Electrocardiographic Localization of Ventricular Tachycardia in Patients with Structural Heart Disease

John M. Miller, MD[a,*], Rahul Jain, MD, MPH[b], Gopi Dandamudi, MD[c], Thomas R. Kambur, MD[c]

KEYWORDS

• Electrocardiogram • Ventricular tachycardia • Exit sites • Mapping

KEY POINTS

- In the presence of scar related to structural heart disease, common sense rules for localizing the source of ventricular complexes and ventricular tachycardia (VT) are not always valid.
- However, reasonably consistent 12-lead patterns of VT exist such that exit sites can be regionalized in up to 75% of VTs in patients with prior myocardial infarction.
- Exit sites of VTs with left bundle branch block (BBB) are more readily regionalized than are VTs with right BBB.
- The presence of a q wave (followed by an r wave) is a strong indicator of epicardial exit sites in non-ischemic cardiomyopathies.

INTRODUCTION

Before the advent of cardiac mapping techniques, the site of origin or source of ectopic complexes from the heart was a matter of interesting conjecture but lacked relevance. When therapeutic options became available, such as surgical ablation for treatment of Wolff–Parkinson–White syndrome and ventricular tachycardia (VT), mapping tools to locate areas responsible for arrhythmias quickly became important. Among these were the ability to correlate the surface electrocardiogram (ECG) during VT with an area in the heart at which ablation could eradicate it. With surgical ablation using a variety of methods, absolute precision of locating the arrhythmogenic tissue was not critical because these techniques (endocardial resection, extensive cryoablation) were able to remove or otherwise destroy large portions of abnormal tissue. With the subsequent development of catheter

Author Disclosures: Dr J.M. Miller reports having received honoraria from Medtronic, St. Jude Medical, Biotronik, Biosense Webster, Boston Scientific and has been a scientific advisor to Abbott/Topera (modest, <$10,000); Dr R. Jain has received honoraria from Biosense Webster; Dr G. Dandamudi has received honoraria from Medtronic and Biosense Webster; Dr T.R. Kambur has no disclosures to report.

[a] Department of Medicine, Krannert Institute of Cardiology, Indiana University School of Medicine, Indiana University, 1800 North Capitol Avenue, E-488, Indianapolis, IN 46202, USA; [b] Department of Medicine, Krannert Institute of Cardiology, Roudebush VA Medical Center, Indiana University School of Medicine, 1000 West 10th Street, Indianapolis, IN 46202, USA; [c] Department of Medicine, Krannert Institute of Cardiology, Indiana University School of Medicine, 1800 North Capitol Avenue, Indianapolis, IN 46202, USA

* Corresponding author.

E-mail address: jmiller6@iu.edu

Card Electrophysiol Clin ■ (2016) ■–■

http://dx.doi.org/10.1016/j.ccep.2016.10.001

ablation techniques, however, precision of localization was more important owing to the more limited amount of damage that could be effected by catheter as opposed to surgical ablation. In this article, we review the ECG features that indicate exit sites of wavefronts from VT reentrant circuits or foci, with an emphasis on patients with structural heart disease (SHD).

GENERAL PRINCIPLES

In patients without SHD, the QRS morphology during VT correlates well with the site of impulse formation (ie, a focus, causal in the vast majority of patients). In this setting, VT with right bundle branch block (RBBB) pattern (defined here as the latter portion of the QRS in lead V1 being a positive deflection) arise from the left ventricle (LV; thus the right ventricle [RV] is activated last), and left bundle branch block (LBBB) pattern VTs (with a negative deflection as the latter half of lead V1) arise from the RV. VTs with tall R waves in inferior leads arise from the anterior/superior aspects of the ventricles and those with R waves in leads 1 and aVL arise from the RV or septal aspect of the LV, and so forth. However, in the presence of SHD, these straightforward relationships between VT morphology and exit sites may be disrupted. This is because SHD in whatever form—myocardial infarction (MI), idiopathic cardiomyopathy (CM), Chagas disease, sarcoid, etc—is generally accompanied by replacement of some amount of myocardium with scar. This has 2 important effects on the QRS complex: first, myocardium that has been supplanted by scar no longer contributes to the contour of the QRS, and second, scar forms a barrier to propagation from 1 portion of the ventricle to another, slowing conduction and altering the QRS in sometimes unpredictable ways. Thus, the QRS complex during VT in a patient with SHD typically differs from VT exiting from a similar location in a patient without SHD by having (1) a lower QRS amplitude, (2) a wider QRS duration, and (3) notches in the QRS rather than smooth contours, indicating change in direction of the wavefront owing to diversion by scar (**Fig. 1**). It is perhaps surprising that, despite the presence of scar and its influence on conduction patterns in residual myocardium, there remains a reasonably consistent relationship between VT QRS morphology and region of exit to the remaining myocardium.

In contrast with the case with VT in the absence of SHD, most cases of VT in patients with SHD have a reentrant mechanism; thus, the cardiac site that is activated at the time of QRS onset in these VTs corresponds with the exit from a circuit. As noted elsewhere, the exit site or region from a VT circuit is typically wider than the diastolic isthmus (which may be >1 cm away). The latter region, from which mid diastolic potentials are recorded during VT, is a more suitable target for ablation than the exit site in most cases, and although the diastolic corridor is near the exit site, they are not at the same location. Thus, in most cases of SHD, the QRS morphology in VT can direct attention to a region near which an appropriate ablation target site (the diastolic corridor) can be found, but usually not the actual ideal ablation target itself.

As noted, LBBB VTs in patients without SHD arise from the RV, RBBB VTs from the LV; however, in patients with the most common forms of SHD (prior MI and idiopathic CM), whereas RBBB VTs almost always exit from the LV, most LBBB VTs also exit from the LV (septum or <1 cm paraseptal). This seems to be related to more rapid spread of activation to and through the RV, which has less disease, than the LV, which has more scarring.

ISCHEMIC HEART DISEASE/PRIOR MYOCARDIAL INFARCTION

The most common setting in which VT occurs is after a prior extensive MI. Historically, myocardial regions damaged by anterior infarction (left anterior descending artery occlusion, usually proximal or mid vessel) include the apex, anterior, and often apical lateral walls and apical one-half of the interventricular septum. Regions damaged by inferior infarction (right or circumflex coronary occlusions) include the inferobasal free wall with variable extension laterally (to the obtuse margin in many cases) and basal septum. These areas are illustrated in **Fig. 2**. VT exit sites tend to occur within or at the periphery of these infarct regions, which are composed of very dense scar with endocardial sparing and "marbling" through the thinned walls with strands of surviving myocardium. Since the mid 1990s, reperfusion therapy has modified this pattern, resulting in varying degrees of myocardial salvage and thus less scar development. Infarction zones tend to be smaller than before the reperfusion era, but exit sites from VT circuits are generally similar.

The vast majority of VTs in post-MI patients are owing to reentry within endocardial circuits[1,2] (or, at least a portion is accessible on the endocardial surface) and exit sites; this suggests that endocardial catheter ablation should have a high likelihood of successful ablation in these cases. A very small proportion (<5%) of VTs have successful ablation