REVIEW

Using the Surface Electrocardiogram to Localize the Origin of Idiopathic Ventricular Tachycardia

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The surface electrocardiogram (ECG) is a useful tool to help identify the sites of origin of ventricular tachycardia (VT). Despite such limitations as chest wall deformity and metabolic and drug effects, the analysis of the QRS morphologic patterns and vectors can discern the site of activation of myocardium. There have been described numerous reports about ECG features of idiopathic left- and right-ventricular VT. In this review, we summarized typical ECG characteristics according to the VT sites of origin based on previous reports, with anatomical considerations of the left and right ventricles, including the outflow tracts and epicardium. (PACE 2012; 35:1516–1527)

electrocardiogram*, *VT*, *Ablation*, *electrophysiology - clinical

Introduction

Idiopathic ventricular tachycardia (VT) is recognized as a ventricular arrhythmia (VA) without an apparent structural heart disease and accounts for approximately 10% of all patients referred for an evaluation of VT.[1] Idiopathic VT commonly arises from the outflow tract (OT) of the right ventricle (RV) and left ventricle (LV), but it also can involve the fascicles of the specialized conduction system in the LV, the mitral and tricuspid annuli, or the anterior and posterior papillary muscles (PPM) in the LV. OT VT can arise from the endocardium of the RVOT and LVOT below the semilunar valves, above the pulmonic valve, or within the aortic sinuses of Valsalva (ASOV). Rarely, idiopathic VT has an epicardial origin. Past decades have seen a tremendous increase in our understanding of the origin and mechanisms of monomorphic VT, as well as in our ability to successfully treat these arrhythmias by catheter mapping and ablation. Central to this progress was the growing interest about the anatomical basis of VAs and the recognition of the role of the 12-lead electrocardiogram (ECG) as a mapping tool (Table I). Prior detailed knowledge about the specific features of the surface ECG regarding different sites of VT origin is essential for choosing the optimal ablation method and improving the chances salient loca ablation success. In this review, we will look at the salient ECG features of idiopathic VT that aid in localization and will guide to design for ablation strategy.

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General Considerations

The QRS complex during idiopathic VT is generated from a distant site of origin followed by spread of activation away from the focus. Some general principles related to ventricular geometry and activation governs the ECG patterns seen in VT. First, left ventricular free wall VT shows a right bundle branch block (RBBB) configuration, while VT exiting from the interventricular septum or right ventricle displays a left bundle branch block (LBBB) configuration. Second, septal exits are associated with narrower QRS complexes consistent with synchronous rather than sequential ventricular activation. Third, basal sites show positive precordial concordance, while negative concordance is seen in apical sites of origin. The QRS axis varies with exit shifts predominantly along a superoinferior axis but right-left shifts can also occur. Anatomical variation is the other main factor that can cause disruption to expected pattern of body surface electrical vector for a given arrhythmia origin. This can arise from translational, rotational, or attitudinal shifts in the normal relationship of the heart to the chest wall, or from variations within the cardiomediastinal anatomy itself. Antiarrhythmic drugs, by affecting

Table I.

Electrocardiographic Features of Idiopathic Ventricular Tachycardias

Localization of VT	BBB	Axis	Precordial Transition	V_1	V_6	I	Other ECG Features
Left ventricle							
ASOV							
LCC	LBBB	Inferior	$\leq V_2$	rS, RS	R	rS	Notched M or W in V_1, QS or RS in lead I
RCC	LBBB	Inferior	$\leq V_3$	rS, RS	R	R	Early transition, broad R in V_2, positive in V_1
LCC/RCC junction	LBBB	Inferior	V_3	qrS	R	R/Rsr'	Notched on the downward deflection in V_1 or w pattern
LVOT							
Septal sites	LBBB	Left inferior	Early	QS/Qr	Rs	Rs	Ratio of QS in II and III > 1
AMC	RBBB	Inferior	None	qR	R	R/Rs	Positive precordial concordance and no S in V_6
MA							
Anterolateral	RBBB	Inferior	Early	R	Rs	QS/rS	Late inferior lead notching; wider QRS, late S wave
Posterior	RBBB	Superior	Early	R	Rs	Rs	Absence of notching in inferior leads
Epicardial							
AIV/GCV junction	LBBB	Inferior	Early	rS/QS	R	rS	Precordial pattern break with abrupt loss of R waves in V_2: MDI > 0.55
Crux	LBBB	Left superior	Early	Variable	R	Rs	Positive concordance V_2-V_6; MDI > 0.55; slurred intrinsicoid deflection
Papillary muscle							
Posteromedial	RBBB	Superior	Variable	rsR	RS	R	Late R to S precordial transition
Anterolateral	RBBB	Inferior	$< V_1$	rsR	RS	S	qR/qr pattern in lead aV_R and rS pattern in lead V_6
Fascicular							
Left posterior	RBBB	Left superior	Early	rsR	RS	Q	Loss of late precordial R waves with more apical exits
Left anterior	RBBB	Right	None	rsR	RS	Q	Similar to posterior fascicular apart from axis
Upper septal	LBBB	Normal or right	V_3	rS	Rs	Rs	Narrow QRS complex with VA dissociation
Right ventricle							
RVOT							
Anterial, septal	LBBB	Inferior	$\leq V_3$	rS	R	rS	Early transition with lead I negative, isoelectric, or multiphasic
Posterior, free wall	LBBB	Inferior	$\geq V_3$	rS	R	R	Late transition; broad late notched inferior leads and lead I positive
Parahisian	LBBB	Inferior	$> V_3$	QS	R	R	Isoelectric or positive aV_L, large R amplitude in I, V_5 and V_6, taller inferior R

continued

Table I.

Continued

Localization of VT	BBB	Axis	Precordial Transition	V_1	V_6	I	Other ECG Features
PA	LBBB	Inferior	$> V_3$	rS/QS	R	rS/QS	$1 > aV_L/aV_R$ ratio of the Q-wave amplitude, $1 <$ R/S ratio in lead V_2
TA							
Septal	LBBB	Inferior	$< V_3$	QS	R	R/r	Positive, isoelectric, or multiphasic in aV_L
Free-wall	LBBB	Variable	V_4-V_5	rS	R	R/r	Notching in limb leads; discordant forces in inferior leads if inferior

AIV = anterior interventricular vein; GCV = great cardiac vein; AMC = aortomitral continuity; ASOV = aortic sinus of Valsalva; RBBB/LBBB = right/ left bundle branch block; LCC = left coronary cusp; RCC = right coronary cusp; OT = outflow tract; MA = mitral annulus; MDI = maximum deflection index; PA = pulmonary artery; TA = tricuspid annulus.

myocardial conduction characteristics, can also affect the surface ECG appearance of VT.

LV or RV Origin?

VTs arising from the LV have a RBBB-like morphology because the LV is activated before the RV; however, VTs arising on or adjacent to the LV septum can have a LBBB morphology if VTs exit from the septum to the RV. Similarly, most VTs arising from the RV will have a LBBB-like morphology. In the OT sites of both ventricles, there are lots of variations and overlaps using these criteria. For instance, the anterior aspect of the LVOT is closely located to the posterior aspect of the RVOT and the ECG morphologies are similar each other. In this review, we explained the ECG characteristics of each idiopathic VT site in detail classifying as both OT and the other ventricular sites including epicardial and fascicular VT (Table II and Fig. 1).

LV Origin Idiopathic VT

Approximately 20–30% of idiopathic VTs arise from the LV. We classified sites of origin of the idiopathic left VT as LVOT (supravalvular, infravalvular, and epicardial), mitral annulus, papillary muscles, and verapamil-sensitive fascicular VT. The LVOT sites were subclassified as supra valvular (ASOV), infravalvular (aortomitral continuity [AMC] and septal-parahisian), and epicardial (anterior interventricular vein/great cardiac vein [AIV/CGV]) site.

Anatomical considerations: It may seem surprising that VT can originate from the aortic sinuses since they are not traditionally thought to be associated with ventricular myocardium. However, crescent fibers of ventricular myocardium that have been identified at the base of the left- and right-aortic sinuses may serve as the underlying arrhythmogenic anatomic structure.[2] By contrast, the base of the non-coronary cusp (NCC) is composed of fibrous tissue, which allows for the atrial input responsible for atrial tachycardia rather than VT,[3] explaining the very rare incidence of VT originating from this location.[4] The anterior aspect of the LVOT, in particular the right coronary cusp (RCC), is closely related to the posterior aspect of the RVOT explaining similar 12-lead ECG characteristics for arrhythmias arising from this origin. The LVOT is in close relationship to the right-sided superior septal region (His). Moreover, since the aortic valve is located more inferior and posterior relative to the pulmonic valve, subtle 12-lead ECG differences result and help to distinguish the VT origin, as discussed in detail below.

LVOT

OT arrhythmias including VT or frequent ventricular premature contractions (VPCs) are the most common form of the idiopathic VT and typically originate from the RVOT but can also occur from the LVOT. Approximately 15–25% of OT arrhythmia cases arise from the LVOT including ASOV.[5–7]

Table II.

Classification of the Idiopathic VT According to the Site of Origin

I. LV Origin VT
- A. LVOT
  - 1. Supravalvular
    - a. ASOV
      - (1) LCC
      - (2) RCC
      - (3) NCC
      - (4) LCC/RCC junction
  - 2. Infravalvular
    - a. AMC
    - b. Septo-parahisian
  - 3. Epicardial
    - a. AIV/CGV
- B. VT arising from other LV sites
  - 1. MA
  - 2. PPM
    - a. Anterolateral PPM
    - b. Posteromedial PPM
  - 3. Crux
- C. Fascicular VT
  - 1. Left posterior fascicle
  - 2. Left anterior fascicle
  - 3. Left upper septal fascicle

II. RV origin VT
- A. RVOT
- B. Parahisian
- C. TA
- D. PA
- E. VT arising from the other sites
  - 1. PPM

III. Epicardial origin VT

LV = left ventricle; RV = right ventricle; OT = outflow tract; ASOV = aortic sinus of Valsalva; LCC = left coronary cusp; RCC = right coronary cusp; NCC = non-coronary cusp; AMC = aortomitral continuity; GCV = great cardiac vein; AIV = anterior interventricular vein; MA = mitral annulus; PPM = papillary muscle; TA = tricuspid annulus; VT = ventricular tachycardia; PA = pulmonary artery.

1. Supravalvular

(a) ASOV. VT arising from the ASOV varies from 9% to 24% of idiopathic VT, depending upon the institution that reported.[8,9] In this arrhythmia, the origin more commonly arises from the left coronary cusp (LCC) than the right, rarely arises from the NCC,[10,11] and once again, is thought to be related to myocardial extensions projecting above the aortic valve into the ASOV (Fig. 2).

(1) LCC: ***ECG features.*** The surface ECG exhibits earlier precordial transition with broader and taller R waves in V_1 or V_2, taller inferior R waves, an S wave in lead I with a characteristic absence of S waves in V_5 and V_6.[12] Ouyang et al. found that in these leads an R-wave duration of >50% of the total QRS duration or an R/S ratio of more than 30% were strongly predictive of an aortic cusp (particularly a LCC) origin.[10] Pace mapping from the LCC showed a characteristic multiphasic notched pattern[13–14] in V_1 with an "M" or "W" pattern, presumably due to transseptal activation after initial LV activation from the LCC.

(2) RCC: ***ECG features.*** RCC pace mapping produces an early precordial transition, with a broad R in V_2 and a longer mean QRS duration. Lead I at this site may also be distinguished from LCC foci by the tendency to have either a QS or rS complex, and RCC foci generate more positive forces in this lead; but, this is variable and depends critically on annular location and orientation.[13] Pacing from RCC by Lin et al.[14] showed that the RCC typically has a left bundle-type pattern with a broad small R wave in V_2 and a precordial transition generally at V_3.

(3) NCC: NCC VT has no pathognomonic ECG pattern and pace mapping here results in atrial capture due to its proximity to the interatrial septum.[14]

(4) LCC/RCC junction: ***ECG features.*** Yamada et al.[15] have described OT VTs arising from the junction of the LCC and RCC. These have a characteristic multiphasic QRS configuration in V_1 and require ablation from the LVOT aspect of the aortic valve cusps. However, it is likely that there is significant overlap between this group and the multiphasic notched V_1 pattern seen in standard LCC foci. Recently, Bala et al. presented that ventricular arrhythmias originate from between the RCC/LCC aortic cusp are common and have a QS morphology in lead V_1 with notching on the downward deflection with precordial transition at lead V_3.[16]

2. Infravalvular

(a) AMC. AMC is located at the superior basal portion of the LV and mainly consists of fibrous tissue. It is embedded between the aortic and mitral valve annuli, bordered by the ventricular septum and anterior wall. The AMC is a recognized, potentially arrhythmogenic area of the heart that can give rise to atrial and ventricular arrhythmias, although it was thought to mainly consist of fibrous tissue. McGuire et al.[17] found cells histologically and electrophysiologically resembling AV junctional cells in this region, and it has been speculated that these cells may contribute to some idiopathic VTs.[18,19]

ECG features. The most distinctive ECG pattern of basal LVOT VT belongs to VT arising from the AMC that characteristically displays a qR pattern in V_1 as a result of the left fibrous trigone deflecting initial electrical activation leftward. Depending on the anatomical factors, however, particularly the position and extent of the trigone, AMC VT may not display this ECG signature and may instead have an RBBB pattern with

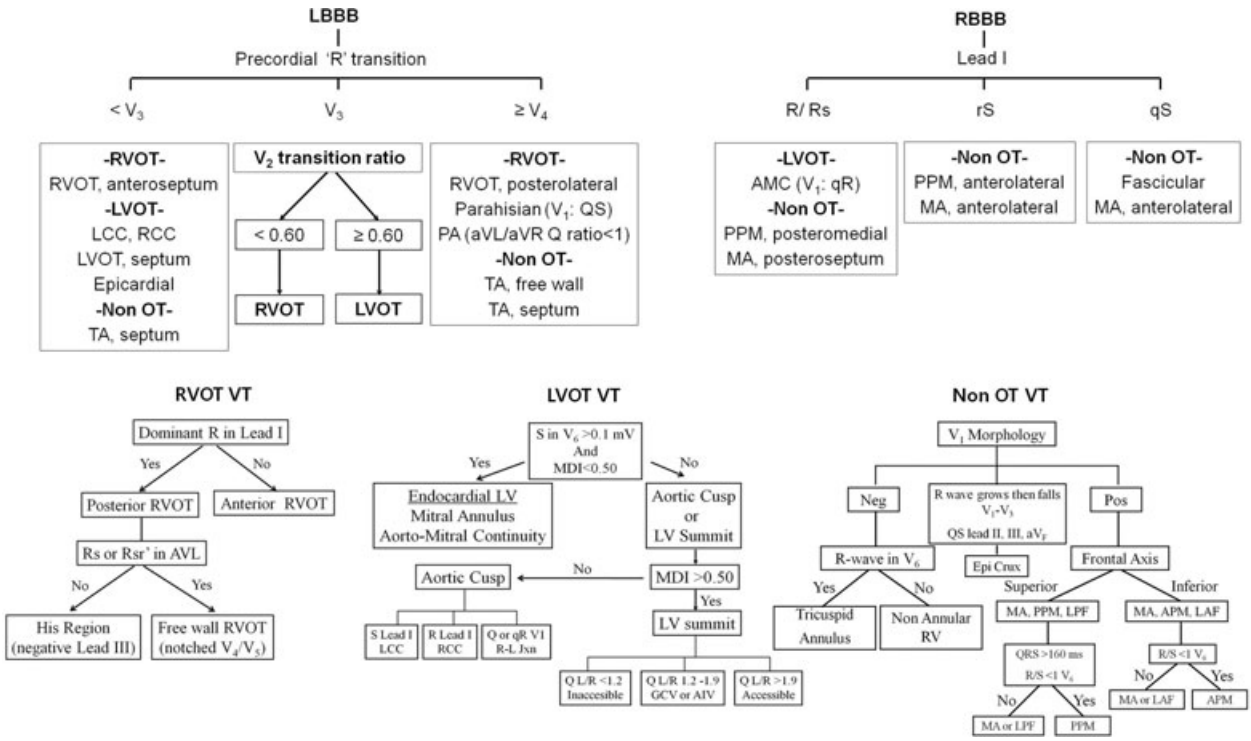

Figure 1. *Useful ECG algorithm to localize site of idiopathic VT using the bundle branch block pattern, precordial "R"-wave transition, and lead I characteristics. OT = outflow tract; LCC = left coronary cusp; RCC = right coronary cusp; R-L Jxn = RCC-LCC junction; TA = tricuspid annulus; PA = pulmonary artery; MA = mitral annulus; PPM = papillary muscle; AMC = aortomitral continuity; MDI = maximal deflection index; GCV = great cardiac vein; AIV = anterior interventricular vein; RBBB/LBBB = right/left bundle branch block.*

positive concordance and no late precordial S waves as described by Kumagai et al.[18] This group also showed that both AMC VT and mitral annulus (MA) VT have earlier transitions than a group of ASOV VTs and that they had longer intrinsicoid deflection times. However, there was no difference in inferior lead R-wave amplitude between the groups. Pacing study from the AMC by Lin et al. described a qR pattern in lead V_1 that was not observed anywhere else in the LVOT.[14]

(b) Septal-parahisian sites. ***ECG features.*** Septal sites in the parahisian region display an LBBB configuration more akin to RV foci and have a dominant R in lead I, usually with left inferior axis. In the RVOT VT, lead I also had a monophasic R wave, but the R wave in lead I was significantly smaller compared to the His group. A QS pattern in lead V_1 and R wave in lead aV_L also were observed more often in the His group than that in the RVOT. A precordial transition zone occurred in lead V_2–V_3. The QRS duration in leads II, III, and aV_F was significantly narrower in the His group than that in the RVOT. The R-wave amplitude in leads V_5 and V_6 in the His group was significantly greater, and the R-wave amplitude in leads III and aV_F was significantly smaller than that in the RVOT.

3. Epicardial

(a) AIV/CGV. The region adjacent to the left sinus of Valsalva (LSOV) is a part of the LV ostium that poses anatomical and technical concerns to electrophysiologists because the endocardial and epicardial sites of VT origin are in close proximity and may be difficult to discriminate. This region may be separated into three parts according to the successful ablation site: the LCC, the AMC, and the epicardial left ventricular summit that may be accessible from the GCV or AIV. Recently, Abularach et al. reported that VT originating near the AIV/CGV can be ablated from the LSOV or adjacent LV endocardium with a Q-wave ratio of aVL/aVR < 1.45 and close anatomical distance <13.5 mm to help identify candidates for successful ablation.[20]

ECG features. A small r wave, or rarely an R wave, is typically observed in lead I during both spontaneous VT and pacing from the AMC, LCC,

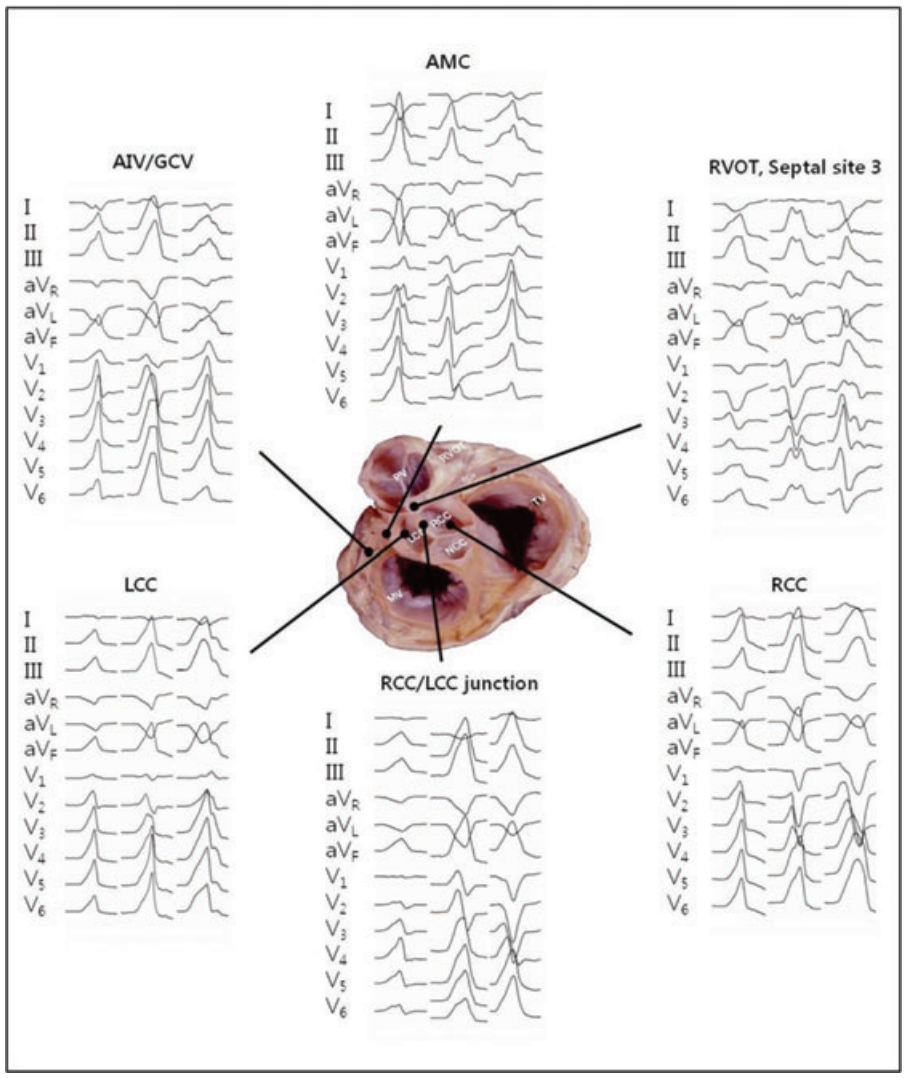

Figure 2. *Aortic sinus cusps image with corresponding examples of ECG morphology. AIV/GCV = anterior interventricular vein/great cardiac vein; AMC = aortomitral continuity; LCC = left coronary cusp; RCC = right coronary cusp; NCC = non coronary cusp; MV = mitral valve; TV = tricuspid valve; RVOT = right ventricular outflow tract.*

GCV, and AIV. The ratio of the Q-wave amplitude in leads aV_L to aV_R is significantly higher in epicardial (CGV and AIV) VT than the AMC VT. An S wave in lead V_5 or V_6 can be observed in any of the LCC VT. The shortest RS interval (interval from the earliest ventricular activation to the nadir of the first S wave in any precordial lead) and maximum deflection index (MDI: interval from the earliest ventricular activation to the peak of the largest amplitude deflection in each precordial lead divided by the QRS duration) are significantly larger in epicardial VT than LCC and AMC VT. When comparing the VT origins successfully ablated by an endocardial approach (in the LCC and AMC) and an epicardial approach (in the GCV and AIV), a shortest RS interval of >121 ms and an MDI of >0.55 predicted an epicardial VT origin with a sensitivity of 67% and 67%, specificity of 69% and 82%, positive predictive accuracy of 33% and 46%, and negative predictive accuracy of 90% and 91%, respectively.[21] Yamada et al. reported that the RBBB, the transition zone, the R-wave amplitude ratio in leads III to II, the Q-wave amplitude ratio in leads aVL to aVR, and the S wave in lead V_6 all accurately predicted the site of origin of the idiopathic VAs originating from the LV summit.[22] Based on these five criteria, the VAs with the RBBB pattern, the earlier precordial transition, the Q-wave amplitude ratio in aVL/aVR > 1.1, and S waves in V_5 or V_6 are likely to be

cured by catheter ablation within GCV, AIV, or the accessible area (Fig. 1).

VT Arising from Other LV Sites

1. Mitral annulus

The MA VT is a wide QRS tachycardia with a delta wave-like beginning of the QRS complex. The MA is located in the posterior portion of the LV and anterior, and anterolateral sites have more predominant occurrence than posteromedial sites.

ECG features. Precordial R-wave transitions earlier than V_2 are suggestive of a LVOT VT.[7] Because the origin of tachycardia in the MA is located in the posterior portion of the LV, distant from the precordial electrodes, the myocardium near the focus is depolarized in a direction toward these electrodes. This could account for the early precordial transition and concordant positive QRS pattern in leads V_2–V_4 of the majority of these tachycardias that arise from the anterolateral MA.[23] Furthermore, in this location the ECG exhibits positive precordial concordance with an RBBB configuration in V_1 usually with late notching in the inferior leads. This has been confirmed with pace mapping studies demonstrating that, compared to septal sites, anterolateral and lateral MA sites of origin exhibit a longer QRS duration along with predominantly negative forces in lead I.[19] Kumagai et al. described a stepwise ECG algorithm for presenting the sites of origin of the MA VT by using earlier precordial transition, S wave in lead V_6, intrinsicoid deflection time, and R-wave polarity in inferior leads.[24] Late notching in the inferior leads or an S wave in lead I, which are found usually in both anterolateral MA and posterior MA VT, may be absent in anterior or posteroseptal MA VT. Tada et al. also proposed an algorithm to predict the precise focus of MA VT based on the QRS-wave configuration on 12-lead ECG recordings with the use of precordial transition, presence of positive QRS polarity, or notching of the R wave in inferior leads.[23] This algorithm correctly identified the origin of MA VT with a sensitivity of 60%, specificity of 99.7%, positive predictive value of 95%, and negative predictive value of 96%.

2. PPM

The PPMs have been suggested to be a potential site for reentry with a contribution in the maintenance of ventricular fibrillation in animal models.[25–27] Recently, the LV PPMs have been reported to be arrhythmogenic in the human heart after myocardial infarction.[28,29] Main characteristics of PPM VT are: (a) RBBB morphology, (b) refractoriness to verapamil and Na^+ channel blockers (which suggests that the conduction system or myocytes sensitive to these antiarrhythmic drugs are not directly involved), (c) tendency for VPCs rather than VT, (d) inducibility with exertion, (e) lack of inducibility with programmed ventricular or atrial stimulation, (f) earliest ventricular activation at the base or middle portion of the LV PPM, (g) absence of high-frequency potentials at the site of origin, and (h) requirement of high RF power to achieve long-term ablation success.[30]

(a) Anterolateral PPM. Anterolateral PPM VTs exhibit RBBB pattern and right inferior axis QRS morphology with an early precordial transition, generally before V_1. qR or qr pattern in lead aV_R and rS pattern in lead V_6 (R/S ratio $<$ 1) are useful features to differentiate from other LV sites. The mean QRS duration during VT or VPCs is 168 ms.[30,31]

(b) Posteromedial PPM. VT with a posterior PPM origin showed RBBB and right or left superior axis QRS morphology. The mean QRS duration during VT was within 160 ms. Monophasic R and qR pattern predominate in lead I. R/S amplitude ratio <1 in lead V_6 was a useful features to differentiate from other LV sites.[30]

3. Crux

Most of these idiopathic epicardial VTs are in close proximity to the coronary venous system at the summit of the LV, at the AIV-GCV junction. The crux is at the epicardial surface of the heart, near the junction of the middle cardiac vein and the coronary sinus (CS).[32]

ECG features. The QRS morphology of this VT shows early precordial transition at or before V_2, in addition to a left superior axis and an MDI $>$ 0.55. Using the Berruezo criteria,[32] all patients had a pseudo-delta wave $\geq$34 ms and intrinsicoid deflection time in lead $V_2 \geq$ 85 ms. Recently, Doppalapudi et al. have described epicardial idiopathic VT arising from the crux of the heart in the pyramidal space adjacent to the posterior descending artery.[34] The inferior leads (II, III, aVF) exhibited deeply negative QS complexes with slurred intrinsicoid deflections, and this is very similar to the pattern of maximal preexcitation seen with manifest posteroseptal pathways that share a similar ventricular insertion into the pyramidal space.

Fascicular VT (Verapamil-Sensitive VT)

The Purkinje networks in the small anatomic structures (e.g., small fibromuscular bans, trabeculae, and small papillary muscles) are important when considering the reentry circuit of verapamil-sensitive left posterior fascicular VT. This

idiopathic arrhythmia, also known as left septal VT or verapamil-sensitive VT, is due to reentry involving altered Purkinje-fibers on the LV aspect of the septum. There are three main variants of this arrhythmia; left posterior, left anterior, and upper septal, each with typical ECG manifestations.

1. Left posterior fascicular VT

ECG features. By far, the most common form is due to reentry involving the left posterior fascicle with an exit at the inferoapical LV septum.[35] This displays an RBBB QRS complex with left superior axis and RS complexes in V_5 and V_6. There is a loss of these late precordial R waves when exits are more apical.

2. Left anterior fascicular VT

ECG features. Uncommon type of verapamil-sensitive fascicular VT, in which the reentry circuit involves the left anterior fascicle. These VTs exhibit an RBBB configuration with right axis deviation.[36]

3. Upper septal fascicular VT

ECG features. Very rare and involves the proximal left bundle branch. It is remarkable for its narrow QRS complex with normal or rightward axis deviation.

Comparison between Fascicular VT and Papillary Muscle Origin VT

PPM-related arrhythmias often involve the distal Purkinje fiber system, so, several different characteristics were documented when compared to fascicular arrhythmias that involve the proximal Purkinje system.[37] ECG features of PPM VT include the lack of discrete Q waves in the lateral or inferior leads and the absence of an rsR' pattern in lead V_1 that is characteristic for fascicular tachycardia. The QRS complex is broader in PPM-related arrhythmias, which probably reflects a site of origin at the Purkinje-myocardial interface, where conduction is slower than in the Purkinje system.

RV Origin Idiopathic VT

This is the commonest form of idiopathic VT and accounts for around 10% of all VT seen in referral centers.[1] Approximately 70–80% of idiopathic VTs arise from the RV. The OT region consists of a number of contiguous structures within a small three-dimensional space. These include the RVOT, the pulmonary artery (PA), and the parahisian region.

RV Outflow Tract (RVOT)

Anatomical considerations. The posteroseptal aspect of the RVOT is adjacent to the RCC region, and the anterior septal surface is adjacent to the anterior margin of the RCC or the medial aspect of the LCC. In young patients, the aortic valve is parallel to the pulmonic valve and perpendicular to the mitral valve. In older patients, the aortic valve may be a more vertical tilted and parallel to the mitral valve. The RVOT region is defined superiorly by the pulmonic valve and inferiorly by the RV inflow tract and the top of the tricuspid valve. The lateral aspect of the RVOT region is the RV free wall, and the medial aspect is formed by the interventricular septum at its base of the RVOT and RV musculature opposite the root of the aorta at the region beneath the pulmonic valve. From the coronal view above the pulmonic valve, the RVOT region is seen wrapping around the root of the aorta and extending leftward. The top of the RVOT may be convex or crescent shaped, with the posteroseptal region directed rightward and the anteroseptal region directed leftward. Occasionally, an extreme convexity of the superior septal RVOT region creates a leftward direction for the most posterior and anterior aspects of the septal RVOT and results in a net negative QRS complex in lead I from either (posterior or anterior) site. The anteroseptal aspect of the RVOT actually is located in close proximity to the LV epicardium, adjacent to the AIV and in proximity to the left anterior descending coronary artery. In addition, the location, rotation, and horizontal position of the heart in the chest cavity may influence surface ECG characteristics. These anatomic considerations are critical for 12-lead ECG analysis and are helpful for predicting the site of origin of OT VT.

ECG features. The RVOT is by far the most common site of origin of idiopathic VT and may account for up to 75% of all OT VT cases. Despite the close anatomical proximity of OT VT sites, the 12-lead ECG remains an outstanding tool for initial mapping. The classical ECG patterns of OT VT include LBBB configuration monomorphic VT with an inferiorly directed frontal plane QRS axis and deeply negative QS complexes in leads aV_L and aV_R. Pace and activation mapping studies have demonstrated several important generalizations in ECG localization of RVOT VT. First, septal sites on the RVOT tend to have narrower LBBB QRS complexes with earlier precordial transition (positive QRS by V_3 or earlier) and higher amplitudes in the inferior leads.[38] In contrast, free wall sites in the RVOT, which account for up to 34% of RVOT VT foci,[1] have later precordial transitions ($\geq V_4$), with broader QRS complexes and notching in the inferior leads.[39] It should be remembered, however, that even from the free wall, conduction velocity in idiopathic OT VT is rapid, and significantly wide QRS

complexes should prompt a thorough evaluation for the presence of structural heart disease and myocardial fibrosis. Second, posterior sites in the RVOT are distinguished by their leftward initial vector that tends to produce a positive QRS complex in limb lead I.[39] Anterior sites have either isoelectric or negative, often multiphasic, forces in lead I.[38] It is important that the limb lead electrodes are placed correctly as the lead I vector will be reversed with posteroseptal sites with electrode placement on the chest rather than the shoulders.[16] While the majority of RVOT VT foci lie at the top of the RVOT within a 1–2-cm craniocaudal band subjacent to the pulmonic valve, they produce a negative QS pattern in lead aV_L.[40] Isoelectric or positive forces in aV_L strongly imply a more caudal site of origin at the base of the RVOT, potentially adjacent to the His bundle.[1,41]

R/S Ratio in Lead V_3

Differential ECG Features between the LVOT and RVOT VT. The presence of an S wave in lead I is helpful to identify an LV origin; however, this ECG characteristic is present also in other sites. Kamakura et al. reported that if the R/S ratio was <1 in lead V_3, the origin was likely located in the RVOT.[40] The reported positive predictive value of this criterion was 100%; however, the negative predictive value was low (50%). Conversely, if the R/S ratio is ≥1 and the initial R-wave amplitude in leads V_1 and V_2 is high, the origin is likely in the LVOT. However, this algorithm does not identify an origin remote from the RVOT and LVOT (Fig. 1).

R-Wave Duration Index/R/S-wave Amplitude Index

Ouyang et al. reported that QRS morphology of idiopathic VT from the ASOV is similar to that of RVOT arrhythmia, and that a longer R-wave duration index (>0.5) and a higher R/S-wave amplitude index (>0.3) in leads V_1 and V_2 are present in VT originating from the aortic sinus of Valsalva.[10] Several more complex ECG algorithms have been developed to classify idiopathic OT VT according to the site of origin, allowing localization of VT origin in up to six different OT sites using seven analysis steps.[42,43]

V_2 Transition Ratio

Although an R/S transition in lead V_3 is common in OT VT, the value of surface ECG criteria in OT VT with R/S transition in V_3 is limited. In patients with R/S transition in lead V_3, stepwise endocardial and epicardial mapping through up to six anatomic approaches, including first RVOT then LVOT, aortic sinus of Valsalva, CS, trunk of the PA, and epicardium can lead to successful radiofrequency catheter ablation.[43] Recently, Betensky et al. found new electrocardiographic criterion, V_2 transition ratio (Fig. 3), for distinguishing LVOT from RVOT origin in patients with lead V_3 precordial transition. They described that a V_2 transition ratio can predict an LVOT origin with 91% accuracy and a VPC/VT precordial transition occurring later than the sinus rhythm transition excluded an LVOT origin with 100% accuracy.[45]

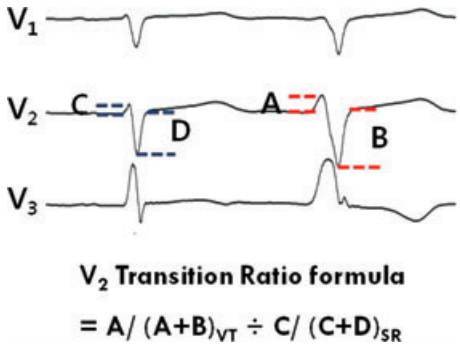

Figure 3. *Electrocardiographic measurements of V_2 transition ratio. VT = ventricular tachycardia; SR = sinus rhythm.*

Parahisian

ECG features. Compared to the RVOT, these parahisian foci also tend to display lower R-wave amplitude (III, aVF) and shorter QRS duration (II, III, aVF) in the inferior leads; larger R-wave amplitude in leads I, V_5, and V_6; and a QS pattern in V_1.[46] Given the close anatomical proximity of the aortic root, parahisian VT may overlap with OT VT arising from the NCC or RCC of the aortic valve, and no specific ECG criteria reliably differentiate these sites.[41]

Tricuspid Annulus (TA)

Another right-sided structure that may give rise to idiopathic VT is the TA.[47] While this does not strictly reside in the RVOT, the majority of TA VTs originate from the anteroseptal aspect in proximity to the His bundle. Thus, there is further overlap between these and parahisian VTs arising either from the base of the RVOT or the NCC.

ECG features. Lead aV_L can be either monophasic positive or multiphasic and of low amplitude, in addition to having the usual left bundle branch configuration with inferior axis. For TA VT arising from the free wall of the valve ring off the septum, a similar configuration is seen but with notching in the limb leads. This situation is analogous to RVOT free wall VT. TA VT arising from the posterolateral portion has discordant

forces in the inferior leads depending on how inferiorly the focus is located. Tada et al. reported that VT/ PVCs arising from the TA demonstrated a LBBB QRS morphology and positive QRS polarity in leads I, V_5, and V_6.[47] No negative component of the QRS complex was found in lead I. Because the origin of the VT arising from the TA was located on the right anterior side of the heart, the myocardium would be depolarized in a direction toward the anode of lead I and leads V_5 and V_6, which might account for the findings.

Pulmonary Artery

Analogous to the arrhythmogenic structure of the pulmonary veins with regard to initiation of atrial fibrillation, the great arteries are invested with variable sleeves of myocardium extending above the semilunar valves.[48] In certain cases these may harbor arrhythmogenic foci and give rise to OT VT.

ECG features. PA VT arises 0.5–2.1 cm cranial to the pulmonic valve and consequently has an ECG appearance similar to typical RVOT VT, sometimes with taller R waves in the inferior leads.[49] However, given that the pulmonary trunk is a more leftward structure than the infundibulum, the ECG may exhibit an earlier precordial transition and deeper QS in aV_L than in aV_R.[50] The R-wave amplitudes on the inferior leads in PA VT are significantly larger than those in RVOT VT. The average aV_L/aV_R ratio of Q-wave amplitude is >1 in PA VT; this is significantly larger than that in RVOT VT. In comparing lead I polarity, QRS morphology shows a QS (rS) pattern in the PA VT, whereas RVOT VT shows an R (Rs) pattern. The transitional zone is often observed at V_4 in both groups, but may also occur from either V_2 or V_3. There is lower R-wave amplitude on lead V_2 in patients of the PA VT, and the R/S amplitude ratio on lead V_2 was significantly larger than that in the RVOT.

VT Arising from Other Sites of RV

PPM: ***ECG features.*** The 12-lead ECG of the RV PPM VTs were analyzed for bundle branch block morphology, axis, QRS width, presence of a notch in V_1–V_6, R-wave pattern in V_1 (rS, QS), and transition point from a predominantly negative S wave to a positive R-wave deflection in the precordium. RV PPM VT has an early transition and is defined as a transition in lead V_4 or earlier. An rS or QS pattern appears in lead V_1 and displays LBBB morphology with an inferior or superior axis. The mean QRS width during ventricular ectopy is over 160 msec. A notch in the precordial leads is present in most PPM VTs. The RV PPM arrhythmias originating in the posterior or anterior PPM more often have a later R-wave transition ($>V_4$) and a superior axis compared with arrhythmias originating from the septal RV PPMs, which more often have an earlier transition ($\leq V_4$) and an inferior axis.

Epicardial Origin VT

VT in this region may arise from perivascular tissue associated with the coronary venous system, particularly at the junction of the GCV and AIV but also from other epicardial sites.[21]

ECG features. While there is no specific ECG feature that is pathognomonic for epicardial OT VT, the surface ECG generally displays an LBBB configuration with an inferior axis and transition usually around V_3. Berruezo et al. developed three ECG criteria in cardiomyopathy patients with sensitivity and specificity ranging between 76% and 95% for predicting an epicardial exit in patients with failed endocardial VT ablation.[33] The first criterion is the presence of a pseudodelta wave in any precordial leads of RBBB VTs, defined as the time from the QRS onset to the earliest rapid deflection of $\geq$34 ms. The second is a delayed intrinsicoid deflection, defined as the time from QRS onset to the peak of the R wave in V_2 of $\geq$85 ms. The last criterion is the shortest RS interval, defined as the time from the first ventricular activation to the nadir of the first S wave in any precordial leads of $\geq$120 ms. Other criteria have been suggested, such as the presence of regional Q waves, indicating a wavefront propagation away from an epicardial site. Bazan et al. developed a regional model of Q-wave patterns that predicted a left ventricular epicardial exit for VT in the absence of prior myocardial infarction.[51] Pace mapping and activation mapping during VT showed that Q waves, as part of the VT morphology, better predicted epicardial exit than the pseudodelta, intrinsicoid deflection or shortest RS criteria described in the predominantly ischemic cardiomyopathy population of the Berruezo et al. study.[33] In particular, anterobasal and anteroapical epicardial VT exits showed Q waves in lead I, and anterobasal sites were especially more likely if there were no inferior Q waves. Inferobasal and inferoapical sites were characterized by inferior Q waves. Daniels et al. quantified this by developing a dimensionless metric called the precordial MDI, which is defined by the shortest time to maximal positive or negative deflection in any precordial lead divided by the QRS duration. A cut-off value of 0.55 had high sensitivity and specificity in discriminating between epicardial foci and other OT sites of origin.[21] Another clue of epicardial LVOT tachycardia is the presence of precordial "pattern break,"[15] also known as R-wave regression/progression, in which there is an abrupt loss

of R wave in V_2 followed by resumption in R waves from V_3 to V_6. Recently, Vallès et al. described a four-step algorithm for identifying epicardial origin VT from basal superior and lateral LV in the setting of nonischemic cardiomyopathy using the presence of inferior q waves, pseudo-delta $\geq$ 75 ms, MDI $\geq$ 0.59, and presence of q wave in lead I. The total sensitivity and specificity of the algorithm in this study population for pace map localization reaches 96% and 93%, respectively.[52]

Conclusion

The surface 12-lead ECG is an important and useful tool for localization of VT site of origin. In this review, we suggested a useful ECG algorithm to localize the site of idiopathic VT using the bundle branch block pattern, precordial "R"-wave transition, and ECG characteristics of leads I and V_1/V_6. Despite some inherent limitations including complex anatomy, the 12-lead ECG remains an indispensible tool for selecting the adequate ablation procedure method in order to save the time and improve ablation success of VT.

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