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Review

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

Masaomi Chinushi, MD[a,*], Osamu Saitou[a], Hiroshi Furushima, MD[b]

[a] Graduate School of Health Science, Niigata University School of Medicine, 2-746 Asahimachi, Chuou-ku, Niigata 951-8518, Japan
[b] Cardiovascular Biology and Medicine, Niigata University School of Medicine, Niigata, Japan

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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,

* Corresponding author. Tel./fax: +81 25 227 0952.
E-mail address: masaomi@clg.niigata-u.ac.jp (M. Chinushi).

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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

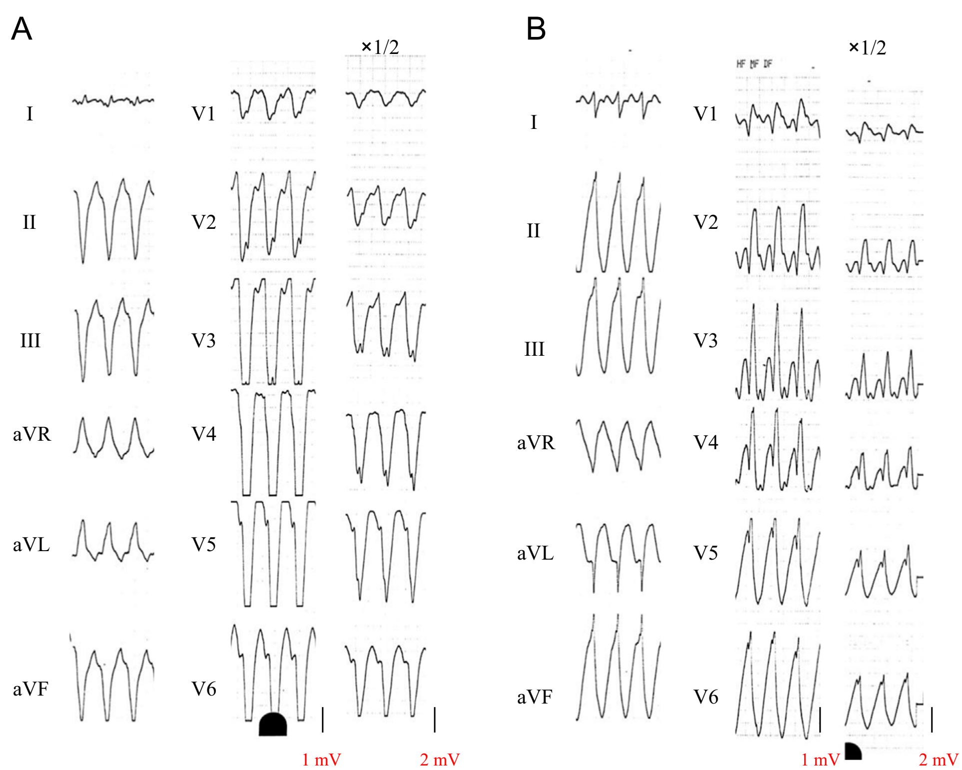

Fig. 1. Ventricular tachycardia (VT). Two VTs were observed in this patient. VT-1 shows a left bundle branch block-like pattern (panel A), and VT-2 shows a right bundle branch block-like pattern (panel B).