Before creating a detailed 3-dimensional map of an arrhythmia, the electrophysiologist should already have a reasonable idea regarding which cardiac chamber contains either the focal site of origin of an automatic tachycardia or the relevant substrate for reentry. Typically, a combination of careful review of the ECG during tachycardia and knowledge of the location of abnormal substrate (such as myocardial infarction, atriotomy scar, etc) will allow focused mapping in the appropriate chamber.

Prior teaching points have already discussed\textsuperscript{1,2} the fact that an isolated “early site of activation” is essentially meaningless for guiding ablation of macroreentry. However, in some circumstances, even for a focal “automatic” tachycardia, the apparently early site is not a suitable site to target for ablation.

Figure 1. Activation map of a focal tachycardia ablated in the supravalvar pulmonic area. In each map, colors reflect activation time relative to the QRS with earliest activation shown as red; yellow, green, blue, and purple indicate progressively later activation. A, Activation map of the right ventricle (RV) with earliest activation at the left anterior right ventricular outflow tract (RVOT) at $-97$ ms before the reference time. B, Activation map of a limited area of the left ventricular outflow tract (LVOT) has been added, showing that earliest activation remains in the RVOT at $-97$ ms. C, Activation map of the proximal pulmonary artery (PA) has been added. Earliest activation is now identified in this supravalvar region at $-106$ ms. D, Activation map of the aortic root (AO) is added. There is no further change in the earliest activation point, which remains above the pulmonary valve, identifying the successful ablation site.
After constructing the activation map during tachycardia before ablation, especially if the earliest site of activation obtained does not appear to be particularly early compared with the surface ECG reference, the following should be considered.

**Overlapping Cardiac Structures**

Mapping a chamber other than the actual chamber where the tachycardia originates will clearly lead to a wrong conclusion. For example, when mapping premature ventricular tachycardias or monomorphic ventricular tachycardia in a structurally normal heart, a detailed map of the right ventricular outflow tract may show the “earliest site” of activation to be in the posterior wall of the right ventricular outflow tract. The true site of origin may, however, be in a coronary cusp or the subvalvar anterior left ventricular outflow tract. The posterior right ventricular outflow tract may appear to be early because a far-field signal is detected on the mapping electrode or because of breakthrough in the right ventricular outflow tract from the wave front originating on the other side of the region. After mapping the left ventricle and coronary cusps and combining the maps, the earliest activation will then shift to the appropriate chamber (Figure 1).

Situations in which similar confusion may arise during mapping of atrial tachycardias include apparent early activation of the posterior right atrium with the true early site of activation in the anterior portion of the right upper pulmonary vein. Simultaneous early activation noted near the Bachmann bundle, the fast pathway region, and the anterior mitral annulus may occur when the true early site is a coronary cusp.

**Fascicular Arrhythmia: Purkinje Fibers**

The role of Purkinje fibers in some ventricular arrhythmias is being increasingly recognized. In addition, idiopathic ventricular fibrillation triggered by focal ventricular ectopy is also well recognized. When mapping fascicular arrhythmias, the earliest ventricular site of activation may simply represent the exit site of the activation wave front where it emerges from the Purkinje system. Thus, mapping the earliest site of ventricular activation is similar to identifying the earliest site of ventricular activation during sinus rhythm and fails to identify the true arrhythmogenic substrate. A potential clue to a tachycardia originating in the Purkinje fascicles is an activation map showing simultaneous early ventricular activation at multiple disparate sites. Mapping between these regions looking for discrete Purkinje potentials may allow identification of the true arrhythmia origin. Fascicle-like signals have also been described in the right and left ventricular outflow tracts. Here again, conduction through these fibers (dead-end tracts) may create the impression of an early ventricular site of activation, but ablation at that site probably
will fail because the true origin of the arrhythmia may be at a distance from the ventricular exit.\textsuperscript{4,9}

The Cul-De-Sacs of the Heart
The early site of cardiac activation also requires careful interpretation when the arrhythmia origin is at the interface of a vessel with the heart. Examples include tachycardias originating from the pulmonary veins, the supra-aortic valve cusps, the superior vena cava, the vein of Marshall, and the pulmonary artery. Contributing to the difficulty when mapping near the cul-de-sacs of the heart is that conduction delay is frequently present between the arrhythmia origin and the cardiac chamber. For example, for premature ventricular tachycardias originating above the pulmonary valve, the early ventricular sites may be seen along the valve annulus in the right ventricle that simply represents multiple breakthrough sites across the valve annulus. However, the true early site of origin will be above the valve, and sometimes, the local electrogram at this true early site may be a further 50 to 60 ms ahead of the right ventricular breakthrough. A very small bipolar electrogram at the true origin can escape attention unless a specific targeted detailed supravalvar map is created.

Deep Myocardial or Epicardial Origin
After a detailed endocardial map with sufficient points has been performed for a focal tachycardia, inconsistencies may be noted. For example, the unipolar electrogram at the “early site” does not show a QS complex or is not deemed

Figure 3. Findings from ventricular tachycardia originating from a left ventricular papillary muscle are shown. Color coding as in Figure 1. A, Electroanatomic activation map of tachycardia shows multiple simultaneously early sites in the region of the inferolateral left ventricle. There was a consistent positive initial deflection (r-wave) on the unipolar signal (U) and early far-field signal on the bipolar (B) electrogram. These sites were similar to where prior ablation was performed and failed. B, Dramatic change in the activation sequence was noted when specific mapping of the papillary muscle was undertaken guided by intracardiac echocardiography to define the anatomy. The previously early sites are now late and earliest activation is identified on the papillary muscle itself (red area beneath the turquoise tag). C, “Mesh” representation of the electroanatomic map shows an early activation on an internal point (blue dot) at the endocardial location of the papillary muscle. Without detailed anatomic definition, these endocardial points that appear to be off the plane of the endocardium may be inaccurately considered as intracavitary and deleted from the map. D, Intracardiac ultrasound imaging from the right atrium to verify the location of specific mapping points and catheter contact on the lateral papillary muscle (LPM). E, Intracardiac ultrasound image showing catheter contact during ablation of papillary muscle. Figure from Liu et al, Heart Rhythm. 5:3.
sufficiently ahead of the arrhythmia p-wave or QRS in the surface ECG. In these cases, the possibility of a deep myocardial or epicardial focus should be considered. Deep tissue or epicardial origin should be specifically considered when an early positive far-field electrogram (initial r-wave) is seen in the unipolar recording (Figure 2), when previously reported clues from the 12-lead ECG are present,10,11 and/or when several neighboring sites are found having relatively equally early far-field electrograms. These findings often warrant specific mapping of the adjacent epicardial surface.

Endocavitary Arrhythmia Origin
As discussed above, the 3-dimensional anatomy of the arrhythmia substrate (endocardium, epicardium, and deep myocardium) should be considered when no specific early site is noted endocardially. In some instances, however, no specifically early site for an automatic tachycardia or the complete circuit of a reentrant tachycardia can be identified even with combined epicardial and endocardial mapping. In some of these cases, the endocardial structures serve as a fourth dimension for arrhythmogenesis.12,13 For example, if a focal left ventricular tachycardia is suspected and detailed endocardial and possibly combined epicardial mapping fail to reveal an early site, specific, ultrasound-guided mapping of the papillary muscles may be required to identify the true origin within the papillary muscle extending into the ventricle. Wave fronts from the arrhythmia focus may exit to various myocardial locations with the relative timing dependent on fiber orientation, relative conduction delay, and exact origin of the tachycardia in the papillary muscle14 (Figure 3). Similar issues also may arise for right ventricular tachycardias originating from the moderator band. This anatomy can also complicate mapping of slow zones of conduction constituting part of a scar-related reentry circuit involving the papillary muscle, especially in the setting of prior myocardial infarction.

Issues germane to both fascicular origin of tachycardia and endocardiatary ventricular tachycardia exist with regard to false tendons. These structures are endocardial but may contain Purkinje fibers. Tachycardia origin within a false tendon may give rise to simultaneous early exit on the left ventricular free wall, left ventricular septum, and/or the papillary muscles.13

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None.

References

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