The principles of using the P wave or QRS to map the site of origin for an automatic tachycardia or the site of early ventricular activation from an accessory pathway can be deduced in a fairly straightforward manner by analyzing the routine electrocardiographic leads. For example, during ventricular tachycardia, a complete negative (QS complex) in lead I strongly suggests origin in the left side of the body and further often signifies epicardial origin because there is no vector of activation that proceeds toward the left shoulder. Using all leads available can usually localize the site of arrhythmia origin to a fairly localized area of atrial or ventricular myocardium.

ECG and Reentry

The principles for ECG localization of an arrhythmia include analyzing the axis, timing of the intrinsicoid deflection, and identifying a lead where the inscribed deflection is completely negative. These analyses are equally relevant when analyzing the P wave of atrial arrhythmias and the QRS complex of ventricular arrhythmias.

In general, identifying the site of origin for an automatic tachycardia or the site of early ventricular activation from an accessory pathway can be deduced in a fairly straightforward manner by analyzing the routine electrocardiographic leads. For example, during ventricular tachycardia, a complete negative (QS complex) in lead I strongly suggests origin in the left side of the body and further often signifies epicardial origin because there is no vector of activation that proceeds toward the left shoulder. Using all leads available can usually localize the site of arrhythmia origin to a fairly localized area of atrial or ventricular myocardium.

Exit Site Versus Focus of Activation

The presumed determinants of the electrocardiographic pattern and reentry primarily involve the transition point between diseased myocardium housing the reentrant circuit and the remainder of what is often more normal myocardium. This transition is usually referred to as the exit site of a reentrant tachycardia. The second determinant is the bystander activation sequence of the remainder of the cardiac chamber (atrium of ventricle). Taking an approach similar to analyzing the electrocardiographic vector for focal source tachycardia, operators use the ECG to estimate the exit site and then, mapping proximate tissue to the likely exit site, identify areas of slow conduction that allow focused activation and entrainment mapping to identify ablation targets. Although this approach is relatively commonplace for ventricular tachycardia, mapping reentrant atrial flutters in postmaze or postablation atria is more complex because the remainder of the atria has unpredictable patterns of activation from previous scars.

Relative Activation

Intracardiac activation, either from point-to-point mapping or, as explained by Shehata et al, using multielectrode catheters, should reflect and provide additional information to P wave (or QRS) analysis. Activation, for example, away from the interatrial septum at the Bachmann bundle region, proceeding sequentially to later sites on the right atrial free wall, would strongly suggest that the entire circuit is in the left atrium. More subtle deductions can be made by comparing the activation sequence at various sites in the chamber of interest to define and correlate with the surface ECG wave broadly whether a mitral isthmus, atrial septal, or left atrial roof-dependent macroreentry circuit is present.
Relevance of the Isoelectric Period on Surface ECG

For reentry to occur, conduction must be sufficiently slow at ≥1 points through the circuit and the circuit must be large enough to allow reentrant conduction after the initially activated tissue has recovered from refractoriness. With large circuit reentry in the atrium, the classic flutter wave with no true isoelectric interval is produced. Visually, it can be difficult to identify a discrete starting point, and a sine-wave–like pattern is produced. However, if a critical region in the circuit or the entire circuit exhibits slow conduction, then a reentrant circuit can be established that involves a relatively small amount of myocardium and is termed microreentry. The smaller the circuit, the more likely an isoelectric interval and less likely a continuous wave pattern is seen on the ECG. With microreentry, analysis of the ECG vector similar to point-source tachycardia can be done and ablation at the anticipated site of origin is often successful. Unlike a focal source tachycardia, however, the electrograms are abnormal showing fragmentation or other evidence of slow conduction at the earliest site of activation that corresponds to the site of origin predicted by surface ECG analysis. Thus, the relative value for P wave analysis (or QRS for ventricular tachycardia) depends on the size of the circuit and is likely most reliable for microreentrant arrhythmias.

Complex Circuits

After a cardiac maze procedure or extensive ablation for atrial arrhythmia, complex atrial reentry circuits may be present. These may involve 2 loops of reentry: figure-of-eight reentry or a primary reentrant circuit with multiple bystander loops. One or more of the components of a complex circuit may be absent at various times during continued initial tachycardia (entrance block or entrainment of a secondary circuit, etc). In these circumstances, the activation sequence remote from the circuit, and hence the P wave morphology, can change while the reentry circuit remains stable and the sole driver of the tachycardia.

Adjunctive and Additive Information Synthesis

Although P wave analysis for reentrant arrhythmia is necessarily complex and the interpreting electrophysiologist needs to be aware of the nuances elegantly brought forward by Shehata et al., the information provided can be valuable. Activation mapping is also complex in the diseased heart with the potential for multiple bystander circuits, and dead end pathways, and it is the rare patient where we can visually identify the primary circuit in these cases from the ECG alone. Similarly, entrainment mapping is also difficult with arrhythmias that may change or alter activation of bystander loops during overdrive pacing attempts, potentially leading to misdiagnosis. A careful and ordered interpretation sequence beginning with the surface ECG then followed by analyzing the activation sequences in the coronary sinus right atrium and other sites in the left atrium, as explained by Shehata et al., along with activation mapping information and targeted entrainment data, maximizes the success for patients with these difficult arrhythmias.

Disclosures

Dr Asirvatham receives no significant honoraria and is a consultant with Abiomed, Atricure, Biosense Webster, Biotronik, Boston Scientific, Medtronic, Spectranetics, St. Jude, Sanofi-Aventis, Wolters Kluwer, Elsevier. Dr Stevenson is coholder of a patent on needle ablation that is consigned to Brigham and Women’s Hospital.

References


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Samuel J. Asirvatham and William G. Stevenson

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