Electrocardiographic Recognition of Epicardial Arrhythmias

Steven M. Stevens, MD[1], David Hamon, MD[1], Ricky Yu, MD, Kalyanam Shivkumar, MD, PhD, Noel G. Boyle, MD, PhD*

KEYWORDS

• Epicardial ablation • Ventricular tachycardia • Accessory pathway

KEY POINTS

- Epicardial access for mapping and ablation has increasingly become a feasible modality for treatment of arrhythmias; therefore, the ability to recognize likely epicardial arrhythmias on electrocardiogram (ECG) is important.
- Classic criteria for identifying epicardial ventricular tachycardia (VT) are: (1) the pseudo-δ wave; (2) the intrinsicoid deflection time; and (3) the shortest RS, all of which are based on the initial QRS portion. Additional criteria include the QRS duration, maximum deflection index, and presence of Q in lead I and absence of Q wave in inferior leads for nonischemic substrates with basal lateral VT focus.
- Despite their applicability, ECG criteria for diagnosis of epicardial VT can vary widely, based on differences in underlying cardiomyopathy, ventricular site of origin, tachycardia cycle length, His-Purkinje conduction, and antiarrhythmic therapy.
- There is no single ECG criterion or unique cutoff value reliable enough to diagnose epicardial VT as a stand-alone assessment; therefore, the entire clinical picture must be considered to identify epicardial arrhythmias.
- Applying ECG criteria is one of several steps in considering an epicardial approach for ablation.

INTRODUCTION

Epicardial interventions in electrophysiology date back to the first bypass tract surgery for Wolff-Parkinson-White syndrome in 1969.[1] Nearly 30 years later, in 1996, pericardial access was moved from the operating room to the electrophysiology laboratory, when Sosa and colleagues[2] described percutaneous pericardial access from the subxiphoid space by performing a dry pericardiocentesis. Epicardial interventions are commonly performed for ablation of ventricular tachycardia (VT), reported as 17% of VT ablations at tertiary centers in a survey carried out by the Heart Rhythm Society and European Heart Rhythm Association.[3] Epicardial ventricular arrhythmias are more common in certain populations, such as those with Chagas cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, nonischemic cardiomyopathy, and ischemic cardiomyopathy with inferior scar.[3] In patients without structural heart disease, epicardial VT may originate in the outflow tracts, septum, and the crux of the heart.[4,5] With increasing feasibility of epicardial interventions, it is important that cardiac electrophysiologists are familiar with the electrocardiographic (ECG)

The authors have nothing to disclose.

UCLA Cardiac Arrhythmia Center, UCLA Health System, David Geffen School of Medicine at UCLA, Los Angeles, CA, USA

[1] The two authors contributed equally to this work.

* Corresponding author. David Geffen School of Medicine at UCLA, 100 UCLA Medical Plaza, Suite 660, Los Angeles, CA 90095-1679.

E-mail address: NBoyle@mednet.ucla.edu

Card Electrophysiol Clin 6 (2014) 603–611

http://dx.doi.org/10.1016/j.ccep.2014.05.007

1877-9182/14/$ – see front matter

recognition of tachycardias that may be most amenable to ablation therapy via an epicardial approach.

VENTRICULAR ARRHYTHMIAS

Several criteria have been published to help identify epicardial exit sites for VT. The epicardial surface has slower conduction compared with the endocardium, which contains the His-Purkinje conduction system. The transmural conduction delay from epicardium to endocardium thus leads an initial slurring or delayed upstroke of the QRS. The following 4 general measurement criteria were initially defined in 2004 by Berruezo and colleagues[6] for VTs with a right bundle branch block morphology pattern, in a series of patients with predominantly ischemic cardiomyopathy:

i. The pseudo-δ wave
ii. Intrinsicoid deflection time
iii. The shortest RS complex
iv. The QRS complex duration

These investigators analyzed the ECG patterns for 14 VTs successfully ablated from the epicardium compared with 27 VTs successfully ablated from the endocardium; a third group consisting of 28 additional VTs with unsuccessful endocardial ablation (presumed epicardial focus) were also studied. In addition, these investigators compared the ECG findings for epicardial and endocardial ventricular pacing in 9 patients undergoing cardiac resynchronization. They determined criteria for these parameters to identify epicardial VT, as described later.

Pseudo-δ Wave

The pseudo-δ wave is the interval of the earliest ventricular activation to the onset of the earliest rapid deflection of the QRS in any precordial lead. Berruezo and colleagues[6] reported that a pseudo-δ cutoff of greater than 34 milliseconds is highly suggestive of an epicardial VT focus, with a sensitivity of 83% and a 95% specificity. This parameter was validated by Bazan and colleagues[7] in a study of 19 epicardial VTs in 15 patients (9 with nonischemic cardiomyopathy), who in addition had endocardial and epicardial pace mapping performed at 5 different sites in the left ventricle (LV). These investigators found that the pseudo-δ was significantly longer from the epicardium than endocardium, and that a cutoff value of 34 milliseconds had 96% sensitivity but only 29% specificity for an epicardial focus. The pseudo-δ can be challenging to measure and may have some variability in interpretation, because onset of the QRS and the first sharp deflection in the precordial leads may be difficult to define. In such cases, other criteria should be used.

Differences between studies may be explained by the nature and the severity of the underlying cardiomyopathy. In cases of previous myocardial infarction, transmural activation time is highly influenced in the scar region. Moreover, endocardial conduction is slower near the scar region and can mimic a pseudo-δ wave. In addition, the specific cardiac region of VT or pacing is probably a critical determinant of this heterogeneity. Bazan and colleagues showed that the pacing site location dramatically influences the ability of those criteria to predict epicardial origin. A pseudo-δ wave of 34 milliseconds or greater was present in almost 40% of patients with endocardial apical inferior pacing, and this increased to 85% when pacing was performed at an endocardial basal inferior site. The percentages were comparable for both regions in epicardial pacing, at greater than 90%. Thus, specificity decreased as the pacing site was changed from apical toward basal regions, which was reproducible with other criteria. This finding may indicate an initial slower conduction in basal rather than apical regions, reflecting the common pattern of predominantly basal fibrosis gradient in nonischemic cardiomyopathy[8] and the distance to the main stems of the His-Purkinje system. Vallès and colleagues[9] reviewed epicardial and endocardial pace maps from the basal superior lateral region in patients with nonischemic cardiomyopathy and VT and then revised the interval criteria, choosing cutoffs that were able to achieve a high specificity of 95% or greater and sensitivity of 20% or greater (**Figs. 1** and **2**).

Intrinsicoid Deflection Time

The intrinsicoid deflection time is the interval from the onset of QRS to the peak of the R wave in lead V2. Berruezo and colleagues[6] found that greater than 85 milliseconds indicates epicardial VT, with 87% sensitivity and 90% specificity. However, in the validation study by Bazan and colleagues,[7] this criterion was found to have low sensitivity (39%) and specificity (24%) values. Again, this lack of reliability may be explained by the integration of all sites of stimulation in the study, because this criterion or cutoff was not appropriate for apical sites of stimulation. In all apical sites, 20% or fewer values were 85 milliseconds or greater during epicardial pacing. Unlike in the earlier study of this group, Vallès and colleagues,[9] in their homogeneous group of patients with dilated cardiomyopathy, paced only in basal superior lateral sites. The same cutoff showed a relatively good