Case Report

# Regional Ablation of Idiopathic Right Ventricular Outflow Tract Tachycardia Guided by Electroanatomical Mapping

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Several studies have reported excellent outcomes using catheter ablation in the right ventricular outflow tract (RVOT) to treat idiopathic left bundle branch block ventricular tachycardia. In the present report a case is presented where a sustained monomorphic RVOT tachycardia originated from multiple contiguous sites in the RVOT, giving rise to a well-circumscribed area with abnormal activity. The arrhythmia was mapped with a non-contact electroanatomical mapping system and the exit sites of the tachycardia were found in the posterolateral aspect of the RVOT. The tachycardia was successfully ablated in that area.

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Idiopathic ventricular tachycardias (VT) by definition occur in patients without identifiable structural heart disease. In the majority of cases they originate near the right ventricular outflow tract (RVOT) as well as from the posteroseptal aspect of the left ventricle. For the treatment of patients with idiopathic, inferior-axis VT resembling left bundle branch block (LBBB) RF energy is usually applied to the RVOT. Pace mapping and, to a lesser extent, activation mapping are used to select the appropriate endocardial site for catheter ablation.[1,2] In patients who cannot be ablated from the right ventricle, specific characteristics of the QRS complex during VT may point to an origin in the left ventricular outflow tract or aortic root.[3-5] Until now, no anatomically well-defined substrate for idiopathic RVOT-VT has been found. It has been suggested that subtle anatomical, structural or functional derangement of the myocardial tissue in the RVOT can lead to the pathophysiologic substrate that triggers abnormal automaticity or triggered activity. In a recent study it was reported that myocardial sleeves may penetrate from the pulmonary artery to the RVOT in some cases and give rise to tachycardias that are analogous to pulmonary vein triggers in atrial tachycardias or atrial fibrillation.[6] Despite this, RVOT tachycardia ablation is currently considered a focal ablation based on the information obtained by conventional mapping.

Electroanatomical mapping with the non-contact system EnSite 3000 (Endocardial Solutions Inc.) allows the anatomical reconstruction of the evaluated cavity along with the calculation of the unipolar potentials from 3600 points of this cavity. The electrophysiologic information is displayed as isopotential maps of the propagation wavefront and as "virtual" unipolar electrograms from the corresponding sites.

In the present report a case is presented where a sustained monomorphic RVOT tachycardia originated from multiple contiguous sites in the RVOT, giving rise to a well-circumscribed area with abnormal activity.

## Case report

A 48-year-old man was evaluated for multiple presyncopal attacks and one wit-

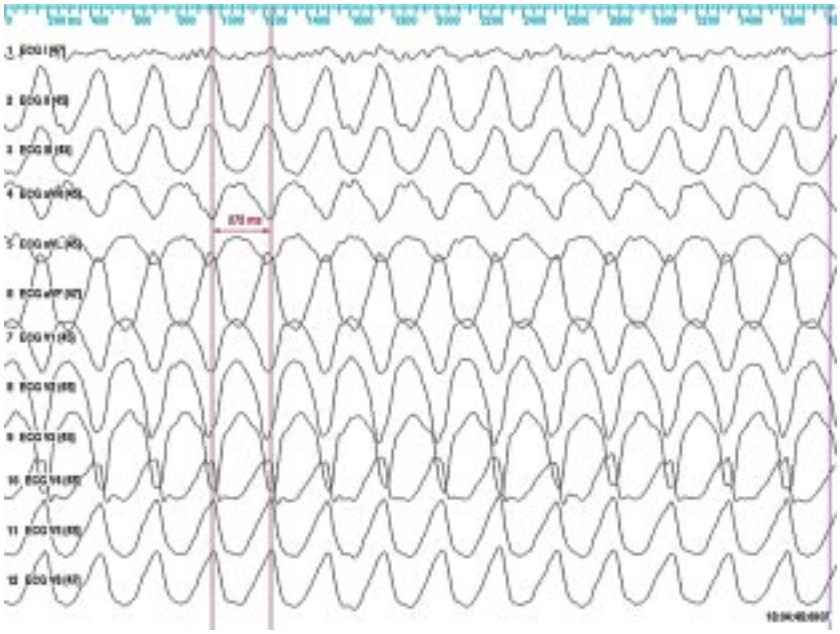

**Figure 1.** Twelve-lead ECG during ventricular tachycardia, demonstrating a left bundle branch block-inferior axis morphology.

nessed syncope during the last 6 months. The patient complained of palpitations that preceded all of these episodes. He underwent 24-hour Holter monitoring that revealed multiple non-sustained and one sustained episode of a wide QRS tachycardia with a rate of 240 /min. His resting ECG was normal with occasional ventricular premature beats having an LBBB-inferior axis morphology. In order to exclude structural heart disease the patient was given a complete evaluation, including an echocardiogram, catheterization with coronary arteriography and a signal-averaged electrocardiogram, all of which were normal. The patient was then scheduled for an electrophysiologic study, which failed to induce any kind of tachycardia despite the use of aggressive pacing protocols and the infusion of high doses of isoproterenol. Interestingly, during the 2-hour study only 3 to 4 ventricular premature beats were recorded. These were insufficient to form an indication or to suggest further therapeutic procedures. The patient was discharged with instructions to return if symptoms recurred.

Twenty days later the patient complained of multiple self-terminating episodes of palpitations with dizziness and he was scheduled for a second electrophysiologic study. During this procedure femoral vein puncture was performed after minimal local anesthesia with lidocaine in order to avoid possible systemic antiarrhythmic actions of the drug. Conventional catheters were introduced via the femoral veins and positioned in the His region and right ventricle. During the infusion of isoproterenol, and despite the complete absence of ventricular extrasystoles initially, a sustained monomorphic VT was induced, with a cycle length of 270 ms and an LBBB-inferior axis morphology, that self-terminated after discontinuation of isoproterenol (Figure 1). The non-contact system EnSite 3000 was then inserted transfemorally and used for the electroanatomical mapping of the tachycardia. Specifically, the multielectrode basket catheter of the system was introduced into the right ventricular outflow tract and positioned just under the pulmonary valve. The geometric reconstruction of the right ventricle was achieved by dragging a mapping catheter along the walls of the cavity and specific anatomic landmarks such as the pulmonary valve edges, the right ventricular apex and the tricuspid valve.

A run of the clinical tachycardia was then recorded by the system after repeat challenge with isoproterenol and analyzed off-line. Under appropriate filtering of the signals in order to exclude repolarization potentials, the ectopic activation sequence of the right ventricle was tracked backwards toward the exit site of the tachycardia. Tracking was accomplished via the isopotential maps provided by the mapping system. The exit site of the tachycardia was mapped in the posterolateral aspect of the RVOT. The virtual electrograms at this site exhibited the characteristic QS morphology of an exit site. However, during

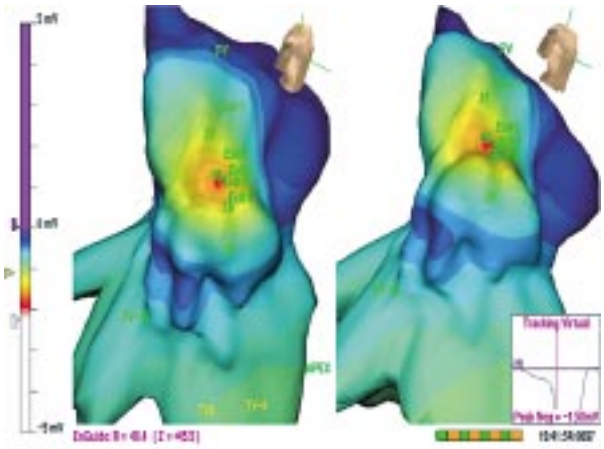

**Figure 2.** Exit sites of the tachycardia within a circumscribed area of the right ventricular outflow tract (RVOT). The white color represents the most negative area at the initiation of the propagation sequence.

the analysis of consecutive complexes of the tachycardia it became evident that the exit site was not stable but moved through contiguous sites within a circumscribed area of the aforementioned wall of the RVOT. Five different exit sites were spotted within this area and ranged anatomically from beneath the pulmonary valve to just above the His bundle area (Figure 2). The local unipolar virtual electrogram was earlier by 15-30 ms and exhibited a QS morphology in the respective beats, with slight variations, and the recording of a small initial r wave for the beats originating from the other exit sites (Figure 3). These small deviations of the exit site of the tachycardia were translated to subtle changes of the surface QRS morphology. Subsequently, in order to delineate the characteristics of the triggering area in the RVOT, the ventricular depolarization was mapped in sinus rhythm. During the analysis it became evident that the abnormal area was characterized by entrance block and revealed small but smooth local electrograms (Figure 4). Pace mapping from various sites in the above area induced a similar but not identical QRS morphology to that of the clinical tachycardia (9-11/12 ECG leads).

Radiofrequency energy was delivered at multiple sites of the above area in a regional ablating fashion (Figure 5). All deliveries within this area were followed by a non-paroxysmal idioventricular rhythm with a QRS morphology identical to that of the tachycardia (Figure 6). In the final check the tachycardia was no longer inducible, despite maximal infusion of isoproterenol and aggressive pacing protocols. The patient remained completely asymptomatic thereafter for a 2 month follow-up period.

## Discussion

With growing experience and expertise, radiofrequency ablation has become a curative procedure for idiopathic ventricular tachycardia. Previous studies using catheter ablation in the RVOT to treat idiopathic LBBB-VT reported excellent outcomes.[1,5,7] Ablation of idiopathic RVOT tachycardias is considered a focal ablation that requires delineation of the tachycardia exit site with activation and pace mapping techniques.

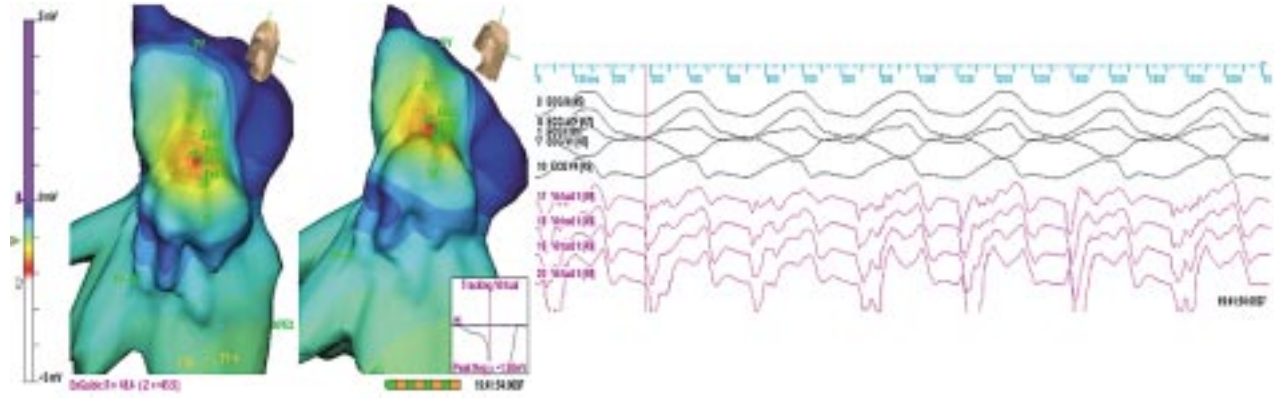

**Figure 3.** The "virtual" electrograms at the exit sites. The QS morphology is characteristic in the unipolar recording, while the morphology becomes rS at sites distally to the exit site, located as shown in the anatomic reconstruction.

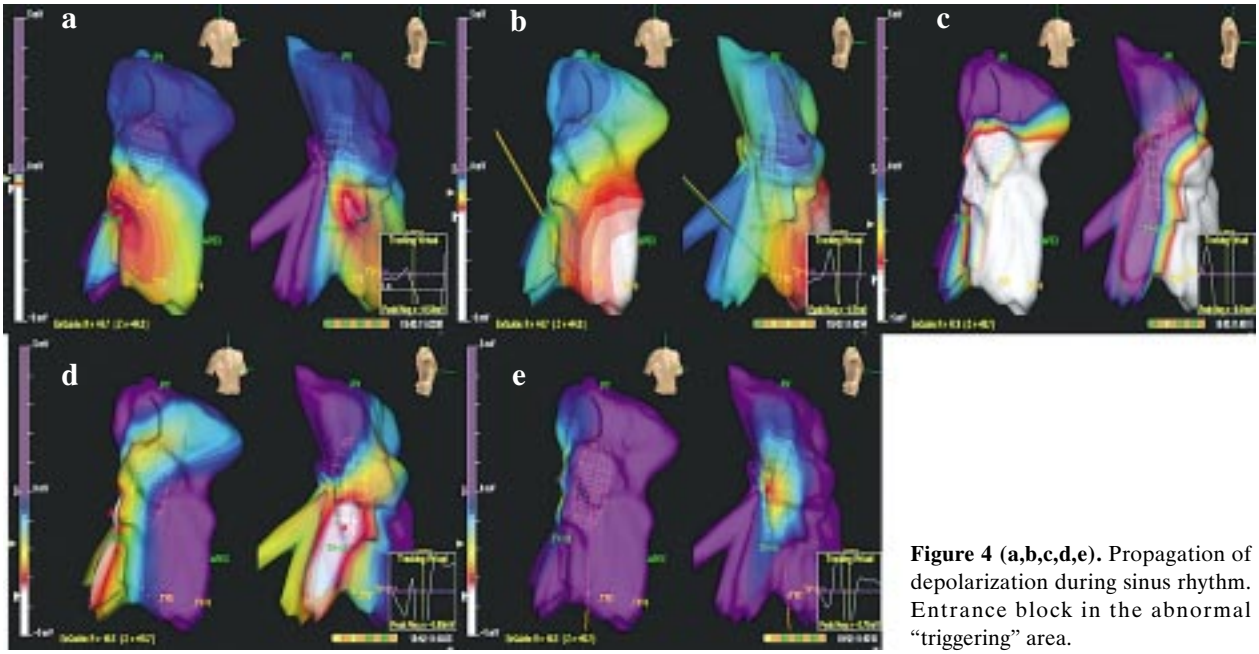


**Figure 4 (a,b,c,d,e).** Propagation of depolarization during sinus rhythm. Entrance block in the abnormal "triggering" area.

However, in the present case report it was evident that the pathophysiologic mechanism of the tachycardia was not supported by a focal spot site but by multiple exit sites within a circumscribed area. This area was characterized by entrance block during sinus rhythm, and during tachycardia it supported multiple alternating "triggering" sites. The presence of an abnormal myocardial area within the concept of idiopathic ventricular tachycardia is difficult to substantiate with contemporary diagnostic tools. However, it has been shown that the embryonic avian and mammalian outflow tract, as well as the outflow tract in adult primitive fish (called conus) and amphibians (called bulbus cordis), is surrounded by myocardium.[8] It has been demonstrated that this myocardium supports semilunar valve function in adult fish and frogs and substitutes for this function in embryonic chicken hearts. In contrast to the myocardium of the atrial and ventricular chambers, this myocardium retains its embryonic features, namely slow propagation of the depolarizing impulse owing to the poor intercellular coupling of the cardiac muscle cells. This characteristic is also a prerequisite for nodal function. In normal mammalian development, the proximal outflow tract myocardium becomes ventricularized by incorporation into the right ventricle, whereas the myocardium of the distal outflow tract disappears. It can be hypothesized that if this ventricularization of myocardium is not complete, remnants persist that may provide the substrate for these tachycardias.

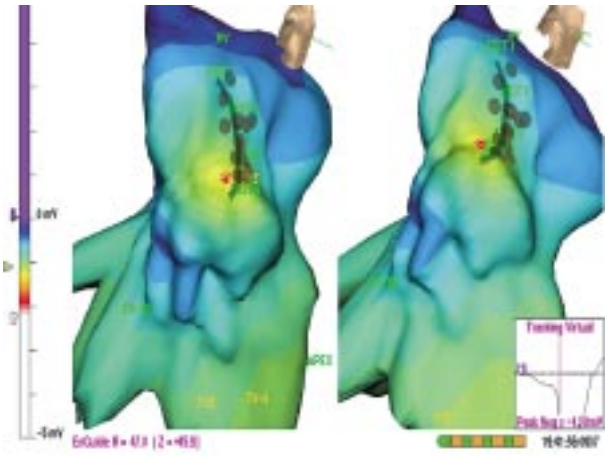

**Figure 5.** Ablation lesions delivered in the "triggering" area of the arrhythmia, depicted as brown spots.

A conventional approach to such a tachycardia would be practically impossible, in view of both its high rate, with subsequent hemodynamic instability of the patient, and the fact that it was paroxysmal and self-terminating. Moreover, even if possible, a conventional approach would result in the location of multiple "early" sites during activation mapping characterized by suboptimal pace mapping. However, non-contact mapping overcame these drawbacks of conventional mapping and successfully guided the ablation procedure.

In conclusion, idiopathic RVOT tachycardias can safely and effectively be ablated under guidance from a non-contact electroanatomic mapping system. It seems that in some cases the triggering site of the

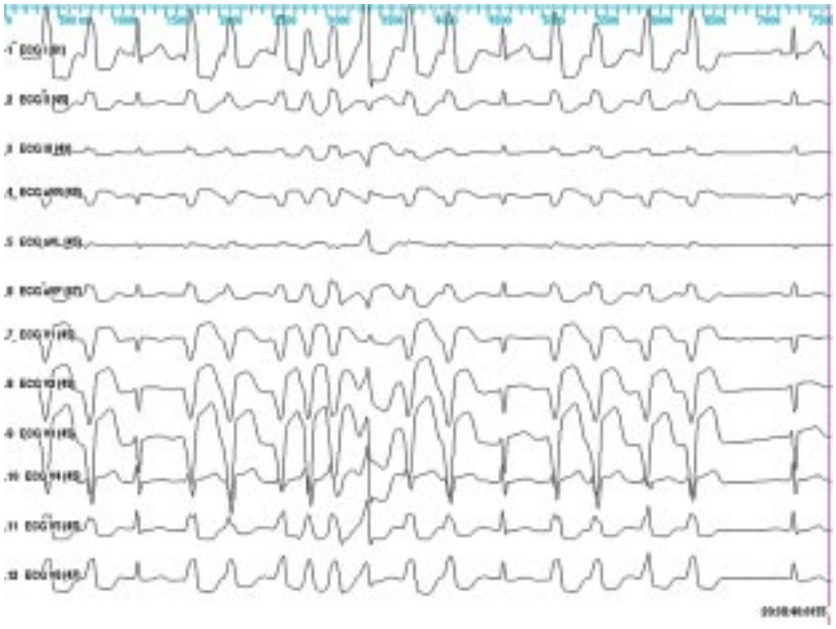

**Figure 6.** Non-paroxysmal idioventricular rhythm during RF application with QRS morphology identical to that of the clinical tachycardia.

arrhythmia may be more complex than a simple focal site. This finding may be associated with ablation failures or recurrences if conventional mapping is the ablation guiding tool.

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