

Catheter Ablation of Idiopathic Ventricular Arrhythmias



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Ventricular arrhythmias (VA) are observed in the setting of structural heart disease. However, in a proportion of patients presenting with VT, the routine diagnostic modalities fail to demonstrate overt myocardial abnormality. These arrhythmias have been called idiopathic VAs. They consist of various subtypes that have been defined by their anatomic location of origin within the heart and/or their underlying mechanism. While the majority of patients are asymptomatic, some experience debilitating symptoms and may develop reversible ventricular dysfunction. Catheter ablation has been traditionally reserved for patients with incapacitating symptoms or progressive ventricular dysfunction. However, as many patients are young, and catheter ablation can be curative in >90% of cases with a low risk (<1%) of serious complications, it is increasingly being offered as a first-line treatment in symptomatic patients. The approach to arrhythmia mapping is guided by the 12-lead electrocardiograph (ECG) morphology of the ventricular tachycardia (VT). Use of three dimensional (3D) electroanatomic mapping systems and intra-cardiac echocardiography are helpful in localising sites for successful ablation.

Keywords

Ventricular tachycardia • Catheter ablation • Idiopathic • Outflow tract

Introduction

Ventricular arrhythmias (VAs) most often occur in the setting of structural heart disease, however, in 10% of patients, they can occur in the structurally normal heart, termed idiopathic VA. Often manifesting in younger patients, idiopathic VAs originate from specific anatomical structures and are caused most frequently by a focal mechanism. The diagnosis is based upon the presence of VA in the absence of myocardial abnormalities on various imaging modalities, including echocardiography, angiography or cardiac magnetic resonance imaging (MRI). Idiopathic VAs can cause debilitating symptoms, as well as leading to left ventricular (LV) systolic dysfunction. Multiple studies have shown that catheter ablation is effective in eliminating idiopathic VA, however, not all VAs are similar and outcomes vary based upon the location

and underlying pathology. Various electrocardiographic and electrophysiologic parameters have been found to be predictive of catheter ablation outcomes.

Sites of Origin of VA

Idiopathic VAs can be categorised by their anatomic location of origin within the heart (Table 1). The anatomic relationship of cardiac structures is complex and a detailed understanding of the anatomy around the site of origin (SOO) of the VA is crucial for planning catheter ablation, and in understanding the potential risks and expected outcomes of catheter ablation. The SOO of idiopathic VAs, and therefore the area of ablation, is suggested by the 12-lead electrocardiograph (ECG).

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Table 1 Idiopathic ventricular arrhythmia origins.

	RV (Right ventricle)	LV (Left ventricle)
Outflow tract region	Pulmonary artery RVOT	Aortic cusp LVOT
– Supra-valvular		LV summit
– Endocardial		
– Epicardial		
Annulus	Tricuspid annulus	Mitral annulus
Fascicles		Left posterior fascicle Left anterior fascicle
Intracavitary	Papillary muscles Moderator band	Papillary muscles
Epicardium	Crux	

Abbreviations: RVOT, right ventricular outflow tract; LVOT, left ventricular outflow tract; LV, left ventricle; RV, right ventricle.

Clinical Presentation

Most idiopathic VAs originate from the outflow tract region, predominantly (80%) originating from right ventricular (RV) outflow tract [1]. Outflow tract VAs typically present in young people, usually between the second to fourth decade of life [2]. Patients can be asymptomatic, and the diagnosis made incidentally on a routine ECG. However, most patients have frequent premature ventricular contractions (PVCs) or non-sustained ventricular tachycardia (VT) that present as palpitations (>50%), chest pain, dyspnoea or presyncope. Syncope is uncommon (<5%). In general, episodes are frequently associated with exertion or emotional stress due to the effect of endogenous catecholamines. The prognosis is generally excellent and the risk of sudden cardiac death (SCD) is very low [3]. However, in a small proportion of patients, significant arrhythmia burden may lead to a reversible form of LV systolic dysfunction [4–6]. Additionally, a malignant variant of “short-coupled” PVCs triggering polymorphic VT has been described [7,8].

RV Outflow Tract VA

Anatomic Relationship and ECG Characteristics

The right ventricular outflow tract (RVOT) lies anterior to the left ventricular outflow tract (LVOT) as it passes superiorly and leftward as the pulmonary artery. The LVOT is located posteriorly and courses rightward to connect to the aortic root. The pulmonic valve is superior, anterior and leftward of the aortic valve [9]. The aortic root is centrally located and in direct contact with the anterior mitral leaflet and the posterior RVOT. Myocardial sleeves can extend above the pulmonic and aortic valves into the great vessels and may be the source of arrhythmias [10,11]. A VT originating from the RVOT has a left bundle branch block (LBBB) pattern with a late transition that begins no earlier than the precordial lead

V₃ [12]. It also displays an inferior axis (i.e. a dominant R wave in inferior leads) (Figure 1) [13,14]. However, because the septal aspect of the RVOT is in close anatomical proximity to the LVOT, the QRS morphology cannot always be solely relied upon to localise the origin of VA (Figure 1). The precordial transition is important in differentiating the two sites of origin [15,16]. If the precordial transition is earlier than V₂, with prominent R waves in V₁ and V₂, the site of origin is more likely to be LVOT and if the precordial transition is V₄ or later, the trigger site is more likely located in the RVOT [13,17]. The QRS duration of VA from the RVOT free wall is wider and shorter with a characteristic ‘notched’ pattern in the inferior leads and a later precordial transition (lead V₄ or later) compared with VA originating from the septum [18]. Characteristic ECG changes could also indicate SOO in LVOT [19,20].

Mapping and Ablation

Catheter ablation is an effective and safe method to permanently cure outflow tract VAs and should be considered as first-line treatment. In experienced centres, the success rate is greater than 90%, with the complication rate less than 1% [21]. Activation mapping is the best method to identify the SOO of VAs (Figure 1). To maximise PVC frequency during the ablation, we discontinue beta blockers, calcium channel blockers, and other anti-arrhythmic medications at least 5 days before ablation. We avoid deep sedation and limit the use of propofol and benzodiazepines and favour remifentanyl, which is rapidly eliminated following discontinuation and has fewer anti-arrhythmic properties [22]. Isoproterenol infusion and burst pacing are used when VA remains infrequent. Using intracardiac echocardiogram (ICE), a three-dimensional geometry of the ventricle is created, and activation points are plotted on this ICE-integrated electroanatomic map. We find ICE to be extremely valuable for understanding anatomic relationships, guiding catheter position and tissue contact, monitoring lesion formation and avoiding/detecting complications (Figure 1). We also create an electroanatomic substrate map at the same time which helps us to identify myocardial scarring [23].

Activation mapping is performed to identify the site of earliest activation breakout, which should be well before the onset of the QRS complex. The unipolar electrogram should have a QS morphology with a steep slew. During mapping, if a wide area has similar activation timing, it either suggests a deep intramural or epicardial origin of VA or breakthrough from an adjacent chamber. Therefore, in this situation, activation mapping of the adjacent structures should be performed. Although a less accurate mapping technique, when the VAs are not frequent enough, we do perform pace mapping at or near the capture threshold with the same coupling interval as the spontaneous VAs. A perfect pace-map match in all 12 surface ECG leads should be sought [24]. We find pace-mapping to be more accurate within the RVOT and less so within the aortic root, particularly in view of the high pacing outputs required here [25]. Irrigated or non-irrigated ablation catheters can be used within the RVOT although sufficient power may be difficult to achieve with solid tip non-irrigate catheters in some

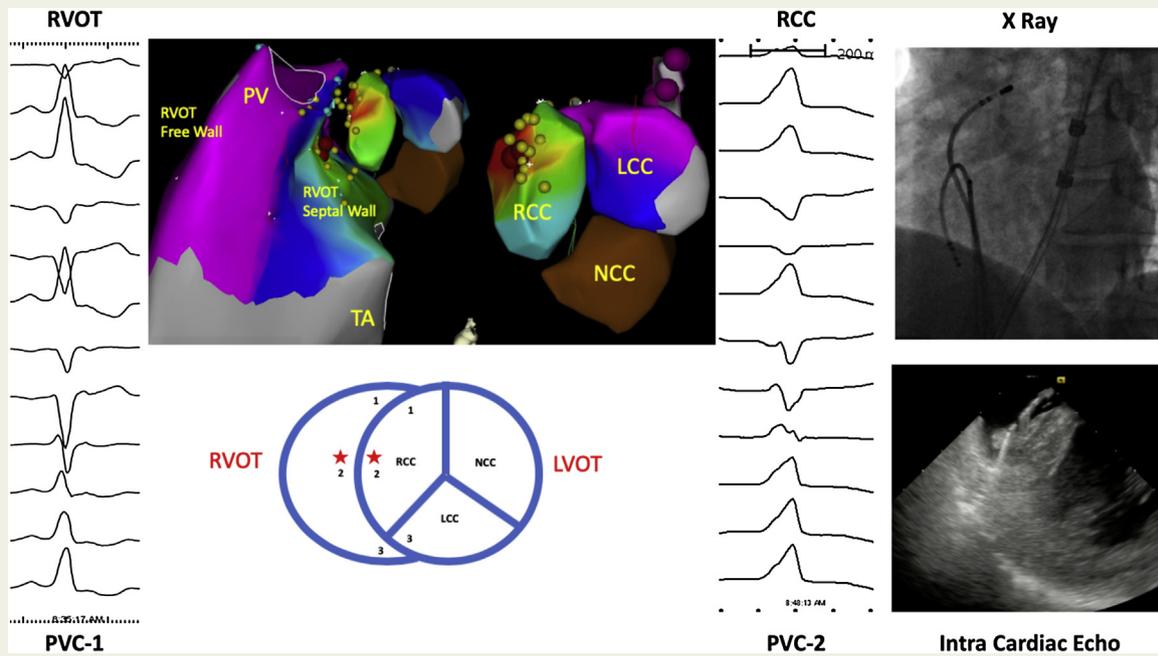


Figure 1 RVOT and LVOT PVC.

PVC-1 with LBBB Inferior Axis V4 transition in precordial leads and rS wave in Lead 1. This PVC was mapped to be earliest at Site 2 of RVOT. Ablation was performed with suppression of PVC-1. However, the PVC returned after 20 min with different morphology (PVC-2). PVC-2 with LBBB, V3 transition in precordial leads, Inferior Axis, Rs in Lead 1 was mapped earliest in RCC. RCC is anatomically very close to Site 2 RVOT. The CARTO 3D merge map of RVOT and RCC shown with earliest activation in respective location for PVC-1 and PVC-2. Also, intracardiac echocardiography utilised for 3D geometry reconstruct and catheter visualisation shows catheter in RVOT Site-2.

Abbreviations: PVC, premature ventricular contractions; LBBB, left bundle branch block; RVOT, right ventricular outflow tract; RCC, right coronary cusp; 3D, three dimensional.

of the deep recesses of this region. However, in the LVOT and coronary venous system, irrigated ablation is required to allow for sufficient radiofrequency energy delivery and to prevent coagulum formation or char.

Because of its anatomical relations, ablation within the LVOT can risk injury to the coronary arteries. When ablating within the right or left aortic sinuses of Valsalva (ASOV), care must be taken to define the location of the coronary ostia, either by ICE or angiography. Coronary injury is uncommon while ablating at the bottom of the ASOV [26]. Additionally, ECG morphology should be carefully examined after lesion delivery to ensure the morphology has not changed as the VA exit may shift. If, indeed, the ECG morphology has changed, mapping should be repeated, including in the adjacent structures (Figure 1). We consider a procedure successful when no further spontaneous VAs are seen, and when VAs cannot be induced with isoproterenol infusion and burst pacing at least one hour following the successful ablation lesion.

Non-Outflow Tract VAs (Mitral and Tricuspid Annular VA)

Ventricular arrhythmias from the mitral annulus (MA) and tricuspid annulus (TA) are rare. If VAs are localised to MA

area, then anterior, anterolateral and posterolateral aspects of the MA are the usual site of origin [27,28]. An origin at the TA is seen in around 8% of patients with idiopathic VTs; 74% of these arise from the septal portion (mostly from the His Bundle region at the superior TA) and 26% from the free wall region [29].

Anatomic Relationship and ECG Characteristics

Mitral annular VAs are characterised by a wide QRS complex and, in some cases, a delta wave-like morphology in the 12-lead ECG. They arise within the posterior aspect of the left ventricle and exhibit a RBBB pattern with monophasic R waves in V₂-V₆ [30]. The success rate of mitral annular VT ablation is comparable to that of outflow tract ablation [31]. Tricuspid annular VAs are characterised by a LBBB morphology and a positive QRS in leads I, V₅ and V₆ [29]. An early precordial R-wave transition by lead V₃ and a QS pattern in lead V₁ are useful indicators for differentiating the origin of VAs from the free wall portion and the septal region of the TA. The QRS duration, Q-wave amplitude in lead V₁-V₃ and notching of the QRS is greater in VT originating from the tricuspid annulus free wall as compared to the septum [29].

Mapping and Ablation

Ventricular arrhythmias originating from the endocardial and epicardial MA can be successfully ablated [27,28]. When the MA VAs cannot be abolished by radiofrequency (RF) application from the endocardium, access via the coronary sinus (CS) for mapping and ablation may be necessary [32,33]. If an epicardial site of origin is not accessible via the CS due to anatomical limitations, an alternative approach is via the pericardial space. Coronary angiography is required to assess the proximity of the coronary arteries to the proposed ablation site whenever transvenous or epicardial ablation is performed.

Mapping VAs originating from TA region is challenging due to poor catheter stability, and we routinely use a long deflectable guiding sheath to achieve adequate contact and stability of the mapping catheter. Occasionally, the VA origin is located close to the AV conduction system. In such cases, mapping in the aortic root at right coronary cusp and non-coronary cusp region should be performed to identify the earliest site and reduce the risk of damage to the AV conduction system. If ablation is performed close to the conduction system, cryo-ablation should be considered. Catheter ablation is more effective for VAs arising from the free wall portion of TA than that from arising the septal portion of the TA [29].

If VAs are frequent, then activation mapping is the best strategy to locate SOO. Pace mapping should be utilised when VAs are infrequent. An atrial electrogram is usually recorded at the successful ablation site of mitral annular and tricuspid annular VAs [27,29]. Depending upon the SOO of MA VAs, either a retrograde or transeptal approach may be required for better mapping and ablation. In either case, ablation should be performed with the ablation electrode in direct contact with the endocardium rather than through the mitral valve itself. There is a potential risk of mitral and tricuspid insufficiency in the catheter ablation of mitral annular and tricuspid annular VAs respectively.

Conduction System and Papillary Muscle VAs

Ventricular arrhythmias originating from the conduction system or involving the specialised conduction system have been described [34,35]. Fascicular VT is the second most common form of idiopathic VT, and accounts for 10–15% of all idiopathic VTs. This VT is seen in the second to fourth decade of life and occurs more often in men [36]. VAs can also originate from intracavitary structures such as papillary muscles (PM) of the RV and LV and the moderator band [37–39].

Anatomic Relationship and ECG Characteristics

Fascicular VT occurs due to a re-entrant mechanism involving the left anterior or posterior fascicle. Left posterior fascicular VT is more common than the anterior and septal fascicular VT. The antegrade limb of these VAs consists of

a zone of slow conduction in the intraventricular left septum from the base to near the apex, with the Purkinje tissue of the fascicles comprising the retrograde limb. The underlying substrate for the region of slow conduction is not clearly defined. False tendons connected to the interventricular septum have been suggested as the anatomical basis for the macro-reentry circuit [40].

Ventricular arrhythmias can also originate from anterolateral [37], posteromedial PM [38], and moderator band of the RV [39]. Papillary muscle VAs are more commonly seen originating from the posteromedial PM than the anterolateral PMs. They often involve the distal Purkinje system, compared to fascicular arrhythmias that involve the proximal Purkinje system. There are distinct ECG characteristics which can help to differentiate these VAs. These VAs are characterised by RBBB and superior or inferior-axis QRS morphology, however, PM VAs lack a discrete Q wave in the lateral or inferior leads, their QRS complex is broader and there is absence of an rsR' pattern in lead V₁ that is characteristic for fascicular tachycardia [41].

Mapping and Ablation

Success rates of ablation of fascicular VT are comparable to outflow tract VT [42]. Ablation is directed at the antegrade late diastolic Purkinje potential [22]. Mapping is performed around the anatomic location of the involved fascicle, seeking a discrete Purkinje potential that precedes the QRS complex during VT [23]. To avoid damage to the proximal conduction system, mapping and ablation should start from an apical site and shift toward a basal site until successful ablation is achieved. If the VA is not inducible at the time of ablation either due to catheter trauma or spontaneously, then a more anatomically based ablation may be required. A linear ablation strategy with lesions set placed perpendicular to the long axis of the LV, approximately midway from the base to the apex in the region of the mid to mid-inferior septum can be performed [24]. However, this approach may be complicated with left posterior fascicular block.

Catheter ablation of papillary muscle VAs is challenging compared to ablation of other VAs. The PMs of the heart are complex intra-cavitary structures and their anatomic and electrophysiological characteristics may determine the outcome of ablation procedures. Additional difficulties arise from the deep location of their sites of origin, multiple VA morphologies due to multiple preferential exits before and during ablation, and poor catheter stability and low achievable contact force. (Figure 2) [26,43]. Guidance using real-time imaging with ICE and integrated electroanatomic mapping is necessary for effective and safe mapping and ablation of PM VAs (Figure 2) [44]. Activation mapping is most reliable in detecting the site of origin. A retrograde transaortic approach or transeptal approach may be utilised for LV access. Pace mapping can provide helpful indications of the breakout points of PM VAs with deep intramural origin. Of note, ablation at the best pace mapped site may not eliminate the VAs as the SOO may be located away from the breakout site. Ablation at the best pace mapped site may change the QRS morphology,

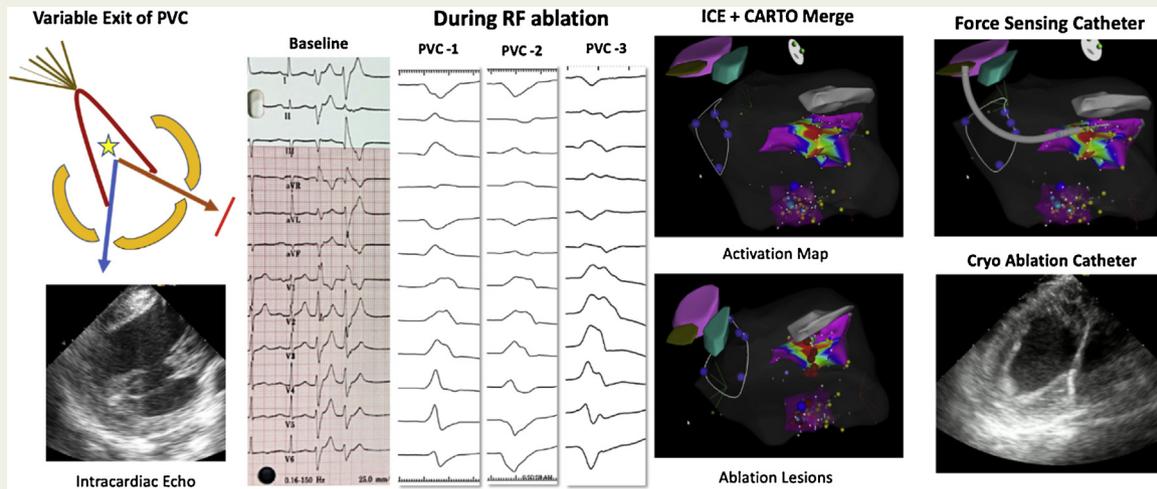


Figure 2 Papillary Muscle PVC.

Baseline ECG shows couplet PVC with PVC-1 RBBB superior axis V3 transition rS in lead 1 (posteromedial papillary muscle) and PVC-2 RBBB inferior axis V5 transition rS in lead 1 (antero-lateral papillary muscle). Intracardiac echocardiography utilised for 3D geometry reconstruct shows papillary muscle and ablation catheter on papillary muscle. The CARTO 3D merge map of papillary muscle shown with earliest activation on antero lateral papillary muscle.

Abbreviations: PVC, premature ventricular contractions; RBBB, right bundle branch block; D, three dimensional; ECG, electrocardiograph.

suggesting a change in the exit site. During pace mapping, the stimulus-to-QRS interval can be utilised to identify preferential exit sites and the SOO of VAs [45]. A low-amplitude ventricular pre-potential or Purkinje potential may be recorded at the successful ablation site [41]. Deeper lesions may be necessary considering intramural origin of many of these VAs. Therefore, we routinely use force sensing irrigated tip ablation catheters to deliver 30–40 W power for 1–2 minutes. Additionally, ECG morphology should be carefully examined after every few lesions to ensure the morphology has not changed as the VA exit may shift. If indeed the ECG morphology has changed, mapping should be repeated. Not infrequently, ablation lesions on both sides of the papillary muscles are often required to eliminate all variations in the QRS morphology. Ablation at the body of PM can be challenging due to poor catheter contact, and at the base due to the risk of steam pop. Use of ICE is critical to ascertain good catheter contact and effective lesion delivery (Figure 2). In cases where catheter stability remains an issue, cryoablation can be utilised to improve catheter stability [46].

Epicardial (Crux and LV Summit) VA

Idiopathic VAs rarely arise from the epicardium of the heart. These epicardial origins are usually seen in the crux of the heart or the LV summit [33].

Anatomic Relationship and ECG Characteristics

The crux is at the epicardial surface of the heart, bordered by the AV groove and posterior interventricular groove,

and is located at the junction of the middle cardiac vein and coronary sinus. Doppalapudi *et al.* have described epicardial idiopathic VT arising from the crux of the heart adjacent to the posterior descending artery [47]. The QRS morphology of these VAs show early precordial transition at or before V_2 , in addition to a left superior axis and a maximum deflection index (MDI) >0.55 . Leads II, III, aVF exhibit deep negative QS complexes with slurred downstroke deflections.

The LV summit is the area of the epicardium that encompasses the most superior aspect of the LVOT and is bound by the left anterior descending coronary artery anteriorly, the left circumflex coronary artery posteriorly and the anterior intraventricular vein (AIV) [25]. Ventricular arrhythmias arising from this area exhibit atypical LBBB morphology with inferior axis (large R waves in leads II, III and aVF) and an early transition in the precordial leads, often with broad R waves in V_1 [48].

Mapping and Ablation

Epicardial VAs can be mapped and ablated via the transvenous or transpericardial route. Detailed understanding of the anatomy of the CS and the adjacent anatomic structures is critical for mapping and ablation of these VAs. Delineation of the CS anatomy by coronary venography allows clear visualisation of the architecture and helps to plan mapping and ablation [49]. The Thebesian valve at CS ostium and the valve of Vieussens that is located between the coronary sinus and great coronary vein may be an obstruction to mapping and ablation [50].

Similar to all other VAs, activation mapping is most reliable in detecting the site of origin of these focal arrhythmias. When an early ventricular activation is recorded within the

middle cardiac vein (MCV) or proximal CS, catheter ablation may be attempted if safe [47]. Ablation in the coronary sinus can be challenging due to proximity to the coronary arteries and inability to provide effective lesions due to high impedance and limited electrode cooling, leading to a rapid rise in electrode temperature. Coronary angiography is recommended to define the location of the coronary arteries and identify a safer area for ablation. Use of an irrigated ablation catheter is recommended with continuous flow at 30 mL/min. If ablation from the CS or MCV is unsuccessful, epicardial mapping via a subxiphoid pericardial approach should be performed. However, several potential complications including pericardial bleeding, injury to the oesophagus, and/or phrenic nerve or pericarditis are possible. Additionally, epicardial fat can be a potential barrier to mapping and effective lesion delivery [51].

For VA arising from the LV summit region, detailed mapping of the distal CS branches (the great cardiac and anterior interventricular veins) and the adjacent structures including the LVOT, the anterior RVOT, and the LV septum should be performed. The presence of a similar early onset of activation in the LVOT and RVOT may suggest deep intramural or epicardial origin. Anatomic proximity enables adjacent sites to be activated simultaneously to the SOO. Ablation in such cases may be required from multiple locations including the coronary veins. Before ablation in the coronary veins, coronary angiography must be performed prior to energy delivery to ensure adequate separation. If coronary artery proximity precludes safe ablation from the coronary veins, ablation should be attempted from adjacent structures including the LV endocardium, left coronary aortic Sinus of Valsalva or from the RVOT. In some circumstances, epicardial ablation may be attempted. The great cardiac vein divides the LV summit into two parts, a superior portion and an inferior portion. The latter may be accessible to epicardial catheter ablation. However, due to the close proximity of the coronary arteries and overlying epicardial fat, the former is completely inaccessible to catheter ablation using the coronary venous and/or epicardial approach [52].

Indications for Catheter Ablation

Currently, radiofrequency ablation of idiopathic monomorphic VAs is indicated in patients who are symptomatic despite antiarrhythmic medication or those who do not wish to take antiarrhythmics. Additionally, patients with significant VA burden (with or without symptoms) and co-existing LV systolic dysfunction represent an important group in which to consider ablation [53]. A large multicentre trial by Latchamsetty et al. found an acute success rate of 84% and a 71% long-term success at 20 months for catheter ablation [54]. The high success rate of catheter ablation with an overall low risk of complications means that this is a reasonable first line approach to patients with this condition.

Conclusion

Idiopathic ventricular arrhythmias occur in patients in the absence of structural heart disease. The clinical course is usually benign; however, some patients may suffer incessant symptoms and some may develop reversible LV systolic dysfunction. Idiopathic VAs present distinctive ECG patterns. Analysis of ECG morphology allows one to predict possible sites of origin of VAs before mapping and to establish an optimal strategy for catheter ablation. Catheter ablation is highly successful with a low complication rate and should be considered first-line in selected patients.

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Conflict of Interest Disclosures

Dr Sanders reports having served on the advisory board of Biosense-Webster, Boston Scientific, CathRx, Medtronic, and St Jude Medical. Dr Sanders reports that the University of Adelaide has received, on his behalf, lecture and/or consulting fees from Biosense-Webster, Medtronic, Boston-Scientific, Pfizer and St Jude Medical. Dr Sanders reports that the University of Adelaide has received, on his behalf, research funding from Medtronic, St Jude Medical, Boston Scientific, Biotronik and Liva Nova. All other authors have no disclosures.

References

- [1] Aliot EM, Stevenson WG, Almendral-Garrote JM, Bogun F, Calkins CH, Delacretaz E, et al. European The Heart Rhythm Association (EHRA); Registered Branch of the European Society of Cardiology (ESC); The Heart Rhythm Society (HRS); American College of Cardiology (ACC); American Heart Association (AHA). EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European The Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and The American Heart Association (AHA). *Heart Rhythm* 2009;6:886–933.
- [2] Buxton AE, Marchlinski FE, Doherty JU, Cassidy DM, Vassallo JA, Flores BT, et al. Repetitive, monomorphic ventricular tachycardia: clinical and electrophysiologic characteristics in patients with and patients without organic heart disease. *Am J Cardiol* 1984;54:997–1002.
- [3] Lemery R, Brugada P, Bella PD, Dugernier T, van den Dool A, Wellens HJ. Nonischemic ventricular tachycardia. Clinical course and long-term follow-up in patients without clinically overt heart disease. *Circulation* 1989;79:990–9.
- [4] Hasdemir C, Ulucan C, Yavuzgil O, Yuksel A, Kartal Y, Simsek E, et al. Tachycardia-induced cardiomyopathy in patients with idiopathic ventricular arrhythmias: the incidence, clinical and electrophysiologic characteristics, and the predictors. *J Cardiovasc Electrophysiol* 2011;22:663–8.

- [5] Baman TS, Lange DC, Ilg KJ, Gupta SK, Liu TY, Alguire C, et al. Relationship between burden of premature ventricular complexes and left ventricular function. *Heart Rhythm* 2010;7:865–9.
- [6] Bottoni N, Quartieri F, Lolli G, Iori M, Manari A, Menozzi C. Sudden death in a patient with idiopathic right ventricular outflow tract arrhythmia. *J Cardiovasc Med (Hagerstown)* 2009;10:801–3.
- [7] Leenhardt A, Glaser E, Burguera M, Nürnberg M, Maison-Blanche P, Coumel P. Short-coupled variant of torsade de pointes: A new electrocardiographic entity in the spectrum of idiopathic ventricular tachyarrhythmias. *Circulation* 1994;89:206–15.
- [8] Viskin S, Rosso R, Rogowski O, Belhassen B. The short-coupled variant of right ventricular outflow tract tachycardia: a not-so-benign form of benign ventricular tachycardia. *J Cardiovasc Electrophysiol* 2005;16:912–6.
- [9] Asirvatham SJ. Correlative anatomy for the invasive electrophysiologist: outflow tract and supraventricular arrhythmia. *J Cardiovasc Electrophysiol* 2009;20:955–68.
- [10] Hasdemir C, Aktas S, Govsa F, Aktas EO, Bozkaya YT, Demirbas MI, et al. Demonstration of ventricular myocardial extensions into the pulmonary artery and aorta beyond the ventriculo-arterial junction. *Pacing Clin Electrophysiol* 2007;30:534–9.
- [11] Gami AS, Noheria A, Lachman N, Edwards WD, Friedman PA, Talreja D, et al. Anatomical correlates relevant to ablation above the semilunar valves for the cardiac electrophysiologist: a study of 603 hearts. *J Interv Card Electrophysiol* 2011;30:5–15.
- [12] Movsowitz C, Schwartzman D, Callans DJ, Preminger M, Zado E, Gottlieb CD, et al. Idiopathic right ventricular outflow tract tachycardia: narrowing the anatomic location for successful ablation. *Am Heart J* 1996;131:930–6.
- [13] Yamada T. Idiopathic ventricular arrhythmias: Relevance to the anatomy, diagnosis and treatment. *J Cardiol* 2016;68:463–71.
- [14] Callans DJ, Menz V, Schwartzman D, Gottlieb CD, Marchlinski FE. 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.
- [15] Betensky BP, Park RE, Marchlinski FE, Hutchinson MD, Garcia FC, Dixit S, et al. The V(2) transition ratio: a new electrocardiographic criterion for distinguishing left from right ventricular outflow tract tachycardia origin. *J Am Coll Cardiol* 2011;57:2255–62.
- [16] Hayashi T, Santangeli P, Pathak RK, Muser D, Liang JJ, Castro SA, et al. Outcomes of Catheter Ablation of Idiopathic Outflow Tract Ventricular Arrhythmias With an R Wave Pattern Break in Lead V2: A Distinct Clinical Entity. *J Cardiovasc Electrophysiol* 2017;28:504–14.
- [17] Ouyang F, Fotuhi P, Ho SY, Hebe J, Volkmer M, Goya M, et al. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp. *J Am Coll Cardiol* 2002;39:500–8.
- [18] Dixit S, Gerstenfeld EP, Callans DJ, Marchlinski FE. Electrocardiographic patterns of superior right ventricular outflow tract tachycardias: distinguishing septal and free-wall sites of origin. *J Cardiovasc Electrophysiol* 2003;14:1–7.
- [19] Bala R, Garcia FC, Hutchinson MD, Gerstenfeld EP, Dhruvakumar S, Dixit S, et al. Electrocardiographic and electrophysiologic features of ventricular arrhythmias originating from the right/left coronary cusp commissure. *Heart Rhythm* 2010;7:312–22.
- [20] Dixit S, Gerstenfeld EP, Lin D, Callans DJ, Hsia HH, Nayak HM, et al. Identification of distinct electrocardiographic patterns from the basal left ventricle: distinguishing medial and lateral sites of origin in patients with idiopathic ventricular tachycardia. *Heart Rhythm* 2005;2:485–91.
- [21] Ling Z, Liu Z, Su L, Zipunnikov V, Wu J, Du H, et al. Radiofrequency ablation versus antiarrhythmic medication for treatment of ventricular premature beats from the right ventricular outflow tract: prospective randomized study. *Circ Arrhythm Electrophysiol* 2014;7:237–43.
- [22] Tondo C, Carbucicchio C, Russo AD, Majocchi B, Zucchetti M, Pizzamiglio F, et al. Idiopathic ventricular tachycardia: transcatheter ablation or antiarrhythmic drugs? *J Atr Fibrillation* 2015;7:1164.
- [23] Nogami A, Naito S, Tada H, Taniguchi K, Okamoto Y, Nishimura S, et al. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. *J Am Coll Cardiol* 2000;36:811–23.
- [24] Lin D, Hsia HH, Gerstenfeld EP, Dixit S, Callans DJ, Nayak H, et al. Idiopathic fascicular left ventricular tachycardia: linear ablation lesion strategy for noninducible or nonsustained tachycardia. *Heart Rhythm* 2005;2:934–9.
- [25] Yamada T, Kay GN. Anatomical consideration in catheter ablation of idiopathic ventricular arrhythmias. *Arrhythmia Electrophysiol Rev* 2016;5:203–9.
- [26] Yamada T, Doppalapudi H, McElderry HT, Okada T, Murakami Y, Inden Y, et al. Electrocardiographic and electrophysiological characteristics in idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: relevance for catheter ablation. *Circ Arrhythm Electrophysiol* 2010;3:324–31.
- [27] Tada H, Ito S, Naito S, Kurosaki K, Kubota S, Sugiyasu A, et al. Idiopathic ventricular arrhythmia arising from the mitral annulus: a distinct subgroup of idiopathic ventricular arrhythmias. *J Am Coll Cardiol* 2005;45:877–86.
- [28] Kumagai K, Yamauchi Y, Takahashi A, Yokoyama Y, Sekiguchi Y, Watanabe J, et al. Idiopathic left ventricular tachycardia originating from the mitral annulus. *J Cardiovasc Electrophysiol* 2005;16:1029–36.
- [29] Tada H, Tadokoro K, Ito S, Naito S, Hashimoto T, Kaseno K, et al. Idiopathic ventricular arrhythmias originating from the tricuspid annulus: Prevalence, electrocardiographic characteristics, and results of radiofrequency catheter ablation. *Heart Rhythm* 2007;4:7–16.
- [30] Tada H, Tadokoro K, Miyaji K, Ito S, Kurosaki K, Kaseno K, et al. Idiopathic ventricular arrhythmias arising from the pulmonary artery: prevalence, characteristics, and topography of the arrhythmia origin. *Heart Rhythm* 2008;5:419–26.
- [31] Wasmer K, Köbe J, Dechering DG, Bittner A, Pott C, Mönnig G, et al. Ventricular arrhythmias from the mitral annulus: Patient characteristics, electrophysiological findings, ablation, and prognosis. *Heart Rhythm* 2013;10:783–8.
- [32] Obel OA, d'Avila A, Neuzil P, Saad EB, Ruskin JN, Reddy VY. Ablation of left ventricular epicardial outflow tract tachycardia from the distal great cardiac vein. *J Am Coll Cardiol* 2006;48:1813–7.
- [33] Baman TS, Ilg KJ, Gupta SK, Good E, Chugh A, Jongnarangsin K, et al. Mapping and ablation of epicardial idiopathic ventricular arrhythmias from within the coronary venous system. *Circ Arrhythm Electrophysiol* 2010;3:274–9.
- [34] Nogami A, Naito S, Tada H, Oshima S, Taniguchi K, Aonuma K, et al. Verapamil-sensitive left anterior fascicular ventricular tachycardia: results of radiofrequency ablation in six patients. *J Cardiovasc Electrophysiol* 1998;9:1269–78.
- [35] Pathak RK, Betensky BP, Santangeli P, Dixit S. Distinct electrocardiographic form of idiopathic ventricular arrhythmia originating from the left bundle branch. *J Cardiovasc Electrophysiol* 2017;28:115–9.
- [36] Nakagawa M, Takahashi N, Nobe S, Ichinose M, Ooie T, Yufu F, et al. Gender differences in various types of idiopathic ventricular tachycardia. *J Cardiovasc Electrophysiol* 2002;13:633–8.
- [37] Yamada T, McElderry HT, Okada T, Murakami Y, Doppalapudi H, Yoshida N, et al. Idiopathic focal ventricular arrhythmias originating from the anterior papillary muscle in the left ventricle. *J Cardiovasc Electrophysiol* 2009;20:866–72.
- [38] Doppalapudi H, Yamada T, McElderry HT, Plumb VJ, Epstein AE, Kay GN. Ventricular tachycardia originating from the posterior papillary muscle in the left ventricle: a distinct clinical syndrome. *Circ Arrhythm Electrophysiol* 2008;1:23–9.
- [39] Sadek MM, Benhayon D, Sureddi R, Chik W, Santangeli P, Supple GE, et al. Idiopathic ventricular arrhythmias originating from the moderator band: electrocardiographic characteristics and treatment by catheter ablation. *Heart Rhythm* 2015;12:67–75.
- [40] Thakur RK, Klein GJ, Sivaram CA, Zardini M, Schleinkofer DE, Nakagawa H, et al. Anatomic substrate for idiopathic left ventricular tachycardia. *Circulation* 1996;93:497–501.
- [41] Good E, Desjardins B, Jongnarangsin K, Oral H, Chugh A, Ebinger M, et al. Ventricular arrhythmias originating from a papillary muscle in patients without prior infarction: a comparison with fascicular arrhythmias. *Heart Rhythm* 2008;5:1530–7.
- [42] Haïssaguerre M, Shoda M, Jais P, Nogami A, Shah DC, Kautzner J, et al. Mapping and ablation of idiopathic ventricular fibrillation. *Circulation* 2002;106:962–7.
- [43] Yamada T, Doppalapudi H, McElderry HT, Okada T, Murakami Y, Inden Y, et al. Idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: prevalence, electrocardiographic and electrophysiological characteristics, and results of the radiofrequency catheter ablation. *J Cardiovasc Electrophysiol* 2010;21:62–9.
- [44] Bunch TJ, Weiss JP, Crandall BG, Day JD, DiMarco JP, Ferguson JD, et al. Image integration using intracardiac ultrasound and 3D reconstruction for scar mapping and ablation of ventricular tachycardia. *J Cardiovasc Electrophysiol* 2010;21:678–84.
- [45] Itoh T, Yamada T. Usefulness of pace mapping in catheter ablation of left ventricular papillary muscle ventricular arrhythmias with a preferential conduction. *J Cardiovasc Electrophysiol* 2018;29:889–99.
- [46] Gordon JP, Liang JJ, Pathak RK, Zado ES, Garcia FC, Hutchinson MD, et al. Percutaneous cryoablation for papillary muscle ventricular arrhythmias after failed radiofrequency catheter ablation. *J Cardiovasc Electrophysiol* 2018.

- [47] Doppalapudi H, Yamada T, Ramaswamy K, Ahn J, Kay GN. Idiopathic focal epicardial ventricular tachycardia originating from the crux of the heart. *Heart Rhythm* 2009;6:44–50.
- [48] Heeger CH, Hayashi K, Kuck KH, Ouyang F. Catheter ablation of idiopathic ventricular arrhythmias arising from the cardiac outflow tracts—recent insights and techniques for the successful treatment of common and challenging cases. *Circ J* 2016;80:1073–86.
- [49] Gensini GG, Digiorgi S, Coskun O, Palacio A, Kelly AE. Anatomy of the coronary circulation in living man; coronary venography. *Circulation* 1965;31:778–84.
- [50] El-Maasarany S, Ferrett CG, Firth A, Sheppard M, Henein MY. The coronary sinus conduit function: anatomical study (relationship to adjacent structures). *Europace* 2005;7:475–81.
- [51] Desjardins B, Morady F, Bogun F. Effect of epicardial fat on electro-anatomical mapping and epicardial catheter ablation. *J Am Coll Cardiol* 2010;56:1320–7.
- [52] Yamada T, McElderry HT, Doppalapudi H, Okada T, Murakami Y, Yoshida Y, et al. Idiopathic ventricular arrhythmias originating from the left ventricular summit: anatomic concepts relevant to ablation. *Circ Arrhythm Electrophysiol* 2010;3:616–23.
- [53] Pedersen CT, Kay GN, Kalman J, Borgreffe M, Della-Bella P, Dickfeld T, et al. EP-Europace, UK. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. *Heart Rhythm* 2014;11:e166–96.
- [54] Latchamsetty R, Yokokawa M, Morady F, Kim HM, Mathew S, Tilz R, et al. Multicenter outcomes for catheter ablation of idiopathic premature ventricular complexes. *JACC Clin Electrophysiol* 2015;1:116–23.