Journal of Arrhythmia 30 (2014) 222–229

Contents lists available at ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Review

Radiofrequency catheter ablation of macroreentrant ventricular tachycardia after corrective surgery for tetralogy of Fallot

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ARTICLE INFO

Article history:
Received 5 February 2014
Accepted 19 February 2014
Available online 4 April 2014

Keywords:
Macroreentrant ventricular tachycardia
Tetralogy of Fallot
Catheter ablation
Isthmus ablation

ABSTRACT

Ventricular tachycardia (VT) may occur in patients after corrective surgery for tetralogy of Fallot (ToF), and this can be a cause of sudden cardiac death. Macroreentrant VT is a unique mechanism in these patients, although other mechanisms are involved in VT development. Owing to advances in electrophysiological knowledge and medical technology, macroreentrant VT after corrective surgery for ToF can be treated by catheter ablation. In the macroreentrant circuit of VT, several critical isthmuses (types 1–4) could be included, and these are supported by anatomical obstacles and operative interventions in the right ventricle. Linear radiofrequency (RF) application through the critical isthmus can terminate and prevent the recurrence of macroreentrant VT. Among the critical isthmuses, the type 1 isthmus (between the right ventricular outflow scar and tricuspid annulus) is the most common, but compared with the other types of isthmuses, it is longer so and has a thicker myocardium. Therefore, higher-energy RF application using irrigation and/or large-tip ablation catheters is usually required to complete the linear conduction block. Since other isthmuses may simultaneously work as critical components of the macroreentrant circuit, detailed mapping is encouraged before starting RF application in the type 1 isthmus. Since long-term evidence of the effectiveness of catheter ablation for VT in patients after ToF repair is limited, hybrid treatment with implantable cardioverter defibrillators (ICDs) would be a reasonable strategy for secondary prevention of cardiac events, such as that in patients with other underlying heart diseases. Indications of electrophysiological study, catheter ablation, and/or ICD therapy for primary prevention of sudden cardiac death should be further examined in high-risk patients after ToF repair.

Contents

1. Background

Tetralogy of Fallot (ToF), which is characterized by (1) a large ventricular septal defect (VSD), (2) obstruction or narrowing of the right ventricular outflow tract (RVOT), (3) overriding of the aorta,

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http://dx.doi.org/10.1016/j.joa.2014.02.007
1880-4276/

and (4) right ventricular (RV) hypertrophy, is the most common form of cyanotic congenital heart disease, accounting for approximately 10% of congenital heart defects [1]. Because of advances in surgical treatment for congenital heart diseases, long-term prognosis of patients with ToF has improved. However, ventricular tachycardia (VT), which may develop late after corrective surgery, is a major cause of sudden cardiac death in these patients [2,3]. A previous study showed that the incidence of VT in these patients is approximately 12%, with an 8.3% risk of sudden death (based on 35 years of follow-up) [4].

The ideal therapeutic strategy for VT in patients after corrective surgery for ToF has not yet been established, and few data are available on the results of long-term management of VT. VT, which develops in these patients, is usually caused by a macroreentrant mechanism supported by a circuit around an anatomical and/or surgical RV obstacle [5,6], although other mechanisms (microreentrant and nonreentrant mechanisms) are also involved. Descriptions of successful catheter ablation for VT in ToF patients first appeared in the early 1990s [7,8]. These began as isolated case reports and have grown to small clinical series. Owing to advances in electrophysiological knowledge and medical technology, the acute success rate of radiofrequency (RF) catheter ablation in VT currently approaches 90%, although recurrence of VT is observed in approximately 20% of the patients [5,6,9–12]. This is probably because the characteristics of arrhythmogenic myocardium and the location, size, and length of the critical part of the reentry circuit vary among patients.

RF catheter ablation for VT after ToF repair is still being developed. In this article, we describe the recent therapeutic approaches for macroreentrant VT in patients after corrective surgery for ToF and discuss reasonable management in such patients.

2. Mapping procedures

Macroreentrant VT, which is supported by anatomical obstacles and surgically related nonexcitable regions (surgical scars or patches), is a unique form of VT and is believed to be the most common mechanism of VT in patients after corrective surgery for ToF [5,6,9–12]. VT due to other mechanisms (microreentrant and/or nonreentrant mechanisms) also occurs in these patients, but mapping and ablation procedures for non-macroreentrant VT are similar to those for other VTs that develop in patients with various other structural heart diseases. Accordingly, in this paper, we focus on the mapping and ablation procedures for macroreentrant VT in patients after ToF repair.

Successful and safe catheter ablation is facilitated by an appreciation for anatomical particularities and surgical details. Morphological analysis of clinically documented VT is also important. In these patients, VT usually shows a left bundle branch block-like QRS morphology and inferior frontal plane axis. A right bundle branch block-like morphology may be present if the exit of the reentry circuit is located on the septal aspect of the RV free wall (Fig. 1). Therefore, preprocedural planning should include a careful review of imaging studies (computed tomography and magnetic resonance imaging), original surgical and interventional records, and 12-lead electrocardiograms (ECGs) of all documented VTs.

Since the reentry circuit of VT is usually supported by complex anatomy and extended arrhythmogenic myocardium, electrical physiological study and catheter ablation should be performed using a three-dimensional (3D) mapping system. Typically, the first step in the procedure is to induce tachycardia with programmed electrical stimulation to confirm the diagnosis and exclude supraventricular tachycardia with aberrancy. If VT is already diagnosed by previous electrophysiological studies, it seems reasonable that voltage (substrate) mapping is first accomplished before VT induction because programmed electrical stimulation may induce hemodynamically unstable (clinically or nonclinically documented) VT, which requires immediate external cardioversion to resume basic rhythm.

Low-voltage myocardium is often identified at the RVOT and septal area of the right ventricle. During voltage mapping, attention should be paid to confirm the presence or absence of a conduction

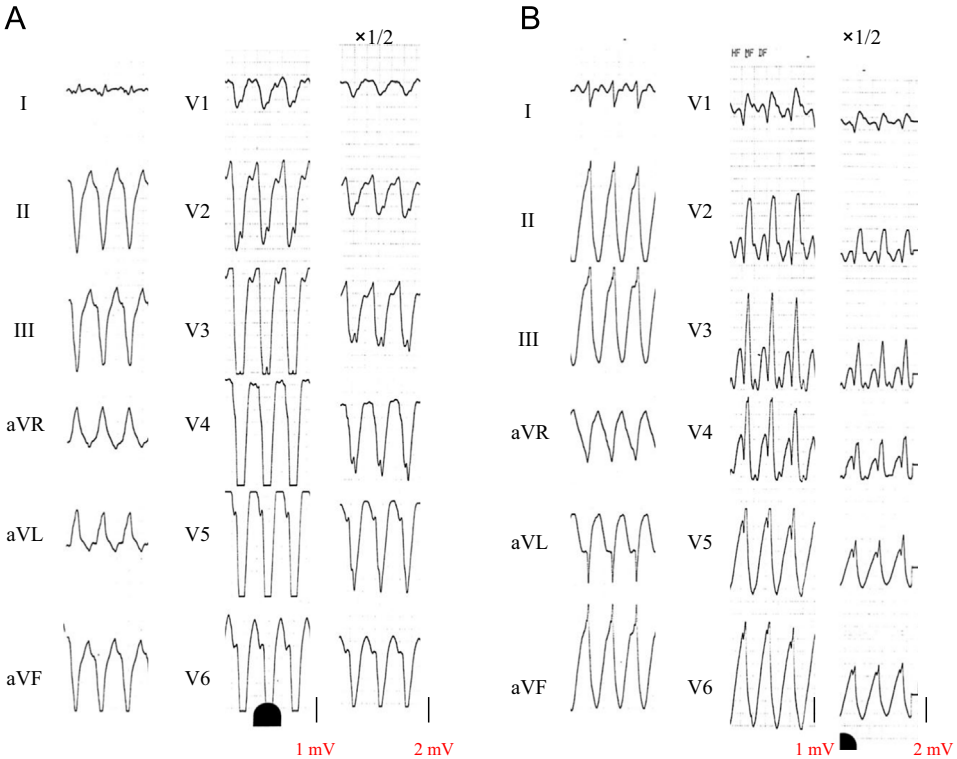

Fig. 1. Ventricular tachycardia (VT). Two VTs were observed in this patient. VT-1 shows a left bundle branch block-like pattern (panel A), and VT-2 shows a right bundle branch block-like pattern (panel B).

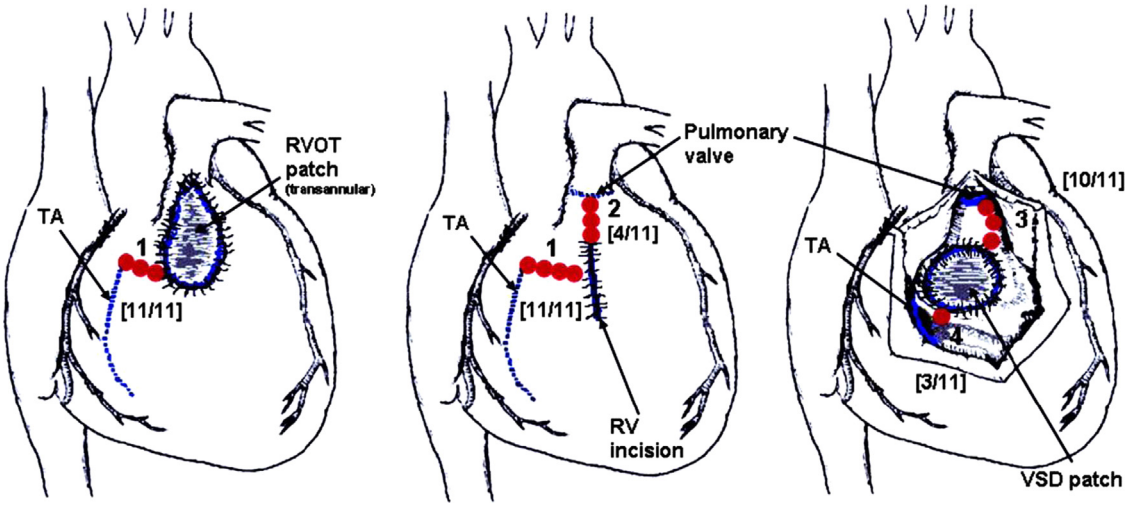

Fig. 2. Schematic representation of the four critical isthmuses for macroreentrant VT after corrective surgery for tetralogy of Fallot. The schema is quoted from Circulation 2007;116:2241–52
(Reference no. [5]: reproduced with permission).

isthmus, which can support the macroreentrant VT circuit in these patients. Four common conduction isthmuses have been proposed in the heart after ToF repair [5] (Fig. 2). Type 1 isthmus is located between the RVOT (or adjacent RV scar) and tricuspid annulus (TA), type 2 isthmus is located between the RVOT scar and pulmonary valve (PV), type 3 isthmus is located between the PV and septal scar, and type 4 isthmus is located between the septal scar and TA. Among them, type 1 isthmuses are the most common, followed by type 3 isthmuses. Type 4 isthmuses are less common, and importantly, a type 2 isthmus does not theoretically exist in patients in whom the RVOT is reconstructed using a transannular patch.

Peak-to-peak bipolar amplitudes are displayed and color coded with electrograms in the 3D mapping system. Local electrograms <1.5 mV are usually defined as low voltage, and electrograms <0.5 mV are defined as very low voltage [13]. At low-amplitude sites, high-voltage pacing (unipolar pacing with 10 mA at a 2-ms pulse width) should be performed to confirm whether the site is an electrically unexcitable scar (EUS) [14]. Anatomical isthmuses are usually defined as areas between the boundaries (such as pulmonary artery=PA, TA, VSD patch, or EUS).

After voltage mapping, programmed electrical stimulation is performed to induce VT, which is highly inducible by such stimulation. During the VT, abnormal local electrograms (fragmentation, late potential, mid-diastolic potential, or continuous activity) are frequently recorded from the wider areas of low-voltage myocardium. However, these abnormal electrograms do not always function as the essential components of the reentry circuit. In patients with hemodynamically tolerated VT, activation and entrainment mapping is performed. If the mechanism of the induced VT is macroreentry, the whole activation sequence can be mapped in the right ventricle [15]. Entrainment pacing from the critical isthmus of the reentry circuit shows concealed fusion between the original VT activation and pacing impulse, and the difference between the postpacing interval and cycle length of VT is <30 ms [16,17]. Diastolic electrical activity is often recorded from the critical isthmus of the VT. A single RF application at the site may terminate the VT, and this suggests that the site is on the essential part of the reentry circuit. However, such a simple termination by RF application is insufficient to prevent VT recurrence, as described later.

For unmappable VT (hemodynamic instability or termination during mapping or entrainment pacing), the locations of isthmuses in the reentry circuit are estimated by the results of pace mapping. Critical isthmuses of the reentry circuit would be the sites where the QRS morphology matches that of the VT (in more than 10 of 12 ECG leads) with an interval from the pacing stimulus to the onset of QRS morphology of >40 ms [18]. If VT can be briefly tolerated, the catheter is moved to the presumed isthmus site during basic rhythm, and the VT is reinduced to confirm whether the position is on the circuit, either by entrainment mapping or by termination during RF delivery [9,19].

In patients after ToF repair, abnormal low-voltage electrograms can be recorded from a very wide area of the right ventricle. In such patients, activation mapping using both multipolar electrode catheters (basket catheters) and ablation catheters would be useful to determine the location of the macroreentrant circuit.

3. Catheter ablation

A reentry circuit is supported by complex anatomy and a thick and/or damaged myocardium; thus, high-energy RF application is usually required to create the conduction block line through the critical isthmus of the reentry circuit in patients after ToF repair. A large-tip (8 mm) ablation catheter (50–60 W) or an open irrigation catheter (30–50 W) can create larger or deeper RF lesions than those created by a standard 4-mm-tip ablation catheter [20,21]. Catheter stability may be improved by using a steerable sheath [22].

As described above, four discrete anatomical isthmuses that often support VT have been identified. Among them, the most common is the type 1 isthmus, which is located between the superior aspect of the TA and an unexcitable scar and/or patch in the free wall of the RVOT. Therefore, in previous studies, first ablation was usually performed on the type 1 isthmus as follows. If the induced VT was hemodynamically stable and entrainment pacing showed the type 1 isthmus to be in the reentry circuit, a line of RF lesions was placed to transect this isthmus. During ablation, the cycle length of VT is generally prolonged, and VT is then terminated. If VT is not stable for mapping, but pace mapping or brief entrainment mapping indicates that the type 1 isthmus is an essential part of the reentry circuit, a line of RF lesions is placed across the type 1 isthmus during sinus rhythm. Successful creation of the linear conduction block through the isthmus would be confirmed by (1) the absence of local capture during pacing along the line, (2) double potentials during basic rhythm (or pacing) along the line, and/or (3) demonstration of the conduction block

through the isthmus during pacing from above or below the line. However, because of RV hypertrophy and a relatively longer length of the isthmus, creation of a complete line conduction block through the type 1 isthmus may be challenging in some patients [23] (Fig. 3). On the other hand, type 2–4 isthmuses are shorter and usually consist of damaged low-voltage myocardium [24,25], which suggests that creation of a conduction block in these isthmuses (types 2–4) is much easier than that in type 1 isthmuses. In addition, if ablation to the type 1 isthmus is unsuccessful, an incomplete conduction block through the isthmus may facilitate VT recurrence. Therefore, before RF application to the type 1 isthmus, we carefully analyze the presence or absence of type 2–4 isthmuses and examine the possibility of whether ablation to these isthmuses (types 2–4) could interrupt the reentrant circuit of VT.

Moore et al. recently described the characterization of the anatomy and histology of VT isthmuses in patients after surgical repair of ToF [24]. In the 27 autopsied hearts, the length and wall thickness of each anatomical isthmus were measured. In that study, the region between the RVOT ventriculotomy site and TA was classified as the type 1A isthmus and that between the RVOT ventriculotomy site and VSD patch was defined as the type 1B isthmus. In patients aged ≥ 5 years at death, type 1A and type 1B isthmuses were present in 88%, type 2 isthmus in 25%, type 3 isthmus in 94%, and type 4 isthmus in 13%. Type 1A isthmus had the greatest dimensions (mean length, 3.9 ± 1.08; thickness, 1.5 ± 0.3 cm), type 1B isthmus had intermediate dimensions (mean length, 2.4 ± 0.8; thickness, 1.1 ± 0.4 cm), and type 2, type 3, and type 4 isthmuses had the smallest dimensions (mean length, 1.5 ± 0.5, 1.4 ± 0.8, and 0.6 ± 0.4 cm, respectively; thickness, 0.5 ± 0.2, 0.6 ± 0.2, and 0.3 ± 0.04 cm, respectively). This study demonstrated that type 1A isthmus was frequently present, but creation of linear transmural RF lesions through the isthmus would be technically difficult even when using irrigation and/or large-tip ablation catheters.

4. Case presentation

To date, we have treated 12 patients with macroreentrant VT after ToF repair. The following are some example cases of our treatment experience [26].

4.1. Case 1

Two clinical VTs (VT1 and VT2) were induced by programmed electrical stimulation, and the cycle lengths of both VTs were 350 ms. VT1 showed left bundle branch block-like morphology, whereas VT2 showed right bundle branch block-like morphology (Fig. 1). The activation wavefront of VT1 circulated counterclockwise around the operation scar, which existed in the RVOT passing through the type 1 and type 2 isthmuses (Fig. 4). On the other hand, the activation wavefront of VT2 propagated clockwise around the same reentrant circuit (Fig. 4). We mapped these two macroreentrant VTs using a single conventional catheter before a 3D mapping system was available for clinical electrophysiological study. A fragmented local potential was recorded from the type 2 isthmus, and rapid pacing from the site demonstrated a concealed entrainment phenomenon. RF application using a 4-mm-tip ablation catheter (50 W) terminated the VT (Fig. 5), but the same VTs were still inducible. Both VTs finally became noninducible after linear RF applications between the PV and operation scar at the RVOT (type 2 isthmus). In this patient, type 1 isthmus also worked as a critical part of the reentry circuit. However, the type 1 isthmus was longer in length and consisted of normal-voltage myocardium. After the RF ablation, no VT recurred

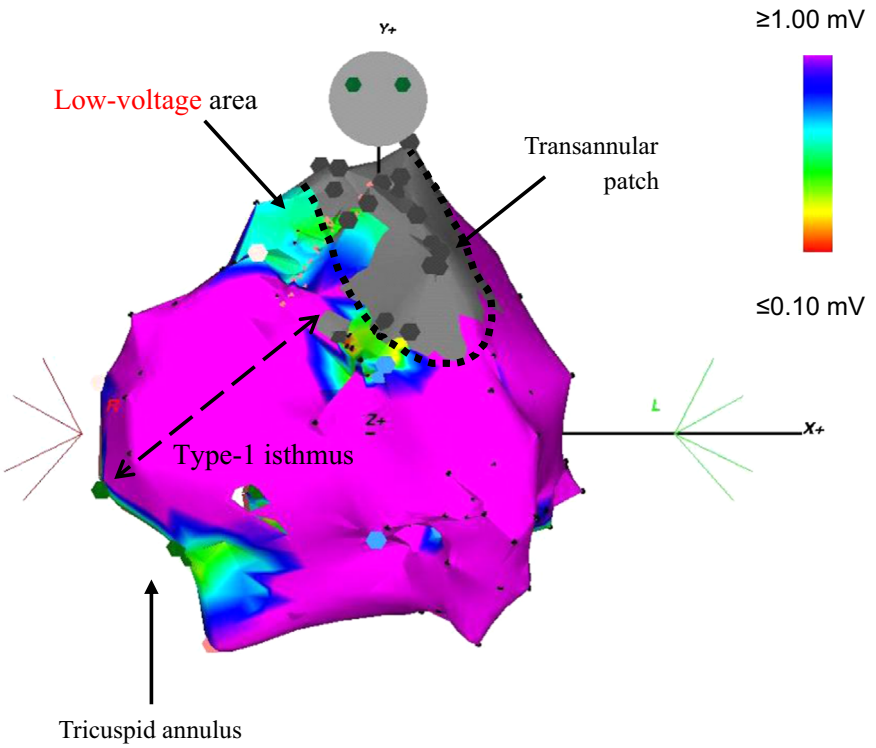

Fig. 3. Voltage mapping of the right ventricle. This case is different from the one in Fig. 1. The outflow tract of this patient was reconstructed using a transannular patch. A dense scar presented in the region of the transannular patch. An area where the endocardial electrograms were < 1.0 mV in amplitude, limited to the area beside the transannular patch. A long type 1 isthmus was present between the transannular patch and tricuspid annulus, and the local electrogram at the isthmus showed normal voltage. The schema is quoted from Internal Medicine 2009;48:1021–3
(Reference no. [23]: reproduced with permission).

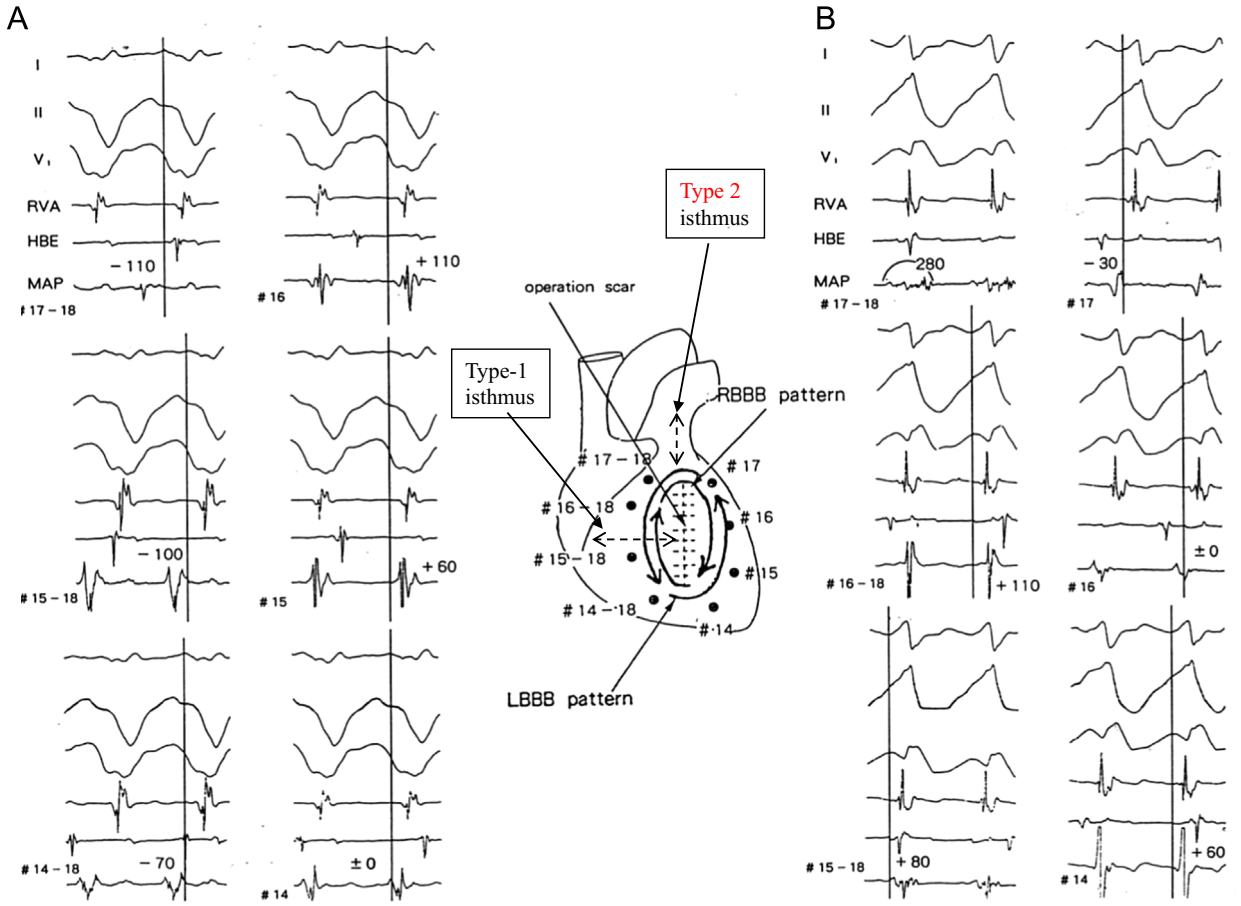

Fig. 4. Activation mapping. This case is the same as the one in Fig. 1. The activation wavefront of VT-1 propagated in a counterclockwise direction around the operation scar in the right ventricular free wall, whereas the activation wavefront of VT-2 circulated along the same obstacle in a clockwise direction. The activation wavefronts of both the VTs passed through the type 1 and type 2 isthmuses, but the type 2 isthmus was shorter than the type 1 isthmus. LBBB-like pattern=left bundle branch block-like pattern, RBBB-like pattern=right bundle branch block-like pattern. RV(apex)=right ventricular apex, HBE=His-electrogram recording site, and Map=mapping catheter. The schema is quoted from Pacing and Clinical Electrophysiology 1997;20:2279–81
(Reference no. [27]: reproduced with permission).

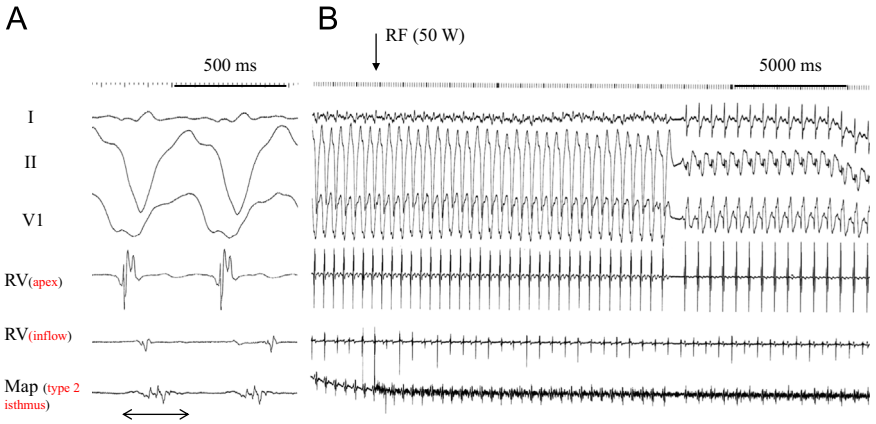

Fig. 5. Catheter ablation. This case is the same as the one in Fig. 1. During VT-1 mapping, a fragmented diastolic potential (marked by a bidirectional arrow) was recorded from the type 2 isthmus (panel A). Using a standard 4-mm-tip ablation catheter, radiofrequency current (50 W) was applied to the site, and the VT terminated after approximately 12 s (panel B). RV(apex)=right ventricular apex, RV(inflow)=right ventricular inflow, and Map=mapping catheter.

during the follow-up period of 20 years. To the best of our knowledge, this is the first case report indicating that RF catheter ablation of a type 2 isthmus successfully treated macroreentrant VT after ToF repair [7].

As described in this patient's case, pleomorphic VT (pleural VTs with monomorphic but different QRS morphologies) can be observed in some patients after ToF repair. Forward and reverse propagation of the activation wavefront through a single macroreentrant circuit is one of the mechanisms of pleomorphic VTs [27]. Diagnosing this mechanism by detailed mapping is important because creation of a single conduction line through the critical isthmus can treat both VTs simultaneously. Pleomorphic VTs can be induced because of the coexistence of pleural macroreentrant circuits in the heart. In such patients, creation of a conduction block line in each critical

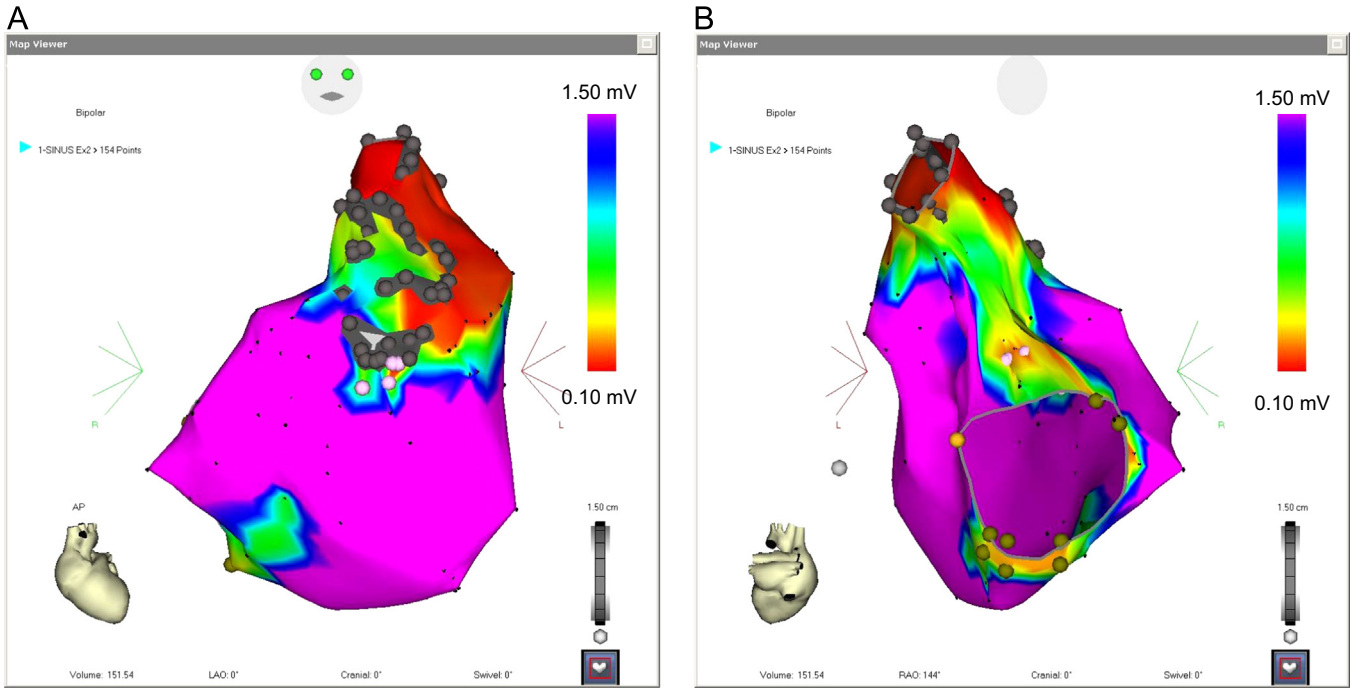

Fig. 6. Voltage mapping in the right ventricle. This case is different from the ones in Figs. 1 and 3. The right ventricular outflow tract was reconstructed using a transannular patch. Wide low-voltage areas (< 1.5 mV) were distributed in the anterior wall (panel A) and septal wall (panel B) of the right ventricle.

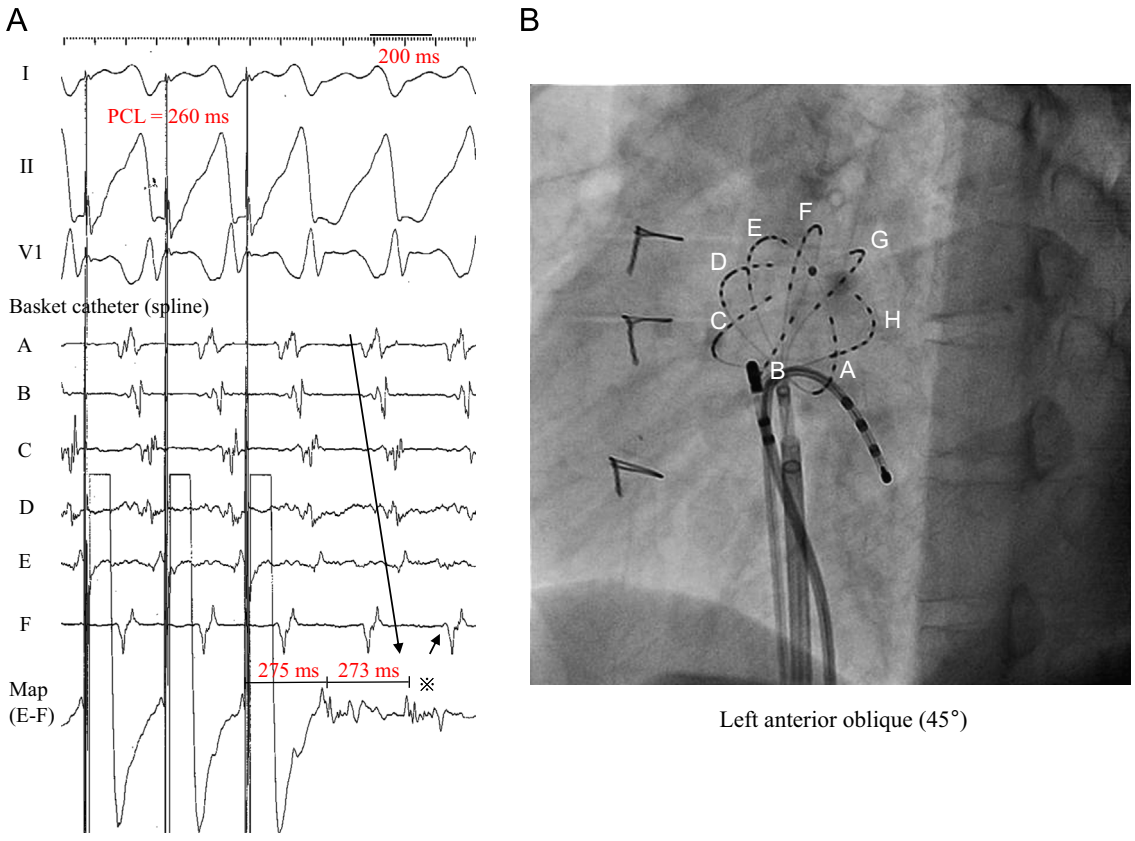

Fig. 7. Activation mapping using an ablation catheter and a basket catheter. This case is the same as the one in Fig. 6. A basket catheter was placed in the inflow area of the right ventricle. The activation sequence of the VT propagated from spline A to spline E, but an activation gap was observed between splines E and F. When the mapping catheter was moved to the site between the splines E and F, a fragmented diastolic potential was recorded. Rapid pacing (at a cycle length of 260 ms) from the site showed a concealed entrainment phenomenon, and the first postpacing cycle length at the site (275 ms) was almost identical to the cycle length of the VT (273 ms). PCL=pacing cycle length, and Map=mapping catheter.

isthmus can eliminate the pleomorphic VTs. Pleomorphic VTs can also be caused by the coexistence of macroreentrant, microreentrant, and nonreentrant VTs. In such patients, treatment of all VTs solely by using catheter ablation is difficult, and hybrid treatment with implantable cardiac defibrillators (ICDs) is usually required, as described below.

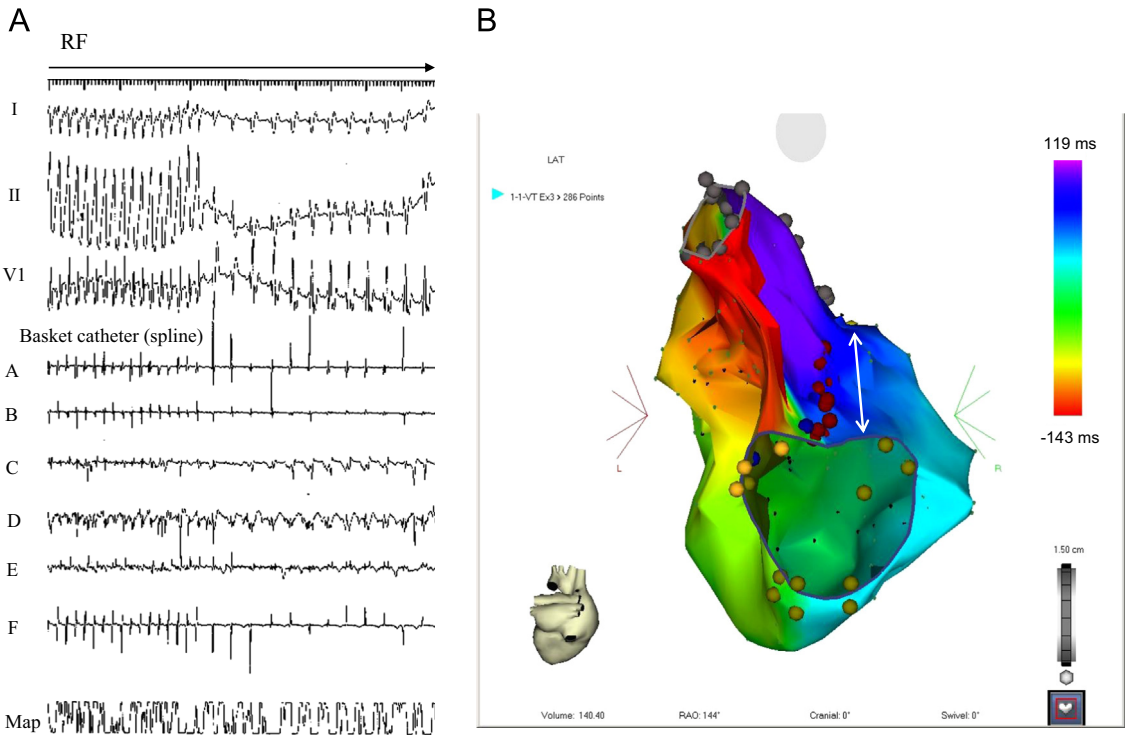

Fig. 8. Catheter ablation and activation mapping. This case is the same as the one in Figs. 6 and 7. Three-dimensional mapping demonstrated that the activation wavefront of the VT was circulating around the tricuspid annulus through the type 4 isthmus (left panel). VT was terminated following radiofrequency (RF) current application to the site showing the concealed entrainment (right panel). Linear RF applications (marked by arrow) were delivered to the isthmus (left panel), and the VT became noninducible. Map = mapping catheter.

4.2. Case 2

In this patient, the RVOT was reconstructed using a transannular patch, and therefore, a type 2 isthmus was not created. Three-dimensional voltage mapping demonstrated wide low-voltage (or scar) areas in the RVOT and RV septum [28] (Fig. 6). On the other hand, local electrograms distributed between the RVOT scar and TA (type 1 isthmus) showed normal voltage. Clinical VT was induced by programmed electrical stimulation with a cycle length of 270–280 ms. We performed activation mapping using an ablation catheter and a basket catheter (Fig. 7) and confirmed that the mechanism of the VT was macroreentry due to the activation wavefront circulating around the TA through the type 4 isthmus (Fig. 8). During the VT, a diastolic fragmented electrogram was recorded from the lower RV septum (type 4 isthmus), and rapid pacing from the site showed concealed entrainment (Fig. 8). RF application to the site (50 W by a 4-mm-tip catheter) terminated the VT, but the same VT was reinduced. After linear RF application through the isthmus, double potentials were recorded on the ablation line, and the VT became noninducible.

5. Role of ICD treatment

VT, which develops in patients after ToF repair, can be treated by catheter ablation. However, the long-term results of catheter ablation have not yet been clarified. As described above, ablation procedures targeting the critical isthmus of the macroreentrant circuit are a standard treatment strategy; however, this strategy recently became the standard approach only after 3D mapping systems became available in clinical electrophysiological studies, and therefore, follow-up results in patients in whom VTs were treated by critical isthmus ablation are limited.

In patients with VT associated with various structural heart diseases, ICD therapy is recommended even if the target VT is successfully ablated and no VT is inducible after catheter ablation [29]. This is because, in such patients, the same VT and/or new VT recurs during the follow-up period because of the presence of an arrhythmogenic myocardium in the heart and/or progression of the underlying heart diseases (prior myocardial infarction, idiopathic dilated cardiomyopathy, arrhythmogenic RV dysplasia, or cardiac sarcoidosis). In addition, ICD therapy is also recommended for primary prevention of sudden cardiac death in patients with an impaired left ventricular function (which is usually indicated by a lower left ventricular ejection fraction of <30–35%) without clinically documented VTs [29–31].

On the other hand, in patients after ToF repair, the left ventricular ejection fraction is usually preserved, although myocardial hypertrophy and/or dilation of the right ventricle persists and could work as an arrhythmogenic substrate long after successful catheter ablation. Therefore, it is reasonable that ICD therapy is recommended in patients with aborted sudden cardiac death and/or ventricular fibrillation. ICD therapy is also recommended in patients in whom programmed electrical stimulation induces nonclinically documented microreentrant and/or nonreentrant VT in addition to the clinically documented macroreentrant VT. In such patients, the arrhythmogenic substrate might be extensively present in the heart. However, indication of ICD therapy is controversial in patients in whom hemodynamically stable clinically documented macroreentrant VT is successfully treated and no other VT is inducible after catheter ablation. We encountered few such patients (Case 1) because these patients were treated before ICD devices were available in our country. Although the clinical outcomes of these patients have been uneventful, supplemental treatment with ICDs may be necessary in such patients because recurrences of VT after successful catheter ablation have been reported. Unlike that for secondary prevention, the effectiveness of ICD therapy and/or

catheter ablation for primary prevention of sudden cardiac death has not yet been established. Although several factors (left and/or RV dysfunction, late corrective surgery, a wider QRS width, and presence of pulmonic valve regurgitation and/or RVOT stenosis) have been proposed as future risks of cardiac events in patients after ToF repair [4,32–35], the risk evaluation method and/or therapeutic approach (using ICDs, catheter ablation, or antiarrhythmic drugs) for primary prevention of sudden cardiac death should be studied further.

6. Summary and conclusions

(1) VT, which develops in patients after ToF repair, includes a macroreentrant mechanism, and its activation wavefront propagates through several critical isthmuses (types 1–4) created by anatomical and surgical obstacles in the right ventricle.
(2) Creation of the linear conduction block through the critical isthmus terminates and prevents the recurrence of macroreentrant VT. However, because of the distribution of the thick myocardium and the long length of the isthmus, higher-energy RF applications using irrigation and/or large-tip ablation catheters are usually required to complete the linear conduction block.
(3) Type 1 isthmuses are the most common, but compared with the other isthmuses, they are longer and have a thicker myocardium.
(4) Long-term evidence of the effectiveness of RF ablation for VT associated with ToF repair is limited. Therefore, supplemental treatment with ICDs is recommended for secondary prevention of cardiac events, especially in patients in whom non-macroreentrant VT is observed clinically and/or induced by programmed electrical stimulation. Indications of electrophysiological study, catheter ablation, and/or ICD therapy for primary prevention of sudden cardiac death in high-risk patients should be further examined.

Conflict of interest

The authors have no conflict of interest to disclose.

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