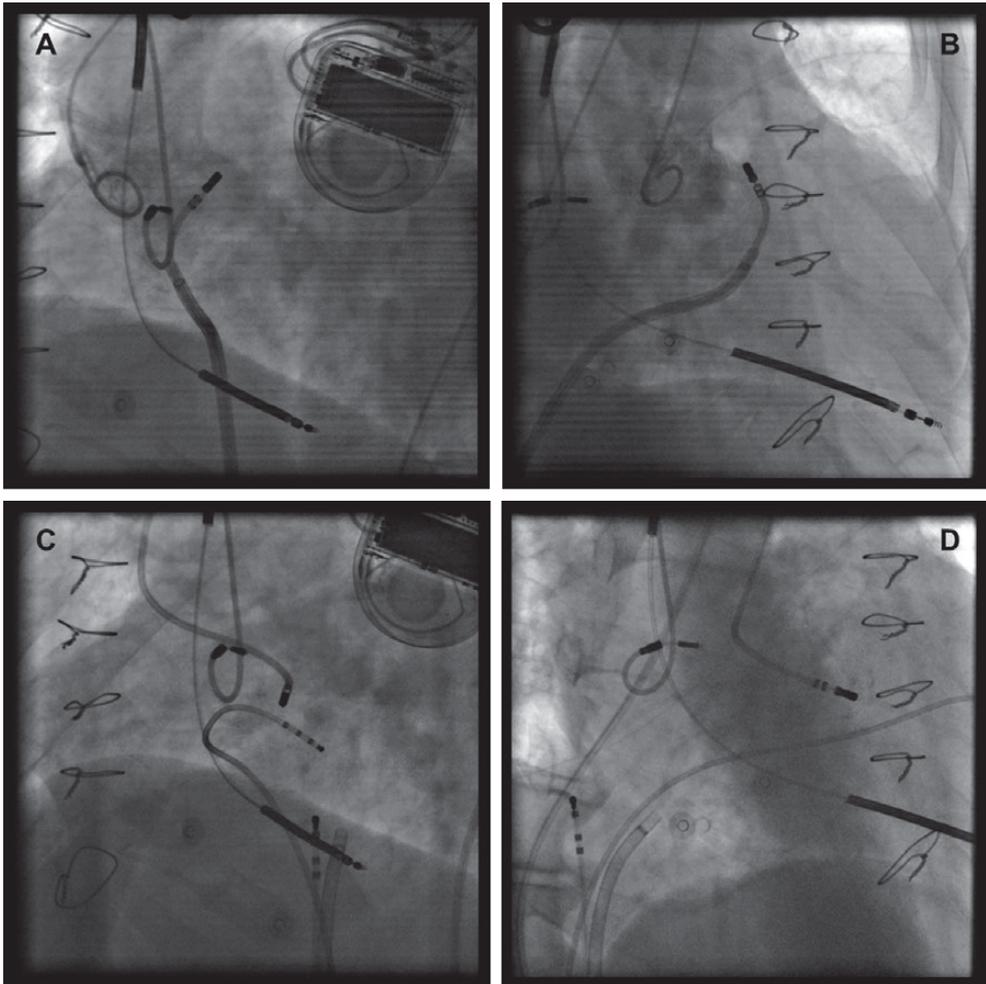


Figure 2. Mapping catheter position on fluoroscopy (case 1). Position of the mapping catheter in the RV during angiography of the aorta in LAO (panel A) and RAO (panel B) view. Position of the mapping catheter in the left coronary cusp of the aorta during VT termination in LAO (panel C) and RAO view (panel D). LAO = left anterior oblique; RAO = right anterior oblique; RV = right ventricle.

Follow-up

In patients without an ICD prior to ablation, an ICD was recommended if indicated according to current guidelines. VA recurrence was defined as documented spontaneous sustained VA and/or symptoms highly suspicious for sustained VA. For patients that were not followed at the two institutes, records of the clinical and device follow-ups including the most recent were obtained.

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Anatomy

In order to gain more insights in the anatomical substrate that may require left-sided RFCA in rTOF, the Leiden collection of malformed hearts was reviewed for rTOF post-mortem specimens. Study of this collection was undertaken in accordance with the local ethics committee and the Dutch regulation for the proper use of human tissue for medical research purposes. Two post-mortem specimens were selected to illustrate (1) the relation between the patch material and the adjacent myocardium and (2) the specific anatomical location and morphology of the infundibular septum.

RESULTS

In 11 of the 28 (39%) consecutive patients with rTOF right-sided RFCA failed to abolish all inducible VTs. In 8 of these 11 patients the VT was mapped to a septal anatomical isthmus (isthmus 3 in 7 pts, isthmus 4 in 1 pt). In four patients a left-sided approach was not taken due to procedural considerations (length of the procedure, poor condition of the patient, inability to re-cross the aortic valve due to position of the aortic root and mild aortic valve stenosis). The presumed reasons for failure of right-sided ablation in these four patients were hypertrophy of the outlet septum in two and a pulmonary homograft in two. In the remaining four patients left-sided mapping and RFCA were performed. These four patients (52 ± 13 years (mean \pm sd), 75% male) were inducible for a median of 1.5 VT (quartiles, 1.0 – 2.0) with a mean VT cycle length of 335 ± 58 msec. Table 1 summarises the baseline characteristics.

Case 1

A 58 year old male had undergone total correction (infundibular resection, transannular patch, patch closure of a perimembranous VSD) at the age of 13 and presented with recurrent, symptomatic, self-terminating broad-complex tachycardias. Echocardiography and MRI revealed severe pulmonary valve regurgitation requiring pulmonary valve replacement (PVR). Before surgery programmed electrical stimulation (PES) and RV EAM were performed. Two fast VTs (cycle lengths 250ms, figure 3) were inducible and substrate mapping identified two anatomical isthmuses: isthmus 1 between the transannular patch and the tricuspid annulus (TA) and isthmus 3 between the pulmonary valve (PV) and the VSD-patch. Both VTs could be mapped to anatomical isthmus 3 with a clockwise activation during VT1 (QR in V1) and counterclockwise activation during VT2 (late transition in precordial lead V_3). As the patient was scheduled for re-surgery, PVR was combined with intraoperative cryoablation

Table 1. Patient characteristics and procedural data.

Patient Characteristics	Patient 1	Patient 2	Patient 3	Patient 4
Age (years)	58	67	45	37
Gender	Male	Female	Male	Male
Prior Shunt	No	No	Yes	No
Age of Repair (years)	13	15	4	3
Type of VSD	Peri-M	Peri-M	Musc	Peri-M
Reoperation	PVR	VSD closure	VSD closure	None
Failed drugs	None	Sotalol	Sotalol Mexiletine	Sotalol
LV ejection fraction	54%	43%	39%	61%
RV function	Moderately Red	Severely Red	Moderately Red	Mildly Red
VT induced (n)	2	1	2	1
Septal isthmus dependent (n)	2	1	1	1
VTCL of induced VT (ms)	245+255	370	370+380	345
Presumed reason right-sided failure	P Homograft	Hypertrophy	VSD-patch	Hypertrophy
Drugs at discharge	None	Sotalol	Sotalol	Nadolol

LV = left ventricular; Peri-M = perimembranous; P homograft = pulmonary homograft; PVR = pulmonary valve replacement; Red = reduced; RV = right ventricular; VSD = ventricular septal defect; VT = ventricular tachycardia; VTCL = VT cycle length.

of isthmus 3. At the postoperatively performed EP study VT1 was again inducible and an ICD was implanted before discharge. Subsequently he had multiple VT episodes terminated by ATP and was intolerant of sotalol. Six months after surgery VT ablation was attempted. Although pacing at isthmus site 3 did not capture, the activation map during sinus rhythm was consistent with continuous conduction through isthmus 3. During VT 1 diastolic activity was recorded at isthmus 3 but RFCA at that region did not terminate VT. These findings suggested that the pulmonary homograft was covering the infundibular septum (figure 4) mimicking scar between the prior pulmonary annulus and the TA. Pacing at the opposite site within the left coronary cusp (LCC, figure 2) captured and resulted in a 12/12 pace-map. During VT mid-diastolic activity was recorded at this site, and RF application terminated VT within 18 seconds and prevented VT re-induction. During 21 months of follow-up the patient had no VT recurrence.

Case 2

A 66 year old female underwent primary repair of TOF at the age of 15 years with resection of severely hypertrophied infundibular muscle, commissurotomy of the PV, partial patch closure of a large perimembranous VSD and suture of a large longitudinal RV incision. Thirteen years later complete closure of the VSD was performed. She subsequently developed recurrent, symptomatic VT terminated by antitachycardia pacing (ATP). During EP study the presumed clinical VT (cycle length 370ms, figure 3) was induced. RV electroanatomic mapping identified

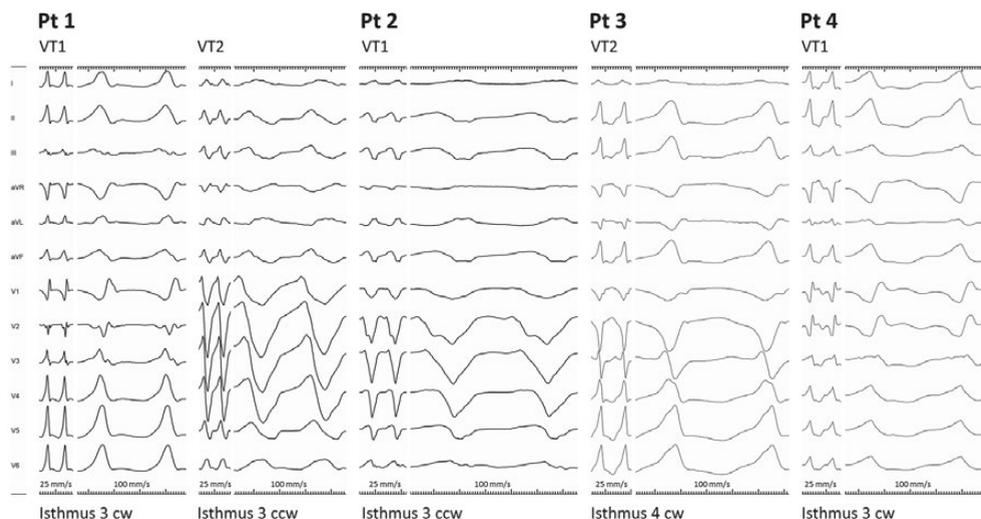


Figure 3. 12 lead ECGs (sweep speed 25mm/s and 100mm/s) of the induced septal VTs in all four cases. In patient one, two and four, the critical reentry site was located at anatomical isthmus 3 (isthmus between VSD-patch and PV). VT1 in patient one and VT1 in patient four demonstrated a clockwise (cw) activation of isthmus 3, please note the QR in lead V1. On the contrary, VT2 in patient one and VT1 in patient two demonstrated a counter clockwise (ccw) activation of isthmus 3, please note the late transition in precordial leads. In patient three, the critical reentry site of VT2 was located at anatomical isthmus 4 (isthmus between VSD-patch and TA) and the VT demonstrated cw rotation through isthmus 4. PV, pulmonary valve; TA, tricuspid annulus; VSD, ventricular septal defect; VT, ventricular tachycardia.

two anatomical isthmuses bordered by EUS; isthmus 1 between a longitudinal EUS area (consistent with the previous anterior RV incision) and the TA and isthmus 3 bordered by an area of EUS in continuity with the TA consistent with the VSD patch and the PV. A good PM for the presumed clinical VT was obtained at isthmus 3. Activation mapping was consistent with counterclockwise propagation (late transition in precordial lead V_6) through isthmus 3. Entrainment mapping at this site revealed a central isthmus site and RF slowed and terminated the VT within 10 seconds, rendering the VT non-inducible. A linear RF lesion was performed to connect the areas of EUS. However, the activation sequence during pacing proximal to the line was still consistent with conduction through the isthmus.

After 3 months VT recurred. At repeat study the same VT was inducible. Although double potentials could be recorded along isthmus 3 during sinus rhythm, differential pacing was consistent with persistent slow conduction through the isthmus. During VT diastolic activity could be recorded within isthmus 3 and RF delivery resulted in delay and late termination of VT, but the VT remained inducible. Mapping at the left side of the region from within the right coronary cusp (RCC) revealed diastolic activity (30 msec before the earliest activation in the RVOT) and entrainment consistent with an isthmus site (figure 5). RF ablation at that site terminated VT within 5 seconds and prevented VT re-induction. Differential pacing

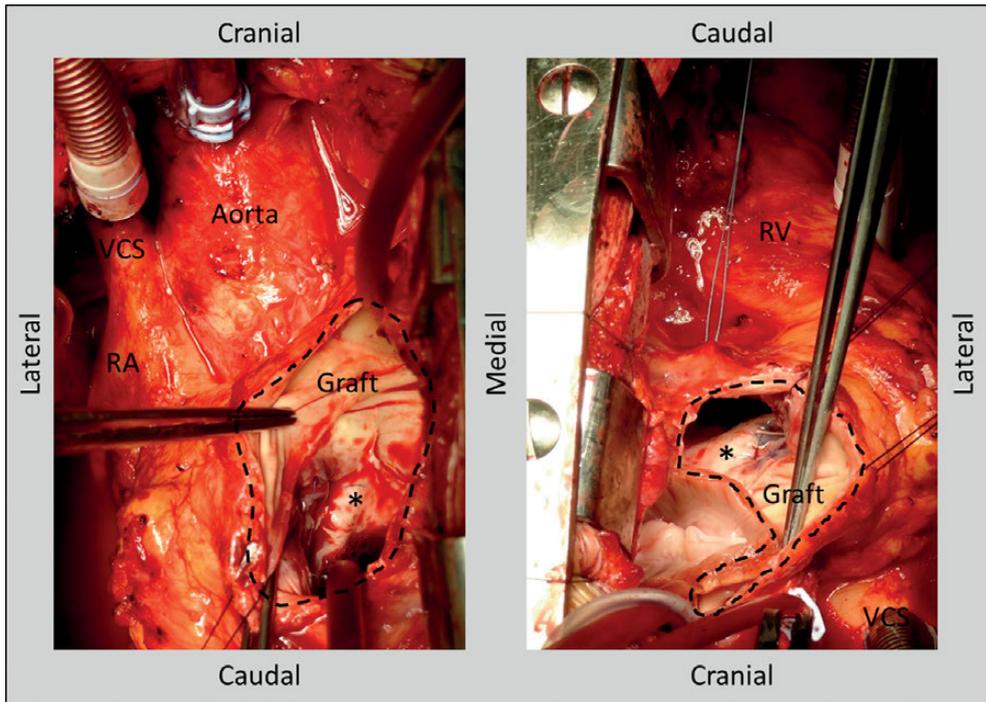


Figure 4. Intraoperative view of pulmonary valve replacement by pulmonary homograft in a patient with repaired Tetralogy of Fallot. Left panel a cranial (top) to caudal view, right panel a caudal to cranial view. The anterior suture line is marked with the dashed line in both pictures. At both panels the pulmonary homograft is held by the forceps. The pulmonary homograft is already posteriorly sutured to the VSD-patch (marked with *). After complete implantation of the homograft, important parts of the RVOT endocardium and in particular the infundibular septum are covered by the homograft. * = VSD-patch; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract; TA = tricuspid annulus; VCS = vena cava superior; VSD = ventricle septum defect.

performed from the RVOT was consistent with conduction block. The patient continued with a low dose of Sotalol and remained free of VT during 15 months of follow-up.

Case 3

A 45 year old male with rTOF (operation reports not available) who had undergone CRT-D implantation for VTs and progressive heart failure was referred for VT ablation after prior ablation attempts in other centers had failed. Two VTs were inducible (VT1, cycle length 370ms and VT2, cycle length 380ms). RV EAM revealed a small low voltage area in the RV free wall perhaps consistent with a prior incision and a second low voltage area at the septum bordered by the TA and an area of EUS in continuity with the PV most likely consistent with a VSD patch. Hence, two anatomical isthmuses were identified: isthmus 1 between the TA and the small free wall scar and isthmus 4 between the VSD-patch and the TA. VT1 could

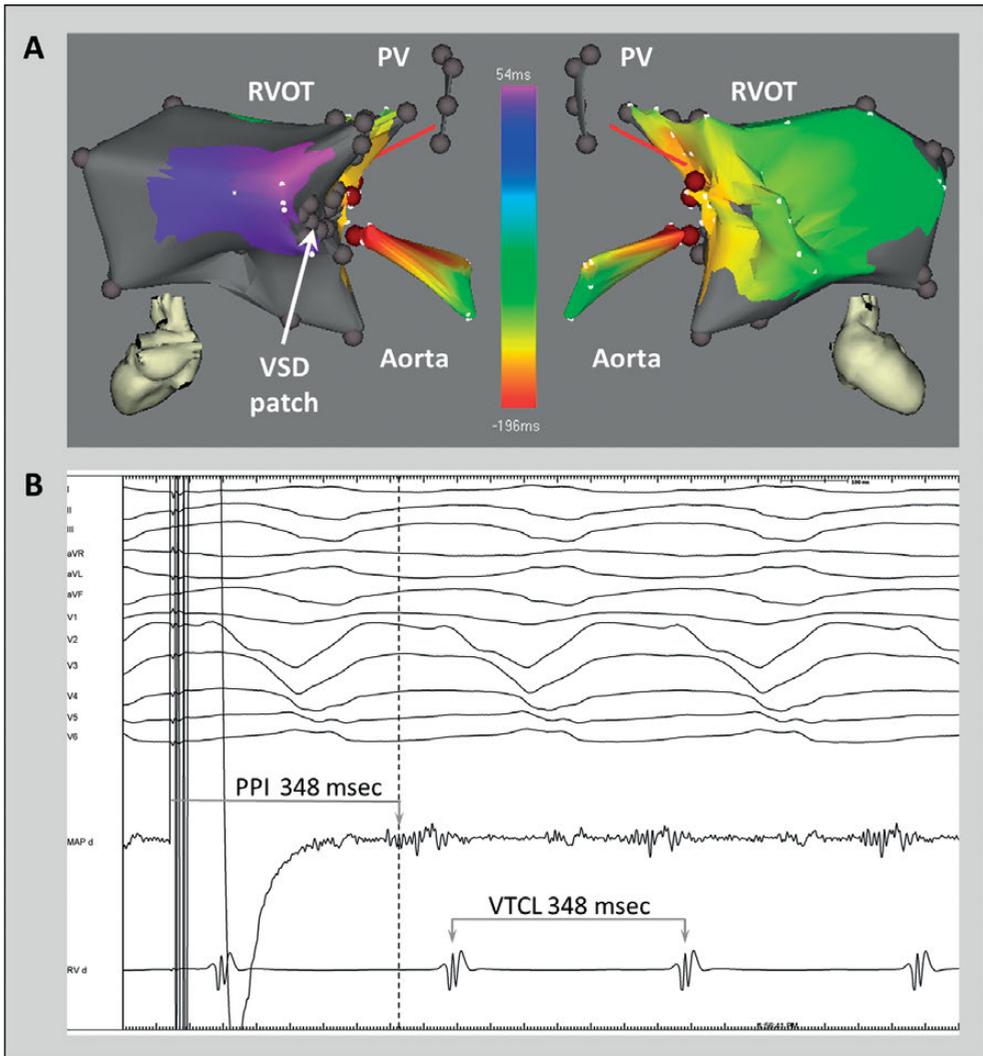


Figure 5. Panel A: Electroanatomical activation map of the clinical VT (activation time color coded according to bar) in left posterior and right lateral views. Earliest RVOT activation was recorded at the anatomical isthmus between the PV and VSD-patch. Diastolic activity preceding RVOT activation by 30 msec could be recorded from the aortic root. Panel B: From the aortic site VT could be entrained with concealed fusion (PPI=VTCL=348ms). RF at this side terminated VT within 5 seconds. MAP d = bipolar recording of the distal mapping catheter; PPI = post pacing interval; PV = pulmonary valve; RVa = right ventricular apex; RVOT = right ventricular outflow tract; VSD = ventricular septal defect; VT = ventricular tachycardia; VTCL = VT cycle length.

be mapped to isthmus 1 and was ablated by connecting the presumed prior incision with the TA.

During VT2 (figure 3), presystolic activity was recorded at the lower edge of the presumed VSD patch with clockwise propagation through isthmus 4. However, the appearance of

the electrogram was consistent with a far-field signal and RF delivery did not terminate VT. At the adjacent LV site a good PM for VT2 was obtained, diastolic activity was present during VT, and a His-bundle electrogram was present in sinus rhythm. RF ablation was performed and resulted, as anticipated, in complete heart block. Thereafter, VT was no longer inducible. Sotalol was continued. He remained free of VT during 40 months of follow-up. He received one ICD shock for ventricular fibrillation after 15 months.

Case 4

A 36 year old male with rTOF (operation report not available) who had undergone ICD implantation and a previous VT-ablation in another center presented with an electrical storm and multiple ICD shocks. The presumed clinical VT (cycle length 345ms) was induced (figure 3). RV EAM identified isthmus 1 between EUS in the anterior RVOT in continuity with the PV and isthmus 3 bordered by EUS in continuity with the TA (VSD patch) and the PV. Entrainment at the ventriculo-infundibular fold was compatible with a VT entrance site and clockwise activation (QR in V1) of isthmus 3. However, RF at this site did not terminate VT. At the opposite side in the aorta, between the RCC and the NCC, a late potential (220 msec after QRS offset) was recorded during SR (figure 6). During VT mid diastolic activity was present and entrainment was consistent with an central isthmus site. A single RF application terminated VT within 10 seconds and prevented subsequent VT induction. To minimize procedural risks, no additional RF applications were performed in the aortic root.

VT recurred after 1 month and a repeat procedure was performed. The previous recorded VT could be induced. Activation mapping confirmed a reentry circuit entrance at the ventriculo-infundibular fold and an exit at the RCC region. A line of RF lesions was placed across isthmus 3 from the RV, the LVOT and the area between the LCC and RCC, abolishing inducible VT. The patient was discharged on nadolol and remained free of VT during 3 months of follow-up.

DISCUSSION

A right-sided RFCA approach for VT in rTOF targeting anatomical isthmuses failed to abolish inducible VT in 39% of our rTOF patients. Of importance, the majority (8/11) of these patients had a proven VT substrate involving anatomical isthmuses located at the infundibular septum. In all 4 patients in whom a left-sided approach was performed after right-sided ablation failure diastolic activity and/or concealed entrainment could be recorded either from the aortic root in muscular continuity and opposite to isthmus 3 (figure 7) or in the LVOT at the left sided inferior border of the VSD patch consistent with isthmus 4. RFCA at these sites terminated VT and/or prevented VT re-induction. The left-sided approach resulted in complete elimination of inducible VT and prevention of VT recurrence during follow-up (20±15 months) without procedure related complications in all cases, although one patient required two left-sided procedures. Based on the operation records, intraoperative and EAM findings the critical isthmus could likely not be transected from the right side due to

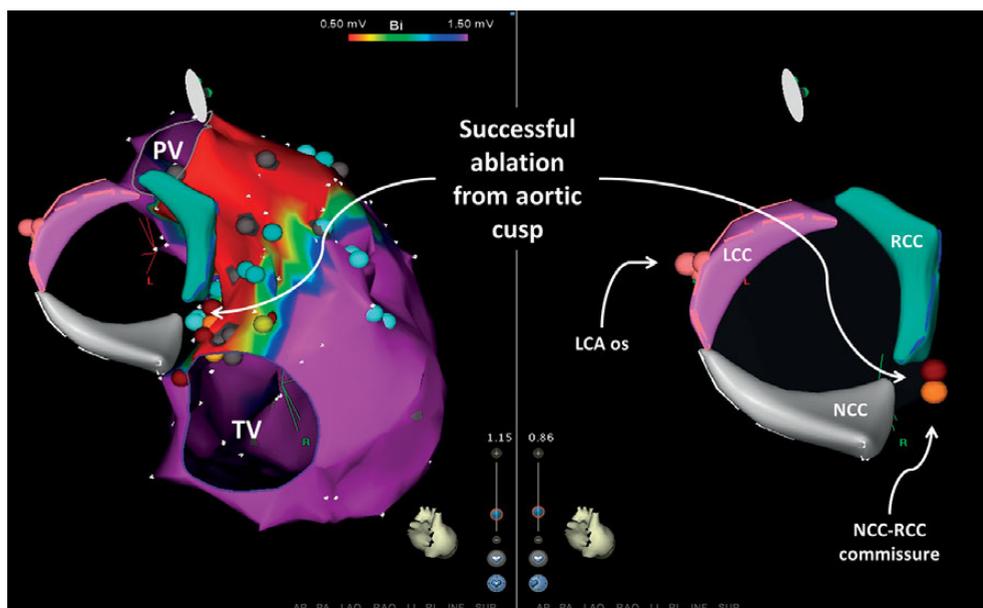


Figure 6. Electroanatomical voltage map (voltage color coded according to bar) of the right ventricle (RV) and coronary cusps in a modified right posterior view. After failed ablation in RV, the aortic root was mapped (cusp anatomy visualized by intracardiac echo using CartoSound® technology). During SR a late potential could be recorded between the NCC and RCC and during VT entrainment was consistent with an isthmus site. A single RF application terminated VT within 10 seconds. LCA = left coronary artery; L/N/RCC = left/non/right coronary cusp; PV= pulmonary valve; TV = tricuspid valve.

hypertrophic myocardium (case 2 and 4), overlying parts of a homograft (case 1) or overlying VSD patch material (case 3).

Anatomical considerations

The outlet septum as important feature of TOF and substrate for VA

TOF is characterized by a (sub)pulmonary stenosis, overriding aorta, VSD and RV hypertrophy.¹⁰ Probably the most important pathologic feature of TOF is anterior displacement of the infundibular septum which results in a subaortic VSD, subpulmonary stenosis and dextroposition of the aorta overriding the ventricular septum. The infundibular septum, i.e. outlet septum, is located between the ventriculo-infundibular fold and the trabeculo septomarginalis and cannot be recognized as a separate structure in the normal heart.^{10, 11} However in TOF, the infundibular septum can be recognized due to its anterior displacement located between the narrowed pulmonary outflow tract and the aortic valve (figure 7 and 8).^{10, 11} The outlet septum in TOF is therefore approachable from the right side (RV and RVOT) and left side (LV, LVOT and aortic root) (figure 7). The thickness of the infundibular septum in TOF can vary from thin fibrous tissue to severely hypertrophied myocardium.^{10, 11}

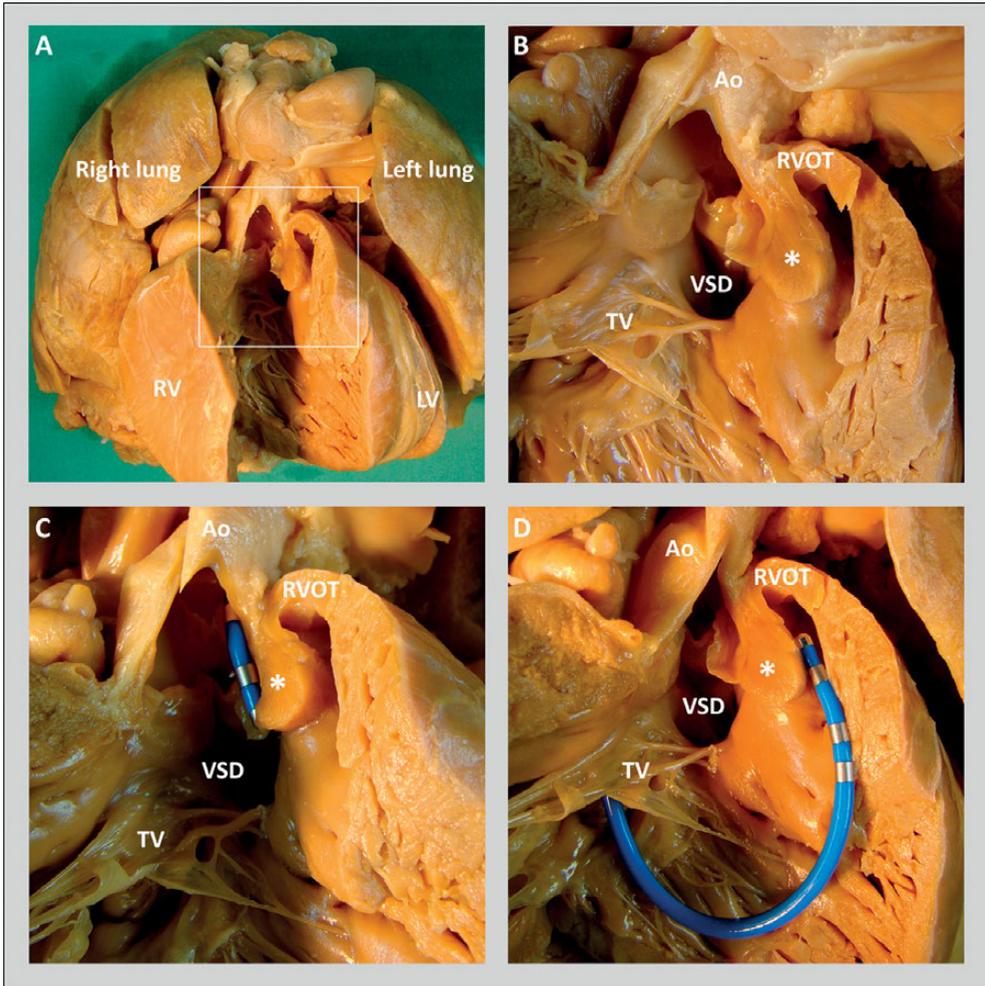


Figure 7. Right ventricular view of a post-mortem specimen with unrepaired Tetralogy of Fallot (4 years old). Panel A provides an overview. Panel B, C and D show the relation between the aorta and outlet septum (*) in detail. The catheter at panel C is positioned in the left coronary cusp. The catheter at panel D is positioned at the pulmonary side of the outlet septum. Please note the spatial relationship between the outlet septum and the sinus of the aorta. Ao = Aorta; LV = left ventricle; RVOT = pulmonary outflow tract; RV = right ventricle; TV = tricuspid valve; VSD = ventricular septal defect; * = outlet septum.

Histological study of the crista supra-ventricularis (i.e. outlet septum) in TOF have demonstrated that (ultra)structural abnormalities (interstitial fibrosis in 65% of the cases) are already present before surgical correction.¹² These (ultra)structural abnormalities and the surgical correction cause myocardial scarring that may provide the substrate for slow conduction and may explain the importance of the infundibular septum for reentry VT.

Prosthetic material and ablation failure

During the primary repair, the VSD is closed with a patch and the (sub)pulmonary stenosis is relieved by resection of the hypertrophied infundibulum and/or a (transannular) patch with additional commissurotomy in some cases.¹³ After correction, the infundibular septum (i.e. the deviated outlet septum) can remain a hypertrophied, muscular structure (figure 8) bordered by PV and VSD (patch). To transect this anatomical isthmus (isthmus 3) ablation from both the right and left side may be required (figure 7 and 8). Pulmonary valve regurgitation, in particular after placement of a transannular patch may require PVR after initial total correction.¹⁴ Mosaic porcine, bovine pericardial, or homograft prostheses are used. Parts of the prostheses may extend towards the VSD patch thereby covering important parts of the infundibular septum (see figure 4). Accordingly the anatomical isthmus between the PV and the VSD may no longer be approachable for catheter ablation from the RV endocardium, and may therefore require a left-sided approach as demonstrated in case 1. Although not observed in our series the combination of graft material and hypertrophy may prevent effective RFCA after PVR even if a combined approach is used. Therefore we feel that preoperative EP study and intraoperative ablation of anatomical isthmuses related to VT is a reasonable consideration even in the absence of spontaneous VTs.

Potential complications

In patients with a muscular VSD, which is present in approximately 20% of TOF patients, the posteroinferior border of the VSD is a muscular rim, which is often only a few millimeters broad. This anatomical isthmus (isthmus 4) is thus bordered by the VSD (patch) and the TA. Importantly, isthmus 4 can be partly covered by the VSD-patch, which is usually stitched some millimeters away from the edge of the rim to avoid damage to the His-bundle.¹³ Therefore a left-sided approach may be required to transect parts of the isthmus covered by the patch. However, due to the proximity of the His-bundle there is a risk of total AV block with the consequent need for (bi)ventricular pacing, as illustrated in case 3.

In this small series no other complications were observed. However, ablation in the aortic sinuses has a risk of coronary injury and the location of the coronary ostia should be visualized by angiography considering the potential for anatomical variation in TOF. RF ablation in the aortic root could also theoretically damage the aortic valve.

CONCLUSION

Although VT in Tetralogy of Fallot is primarily right ventricular in origin, ablation from the left ventricle or aorta is required to interrupt some anatomical isthmuses bordering on the infundibular septum. Our limited series suggests that this approach may be of benefit to a significant number of patients in whom a right-sided approach fails. The proximity of the coronary arteries and the cardiac conduction system needs to be considered.

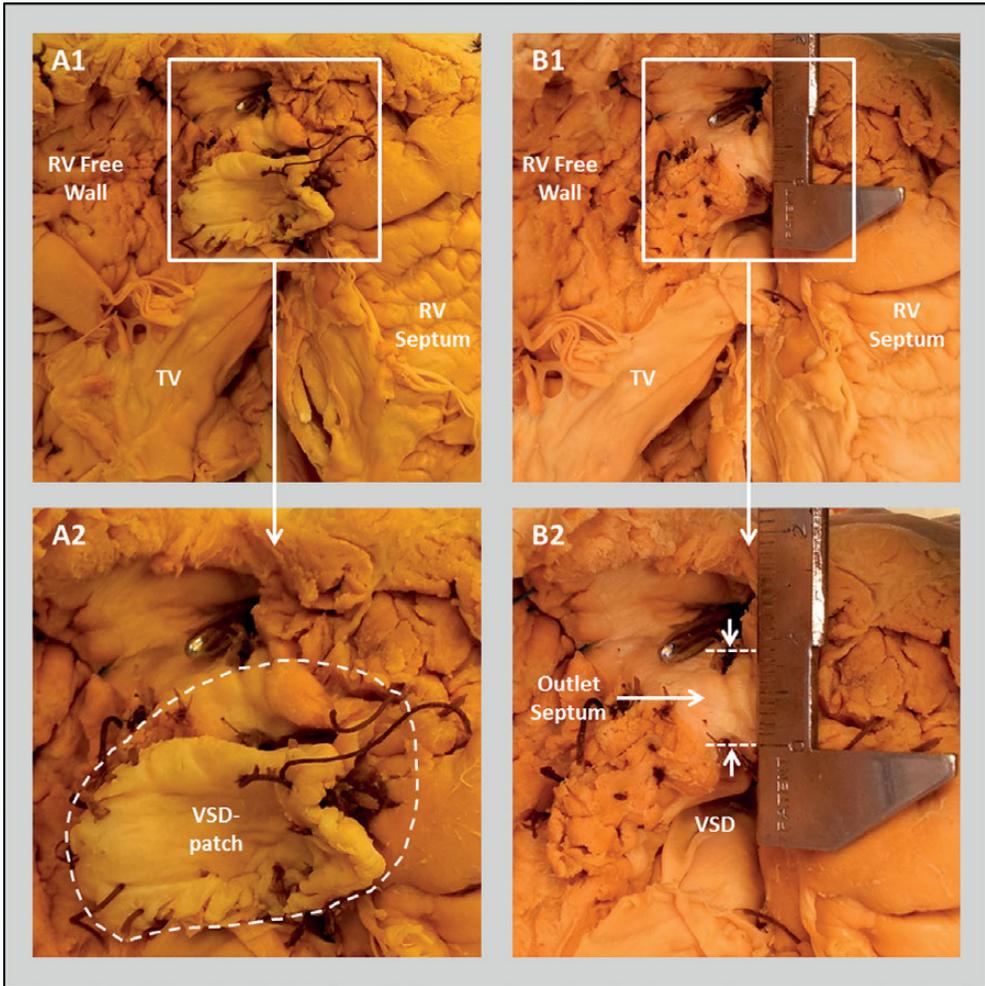


Figure 8. Right ventricular view of a post-mortem specimen with repaired Tetralogy of Fallot (24 years old). The correction consisted of infundibular resection and patch closure of a perimembranous VSD. Panel A1 and B1 provide an overview, panel A2 and B2 show the infundibulum and VSD in detail. The prior location of the VSD-patch (partly removed) is illustrated with the dashed circle in panel A2, the VSD-patch is removed in panel B1-2. A probe was inserted in the pulmonary artery (*not visible*) and advanced through the pulmonary valve (*not visible*) into the infundibulum (*visible*). The probe is located on the pulmonary side of the outlet septum, i.e. the isthmus between the VSD and pulmonary valve. B2, the outlet septum is marked by two dashed lines and perpendicular directed arrows, please note the thickness of the septum (9 mm) even after the correction. RV = right ventricular; TV = Tricuspid Valve; VSD = ventricular septal defect.

ACKNOWLEDGMENTS

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CLINICAL PERSPECTIVE

Ventricular arrhythmias are an important cause for late morbidity and mortality in a growing population of adults with repaired Tetralogy of Fallot. The majority of these ventricular arrhythmias are re-entrant ventricular tachycardias (VTs), which are related to anatomic isthmuses usually approached from the right ventricle. These VTs can be successfully treated with radiofrequency catheter ablation (RFCA) provided that the anatomic isthmus can be completely transected. In the present study performed in 28 consecutive repaired Tetralogy of Fallot patients, right-sided RFCA failed to abolish the VTs in 11 (39%) patients. Of importance, in 8 of 11 patients, the VT was mapped to a septal anatomic isthmus. In 4 of 8 patients, an additional left-sided approach was performed, and in all 4, the anatomic isthmus could be successfully transected from the left, resulting in noninducibility and freedom of VT recurrence during follow-up. RFCA was performed from the aortic root in 3 patients and in 1 patient, with a biventricular implantable cardioverter-defibrillator, from the left side of the septum in proximity to the His-bundle, followed by anticipated atrioventricular block. Based on the demonstrated anatomic and intraoperative observations, right-sided RFCA failure was likely because of the septal hypertrophied myocardium, the overlying pulmonary homograft, and overlying ventricular septal defect patch. A left-sided ablation approach should be considered in repaired Tetralogy of Fallot patients in whom right-sided RFCA of a septal anatomic isthmus failed. Knowledge of the anatomy, the type of repair, and reoperations are helpful to plan the procedure and to anticipate potential complications.

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