Posted on Authorea 1 Apr 2022 — The copyright holder is the author/funder. All rights reserved. No reuse without permission. — https://doi.org/10.22541/au.164882013.31335659/v1 — This a preprint and has not been peer reviewed. Data may be preliminary.

Don't Forget the Sinuses: an important site for some infarct-related VT

Gregory E. Supple[1]

[1]University of Pennsylvania

April 1, 2022

Abstract

The aortic sinuses of Valsalva are an important ablation site in non-ischemic substrates and in patients with idiopathic ventricular arrhythmias. Siontis and colleagues have demonstrated that these sites should also be considered for ablation in patients with infarct-related inferior axis VT. Low voltage in the aortic sinuses of Valsalva or in the sub-aortic region should prompt further evaluation of these regions for ablation.

Title:

Don't Forget the Sinuses: an important site for some infarct-related VT

Author Name:

Gregory E. Supple, MD

Author Affiliations:

University of Pennsylvania Perelman School of Medicine, Philadelphia, PA

Corresponding author:

Gregory E. Supple, MD

Corresponding author contact information:

Email: gregory.supple@pennmedicine.upenn.edu

Phone: 215-615-3811

Fax: 215-662-2879

Address: Founders 9

3400 Spruce St.

Philadelphia, PA 19104

Funding: none

Disclosures: none

Subject terms:

VT ablation, aortic sinuses of Valsalva, ischemic cardiomyopathy

Keywords:

Posted on Authorea 1 Apr 2022 — The copyright holder is the author/funder. All rights reserved. No reuse without permission. — https://doi.org/10.22541/au.164882013.31335659/v1 — This a preprint and has not been peer reviewed. Data may be preliminary.

VT ablation, aortic sinuses of Valsalva, ischemic cardiomyopathy, aortic cusps, ventricular tachycardia

Abstract:

The aortic sinuses of Valsalva are an important ablation site in non-ischemic substrates and in patients with idiopathic ventricular arrhythmias. Siontis and colleagues have demonstrated that these sites should also be considered for ablation in patients with infarct-related inferior axis VT. Low voltage in the aortic sinuses of Valsalva or in the sub-aortic region should prompt further evaluation of these regions for ablation.

Catheter ablation of ventricular tachycardia from the aortic sinuses of Valsalva (SoV) has been performed for about 20 years. It has been recognized as an important site to access both idiopathic ventricular arrhythmias (VA) from the region of the outflow tracts and LV summit[1-3], as well as myocardial substrate for reentrant VA in patients with dilated non-ischemic cardiomyopathy (NICM) and scarring in the basal septum and peri-aortic and mitral valve regions[4]. From within the aortic cusps, we may be accessing myocardial extensions to the valvular plane[5], or the adjacent myocardial tissue of the LV ostium[6]. While it is important to avoid injury to the ostia of the coronary arteries and aortic cusps, we have learned that ablation of the myocardial tissue can be performed safely through the valve tissue without resulting in valvular dysfunction, and when the ablation is performed in areas in close proximity to the myocardium, this is typically a safe distance from the coronary arteries to avoid damage—either in the base of the cusp, well below the ostium; or higher in the commissures or junction of the cusps.

Ablation of idiopathic VA and non-ischemic substrate from within the SoV has expanded over the years as we have learned to safely perform ablation within these regions, supported by electroanatomic mapping and intracardiac echocardiography to better visualize and understand the relevant anatomy being targeted. While the sinuses have become a common site of evaluation and ablation for idiopathic and non-ischemic substrates, it has not previously been identified as a common site for ablation of ventricular tachycardia in patients with infarct-related ventricular tachycardia (VT). In this issue of the *Journal of Cardiovascular Electrophysiology* , Siontis and colleagues report on the findings of a cohort of patients with ischemic cardiomyopathy and VT ablated from the sinuses of Valsalva, comparing them to a cohort of patients with idiopathic VT ablated from the sinuses as well as patients with ischemic VT without VT originating from the sinuses[7]. This cohort represented patients from a single referral center for VT ablation: 13 patients were identified based on successful ablation of ventricular tachycardia from an aortic sinus of Valsalva, from a series of 217 consecutive patients with post-infarction VT undergoing ablation at their center between 2006 to 2018. These 13 patients were compared to two cohorts of randomly selection patients with 1. idiopathic ventricular arrhythmias and 2. infarct related VT without ablation performed in the sinuses of Valsalva.

The study identified several notable findings. First, when compared to their reference cohort of infarct-related VT ablation patients, the patients with VT ablated from the SoV were more likely to present as repeat ablation, suggesting that this important substrate may have been missed during an initial VT ablation. During these procedures, these patients also had a lower number of induced VTs which is unsurprising if more of them were repeat ablations.

Second, the QRS morphology of the VT in the study group was frequently different compared to the classic morphologies that have been described to localize site of origin of idiopathic VT ablated from the sinuses of Valsalva. This is likely due to a combination of factors: in the setting of significant substrate, the VT isthmus is frequently a significant distance from the exit, thereby generating a morphology that would be predicted to be remote from the aortic cusps. Additionally, just as significant scarring and substrate alters the normal sinus QRS morphology, VT morphology can similarly be affected. These unexpected morphologies may help explain why the SoV represent an underassessed substrate in infarct-related VT patients. The morphologies of these VTs are provided in the first figure in the article, and the authors suggest based on their findings that with any inferior axis VT, mapping in the SoV should be considered even when the precordial transition is later than would be typically expected.

Third, some of these patients had infarct scar that was contiguous with the subaortic valve region, but some had an isolated scar in this region. Their second figure demonstrates an example of each type. Similarly,

Posted on Authorea 1 Apr 2022 — The copyright holder is the author/funder. All rights reserved. No reuse without permission. — https://doi.org/10.22541/au.164882013.31335659/v1 — This a preprint and has not been peer reviewed. Data may be preliminary.

while the median bipolar voltage in the SoV in patients with idiopathic VA was 1.06 mV, in the infarct patients the median voltage was 0.33 mV. While normal bipolar voltage range in the SoV has not been previously reported in large series, these data suggest that while normal may be lower than the standard >1.5 mV bipolar voltage noted in normal ventricular myocardium, patients with scar or substrate adjacent to the SoV still result in lower voltage than would be seen in healthy patients.

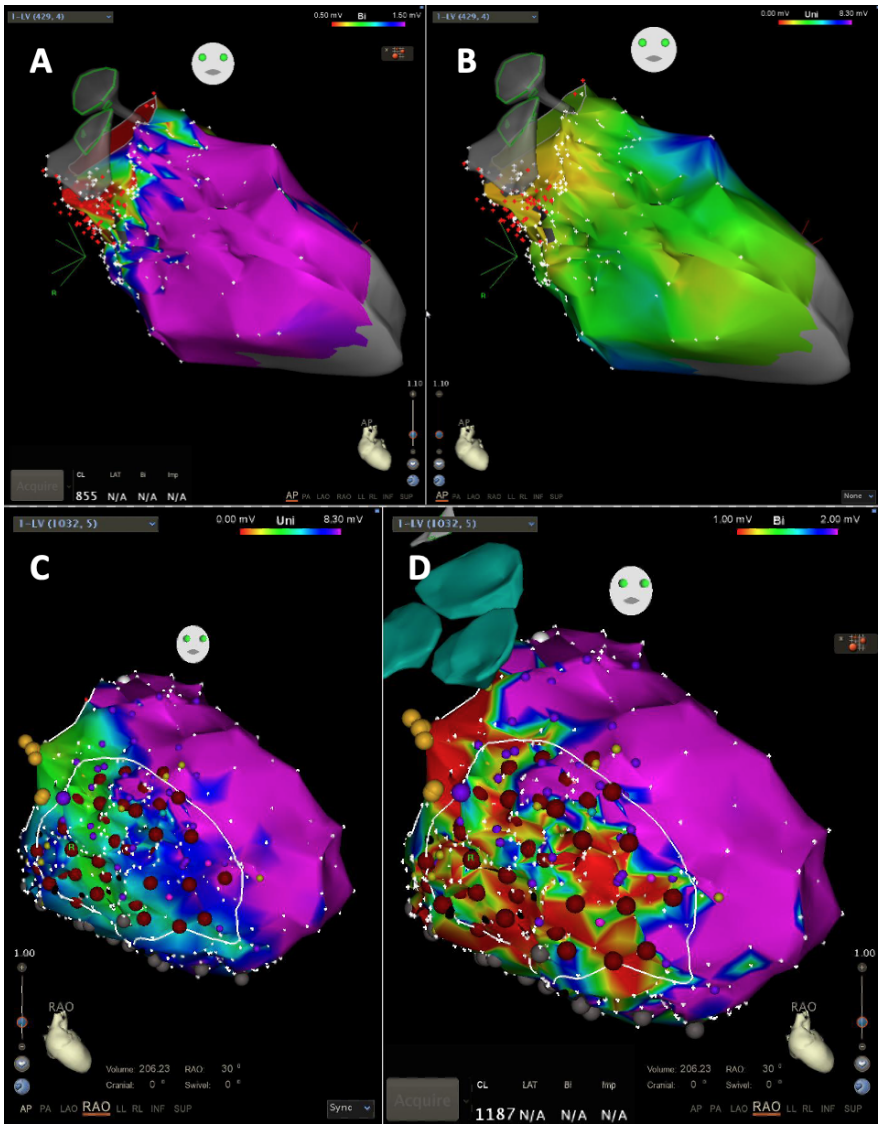
An important question that remains to be answered is if the VT substrate in these study patients is related to an infarct, or if this represents a mixed cardiomyopathy with non-ischemic substrate responsible for these VTs. Reports on the incidence of combined non-ischemic and ischemic cardiomyopathy substrate are variable. A prior study from our center identified 1.2% (9 of 732) patients with prior infarction who had non-ischemic substrate and VTs at the time of ablation. All these patients had VT from basal peri-valvular substrate, however only 4 of the 9 were from the LV outflow tract or SoV[8]. In distinction, some prior MRI imaging work has identified up to 19% prevalence of a nonischemic scar pattern in patients who had ischemic cardiomyopathy[9]. MRI and late gadolinium enhancement-based series have previously been conducted to differentiate between ischemic cardiomyopathy and NICM, and these studies have reported up to 13% prevalence of subendocardial and/or transmural scar pattern in patients without coronary artery stenosis that was indistinguishable from patients with CAD[10,11]. Alternatively, some patients with CAD may have mid-wall late gadolinium enhancement consistent with NICM[9,11]. Other studies monitoring for development of ischemic disease in NICM identified 4% of 139 patients over three years who developed MI or ischemic disease requiring revascularization. These mixed results highlight the challenges in understanding and identifying when patients may have both ischemic and non-ischemic substrate responsible for ventricular tachycardia.

In this study, the authors point out that the SoVs are known to have vasculature (conus artery branches to the right SoV and early septal perforators for the left) which may result in extension of infarction to these regions, particularly when it is contiguous with other infarction territory. Conversely, in patients with peri-annular substrate that is distinct from the infarct scar, this may be more likely non-ischemic. Unipolar voltage mapping in such cases may help determine if there are more prominent abnormalities compared to bipolar mapping, which is often suggestive of mid-myocardial scar (Figure 1).

Regardless of the cause of the substrate, Siontis and colleagues are to be commended for this work which highlights an entity that may be underrecognized for a small subset of patients with ischemic VT undergoing ablation. While it is relatively straightforward to sample voltage and pace from the SoV during ablation with retrograde aortic access, this is more challenging if a transseptal approach is utilized. Nonetheless, low voltage in the SoV or inferior axis VTs in ischemic cardiomyopathy patients should prompt the electrophysiologist to consider if ablation is warranted in the SoV. Pattern recognition of VT morphology and substrate location often helps guide a VT ablation, and this information should be added to the patterns we are watching for in the electrophysiology lab.

Figure 1: Top, a patient with non-ischemic substrate and a small basal septal bipolar scar (A), with much more prominent and diffuse unipolar voltage abnormality of the entire LV septum (B); Bottom, a patient with septal myocardial infarction and a unipolar voltage abnormality (C) that closely mirrors the bipolar scar (D).

Posted on Authorea 1 Apr 2022 — The copyright holder is the author/funder. All rights reserved. No reuse without permission. — https://doi.org/10.22541/au.164882013.31335659/v1 — This a preprint and has not been peer reviewed. Data may be preliminary.



References

1. Ouyang F, Fotuhi P, Ho SY, et al. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: electrocardiographic characterization for guiding catheter ablation.*J Am Coll Cardiol.* 2002;39(3):500-508.
2. Kanagaratnam L, Tomassoni G, Schweikert R, et al. Ventricular tachycardias arising from the aortic sinus of valsalva: an under-recognized variant of left outflow tract ventricular tachycardia. *J Am Coll Cardiol.* 2001;37(5):1408-1414.
3. Yamada T, McElderry HT, Doppalapudi H, et al. Idiopathic ventricular arrhythmias originating from the aortic root prevalence, electrocardiographic and electrophysiologic characteristics, and results of radiofrequency catheter ablation. *J Am Coll Cardiol.*2008;52(2):139-147.
4. Muser D, Santangeli P, Castro SA, et al. Long-Term Outcome After Catheter Ablation of Ventricular Tachycardia in Patients With Nonischemic Dilated Cardiomyopathy. *Circ Arrhythm Electrophysiol.* 2016;9(10).
5. Hasdemir C, Aktas S, Govsa F, et al. Demonstration of ventricular myocardial extensions into

Posted on Authorea 1 Apr 2022 — The copyright holder is the author/funder. All rights reserved. No reuse without permission. — https://doi.org/10.22541/au.164882013.31335659/v1 — This a preprint and has not been peer reviewed. Data may be preliminary.

the pulmonary artery and aorta beyond the ventriculo-arterial junction. *Pacing Clin Electrophysiol.*2007;30(4):534-539.

6. Yamada T, Litovsky SH, Kay GN. The left ventricular ostium: an anatomic concept relevant to idiopathic ventricular arrhythmias.*Circ Arrhythm Electrophysiol.* 2008;1(5):396-404.
7. Siontis KC, Njeim M, Dabbagh GS et al. Ventricular Tachycardia Targeted in the Aortic Sinuses of Valsalva in Patients with Prior Myocardial Infarction. *J Card Electrophysiol.* 2022.
8. Aldhoon B, Tzou WS, Riley MP et al. Nonischemic cardiomyopathy substrate and ventricular tachycardia in the setting of coronary artery disease. *Heart Rhythm* . 2013 Nov;10(11):1622-7.
9. Gao P, Yee R, Gula L, Krahn AD, et al. Prediction of arrhythmic events in ischemic and dilated cardiomyopathy patients referred for implantable cardiac defibrillator: evaluation of multiple scar quantification measures for late gadolinium enhancement magnetic resonance imaging. *Circ Cardiovasc Imaging* . 2012 Jul;5(4):448-56.
10. Soriano CJ, Ridocci F, Estornell J, et al. Noninvasive diagnosis of coronary artery disease in patients with heart failure and systolic dysfunction of uncertain etiology, using late gadolinium-enhanced cardiovascular magnetic resonance. *J Am Coll Cardiol*2005;45:743–748.
11. Bello D, Shah DJ, Farah GM, et al. Gadolinium cardiovascular magnetic resonance predicts reversible myocardial dysfunction and remodeling in patients with heart failure undergoing beta-blocker therapy.*Circulation* 2003;108: 1945–1953.

Posted on Authorea 1 Apr 2022 — The copyright holder is the author/funder. All rights reserved. No reuse without permission. — https://doi.org/10.22541/au.164882013.31335659/v1 — This a preprint and has not been peer reviewed. Data may be preliminary.