Idiopathic Ventricular Outflow Tract Arrhythmias: Avoid the Use of a Sledgehammer to Crack a Nut

Roy John[1] and Alexander Perino[1]

[1]Stanford University School of Medicine

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Abstract

Ventricular outflow is a common site for idiopathic PVCs and repetitive ventricular arrhythmias. Sites of origin of these arrhythmias may vary from the sites of earliest activation mapped. Better definition of the site of origin can help avoid unnecessary large volume ablation to suppress these arrhythmia.

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Roy M. John, MBBS, PhD

Alexander C. Perino, MD

Department of Medicine

Stanford University School of Medicine

Palo Alto, CA 94304

Address for correspondence:

Roy M. John, MD, PhD

Stanford School of Medicine

Center for Academic Medicine

453 Quarry Road Room 335

Palo Alto, CA 94304

Email: royjohn@stanford.edu

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The right (RV) and left ventricular (LV) outflow tracts (OT) are common sources of premature ventricular contractions (PVCs) and repetitive ventricular tachycardia (VT) in structurally normal hearts. Most of these arrhythmias (approximately, 70%) are mapped to early sites just beneath the pulmonary valves in the

RV outflow tract. On the left ventricular side, sites of successful ablation are more widely distributed. It spans a course extending from the aortic sinuses, interleaflet triangles usually between the left and right valve leaflets, the infra-aortic area on the endocardium to the epicardial region beneath the coronary venous system overlying the LV summit. Successful suppression of arrhythmia can be achieved by application of radiofrequency energy in a variety of locations along this course, occasionally, well away from the site of recorded earliest activation. More recently, distal coronary venous systems have been targeted with alcohol to ablate the LV summit region.[1]

The exact sites of origin of these arrhythmias, particularly left sided OTs, are therefore controversial. It is likely that the majority originate in strands of muscles that course from the interleaflet triangle and adjacent areas of ventricular-arterial (V-A) junction and insert into the ventricular summit. The myocardial network in the OTs is complex.[2] Exit sites of triggered activity generated by these muscle strands are governed by fiber orientation and fibrous insulation from surrounding myocardium. The premise of insulation is supported by the fact that during sinus rhythm, discrete potentials with an isoelectric delay can be recorded from the aortic sinuses.[3] During PVCs, these potentials precede ventricular activation by 30-40ms (figure 1) and ablation is usually successful in the region. However, achieving stability in the LV outflow interleaflet triangle to record consistent high frequency signals is difficult. In addition, conventional ablation catheters with a 3.5 mm tip electrode may not have the resolution to discern these signals. Often, mapping in the distal great cardiac vein or anterior interventricular branch of the coronary sinus may record early electrograms (-30ms or more presystolic). Despite such early activation, arrhythmia suppression can still be achieved by ablation at the immediate infra-aortic region by interrupting the course of the muscle fibers (figure 2). The immediate infra-aortic V-A junction is relatively thin, measuring 5mm, and allow for transmural ablation.[4]

Pre-procedural determination of site of origin between the right and left ventricles is helpful in assessment of risks and benefits of intervention and procedural planning. In general, most PVCs or VT with left bundle branch block morphology with precordial transition (the first precordial lead with R/S ratio >1.0) no earlier than lead V4 and inferiorly directed axis, have an RVOT origin. Precordial transition earlier than V3 suggests an LVOT origin. To account for cardiac rotation, precordial R/S transition during sinus rhythm can be compared with that of the arrhythmia. In LVOT arrhythmias, precordial transition tends to occur in the same or earlier precordial lead than in sinus rhythm, whereas it occurs later in RVOT arrhythmias. Transition in V3, particularly abrupt transition between V2 and V3, is highly indicative of an origin from the interleaflet triangle between the left and right aortic cusps with LV summit exit. [4,5]However, the complex anatomy of the OT and variable exits that are often distinct from origins, preclude precise localization from ECG alone and sequential mapping of the RVOT, coronary sinus, aortic root and LVOT is often necessary for precise definition. Other features such as variability of coupling interval of the PVCs has been proposed as a distinguishing feature. Given the more complex course of LV outflow myofibers, greater variability of coupling interval (> 60 ms variation) is observed in arrhythmias that emerge out of the aortic sinuses.[6]

In this issue of the Journal, Waight et al. present a study of analysis of Holter monitors to assess hourly variability in OT ectopy as the predictor of site of origin of OT arrhythmias in structurally normal hearts.[7] The gold standard for site of origin was successful suppression with ablation. In a derivative cohort of 40 patients, a coefficient of variation of hourly PVCs >0.7 and the presence of any hour with <50 PVCs were found to be predictive of an RVOT site of origin. In a validation cohort of 29 patients, these parameters prospectively identified the site of origin in close to 80 to 90% of patients. Any hour with <50 PVCs, offered the highest probability of the arrhythmia being of RVOT origin (Youden Index of 78). Arrhythmias of LVOT origin, on the other hand, had a more even hourly distribution. RVOT PVCs had the highest variability in the hours between and 0600 and 1200 hours. These are interesting and novel observations in keeping with the known disproportionate variability in work-load on the RV compared to the LV with rest and exercise. Limitations of the study should include ambulatory monitoring limited to only 24 hours. PVCs demonstrate substantial daily variation. Recent studies have suggested greater accuracy in detection of PVC burden with 6-7 days of monitoring.[8] In addition, the terminology "site of origin" is not strictly applicable as site of successful ablation can be anywhere along the course of a conducting muscle fiber and not necessarily the site of origin.

The authors offer variability in parasympathetic and sympathetic activation as potential mechanism for RVOT PVC variability. An additional mechanism worth investigation may be the influence of mechanical stretch on frequency of triggered automaticity, the proposed mechanism for outflow arrhythmias in normal hearts. Compared to the LV, the RV mechanical response to rest and exercise has far greater variability. Mechanical myocardial stretch during diastole is known to generate triggered activity and PVCs.[9]

Given the growing importance of PVC and repetitive VT on ventricular function, the current paper adds significantly to the gross differentiation between the sites of origin. What needs better definition is the exact anatomical relationship between the origins and exits points for these arrhythmias such that indiscriminate and large volume ablation of ventricular muscle tissue can be avoided. We shouldn't have to use a sledgehammer to crack a nut.

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Figure legends:

Figure 1: Delayed activation recorded in left coronary sinus during PVC. The electrograms marked by the blue arrows represent delayed activation of local muscle bundle during sinus rhythm. The delay is likely due to insulation by surrounding fibrous tissue of the ventriculo-aortic junction. The more rounded early electrogram that correspond in timing to the QRS is the electrogram generated by activation of larger muscle of the LV summit. During PVC (second complex in the trace), this electrogram represents earliest activation (black arrow, 40ms pre-systolic) suggesting site of origin of the PVC. PVC = premature ventricular contraction.

Figure 2: Electroanatomic map in the left anterior oblique view with caudal tilt of the ventricular outflow region showing the RV outflow, aortic leaflets and coronary sinus. Earliest activation (red, early; purple, late) is registered in the distal great cardiac vein overlying the LV summit. Ablation suppressed PVCs in the infra-aortic region (blue solid circle) below the commissure of the right and left aortic valve leaflets where electrograms were indistinct. RCC = Right coronary cusp; LCC = left coronary cusp; NCC = non coronary cusp.

Figure 1

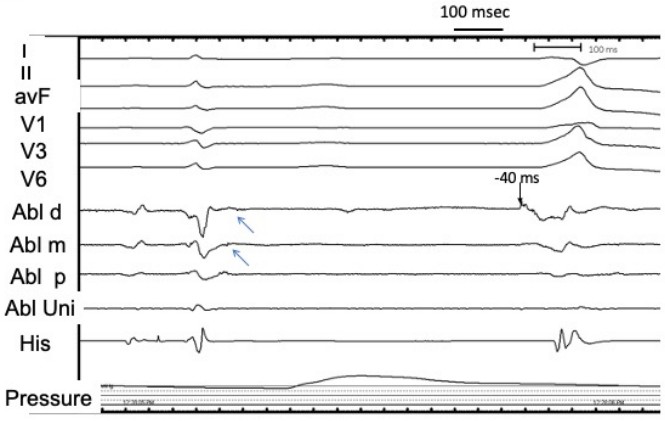

Figure 2