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REVIEW

The Surface Electrocardiograph in Ventricular Arrhythmias: Lessons in Localisation

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The 12-lead electrocardiograph (ECG) is of critical importance both in the diagnosis of wide complex tachycardia and in the further classification, characterisation and management of ventricular arrhythmias. With its diligent application and interpretation, remarkable precision can be achieved in the localisation of the site of origin of ventricular arrhythmias and this may have major implications in the care of these patients. This review discusses the technical, anatomic and mechanistic basis of ECG interpretation in ventricular arrhythmias.

Keywords Electrocardiography • Ventricular tachycardia • Ventricular arrhythmia

Introduction

Despite several decades of major technological advances in tachycardia diagnosis and mapping, it is remarkable that no single tool has surpassed the standard 12-lead electrocardiograph (ECG) for the fundamental characterisation and analysis of ventricular arrhythmias (VA). No one other investigation provides as much critical information to guide the overall management of the patient with ventricular tachycardia (VT), both during the acute and often unstable clinical presentation, as well as during subsequent treatment. This is because so much of the current understanding of VA mechanism, classification and prognosis is intimately related to the ECG findings. All this from an inexpensive, non-invasive and essentially risk-free test that can be performed rapidly at the bedside. Additionally, for localisation and definitive elimination of VT by catheter ablation, the surface ECG forms the basis of all conventional and advanced mapping methods, not least by acting as a reliable fiducial reference of the onset and origin of ventricular activation.

This review examines the use of the 12-lead ECG in the diagnosis and management of patients with monomorphic ventricular arrhythmias.

Wide Complex Tachycardia: Factors Favouring VT

Given that a tachyarrhythmia originating in the ventricle at least partially circumvents the normal sequence of rapid biventricular myocardial depolarisation by the His-Purkinje system, VT essentially always presents as a wide complex tachycardia (WCT) with QRS duration of >110 ms [1]. The diagnostic methodology and approach to WCT interpretation is detailed elsewhere [2] but it is worth emphasising several important principles here.

1. In view of the relative rarity of preexcited tachycardias (those in which there is any antegrade conduction over a manifest bypass tract), pacemaker-related tachycardias, drug and electrolyte related tachycardias and artefact, in practice the most common diagnostic possibilities for WCT lie between (a) VT; or (b) supraventricular tachycardia (SVT) with some degree of functional conduction delay or block in the His-Purkinje system (aberrance).
2. The clinical condition of the patient is of entirely no relevance in making a diagnosis with up to 85% of conscious WCT patients having VT as their arrhythmia [3].

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3. The presence of ventricular fibrosis, either confluent from prior myocardial infarction or more diffuse due to dilated cardiomyopathy, strongly favours VT as the diagnosis.
4. Although it is self-evident that the haemodynamically unstable patient needs to be electrically cardioverted immediately, the reasonably well-perfused patient should be assessed closely for any of the several (overall uncommon) findings that are essentially diagnostic for VT. These include (a) clinical signs of ventriculoatrial (VA) dissociation such as canon A waves in the jugular venous pulse or a variable intensity of the first heart sound; (b) electrocardiographic VA dissociation; (c) negative precordial concordance (positive concordance may be seen with some preexcited tachycardias); (d) a narrower QRS width during tachycardia than in sinus rhythm traces (if available); and (e) a WCT morphology displaying a contralateral bundle branch block pattern to a bundle branch block present in sinus rhythm traces (if available).
5. The morphology of the QRS complex is of great importance in making the correct diagnosis for any WCT, and its de-emphasis in contemporary ECG education is regrettable. While multiple algorithms exist, an important general principle relates to the limited number of stereotypical patterns that His-Purkinje aberrance can adopt. Thus great caution should be exercised in making a diagnosis of SVT in any WCT patient who does not exhibit a typical bundle branch block and/or fascicular block pattern [1].
6. The use of adenosine as a diagnostic manoeuvre is of limited utility given that many forms of focal idiopathic VT (particularly those due to triggered activity) can be terminated by adenosine while many supraventricular tachycardias may not be. In addition, despite its usually brief duration of action, adenosine has the potential to cause coronary steal in patients with coronary artery disease, who are overrepresented in cohorts of patients presenting with WCT.

General Principles of VT Electrocardiography

By virtue of the QRS morphology, the 12-lead ECG provides a panoramic, omniplanar, multi-vectorial view of the global wavefront of ventricular myocardial activation, including, critically, its site of initiation. For idiopathic focal VT occurring in the absence of overt manifest structural heart disease, this site is the point source of origin of the arrhythmia. In the much more frequent setting of scar-related VT, this site is the exit of a re-entrant activation wavefront from a constrained diastolic isthmus located within electrically silent myocardial scar.

The key ECG lead in the initial regionalisation, and indeed classification, of VT origin is V_1. This is because it is located nearly orthogonal to the septal plane and is, not dissimilarly to the situation in the atrium [4], best able to resolve initial right verses left sided activation. When V_1 has a net positive QRS (R>s), the VT is considered to have right bundle branch block (RBBB) configuration. Conversely, net negative QRS (r<S) defines a left bundle branch block (LBBB) configuration. This is not to imply that these configurations look anything like typical bundle branch block patterns, but they are a helpful part of VT nomenclature.

Given the overall uniformity of human ventricular topology and its relationship to the chest wall (where the surface ECG electrodes are applied in the standard positions), a few general principles are useful in VT localisation:

1. With some rare exceptions only, VTs of RBBB configuration arise from the left ventricle (LV) while VTs of LBBB configuration arise from the septum or from the free wall of the right ventricle (RV).
2. Septal origins of VT give rise to simultaneous rather than sequential activation of the LV and RV, and hence are associated with narrower QRS widths.
3. Basal sites of origin in the ventricle have a net activation vector towards the overlying chest wall electrodes and hence show earlier transitions to QRS positivity in the precordial leads, or indeed in the extreme case of some annular origins, full positive precordial concordance. Conversely, apical sites have early transitions to QRS negativity given that their activation vector points away from the chest wall. In the extreme case of some apical VTs, this may result in complete negative precordial concordance.
4. Whilst the QRS axis in the frontal plane generally reflects VT origin along a craniocaudal extent, given the way the ventricles spiral around each other, it may also reflect right–left shifts in wavefront origin.

The precision with which the ECG can localise the origin or exit of a VA is subject to a host of possible confounding factors [Table 1]. In light of these considerations, it can be appreciated that the optimal scenario for ECG-based localisation of VT origin is in the setting of a point focus of activation in a patient with a structurally normal heart and chest wall, who is on no conduction-slowing medications.

Table 1 Limitations of the ECG in localisation of ventricular arrhythmias.

Significant ventricular fibrosis, particularly large confluent scar
Significant ventricular dilatation, hypertrophy or infiltration
Congenital heart disease
Translational, attitudinal, or rotations shifts of ventricle relative to chest wall
Chest wall deformity (e.g. pectus excavatum)
Antiarrhythmic medication
Incorrect ECG lead hook-up (reversal of limb or precordial lead cables)
Incorrect ECG electrode placement (particularly limb electrodes on chest wall)

Abbreviation: ECG, electrocardiogram.

This is indeed the case in most patients with idiopathic VT and, not surprisingly, ECG prediction of the site of origin is remarkably accurate in these cases. Conversely, in patients with large confluent anterior LV scars, even septal versus free wall regionalisation of VT exits may be difficult.

Needless to say, the ECG can only be useful for localisation of a monomorphic ventricular arrhythmia if it is recorded during the arrhythmia. Whilst this is not an issue in patients with tolerated monomorphic VT, unstable patients may need to be shocked urgently and it may not be possible to record a full 12-lead ECG, although every effort should be made to do so. Failure to record the ECG during the actual arrhythmia is an even bigger problem when conventional four-channel, 4 × 3 lead recordings are used to try to capture isolated ventricular ectopy or runs of non-sustained VT. Many patients with idiopathic VT have high baseline levels of ambient ectopy arising from the same focus and (as discussed elsewhere in this issue) there are several emerging indications for treatment of ventricular ectopics. If only one premature ventricular complex (PVC) occurs during the 10 second recording window of a four-channel trace, then the QRS morphology of the ectopic may be available on as few as three (and no more than four) ECG leads. For this reason, our preferred mode of ECG recording is the running 12-lead rhythm strip which guarantees a 12-lead ECG morphology for all isolated ectopics or other non-sustained events [Figure 1].

Classification of Focal Idiopathic VA

The vast majority of idiopathic VT in patients without overt heart disease arises from a focal point source of activation. The electrophysiologic mechanism is generally triggered activity in the form of delayed afterdepolarisations (DADs) due to intracellular diastolic calcium overload [5]. Smooth, rapid wavefront propagation through the structurally normal ventricle means the ECG is of great importance (and reliability) in understanding the site or origin of the arrhythmia.

Idiopathic VT was initially conceived of as an infundibular problem and was even known by the limiting term, 'RVOT (right ventricular outflow tract) VT'. It is apparent that the cellular foci from which idiopathic VTs arise are not randomly scattered throughout the ventricles but are clustered in well described sites of anatomic heterogeneity. Although the RVOT probably accounts for the majority of sites of origin [6], referral bias in data from tertiary centres probably underestimates this proportion and contemporary population based epidemiological studies are lacking. The last decade has seen the systematic description by multiple groups of various characteristic sites of origin of idiopathic VT, both right and left sided, and from outflow and non-outflow regions of the ventricles. Since the ECG features suggestive of right versus left sided origins can be subtle with significant overlap present, it is more helpful to initially classify idiopathic VT into outflow tract and non-outflow tract forms. Outflow tract VT (OTVT), regardless of its precise site of origin, is characterised by an inferior frontal plane QRS axis with variable but overall negative QS-type complexes in aVR and aVL. Non-outflow tract VTs conversely have a superiorly directed QRS axis.

Outflow Tract Idiopathic VA

The anatomy of the outflow tracts must be properly understood to appreciate the ECG patterns seen in OTVT. The

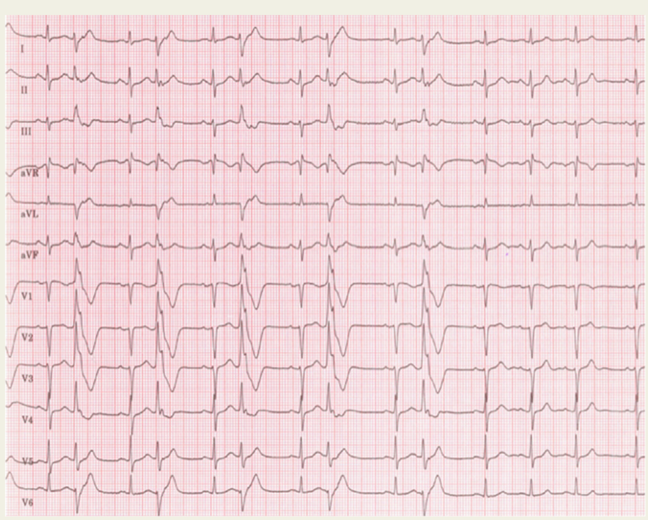

Figure 1 Running 12-channel rhythm strip recording a 12-lead ECG morphology of isolated ventricular ectopics in a bigeminal pattern arising from anterolateral papillary muscle. Note that the run of ventricular bigeminy ends before the end of the trace and if a conventional 4 × 3, 4-channel ECG had been recorded then, only the sinus rhythm QRS morphology would be available.
Abbreviation: ECG, electrocardiogram.

outflow tracts are basally located, and the central structure of the heart, namely the aortic root, forms a useful reference point to consider the anatomic relations here [Figure 2]. Due to the pattern of folding of the embryonic bulbis cordis, the RVOT (infundibulum) wraps anteriorly around the aortic root and LV summit, leftward of the subjacent interventricular septum (IVS). Since the pulmonary valve is located around 2 cm cranial of the aortic valve, much of the so-called 'septal' aspect of the RVOT is supravalvular and is applied to the aortic root, not the IVS. The anteroseptal aspect of the crescent-shaped RVOT is the most leftward part of it and sits overlying the left aortic sinus of Valsalva (ASOV) and the LV summit. The posteroseptal aspect is in close relationship to the right ASOV. The distal muscular RV infundibulum supports the pulmonary valve cusps at the ventriculoarterial junction and extends further to become the sinotubular pulmonary artery. Here it comes into close proximity to the left atrial appendage.

In the majority of people, including those with no clinical VT, there are variable myocardial extensions from the RVOT beyond the infundibular conus and sinotubular junction and into the wall of the pulmonary artery (above the pulmonary sinus cusps) and these may be arrhythmogenic [7,8]. Ablation of VA from the pulmonary sinus cusp (PSC) probably targets these extensions, and must be undertaken with particular care in the left PSC where proximity to the left coronary artery is a consideration [8,9]. Myocardial extensions are also commonly noted above the right ASOV more than the left ASOV, and only rarely in the non-coronary ASOV [8].

As described above, the basic ECG pattern of outflow tract VT is that of a LBBB configuration with inferior axis. Whilst this is the classical pattern seen in most forms of RVOT and pulmonary artery VT, various left-sided sites of origin may also exhibit a similar pattern. Thus, particularly from an ablation perspective, the most important initial consideration in interpreting OT VT morphologies is distinguishing right-sided from left-sided sites of origin. Given the overlapping anatomic relations described above, this is not always straight-forward although some general principles apply. Most helpful is generally the precordial QRS transition, with r<S to r>S reversal occurring earlier with left-sided foci. The best reference for this transition during VT or ectopy is the sinus rhythm precordial transition [Figure 3]. This was quantified in a metric called the V_2 transition ratio by Betensky et al. and although a measured ratio of ≥ 0.6 is highly predictive of a left-sided origin, in many cases, a visual comparison of the VT and sinus rhythm transitions suffices [10]. In an elegant study, Zhang et al. expanded on this concept by incorporating a posterior lead, V_8, to add further right-left discriminating information. They controlled for the R wave amplitude in V_4 and compared this ratio to that present in sinus rhythm, finding that this algorithm performed well for detecting left-sided foci [11].

After establishing the likely side of the arrhythmogenic focus, further ECG clues localise particular sites of origin on each side. Each of these clues is based on specific anatomic considerations. On the right side, the septal aspect of the

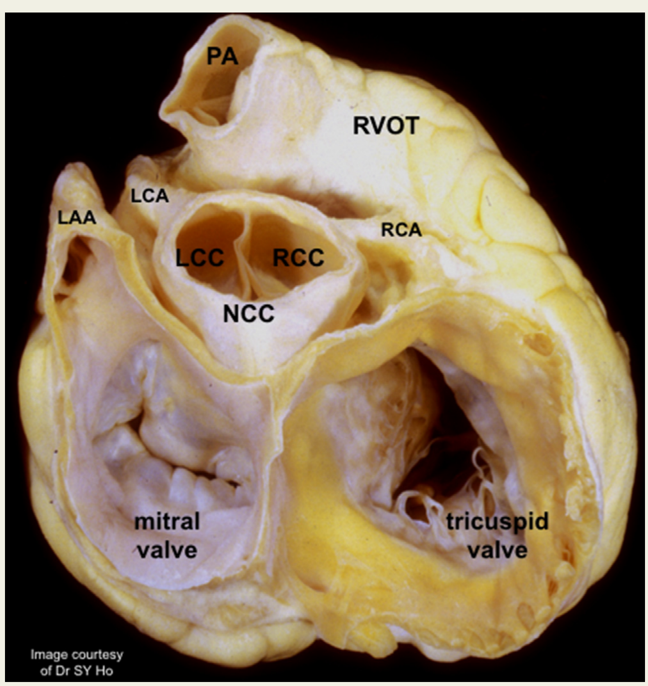

Figure 2 Outflow tract anatomy from a basal view after removal of the atria. Relations described in text.
Abbreviations: PA, pulmonary artery; RVOT, right ventricular outflow tract; LAA, left atrial appendage; LCA, left coronary artery; RCA, right coronary artery; LCC, left coronary cusp of aortic sinus of Valsalva; RCC, right coronary cusp; NCC, non coronary cusp.

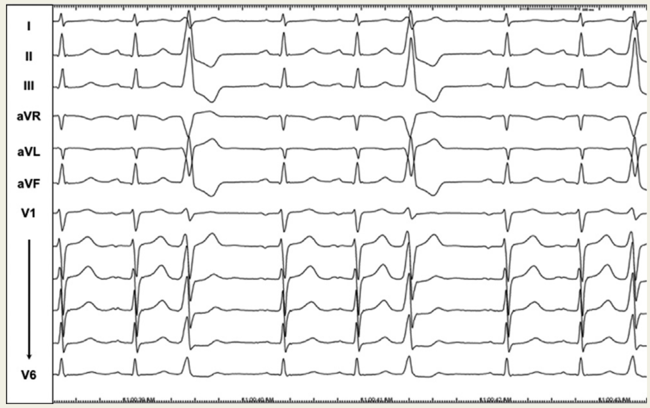

Figure 3 A 12-lead ECG showing ventricular ectopy arising from the outflow tract with positive forces in the inferior leads II, III and aVF. Features against an origin in the right ventricular outflow tract include the early precordial transition in the ectopic relative to the sinus transition, the negative ratio of QS amplitude in aVR to aVL, and the broad prominent R in V_1. This ectopic focus was ablated in the left aortic sinus of Valsalva.
Abbreviation: ECG, electrocardiogram.

RVOT [12], the free wall of the RVOT [12], the parahisian region [13,14], and the right, left and anterior pulmonary sinus cusps of the pulmonary artery [9] all have specific features that are summarised in Table 2. The specific features of the various left-sided sites of origin, namely the left ASOV [15], the right ASOV [16], the left/right commissural junction of the ASOV [17], the aortomitral continuity [18], the antero-lateral mitral annulus [19] and the LV summit [20] are outlined in Table 3.

Non-Outflow Tract Idiopathic VT

As a corollary to the outflow sites described above, non-outflow tract idiopathic VAs generally have an overall superior axis with negative (or discordant) forces in leads II, II and aVF (Table 4). Similar electrophysiologic mechanisms drive these arrhythmias with triggered activity being prevalent. However, there is one important exception, a rare form of VT designated variably fascicular VT, left septal VT or verapamil-sensitive VT. Although an idiopathic VA occurring in structurally normal hearts, the operative mechanism in fascicular VT is re-entry involving the posterior fascicle [21] [Figure 4]. Also listed for completeness in Table 4 are two other forms of Purkinje-system re-entry, namely bundle branch re-entry VT and interfascicular re-entry, although these are not idiopathic VTs and instead usually occur in patients with significant structural heart disease.

Stereotypical sites of origin in regions of heterogeneity are again the rule in focal non-outflow tract idiopathic VA, and the commonest of these with their associated ECG features are listed in Table 4. Anatomically important structures here include the posteromedial papillary muscle [22], the

Table 2 **Specific ECG features of right-sided idiopathic outflow tract VA foci.**

Site of Origin	BBB	Axis	V_1	V_6	Other features
Septal RVOT	LBBB	Inferior	rS	R	Early transition $<V_4$; lead I negative; positive or multiphasic
Free wall RVOT	LBBB	Inferior	rS	R	Later transition; broad inferior QRS with notching on downstroke
Parahisian RVOT	LBBB	Inferior discordance	QS	R	Early transition with isoelectric or positive aVL; large R in lead I
Anterior PSC	LBBB	Inferior	rS	R	Narrower QRS, larger R in II, III, aVF; QS in lead 1; transition V_4 or V_3
Left PSC	LBBB	Inferior	rS	R	Similar features to anterior PSC
Right PSC	LBBB	Inferior	rS	R	Wider QRS, large R in lead I, low amplitude inferior lead R with II > III, Q wave in aVR > Q wave in aVL

Abbreviations: ECG, electrocardiogram; RVOT, right ventricular outflow tract; PSC, pulmonary sinus cusp; VA, ventricular arrhythmia; LBBB, left bundle branch block.

Table 3 Specific ECG features of left-sided idiopathic outflow tract VA foci.

Site of Origin	BBB	Axis	V_1	V_6	Other features
Left ASOV	LBBB or RBBB	Inferior	rS, 'M' or 'W'	R	Early transition $<V_3$; lead I QS or rS; lead aVL QS
Right ASOV	LBBB	Inferior	rS	R	Early transition $<V_3$; broad R in V_2; lead I positive
Commissural L/R ASOV junction	LBBB	Inferior	QS	R	Notch on downstroke of V_1 QS complex
Aortomitral continuity	RBBB	Inferior	qR	R or RS	Basal precordial transition with qR in V_1; lead I biphasic
Mitral annulus	RBBB	Inferior	R	Rs	Basal precordial transition; lead I negative; inferior lead notching
LV summit*	Atypical LBBB/RBBB	Inferior	rS	R	Broad r in V_1; precordial transition pattern break; MDI $>$ 0.55

Abbreviations: ASOV, aortic sinus of Valsalva; LV, left ventricular; MDI, maximum deflection index; ECG, electrocardiogram; LBBB, left bundle branch block; RBBB, right bundle branch block.
*Epicardial site.

Table 4 Specific ECG features of non-outflow tract VA.

Site of Origin	BBB	Axis	V_1	V_6	Other features
Posteromedial LV papillary muscle	RBBB	Left superior	Rr′	RS	R $>$ r′ in V_1, apical transition, wider QRS
Anterolateral LV papillary muscle	RBBB	Right superior	Rr′	RS	R $>$ r′ in V_1, inferior discordance, apical transition, wider QRS
Fascicular left septal VT	RBBB	Left superior	rR′	RS	r $>$ R′ in V_1, QRSd $<$ 133 ms, more basal transition; RBBB with VA dissociation
Bundle branch re-entry VT	LBBB	Variable	QS	R	Typical LBBB morphology with VA dissociation
Interfascicular re-entry	RBBB	Right	rR′	RS	Narrow QRS, variable transition
Mitral annulus	RBBB	Variable	R	Rs	Positive precordial concordance
Tricuspid annulus	LBBB	Variable	R	Rs	Variable axis/transition; lead I positive
RV papillary muscles	LBBB	Left superior	QS	rS or QS	Late transition LBBB pattern
Moderator band	LBBB	Superior	QS	rS or QS	Very late transition LBBB pattern
Crux*	LBBB or RBBB	Superior	rS	R or rS	Right or atypical LBBB; variable transition with precordial pattern break

Abbreviations: ECG, electrocardiogram; LBBB, left bundle branch block; RBBB, right bundle branch block; RV, right ventricular; VT, ventricular tachycardia; LV, left ventricular.
*Epicardial site.

anterolateral papillary muscle [22], posterolateral mitral annular sites [19], tricuspid annular sites [23], right ventricular papillary muscles [24], the moderator band [25] and the crux [26].

Epicardial Idiopathic VT

While most idiopathic VT arises from well described endocardial foci, two regions in particular have a predilection for epicardial VT breakouts. The more common of these is the region known as the LV summit, the highest point on left ventricular epicardium. It is bounded cranially by the bifurcation of the left main coronary artery, and the great cardiac vein traverses across its caudal aspect. Idiopathic VT foci here display outflow type characteristics with LBBB-type morphology (albeit often with a broad R in V_1 and V_2) and right inferior frontal plane axis. Due to their remoteness from the endocardially located Purkinje system, epicardial foci tend to have delayed intrinsicoid deflections particularly in the precordial leads and this was quantified into a metric called the maximum deflection index by Daniels et al. [27]. Additionally, since these summit foci lie closest to lead V_2, a characteristic precordial

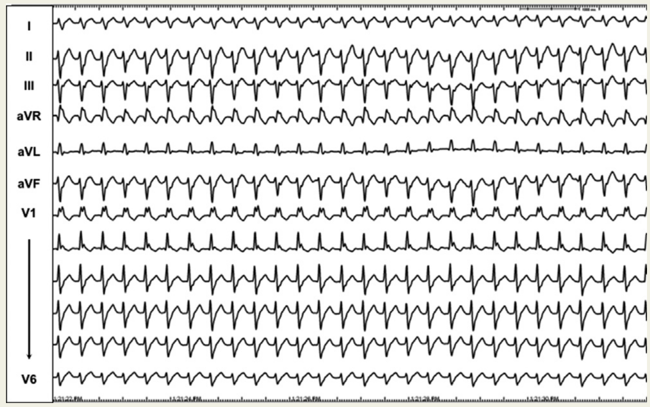

Figure 4 A broad complex tachycardia with right bundle branch block configuration, apical transition and superior axis. VA dissociation is best appreciated in the inferior leads and excludes SVT with aberrance. This is fascicular VT which is an idiopathic VT in structurally normal hearts. However, it is not due to a focal mechanism but rather re-entry involving the left posterior fascicle.
Abbreviations: VA, ventricular arrhythmia; VT, ventricular tachycardia; SVT, supraventricular tachycardia.

transition pattern break may be observed with net negative forces observed in V_2 relative to V_1 and V_3 [28]. Potential coronary artery proximity makes ablation of these foci challenging.

The second typically epicardial breakout of idiopathic VT is from the crux of the heart, apical of the pyramidal space. The basic ECG pattern here is a LBBB configuration with left superior axis but the inferior leads here display markedly negative QS complexes. These foci also typically exhibit a prolonged maximum deflection index (MDI) >0.55 and may display precordial V_2 pattern break also [26]. Proximity to the posterior descending coronary artery is a particular concern in the ablative management of epicardial crux VT.

Post-Myocardial Infarct VT

In the setting of structural heart disease associated with ventricular scarring, VT is generally due to myocardial re-entry. The most well understood example of this is in the confluent subendocardial scar caused by prior myocardial infarction (MI). The machinery for re-entrant post-MI VT circuits is located within the dense scar zone where surviving myocyte bundles can become organised into conducting channels. During VT, these channels form the constrained diastolic isthmus through which the activation wavefront passes in between successive QRS complexes. Since it lies within electrically silent dense scar, the diastolic corridor has no representation on the surface ECG and the QRS during VT is instead inscribed when the tachycardia wavefront exits from the isthmus and propagates into the surrounding normal myocardium to activate the rest of the ventricles. The confluent scar in these patients alters wavefront conduction substantially and can cause electrical 'holes' that reduce the accuracy of the ECG in predicting the VT exit compared to patients with focal idiopathic VT in structurally normal hearts.

It should be noted here that the use of the term 'ischaemic VT' is best avoided given its ambiguity. In the setting of acute ischaemia or infarction, VT is usually polymorphic or pleomorphic, and is generally due to triggered activity or abnormal automaticity. Sustained monomorphic VT usually occurs in remote infarct scars that have matured in specific ways to allow for stable re-entrant circuits to form [29].

As in all patients with scar-related VT, it is critical to appreciate the underlying substrate and its extent in order to best interpret the ECG during VT in the post-infarct setting. Patients with anterior MI scars develop characteristically different VT morphologies than patients with inferior MI scars and those with non-ischaemic cardiomyopathy.

In view of the typical locations of anterior and inferior infarct scars, the vast majority of post-MI VT circuits are located in the LV. Thus LBBB configurations of VT in this setting imply a septal LV exit while LV free wall exits display a RBBB morphology [Figure 5]. Cranial and caudal exits can be distinguished by examining the frontal plane axis, while apical and basal exits affect the precordial transition [30]. Inferior infarct VTs generally have more basal exits and thus more positive QRS complexes in the precordial leads. Thus, for example, an inferior infarct-related VT circuit exiting from the basolateral scar border would be expected to display a RBBB configuration with right superior axis and a late transition to negative QRS complexes in the precordial leads. Conversely, septal VT exits from an inferior MI scar show a LBBB morphology with left superior axis and an earlier transition to R waves in the precordial leads. Prediction of VT exit in anterior infarcts is more difficult due to the larger scar mass, particularly the more apical exits in which case even septal/lateral regionalisation may not be accurate. Additionally, RBBB morphology VTs with superior axis are especially difficult to localise as multiple LV exits are possible in anterior infarct patients with this VT pattern [30].

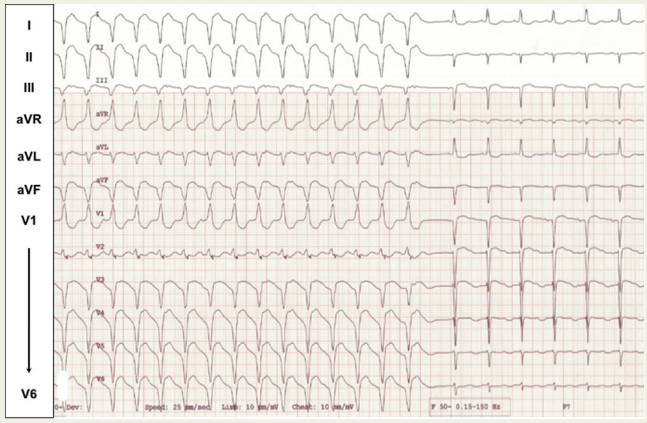

Figure 5 A 12-lead rhythm strip showing termination of a post-infarct VT to sinus rhythm. The VT is of right bundle right superior axis configuration and is exiting at the lateral border of a large anteroapical post-infarct scar. Notice the extensive Q waves which are more prominent in VT than in sinus rhythm.
Abbreviation: VT, ventricular tachycardia.

Non-Ischaemic Dilated Cardiomyopathy VT

As a rule, the substrate for monomorphic VT in non-ischaemic cardiomyopathy (NICM) is located in a basal periannular distribution and can affect the entire transmural extent of the LV myocardium in this region. There are generally two patterns of basal substrate seen in NICM, although they are not mutually exclusive. The first of these is centred around the basolateral LV and these patients have a high preponderance of epicardial substrate [31,32]. Their sinus rhythm ECGs may show a prominent S in V_6 and a significant r wave in V_1 [33]. The second pattern displays a predominant basal septal intramural involvement with minimal epicardial fibrosis and a tendency to early conduction system disease [32,34]. Not surprisingly, the characteristic VT morphologies seen in each pattern of scarring differ with a greater likelihood of epicardial exit configurations (see below) in the former and a tendency to seeing deep septal and pre-aortic exits in the latter. However, regardless of the precise pattern of scarring, the majority of NICM-related VTs have basal configurations with earlier transitions to precordial QRS positivity. Apical configurations of VT with late precordial transitions suggest the presence of a larger basal to apical scar (except in the uncommon but important situation of bundle branch re-entry) and Frankel et al. demonstrated a worse prognosis in NICM patients with these VTs [35].

Although uncommon overall, the most important VT that is most often seen in the context of NICM (but can be seen, rarely, even in normal hearts) is bundle branch re-entry (BBR) VT. In its typical form, this is due to a re-entrant wavefront of activation travelling down the right bundle branch, across the septum and then retrogradely up the left bundle branch, hence producing its stereotypical LBBB QRS morphology usually with apical transition. These patients generally have significant conduction system disease at baseline, often with a LBBB that appears similar in sinus rhythm and VT [Table 4]. A reverse typical form may be seen with retrograde activation up the right bundle branch and antegrade activation over the left bundle branch, consequently producing a RBBB QRS configuration. It is the rule that BBR VT is rapid given the small slow zone and partially excitable gap, as well as the fact that rapidly conducting Purkinje tissue makes up the majority of the circuit [36]. The recognition of BBR VT is important in view of the fact the it can readily be eliminated with focal ablation of the right (or less commonly, left) bundle branch. Although BBR VT is curable in this manner, patients' risk of recurrent myocardial scar-related VT and arrhythmic or heart failure death is determined by the severity of the underlying NICM and hence ICD implantation is usually indicated in these patients.

Epicardial Scar-Related VT

Although the majority of post-infarct VT circuits are located subendocardially, a substantial proportion of the re-entrant VTs in NICM have circuit components located intramurally or on the epicardium. When VT circuits exit on the epicardium, particular QRS signatures may be seen on the 12-lead ECG. These are predicated on two anatomic facts. Firstly, the rapidly conducting Purkinje system is a predominantly subendocardial network and is engaged much later by epicardial breakouts than by endocardial ones. This has the effect of broadening the QRS, increasing the duration of the intrinsicoid deflection and slurring its initial forces (so called pseudo-delta waves). Along with the MDI described above, these latter are known as the interval criteria for epicardial VT exit. In the initial description, a pseudo-delta wave of $\geq$34 ms, an intrinsicoid deflection of $\geq$85 ms and an RS complex duration of $\geq$121 ms had reasonable sensitivity and specificity for predicting and epicardial VT origin [37].

Secondly, from the point-of-view of the respective regional surface ECG leads, the transmural thickness of the ventricular wall will be depolarised in the same direction by an epicardial exit such that the initial transmural forces giving rise to small R waves with a corresponding endocardial exit will be lost. Similarly, the small Q waves seen in distant leads during endocardial VT may be lost with epicardial exits [38,39]. These signatures of epicardial VT origin are known as the morphology criteria. They only apply in non-ischaemic settings as the presence of prior MI scar may generate confounding Q waves.

An algorithm combining interval and morphology criteria has displayed the best published performance in predicting an epicardial exit for VT [40]. It must be stressed, however, that ECG features suggestive of an epicardial VT origin are but one of many factors in deciding whether to pursue pericardial access and epicardial mapping. The underlying substrate, imaging findings, prior failed endocardial ablation and prior pericardial disease or instrumentation may all impact the decision in addition to the ECG.

Conclusions

The 12-lead ECG is a simple, reproducible and accurate tool for the initial localisation of the origin of both focal and re-entrant forms of VT. While it performs best in the setting of idiopathic VT in structurally normal hearts, it is also of great utility in diagnosing re-entrant scar-related VT in both ischaemic and non-ischaemic cardiomyopathy. Although numerous metrics and highly detailed algorithms exist, a good first-order localisation of the origin of most VT can be made with an understanding of cardiac anatomy and the intelligent application of a few simple principles.

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