

<sup>1</sup>Department of Cardiology, Asklepios Klinik St. Georg, Hamburg, Germany,

# Left Ventricular Outflow Tract Tachycardia Including Ventricular Tachycardia from the Aortic Cusps and Epicardial Ventricular Tachycardia

K.R. Julian Chun, Kazuhiro Satomi, Karl-Heinz Kuck, Feifan Ouyang, Matthias Antz<sup>1</sup>

## Key Words:

Ventricular tachycardia · Catheter ablation · Left ventricular outflow tract · Aortic cusp · Epicardial VT · ECG

Herz 2007;32:226–32

DOI 10.1007/s00059-007-2977-0

## Schlüsselwörter:

Ventrikuläre Tachykardie · Katheterablation · Linksventrikulärer Ausflusstrakt · Aortensinus · Epikardiale VT · EKG

## Abstract

Idiopathic outflow tract ventricular tachycardia (VT) can arise from the right (RVOT) or left ventricular outflow tract (LVOT). The electrocardiographic (ECG) pattern of RVOT VT is typical in most patients, showing a monomorphic left bundle branch block (LBBB) QRS morphology with an inferior axis. Radiofrequency catheter ablation can be performed with a high success rate and provides a curative therapeutic approach. However, not all VTs with LBBB and inferior axis can be ablated from the RVOT. It has become apparent that

LVOT VTs including VT originating from the aortic sinus of Valsalva or epicardium represent underrecognized VT entities which are also amenable to successful catheter ablation. Twelve-lead ECG criteria can contribute to distinguish between sites of VT origin.

LVOT arrhythmias represent an increasingly recognized VT entity which can be safely and successfully treated by catheter ablation. Identification of VT origin using ECG criteria and differentiation of LVOT versus RVOT origin is essential in the careful planning of the ablation strategy.

## Linksventrikuläre Ausflusstrakttachykardien inklusive ventrikulärer Tachykardien vom Aortensinus und epikardialer ventrikulärer Tachykardien

### Zusammenfassung

Idiopathische ventrikuläre Ausflusstrakttachykardien (VTs) können sowohl im rechtsventrikulären (RVOT) als auch im linksventrikulären Ausflusstrakt (LVOT) entstehen. Das Elektrokardiogramm (EKG) einer RVOT-VT zeigt typischerweise einen monomorphen Linksschenkelblock mit einer inferioren Achse. Die Katheterablation ist als kurativer Ansatz mit einer sehr hohen Erfolgsrate gut etabliert. LVOT-VTs einschließlich VTs aus dem Bereich der Aortensinus oder mit epikardialem Ursprung stellen eher seltener diagnostizierte VT-Enti-

täten dar, die allerdings ebenfalls mittels Katheterablation erfolgreich behandelt werden können. Zur Unterscheidung zwischen VT-Ursprung im RVOT und LVOT können Zwölf-Kanal-EKG-Kriterien verwendet werden.

Arrhythmien aus dem LVOT können sicher und erfolgreich mit Katheterablation behandelt werden. Die Identifikation des VT-Ursprungs mittels EKG-Kriterien zur Differenzierung zwischen einem VT-Ursprung im LVOT und RVOT ist für die sorgfältige Planung der Katheterablationsstrategie notwendig.

### Introduction

Idiopathic ventricular tachycardia (VT) is recognized as a ventricular arrhythmia without an apparent structural heart disease and accounts for approximately 10% of all patients referred for an evaluation of VT [1]. Idiopathic VT can present as a repetitive monomorphic VT (RMVT) characterized by frequent short salvos of monomorphic nonsustained VT or frequent ventricular premature contractions (VPCs; Figure 1). It was first observed by Gallaverdin in 1922 [2] and is variously described in the literature as RMVT, right ventricular outflow tract (RVOT) VT, adenosine-sensitive VT, exercise-induced VT, or catecholamine-induced VT. Outflow tract arrhythmia (VT or frequent

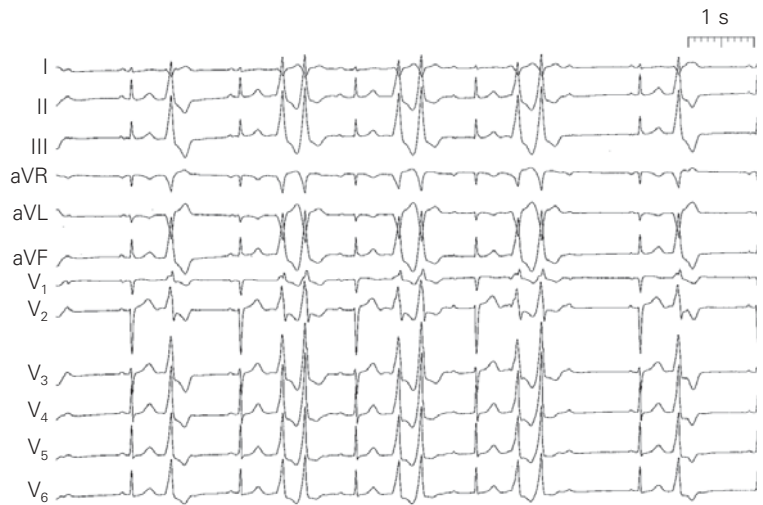
VPCs) is the most common form of idiopathic VT and typically originates from the RVOT but can also occur from the left ventricular outflow tract (LVOT), the aortic sinus of Valsalva and the epicardial surface of the ventricles [3–5]. Approximately 10–15% of cases arise from the LVOT [6–8].

The electrocardiographic (ECG) pattern of RVOT VT is typical in most patients, showing a monomorphic left bundle branch block (LBBB) QRS morphology with an inferior axis (Figure 2). Radiofrequency (RF) catheter ablation can be performed with a high success rate and provides a curative approach [9–11]. Recently, it has become apparent that LVOT VTs including VT originating from the aortic

sinus of Valsalva or the epicardium represent an underrecognized VT entity which is also amenable to successful catheter ablation [3].

**Anatomy**

It may appear surprising that VT can originate from the aortic sinuses, not traditionally thought to be associated with ventricular myocardium. However, crescent fibers of ventricular myocardium have been identified in particular at the base of the left and right aortic sinuses [12] which may serve as the underlying arrhythmogenic anatomic structure. By contrast, the base of the noncoronary cusp is composed of fibrous tissue [13] explaining the very rare incidence of VT originating from this location [14]. The anterior aspect of the LVOT, in particular the right aortic cusp, is closely related to the posterior aspect of the RVOT explaining similar twelve-lead ECG characteristics for arrhythmias arising from this origin. The LVOT is in close relationship to the right-sided superior septal region (His). Moreover, the aortic valve is located more inferior and posterior in comparison to the pulmonary valve, therefore, subtle twelve-lead ECG differences result and help to distinguish the VT origin which is discussed in detail below.



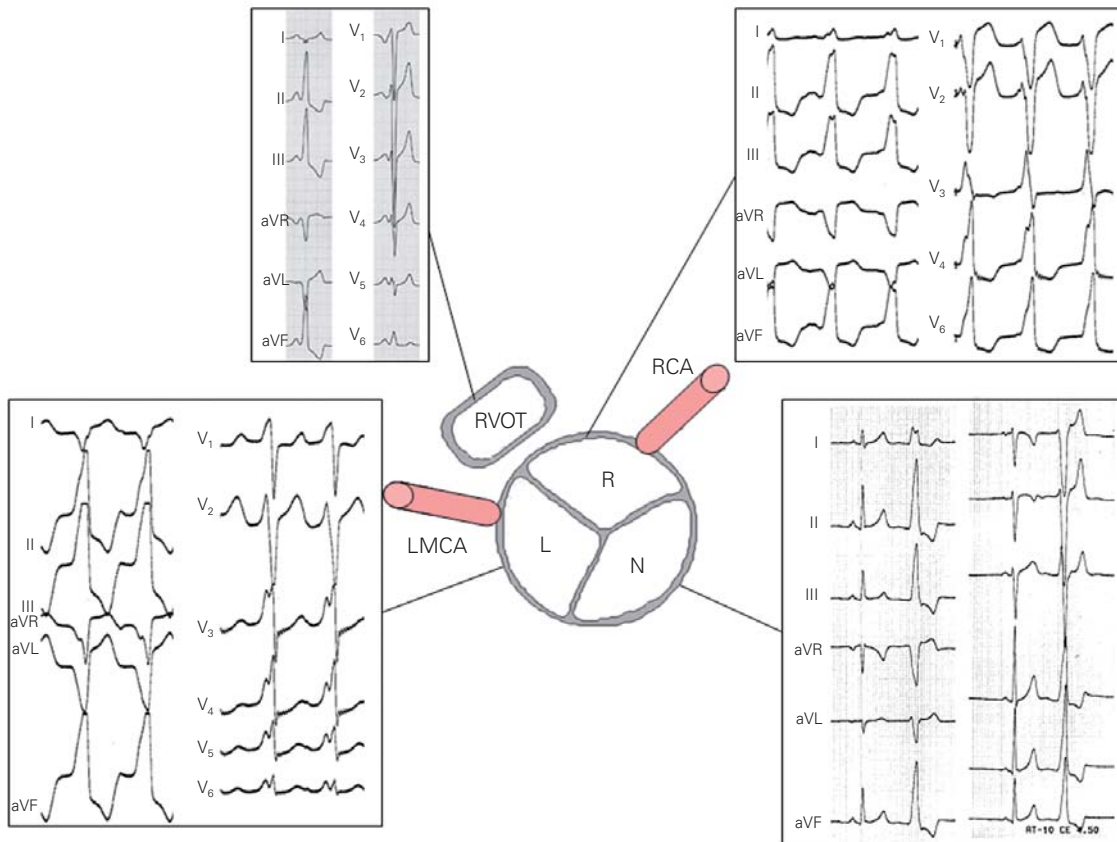
**Figure 1.** Repetitive monomorphic ventricular extrasystoles originating from the left aortic sinus cusp.

**Abbildung 1.** Repetitive monomorphe ventrikuläre Extrasystolen aus dem Bereich des linken Aortensinus.

**Ventricular Tachycardia Mechanism**

Outflow tract VT can be induced in the electrophysiological (EP) laboratory, although usually not with pro-

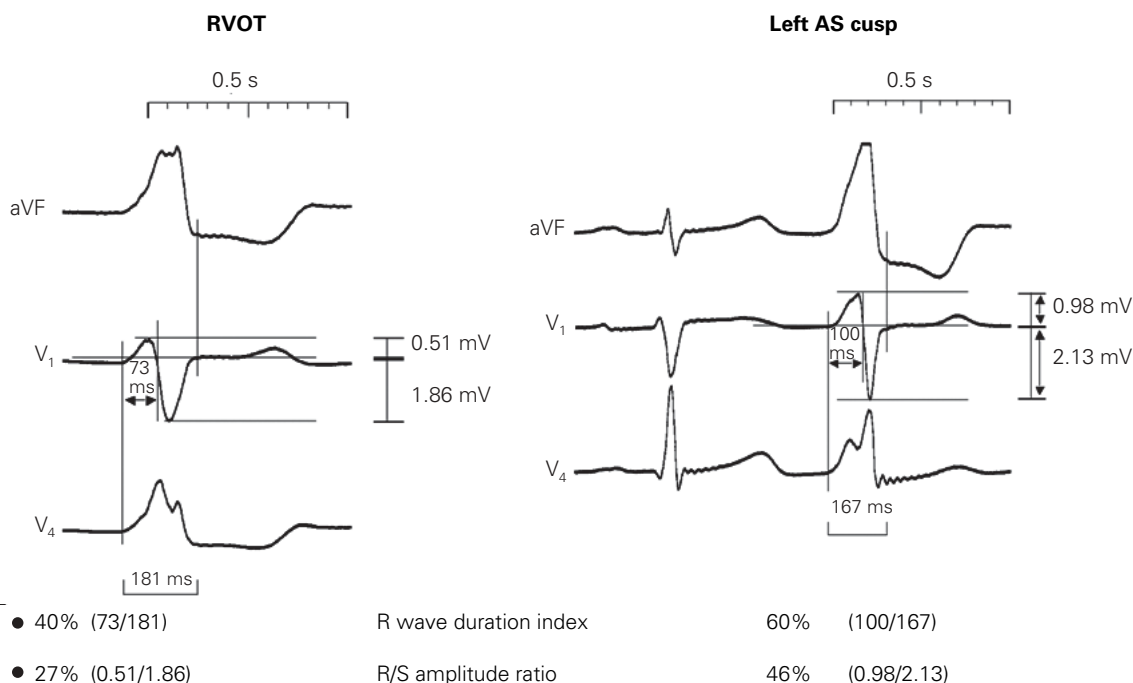
**Figure 2.** Schematic drawing of the aortic sinus cusp and the related anatomic structures with corresponding ECG morphology examples. L: left coronary cusp; LMCA: left main coronary artery; N: noncoronary cusp; R: right coronary cusp; RCA: right coronary artery; RVOT: right ventricular outflow tract.



**Abbildung 2.** Schematische Darstellung der Aortensinus mit den benachbarten anatomischen Strukturen und dazu korrespondierenden typischen EKG-Beispielen. L: linke Koronartasche; LMCA: linker Hauptstamm; N: nichtkoronare Tasche; R: rechte Koronartasche; RCA: rechte Koronararterie; RVOT: rechtsventrikulärer Ausflusstrakt.

**Figure 3.** ECG analysis and calculation examples for “R wave duration index” and “R/S amplitude ratio”. AS: aortic sinus; RVOT: right ventricular outflow tract.

**Abbildung 3.** EKG-Analyse mit Beispielen zur Berechnung des „R-Zacken-Dauer-Index“ und der „R/S-Amplituden-Ratio“. AS: Aortensinus; RVOT: rechtsventrikulärer Ausflusstrakt.



grammed stimulation [15]. In most patients, sustained or nonsustained episodes occur in response to burst pacing and are greatly facilitated by the infusion of isoproterenol, atropine or aminophylline [16]. These EP observations suggest that the triggered activity by delayed afterdepolarizations rather than reentry is the mechanism in RMVT. Outflow tract VT is typically provoked by physical or emotional stress [15] and terminates in response to verapamil, adenosine and enhanced vagal tone which all lead to decreased stimulated intracellular calcium levels [17, 18]. Interestingly, RVOT and LVOT appear to share the same arrhythmogenic mechanism [19] and termination of VT by adenosine is considered to be diagnostic of a cyclic adenosine monophosphate-(cAMP)-dependent mechanism mediated by triggered activity dependent on delayed afterdepolarizations [20]. Interestingly, an inhibitory G protein mutation causing inhibition of adenylyclase leading to increased intracellular cAMP has been identified as one VT mechanism [21]. However, the lack of specificity and the absence of a uniform response support the general consensus that the VT mechanism is incompletely characterized and probably varies among individual patients.

**Clinical Presentation**

In general, outflow tract VT occurs more frequently in women than men [22] and manifests at a relatively early age [23–26] with symptoms due to RMVT or VPCs such as palpitations, lightheadedness or pre-

syncope [15, 27, 28]. Most arrhythmias are usually nonsustained, but in some patients additional episodes of sustained VT [25] and in others only sustained VT have been observed [15, 29]. Fortunately, the prognosis is almost uniformly benign [15, 23, 24, 28, 30] suggesting a nonprogressive underlying pathophysiological process and that the tachycardia itself does not represent an early manifestation of an occult heart disease. More recently, however, a malignant variant of RMVT has been identified [31, 32]. In these patients, VPCs were closely coupled to prior beats [31] and it was speculated that relatively early triggered beats in the vulnerable period of the repolarization phase resulted in ventricular fibrillation. The prevalence of this malignant variant remains, however, unclear (see article by Tilz et al. in this issue).

**ECG Criteria: RVOT versus LVOT**

The twelve-lead ECG can help to differentiate between RVOT and LVOT aortic sinus cusp arrhythmia origin which both present with an inferior axis and an LBBB morphology. According to Ouyang et al. [3], ECG analysis should be performed with regard to the following criteria: (1) R wave duration in lead V<sub>1</sub> and V<sub>2</sub>, determined from the onset of the QRS to the transition point between the R wave and the isoelectric line, (2) R wave duration index, calculated as a percentage by dividing the QRS complex duration by the longer R wave duration in lead V<sub>1</sub> or V<sub>2</sub>, (3) R/S wave amplitude ratio, measured from the peak of R and na-

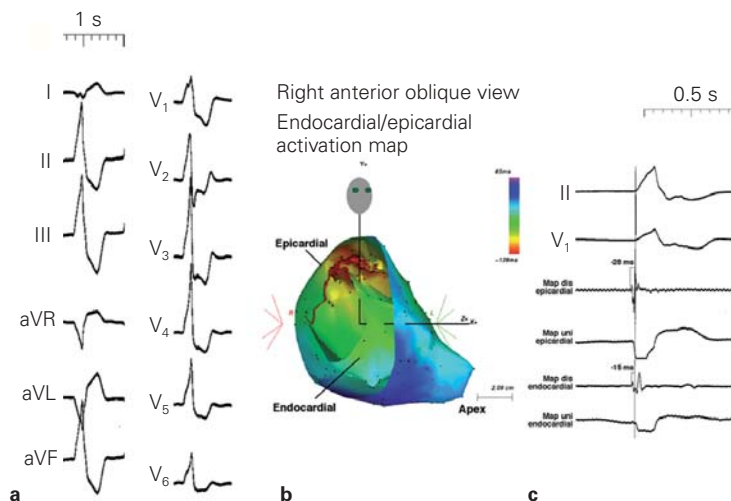
dir of S to the isoelectric line, expressed as a percentage (measured in leads  $V_1$  and  $V_2$ , and then the larger value is used; Figure 3), and (4) R/S wave transition. In LVOT aortic cusp VT origin, the R wave duration and R/S wave amplitude ratio (lead  $V_1$  or  $V_2$ ) were significantly increased compared to RVOT VT origin. Ouyang et al. suggested “cutoff” values (R wave duration index  $\geq 50\%$  and R/S wave amplitude ratio  $\geq 30\%$ ) (which allowed the identification of an LVOT aortic cusp origin in 6/7 cases (Table 1). In the one remaining patient (R wave duration index 46%, R/S wave amplitude ratio 27%), the VT did arise from the right aortic cusp which is closely related to the posteroseptal section of the RVOT (Figure 2), thus mimicking an RVOT origin. By contrast, in LVOT VT originating from below the aortic valve, a right bundle branch block (RBBB) morphology is observed. Rarely, LVOT VT can also originate from epicardial sites (Figure 4) which can be associated with a so-called pseudo-delta wave (widening of the initial QRS complex) in the twelve-lead ECG [33].

### Therapy

The decision to treat LVOT VT depends on the frequency and severity of symptoms. If clinical symptoms are absent, infrequent or mild, treatment is not mandatory. If, by contrast, symptoms are associated with presyncope, syncope or frequent debilitating extrasystoles, a more aggressive therapeutic strategy should be chosen. Therapeutic decisions should consider that many patients are young and otherwise healthy. As a result, catheter ablation may therefore be preferable to chronic administration of antiarrhythmic drugs. Moreover, the concept of a so-called tachycardia-induced cardiomyopathy has been established. Frequent or incessant tachycardias can lead to a progressive ventricular dysfunction which is reversible after rate control or successful catheter ablation [34]. Therefore, frequent or incessant outflow tract VTs with left ventricular dysfunction should be considered an indication for catheter ablation, if these patients do not exhibit any underlying causes of left ventricular dysfunction such as coronary heart disease [35].

### Medical Treatment

Medical treatment pursues two aims: (1) acute termination of the arrhythmia, and (2) prevention of recurrence. For termination, adenosine, verapamil or  $\beta$ -blockers can be successfully administered which all interfere with the cAMP-dependent intracellular calcium level [16, 17, 36–38]. For prevention of recurrence,  $\beta$ -blockers or verapamil are often used as first-line agents. Side effect profiles for both drugs are mild in comparison to specific antiarrhythmic



**Figures 4a to 4c.** Subepicardial left focal VT in a patient with repetitive VT. a) Twelve-lead ECG morphology with widening of the initial QRS complex (see text). b) Focal activation on the clipped endocardial and epicardial CARTO maps during tachycardia. The VT was only terminated by irrigated epicardial RF applications in the left ventricular outflow tract. The red dots indicate endocardial and epicardial ablation sites. c) Tracings are ECG leads II,  $V_1$ , and intracardiac electrograms recorded from the mapping catheter at the epicardial (Map dis epicardial) and endocardial (Map dis endocardial) sites with earliest activation during tachycardia. Note that the earliest activation recorded from the epicardial site preceded that from the endocardial site by 13 ms. A steep downslope in the unipolar electrogram (Map uni) was only present in the epicardial recording.

**Abbildungen 4a bis 4c.** Subepikardiale linksventrikuläre VT bei einem herzgesunden Patienten mit repetitiver VT. a) Zwölf-Kanal-EKG mit initialer Verbreiterung des QRS-Komplexes (s. Text). b) Fokale Aktivierung im endokardialen und epikardialen VT-Map. Die VT konnte nur durch gekühlte epikardiale RF-Applikationen im linksventrikulären Ausflusstrakt terminiert werden. Die roten Punkte markieren die endokardialen und epikardialen Ablationsorte. c) EKG-Ableitungen II und  $V_1$  sowie intrakardiale Elektrogramme vom Mapping-Katheter am Ort der frühesten Aktivierung während VT, und zwar von epikardial (Map dis epicardial) und endokardial (Map dis endocardial). Zu beachten ist, dass die früheste Aktivierung epikardial der frühesten Aktivierung endokardial um 13 ms vorausging. Ein steiler negativer Ausschlag im unipolaren Elektrogramm (Map uni) konnte nur von epikardial aufgezeichnet werden.

agents. However, success rates are low ranging from 25–50% which is also comparable for class Ic drugs such as flecainide [39].

### Catheter Ablation

Due to the limited efficacy and side effects of the chronic antiarrhythmic medical treatment, radiofrequency (RF) catheter ablation has been increasingly established in the therapy of outflow tract VT and can be associated with a high success and low complication rate [6, 10, 11, 36, 40, 41]. LVOT VT can originate from several sites including the superior basal region of the left interventricular septum, the aorto-mitral continuity, mitral annulus and, in particular, from the different aortic sinus cusps and epicardial

**Table 1.** Suggested ECG criteria to differentiate between right (RVOT) and left ventricular outflow tract (LVOT) aortic cusp arrhythmia origin according to Ouyang et al. [3]. More detailed description is given in the text.

**Table 1.** EKG-Kriterien zur Unterscheidung zwischen einem Ursprung der ventrikulären Tachykardie im rechtventrikulären Ausflusstrakt (RVOT), linksventrikulären Ausflusstrakt (LVOT) und Aortensinus nach Ouyang et al. [3]. Weitere Details sind im Text beschrieben.

| Formula for calculation                                      |                                      | RVOT                           | LVOT<br>Aortic cusp            |
|--|--------------------------------------|--------------------------------|--------------------------------|
| R wave duration index (V <sub>1</sub> and V <sub>2</sub> )   | R wave duration/QRS complex duration | < 50%                          | ≥ 50%                          |
| R/S wave amplitude ratio (V <sub>1</sub> or V <sub>2</sub> ) | Peak R wave/nadir S wave             | < 30%                          | ≥ 30%                          |
| R/S wave transition  |                                      | V <sub>3</sub> -V <sub>4</sub> | V <sub>2</sub> -V <sub>3</sub> |

sites. Therefore, approximation of the VT origin using twelve-lead ECG criteria is essential in the planning of the optimal ablation strategy [3] (Figure 2), in order to decide what mapping access is required (venous, arterial or even epicardial access). If the clinical VT does not occur spontaneously and is not inducible during baseline state, isoproterenol (i.v.) is administered to provoke the arrhythmia.

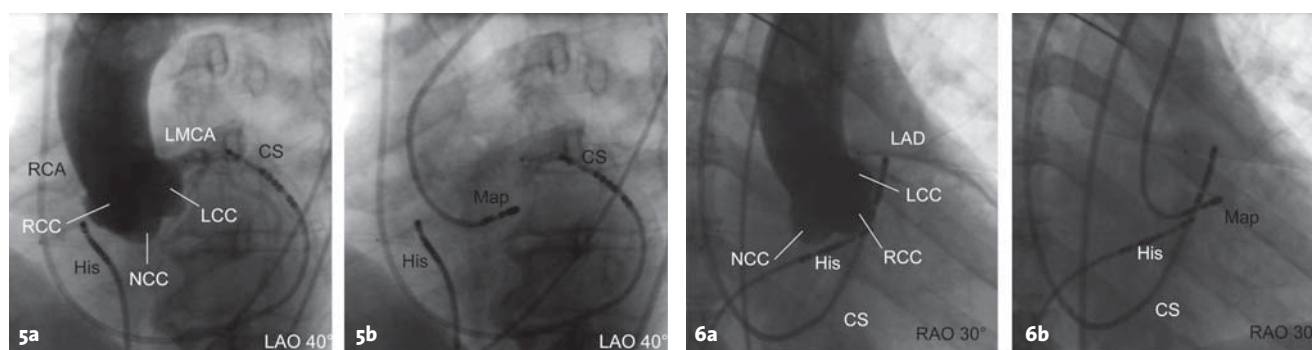
If the distal anterior coronary sinus recording shows very early local activation during VT/VPC, its origin from the left coronary cusp should be considered while early local activation during VT/VPC in the proximal coronary sinus or His bundle region suggests an either right coronary cusp or RVOT origin.

With frequent VT/VPC, activation mapping can be performed: activation mapping identifies the local

bipolar ventricular electrograms preceding the onset of the QRS complex. Interestingly, in RMVT from the left aortic sinus cusp two ventricular activation components have been observed which were lacking in RVOT VT [3]. The unipolar signal should exhibit a sharp QS morphology indicating the activation moving away from the electrode. The arrhythmia origin and ablation target are defined by the earliest activation site. If VT/VPC is infrequent, also pace mapping (comparing spontaneous vs. stimulated twelve-lead ECG QRS morphologies) can be helpful to identify the site for ablation, although this technique has limitations with regard to resolution. Three-dimensional electroanatomic reconstruction systems (CARTO, Biosense Webster, NAVX, St. Jude Medical) can contribute to facilitate catheter navigation and successful ablation (Figure 4).

For LVOT VT, RF energy should be delivered under continuous fluoroscopy to avoid coronary artery injury (aortic cusp: target temperature: 55 °C, 15–30 W, 120 s; LVOT below the aortic valve: target temperature: 55 °C, 30–40 W, 120 s). Before application of RF energy at epicardial sites (irrigated tip flow rate 17 ml/min, target temperature: 43 °C, 20–30 W, 110 s), a coronary angiogram should be performed to localize coronary arteries.

A possible explanation for a previous failed LVOT VT RF ablation is that the arrhythmia arises from an epicardial (Figure 4) rather than an endocardial focus (see ECG criteria above). In one report of previously failed ablation, subxiphoid instrumentation for an epicardial access resulted in successful ablation in 17/24 patients, while six of the remaining seven patients were ablated from the left aortic cusp



**Figures 5a and 5b and 6a and 6b.** Aortography reveals major anatomic landmarks in the aortic root in LAO (5a and 5b) and RAO projections (6a and 6b). The mapping catheter is located directly underneath the left coronary cusp. CS: coronary sinus; His: His catheter; LAD: left anterior descending; LAO: left anterior oblique; LCC: left coronary cusp; LMCA: left main coronary artery; Map: mapping catheter; NCC: noncoronary cusp; RAO: right anterior oblique; RCA: right coronary artery; RCC: right coronary cusp.

**Abbildungen 5a und 5b und 6a und 6b.** Die Kontrastmitteldarstellungen in der Aortenwurzel zeigen die verschiedenen Aortensinus in LAO- (5a und 5b) und RAO-Projektionen (6a und 6b) sowie die Abgänge der Kranzarterien. Der Mapping-Katheter liegt direkt unterhalb des linken Aortensinus. CS: Koronarvenensinus; His: His-Katheter; LAD: Ramus interventricularis anterior; LAO: linksanteriore Schrägprojektion; LCC: linke Koronartasche; LMCA: linker Hauptstamm; Map: Mapping-Katheter; NCC: nichtkoronare Tasche; RAO: rechtsanteriore Schrägprojektion; RCA: rechte Koronararterie; RCC: rechte Koronartasche.

[14]. Applying these techniques in conjunction with the twelve-lead ECG criteria to approximate the arrhythmia origin, LVOT VT can be safely and successfully ablated in many patients.

### Complications

RF catheter ablation is generally associated with a low complication rate. However, especially when ablating in the region of the aortic cusps some special care should be taken with regard to the coronary arteries. The aortic root, the ostia of the right coronary artery (RCA) and left main coronary artery (LMCA) can be visualized by angiography allowing further anatomic classification of the aortic cusp sinuses (Figures 5 and 6). The LMCA can be selectively cannulated as a marker and used as a protection in the case of catheter dislodgment during RF application. In a small series, the mean distance between the LMCA and the ablation site in the left aortic cusp was 12.2 mm, and the mean distance between the RCA and the ablation site in the right aortic cusp was 9.2 mm. In all cases, RF ablation was performed without complications [3]. Coronary angiography can be performed immediately after the ablation procedure to rule out spasm, dissection or thrombus. Postablation care should also include transthoracic echocardiography to rule out aortic valve damage.

### Conclusion

LVOT arrhythmias represent an increasingly recognized VT entity which can be safely and successfully treated by catheter ablation. Identification of the VT origin using ECG criteria and differentiation versus RVOT is essential in the careful planning of the ablation strategy.

**Conflict of interest:** None. The authors declare that they had no financial or personal relations to other parties whose interests could have affected the content of this article in any way, either positively or negatively.

### References

- Brooks R, Burgess JH. Idiopathic ventricular tachycardia. A review. *Medicine (Baltimore)* 1988;67:271–94.
- Gallaverdin L. Extrasystolie ventriculaire à paroxysmes tachycardiques prolongés. *Arch Mal Coeur* 1922;15:298–306.
- Ouyang F, Fotuhi P, Ho SY, et al. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: electrocardiographic characterization for guiding catheter ablation. *J Am Coll Cardiol* 2002;39:500–8.
- Kanagaratnam L, Tomassoni G, Schweikert R, et al. Ventricular tachycardias arising from the aortic sinus of valsalva: an under-recognized variant of left outflow tract ventricular tachycardia. *J Am Coll Cardiol* 2001;37:1408–14.
- Kamakura S, Shimizu W, Matsuo K, et al. Localization of optimal ablation site of idiopathic ventricular tachycardia from right and left ventricular outflow tract by body surface ECG. *Circulation* 1998;98:1525–33.
- Coggins DL, Lee RJ, Sweeney J, et al. Radiofrequency catheter ablation as a cure for idiopathic tachycardia of both left and right ventricular origin. *J Am Coll Cardiol* 1994;23:1333–41.
- Lerman BB, Stein KM, Markowitz SM. Mechanisms of idiopathic left ventricular tachycardia. *J Cardiovasc Electrophysiol* 1997;8:571–83.
- Callans DJ, Menz V, Schwartzman D, et al. Repetitive monomorphic tachycardia from the left ventricular outflow tract: electrocardiographic patterns consistent with a left ventricular site of origin. *J Am Coll Cardiol* 1997;29:1023–7.
- Morady F, Kadish AH, DiCarlo L, et al. Long-term results of catheter ablation of idiopathic right ventricular tachycardia. *Circulation* 1990;82:2093–9.
- Calkins H, Kalbfleisch SJ, El-Atassi R, et al. Relation between efficacy of radiofrequency catheter ablation and site of origin of idiopathic ventricular tachycardia. *Am J Cardiol* 1993;71:827–33.
- Klein LS, Shih HT, Hacken FK, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. *Circulation* 1992;85:1666–74.
- Anderson RH. Clinical anatomy of the aortic root. *Heart* 2000;84:670–3.
- Sutton JP, Ho SY, Anderson RH. The forgotten interleaflet triangles: a review of the surgical anatomy of the aortic valve. *Ann Thorac Surg* 1995;59:419–27.
- Schweikert RA, Saliba WJ, Tomassoni G, et al. Percutaneous pericardial instrumentation for endo-epicardial mapping of previously failed ablations. *Circulation* 2003;108:1329–35.
- Buxton AE, Waxman HL, Marchlinski FE, et al. Right ventricular tachycardia: clinical and electrophysiologic characteristics. *Circulation* 1983;68:917–27.
- Lerman BB, Stein K, Engelstein ED, et al. Mechanism of repetitive monomorphic ventricular tachycardia. *Circulation* 1995;92:421–9.
- Lerman BB, Belardinelli L, West GA, et al. Adenosine-sensitive ventricular tachycardia: evidence suggesting cyclic AMP-mediated triggered activity. *Circulation* 1986;74:270–80.
- Lerman BB. Response of nonreentrant catecholamine-mediated ventricular tachycardia to endogenous adenosine and acetylcholine. Evidence for myocardial receptor-mediated effects. *Circulation* 1993;87:382–90.
- Iwai S, Cantillon DJ, Kim RJ, et al. Right and left ventricular outflow tract tachycardias: evidence for a common electrophysiologic mechanism. *J Cardiovasc Electrophysiol* 2006;17:1052–8.
- Song Y, Thedford S, Lerman BB, et al. Adenosine-sensitive afterdepolarizations and triggered activity in guinea pig ventricular myocytes. *Circ Res* 1992;70:743–53.
- Lerman BB, Dong B, Stein KM, et al. Right ventricular outflow tract tachycardia due to a somatic cell mutation in G protein subunit  $\alpha_{1z}$ . *J Clin Invest* 1998;101:2862–8.
- Nakagawa M, Takahashi N, Nobe S, et al. Gender differences in various types of idiopathic ventricular tachycardia. *J Cardiovasc Electrophysiol* 2002;13:633–8.
- Deal BJ, Miller SM, Scagliotti D, et al. Ventricular tachycardia in a young population without overt heart disease. *Circulation* 1986;73:1111–8.
- Lemery R, Brugada P, Della Bella P, et al. Nonischemic ventricular tachycardia: clinical course and long-term follow-up in patients without clinically overt heart disease. *Circulation* 1989;79:990–9.
- Mont L, Seixas T, Brugada P, et al. The electrocardiographic,

**Address for Correspondence**

Matthias Antz, MD  
 Department of  
 Cardiology  
 Asklepios Klinik  
 St. Georg  
 Lohmühlenstraße 5  
 22099 Hamburg  
 Germany  
 Phone (+49/40)  
 181885-3616, Fax -4435  
 e-mail: jongichun@  
 t-online.de

- clinical and electrophysiologic spectrum of idiopathic monomorphic ventricular tachycardia. *Am Heart J* 1992; 124:746–53.
26. Ohe T, Shimomura K, Aihara N, et al. Idiopathic sustained left ventricular tachycardia: clinical and electrophysiologic characteristics. *Circulation* 1988;77:560–8.
  27. Proclemer A, Ciani R, Feruglio GA. Right ventricular tachycardia with left bundle branch block and inferior axis morphology: clinical and arrhythmological characteristics in 15 patients. *Pacing Clin Electrophysiol* 1989;12:977–89.
  28. Rahilly GT, Prystowsky EN, Zipes DP, et al. Clinical and electrophysiologic findings in patients with repetitive monomorphic ventricular tachycardia and an otherwise normal electrocardiogram. *Am J Cardiol* 1982;50:459–68.
  29. Markowitz SM, Litvak BL, Ramirez de Arellano EA, et al. Adenosine-sensitive ventricular tachycardia. Right ventricular abnormalities delineated by magnetic resonance imaging. *Circulation* 1997;96:1192–200.
  30. Froment R, Gallavardin L, Cahen P. Paroxysmal ventricular tachycardia; a clinical classification. *Br Heart J* 1953;15:172–8.
  31. Viskin S, Rosso R, Rogowski O, et al. The “short-coupled” variant of right ventricular outflow ventricular tachycardia: a not-so-benign form of benign ventricular tachycardia? *J Cardiovasc Electrophysiol* 2005;16:912–6.
  32. Noda T, Shimizu W, Taguchi A, et al. Malignant entity of idiopathic ventricular fibrillation and polymorphic ventricular tachycardia initiated by premature extrasystoles originating from the right ventricular outflow tract. *J Am Coll Cardiol* 2005;46:1288–94.
  33. Berruezo A, Mont L, Nava S, et al. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation* 2004;109:1842–7.
  34. Grimm W, Menz V, Hoffmann J, et al. Reversal of tachycardia induced cardiomyopathy following ablation of repetitive monomorphic right ventricular outflow tract tachycardia. *Pacing Clin Electrophysiol* 2001;24:166–71.
  35. Nerheim P, Birger-Botkin S, Piracha L, et al. Heart failure and sudden death in patients with tachycardia-induced cardiomyopathy and recurrent tachycardia. *Circulation* 2004;110:247–52.
  36. Wilber DJ, Baerman J, Olshansky B, et al. Adenosine-sensitive ventricular tachycardia: clinical characteristics and response to catheter ablation. *Circulation* 1993; 87:126–34.
  37. Sung RJ, Shapiro WA, Shen EN, et al. Effects of verapamil on ventricular tachycardias possibly caused by reentry, automaticity, and triggered activity. *J Clin Invest* 1983;72:350–60.
  38. Sung RJ, Keung EC, Nguyen NX. Effects of beta-adrenergic blockade on verapamil-responsive and verapamil-irresponsive sustained ventricular tachycardias. *J Clin Invest* 1988; 81:688–99.
  39. Lerman BL, Stein K, Markowitz SM et al. Ventricular tachycardia in patients with structurally normal hearts. In: Zipes D, Jalife J, eds. *Cardiac electrophysiology: from cell to bedside*, 4th edn. Philadelphia: Saunders, 2004:668–82.
  40. Timmermans C, Rodriguez LM, Crijns HJ, et al. Idiopathic left bundle-branch block-shaped ventricular tachycardia may originate above the pulmonary valve. *Circulation* 2003;108:1960–7.
  41. Rodriguez LM, Smeets JL, Timmermans C, et al. Predictors for successful ablation of right- and left-sided idiopathic ventricular tachycardia. *Am J Cardiol* 1997;79:309–14.