An increasingly used technique for ablation in patients with structural heart disease is substrate mapping. At times, because of the multiplicity of arrhythmia-induced or hemodynamic intolerance in tachycardia, activation or entrainment mapping is not feasible. In these cases, a map of the arrhythmogenic substrate (cataloguing of scar, abnormal myocardium, and normal myocardium during sinus rhythm or a stable arrhythmia) may allow detection of arrhythmogenic channels for the various tachyarrhythmias and can be targeted for ablation1,2 (Figure 1).

The unipolar and bipolar signals derived from mapping not only convey timing information used to construct the activation map, but, in addition, the amplitude of the electrogram (voltage) recorded from mapping catheter in contact with the myocardium reflects the presence of functioning myocardium beneath the electrode. Electrogram voltage is reduced in areas of fibrous scar and scar intermixed with myocytes, which is the substrate for many scar-related arrhythmias.

A substrate map is a compilation of the voltage amplitudes of all contact points with the mapping catheter in a cardiac chamber. A coded color range is assigned that indicates the peak-to-peak amplitude of the signal; for example in Figure 1, red is low voltage, and the voltage progressively increases moving from yellow, to green, to purple. By adjusting this color scale manually, the operator can visualize areas of low voltage and compare these sites with scar and relatively normal myocardium.3,4

The definition of scar myocardial tissue by voltage criteria comes from studies in animal models of healed myocardial infarction5,6 after performing detailed substrate maps and comparing the bipolar electrogram amplitudes characteristics in areas of infarcted myocardium, border zone, and normal myocardium. Subsequently, the infarct characterization by electroanatomic mapping was correlated with histopathologic analyses. Validation studies in humans after high-density electroanatomic endocardiac mapping with cardiac MRI (as the gold standard of scar imaging) have confirmed 1.5 mV as the bipolar amplitude threshold.7 A slightly lower threshold is accepted for endocardial mapping of the right ventricle. For epicardial mapping, the distribution of the subepicardial fat should be taken into account, mainly at the ventricular base and around the distribution of the epicardial coronary arteries.

As described previously, with activation mapping, the position and consistency of gaining and annotating the voltage signals is paramount in creating an accurate and reliable substrate map.

Catheter Contact
Critical to accurate substrate mapping is obtaining adequate electrode-tissue contact when cataloguing the voltages from the derived signals. Combining fluoroscopy, intracardiac echocardiography, and analysis of the near-field components of the signal must be carefully executed to create a valid substrate map.

Bipolar Versus Unipolar Signals
As discussed in a previous segment in this series on activation mapping,3 unipolar signals inherently include substantial far-field electric activation from depolarization of distant tissues and thus are probably not as reliable as bipolar recording for determining the characteristics the tissue in contact with the recording electrode.4 Although bipolar signals reduce the far-field signal components, there are significant causes for error. The actual signal may be recorded from the proximal electrode, which may be in contact with relatively normal myocardium, while the catheter tip itself, which is the anatomic position annotated in the three-dimensional map, may be at a site of scar.

The amplitude of the bipolar signal itself is determined by several factors. The direction of activation of the wave front in relation to the orientation of the distal bipole will determine the amplitude of the signal. Thus, when an activation
wave front is parallel to the recording bipole, the amplitude will be relatively large. This is a desirable scenario for determining viability of myocardial tissue being mapped. On the other hand, when the activation wave front is perpendicular to the bipole, the bipolar signal will be smaller at the same site being mapped and theoretically may be zero. Thus, viable myocardium that is potentially arrhythmogenic may be misconstrued to be scar tissue.

Validation of Scar
An integral part of adequate substrate mapping is appropriate delineation of dense, electrically unexcitable scar versus viable but abnormal myocardium and distinguishing these from normal myocardium. There is no reliable lower voltage limit that distinguishes electrically unexcitable scar from areas of scar intermixed with viable myocardium. Generally, dense scar should be considered present only when no

reliable and reproducible electrogram (seen with each cardiac cycle) is present. Even very small (0.05 mV) near-field signals seen with each cardiac cycle should be considered an indication of viable (but diseased) myocardium that may represent an arrhythmogenic channel.

Furthermore, considering all sites with any signal as viable can result in erroneous labeling of unexcitable scar as viable tissue. This particularly occurs in the ventricle when an infarcted area is close to normal myocardium, possibly on an endocavitary structure (papillary muscle) or a neighboring chamber (Figure 1). The bipolar signal may record electric activation of the neighboring structure lying adjacent and assign the voltage value of that structure to the catalogued catheter contact site (scar).

One method to validate the presence of unexcitable scar uses pacing at the mapping site. True dense fibrous scar tissue devoid of viable myocardium cannot be captured with high pacing output, typically. Thus, pacing at 10 mA or higher, resulting in failure to capture, probably represents unexcitable myocardium and has been used to validate scar tissue.

This technique does have significant limitations. For example, when pacing at high output, surrounding tissue may be captured, and when the catheter lies adjacent to an endocavitary structure such as a papillary muscle, capture of the papillary muscle may mistakenly underdiagnose the underlying scar (Figure 2). Careful assessment of catheter contact, correlation with fluoroscopy and intracardiac echocardiography, correlation of both bipolar and unipolar signals, and finally, checking for overlapping chambers and endocavitary structures are important for creation of accurate substrate maps.

Endocavitary Structure Points Versus Intracavitary Points Caused by Poor Catheter Contact
The role of endocavitary structures such as the papillary muscles in arrhythmogenesis is now well accepted. Therefore, one important differentiation to make during construc-
tion of a substrate map is to determine if a point that looks intracavitary is at the papillary muscle or it is due to lack of catheter contact with myocardium (mapping catheter is “floating” in the cavity). In addition to the use of supporting imaging (catheter movement and location by fluoroscopy, intracardiac echocardiography), analysis of the local bipolar electrogram is also helpful (Figure 3).

**Epicardial Substrate Mapping**

Epicardial substrate mapping generally follows the same principles described above for endocardial mapping. The absence of the equivalent of endocavitary structures and usually easily obtained catheter contact facilitates epicardial substrate mapping. However, an important confounding variable is the presence of epicardial fat (Figure 4). At regions of fat, low-amplitude signals may be recorded, suggesting arrhythmogenic tissue or scar, when the underlying myocardium may actually be normal.

Knowledge of the usual patterns of fat distribution (along the annulus and the anterior and posterior intraventricular grooves) may help exclude this possibility. In addition, analyzing near-field bipolar electrogram amplitudes and judicious use of pacing to document unexcitable underlying tissue before cataloguing scar is particularly relevant in defining epicardial substrate. Although thick regions of fat reduce electrogram amplitude, fat is not expected to cause delayed or fractionated electrograms, which may therefore be a more reliable indication of epicardial arrhythmogenic substrate.

**Disclosures**

None.

**References**


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