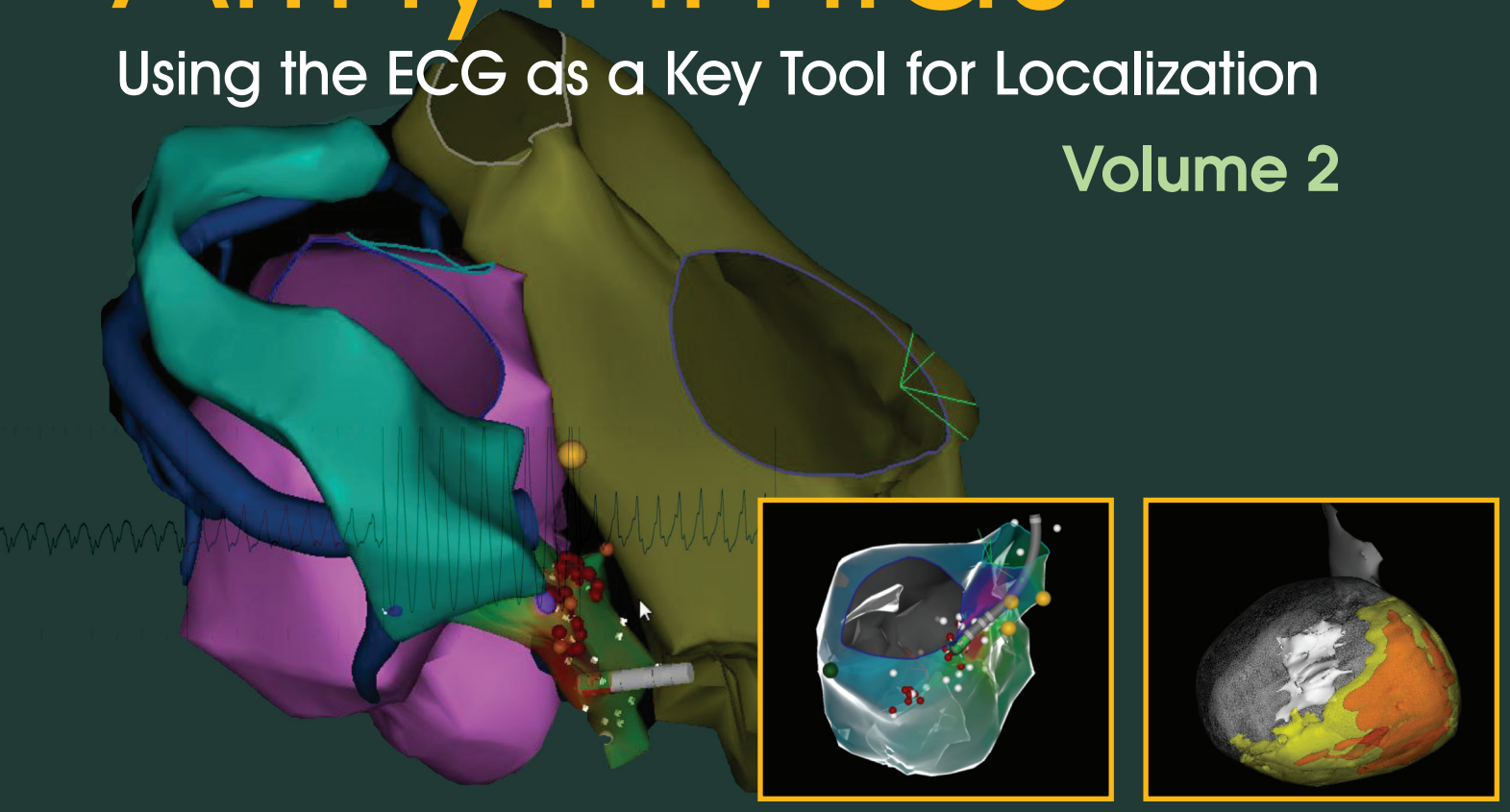
THE ORIGINS OF Ventricular Arrhythmias

Using the ECG as a Key Tool for Localization

Volume 2



Frank M. Bogun, MD

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PREFACE

The first volume of these books highlighted the value of the 12-lead ECG for the localization of sites of origin or exit sites of ventricular arrhythmias. My intent was to make electrophysiology fellows in training aware of certain ECG patterns that may help to localize the origin of arrhythmias. For this second volume, I chose to also emphasize the anatomic relationship of arrhythmia origins. Therefore, the tracings are complemented by cardiac magnetic resonance imaging and real-time echocardiographic data, in order to help visualize the actual location of ablation target sites for each case. The value of appropriate imaging in addition to the 12-lead ECG is highlighted throughout. Many of these cases were challenging; the patients had failed ablation procedures elsewhere, and therefore the approach taken by the operator to reach a particular area may be of particular interest to the reader. I hope that this book will aid in the understanding of the localizing value of the 12-lead ECG for ventricular arrhythmias, which is key for successfully targeting these arrhythmias using catheter ablation.

This book presents the cases as a series of "unknowns." For the reader interested in reviewing a specific type of case, or the answers to the questions, please note that there is an appendix provided at the end of the book that identifies the origin of the VT or PVC.

ABBREVIATIONS

AMC	aortomitral continuity
Ao	aortic
ASD	atrial septal defect
AVI	anterior interventricular vein
BBRVT	bundle branch reentry VT
CMP	cardiomyopathy
CMR	cardiac magnetic resonance imaging
CVS	coronary venous system
EAM	electroanatomic map
EF	ejection fraction
EGM	electrogram
GCV	great cardiac vein
HPS	His-Purkinje fiber system
HRA	high right atrium
IA	inferior axis
IHD	ischemic heart disease
LAD	left anterior descending artery
LBBB	left bundle branch block
LBBBIA	left bundle branch block inferior axis
LM	left main coronary artery
LVEF	left ventricular ejection fraction
LVOT	left ventricular outflow tract
MA	mitral annulus/annular
MB	moderator band
MCV	middle cardiac vein
MVA	mitral valve annulus
NICM	nonischemic cardiomyopathy
NSVT	nonsustained VT
NYHA class	New York Heart Association functional class
PA	pulmonary artery, pulmonic annulus
PAP	papillary muscle
PVC	premature ventricular complex
RBBB	right bundle branch block
RF	radiofrequency
RVA	right ventricular apex
RVOT	right ventricular outflow tract
S-QRS	stimulus-QRS interval

SA	superior axis
SV	sinus of Valsalva
TVA	tricuspid valve annulus
VA	ventricular arrhythmia

GLOSSARY

Pace mapping: Pacing from the catheter tip of the mapping catheter aiming to replicate the morphology of a targeted ventricular arrhythmia. For reentrant VTs, a matching pace map indicates an exit site and for idiopathic VAs or focal arrhythmias, it indicates the site of origin.

CASE 1

CLINICAL HISTORY

A 58-year-old man presented with frequent premature ventricular complexes (PVCs). His PVC burden was 22% and his left ventricular ejection fraction (LVEF) was 45%. Cardiac magnetic resonance imaging (CMR) showed an intramural scar in the anterobasal septum. His PVCs are shown in **Figure 1-1**. He also had easily inducible VT with the morphology shown in **Figure 1-2**.

Questions

What is the origin of the PVCs and the VT? What should be done about the VT?

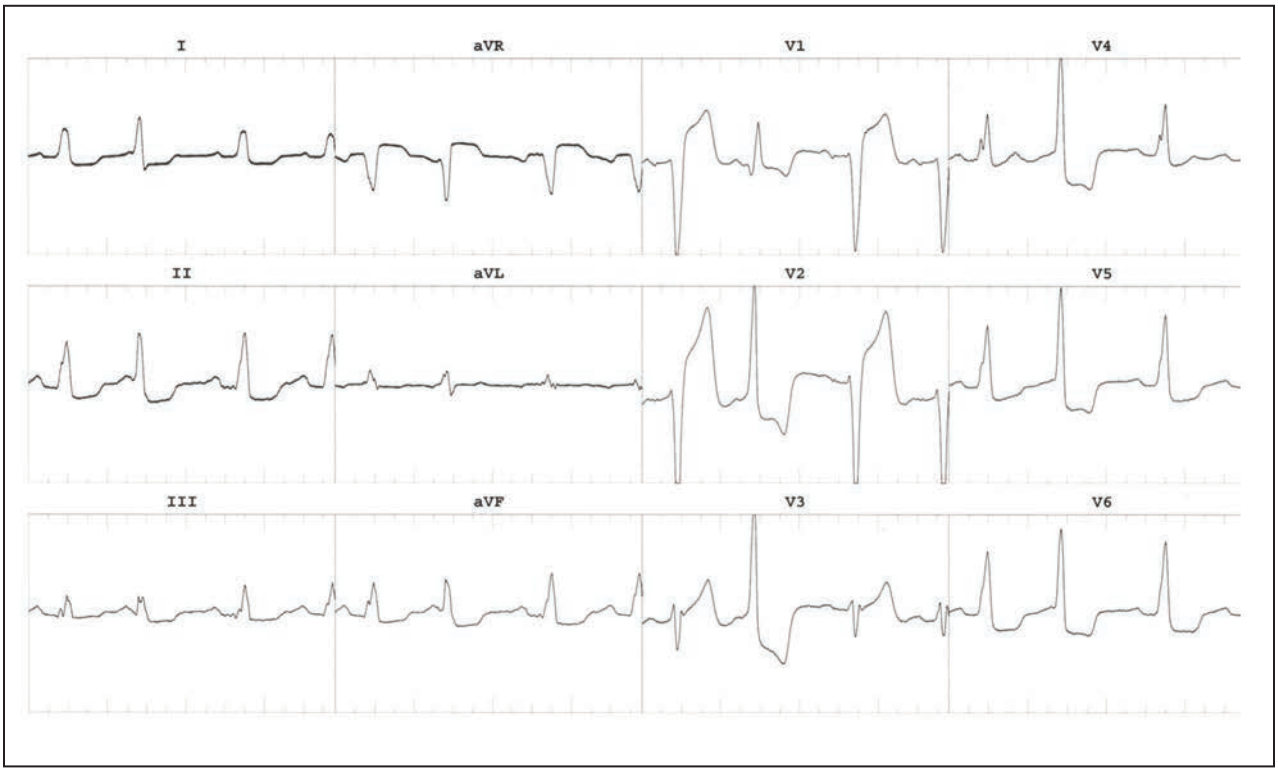

Figure 1-1 A 12-lead ECG of PVC.

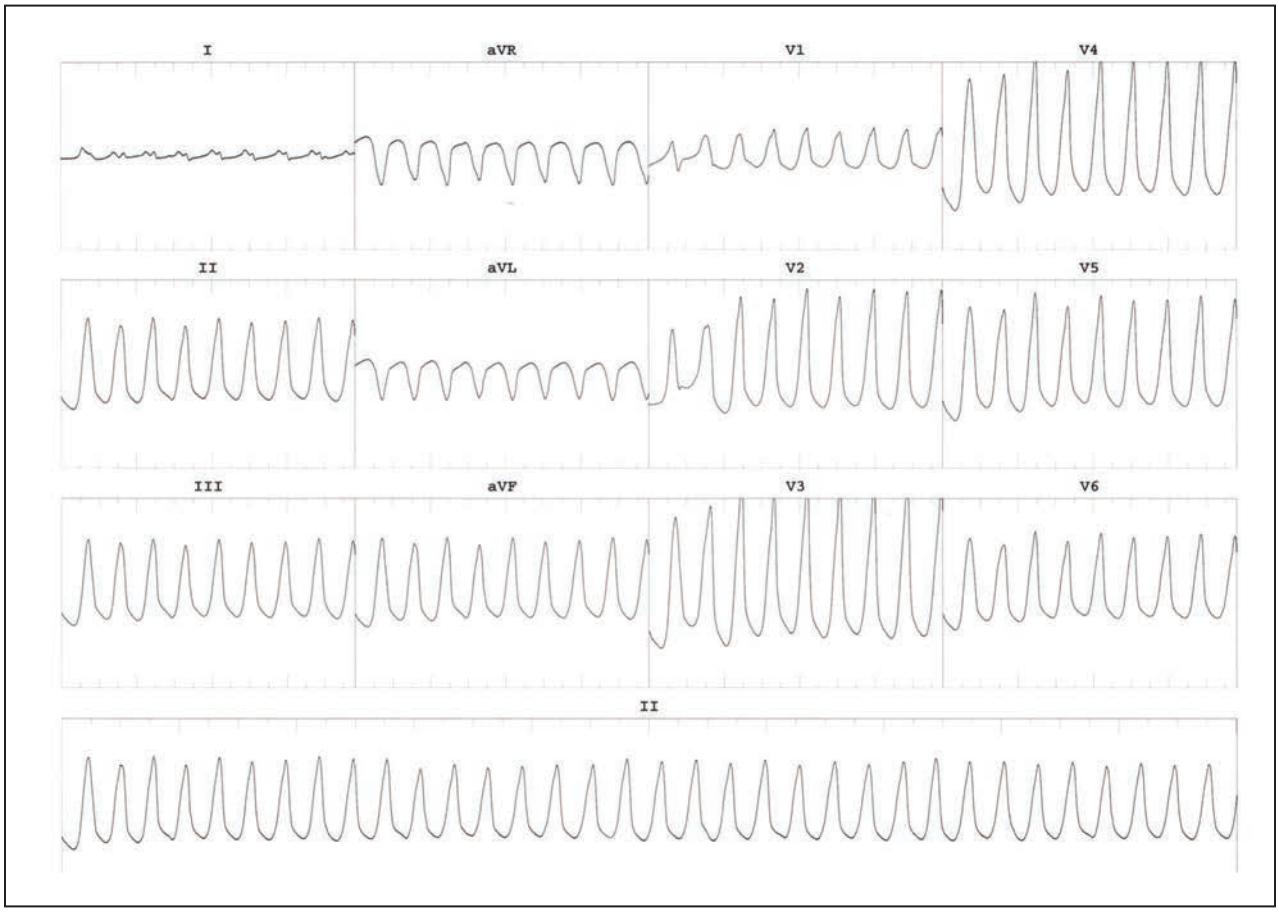

Figure 1-2 A 12-lead ECG of induced VT.

Answer

The PVCs as well as the VT were intramural in origin. The exit site of the PVC was targeted in the anterior basal LV. There was no matching pace map at the site with the earliest activation time. Characteristics suggesting a basal origin include the positive concordance in the precordial leads. There is a qR complex in V1 that suggests an origin in the aortomitral continuity (AMC), indicating that the initial vector goes away from lead V1, which is typical for this origin. However, other characteristics support a different origin: r wave in lead aVL and large R in I as well as relatively narrow QRS support an origin closer to the conduction system. The qR pattern is specific[1] but not sensitive[2,3] for an origin in the aortomitral continuity. The effective ablation site for the PVC was located at the AMC and coincided with the site of earliest site of activation.

The VT in comparison originates or exits from a more superior and more leftward position: inferior axis, positive R-wave concordance, R-wave amplitude in inferior leads higher, aVL more negative, R wave in lead III more positive.

For the VT there were matching pace maps in the left sinus of Valsalva (LSV) (shorter S-QRS interval, but still relatively long S-QRS interval) and just below the LSV (long S-QRS interval, **Figures 1-3** and **1-4**). The intramural origin of the VT is based on the observation that a short

matching S-QRS was not obtained, the S-QRS intervals were all relatively long: 85 ms for the LSV and 130 ms below the LSV. The exit was not identified, the matching pace maps with long S-QRS interval indicate capture of tissue remote to the exit. The intramural scar in this region extending to the aortic valve supports an intramural origin. Ablation at the sites with matching pace maps did render the VT noninducible. An ICD was implanted the following day. Patients with frequent PVCs and scar tissue should have programmed stimulation for risk stratification and in the presence of inducible VT, even in the absence of a history of VT, should undergo ICD implantation because of high VT recurrence rates in the setting of inducible VTs in the presence of scarring.[4,5]

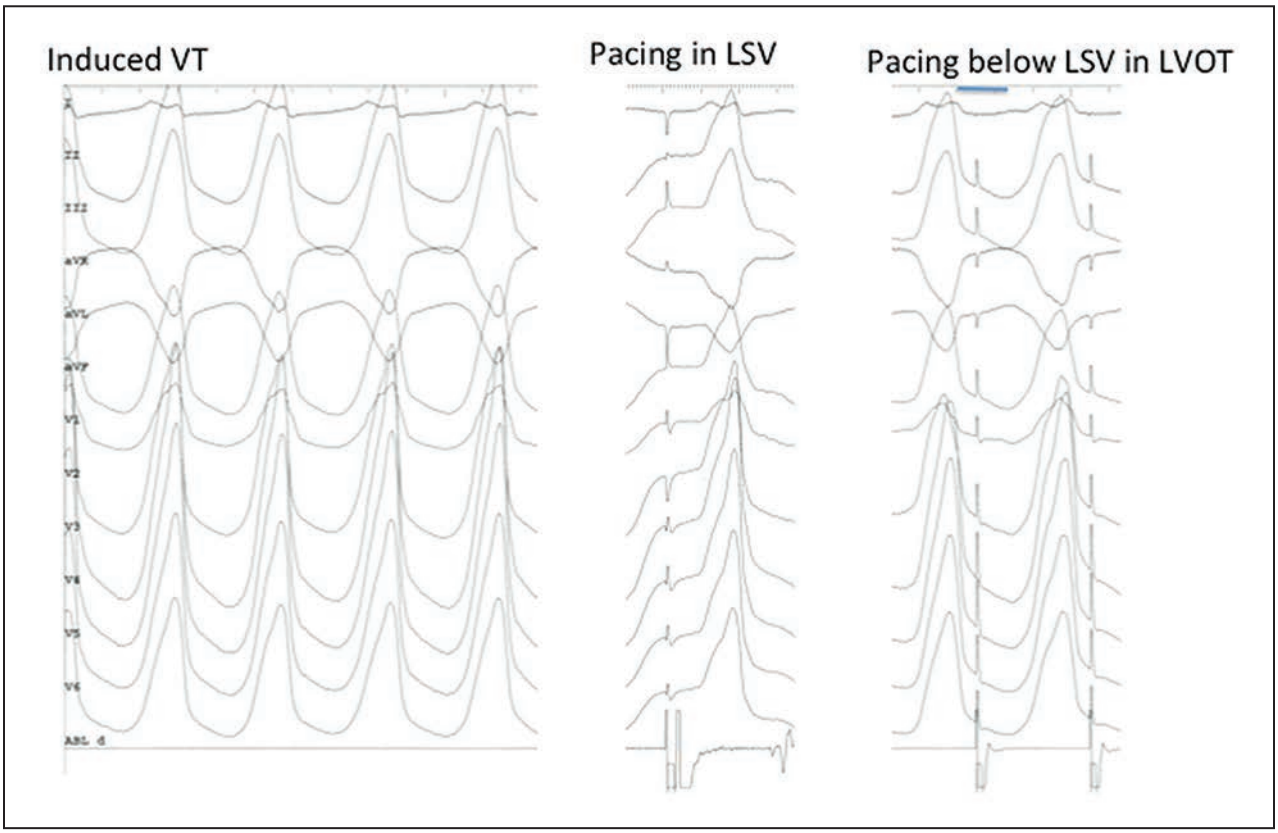

Figure 1-3 **Left panel:** A 12–lead ECG of inducible VT. **Middle panel:** Pace mapping morphology when pacing is performed from the aortic aspect of the left sinus of Valsalva (LSV). There is match with the VT morphology. Note the relatively long stimulus-QRS interval of 85 ms. **Right panel:** Pace map performed from the ventricular aspect of the LSV. The S-QRS interval is long with 130 ms. There is a matching pace map with the inducible VT.

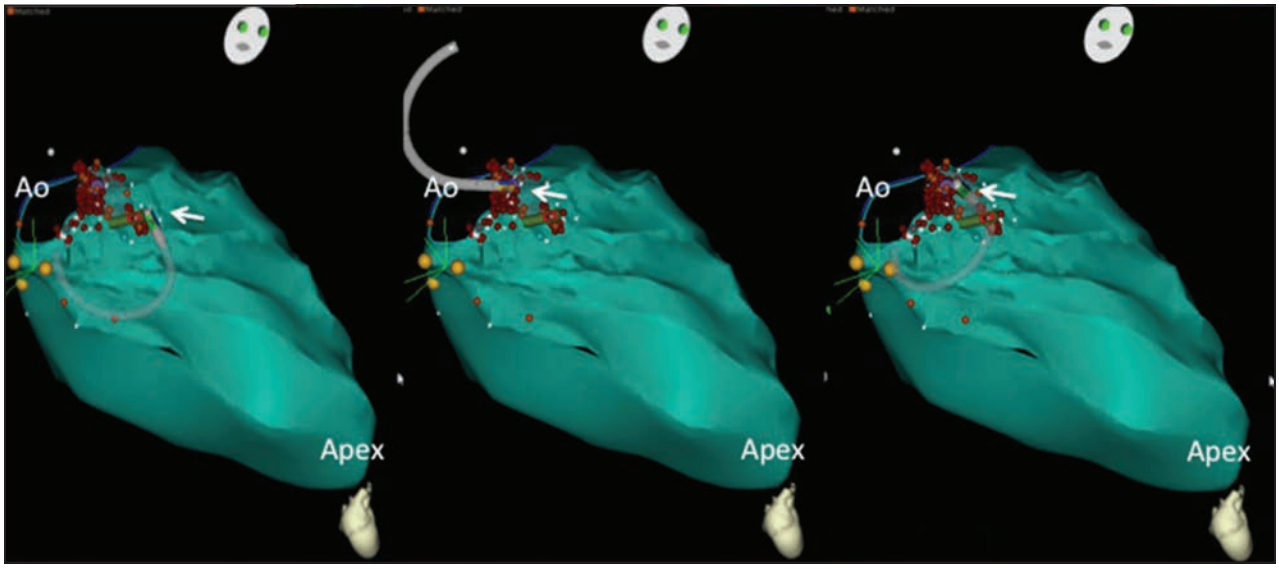

Figure 1-4 **Left panel:** 3D reconstruction of echocardiographic contours (**green**) of the left ventricle from an anterior view showing the aortic (Ao) valve and the left ventricular apex. The catheter location where the PVC was eliminated is shown (**arrow**). **Middle panel:** 3D reconstruction of echocardiographic contours (**green**) of the left ventricle from an anterior view showing the AV and the left ventricular apex. The catheter location where pace mapping was performed (Figure 1-3) is shown. **Right panel:** 3D reconstruction of echocardiographic contours (**green**) of the left ventricle from an anterior view showing the AV and the left ventricular apex. The catheter location where pace mapping was performed (Figure 1-3) is shown.

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5. Ghannam M, Siontis KC, Kim MH, et al. Risk stratification in patients with frequent premature ventricular complexes in the absence of known heart disease. *Heart Rhythm*. 2019; Epub ahead of print, September 30, 2019.

CASE 8

CLINICAL HISTORY

A 62-year-old man presented with frequent asymptomatic PVCs and a history of aortic valve replacement seven years earlier with a bioprosthesis. His PVC burden was 22% and his ejection fraction (EF) was 45% (**Figure 8-1**). He denied syncope or presyncope. A cardiac MRI showed a predominantly intramural scar.

Questions

Where is the origin of the VT? What would be the next step after ablation of his ventricular arrhythmias?

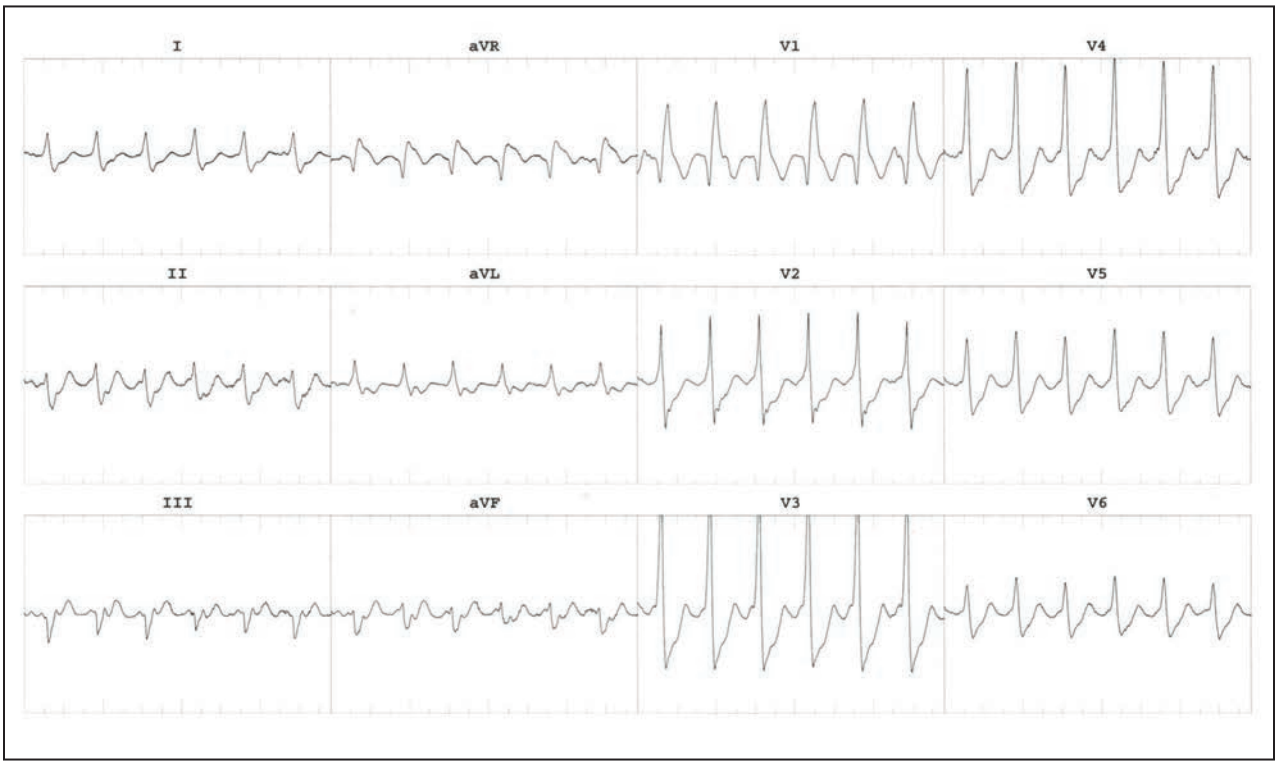

Figure 8-1 A 12-lead ECG of the patient's VT.

Answer

The origin is the intramural LVOT close to the conduction system. The qR in lead V1 suggests an origin close to the aortomitral continuity. The superior axis, however, indicates that the origin is not anterior but more likely in a posterior location. The sizable R wave in lead I and the R wave in aVL together with the relatively narrow QRS complex indicate an origin closer to the conduction system. Indeed, when pacing from a site 2–3 mm posterior to the His position (**Figure 8-2** and **Figure 8-3**), a site with a matching pace map is identified. Note the long stimulus-QRS interval indicating that this is not the exit site but a site remote from the exit, which is likely located deeper, corresponding to a predominantly intramurally located scar. The patient had frequent PVCs at the onset of the ablation procedure. Programmed ventricular stimulation was performed for risk stratification because of the presence of scarring. We were able to induce four different monomorphic VTs, the present VT being one of them. We targeted both VTs and frequent PVCs. His PVCs as well as his VTs had intramural origins, and radiofrequency (RF) energy was guided by activation mapping and pace mapping. Postablation, his VTs were no longer inducible and the predominant PVC was not seen anymore. After the ablation, the patient underwent ICD implantation. He subsequently had antitachycardia pacing for one of the VTs. His EF normalized during follow up. Programmed ventricular stimulation is important for risk stratification in patients with structural heart disease, as illustrated in this case. Patients with inducible VT do have VT recurrences, as demonstrated in several reports, even though they may have preserved left ventricular function and no history of prior VT.[1,2] ICD implantation should strongly be considered.

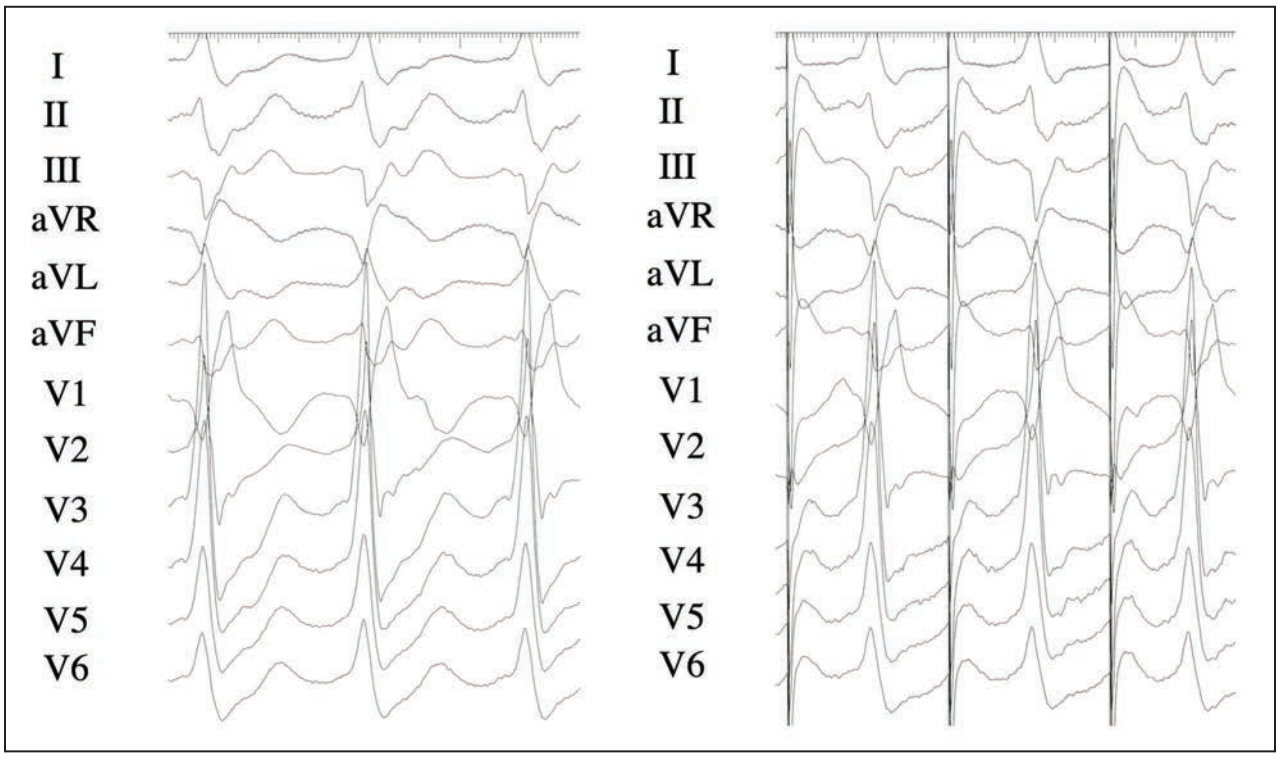

Figure 8-2 **Left panel:** A 12-lead ECG of the targeted VT. **Right panel:** The pace map at this site matched the induced VT. Note that there is a long stimulus–QRS interval measuring 180 ms.

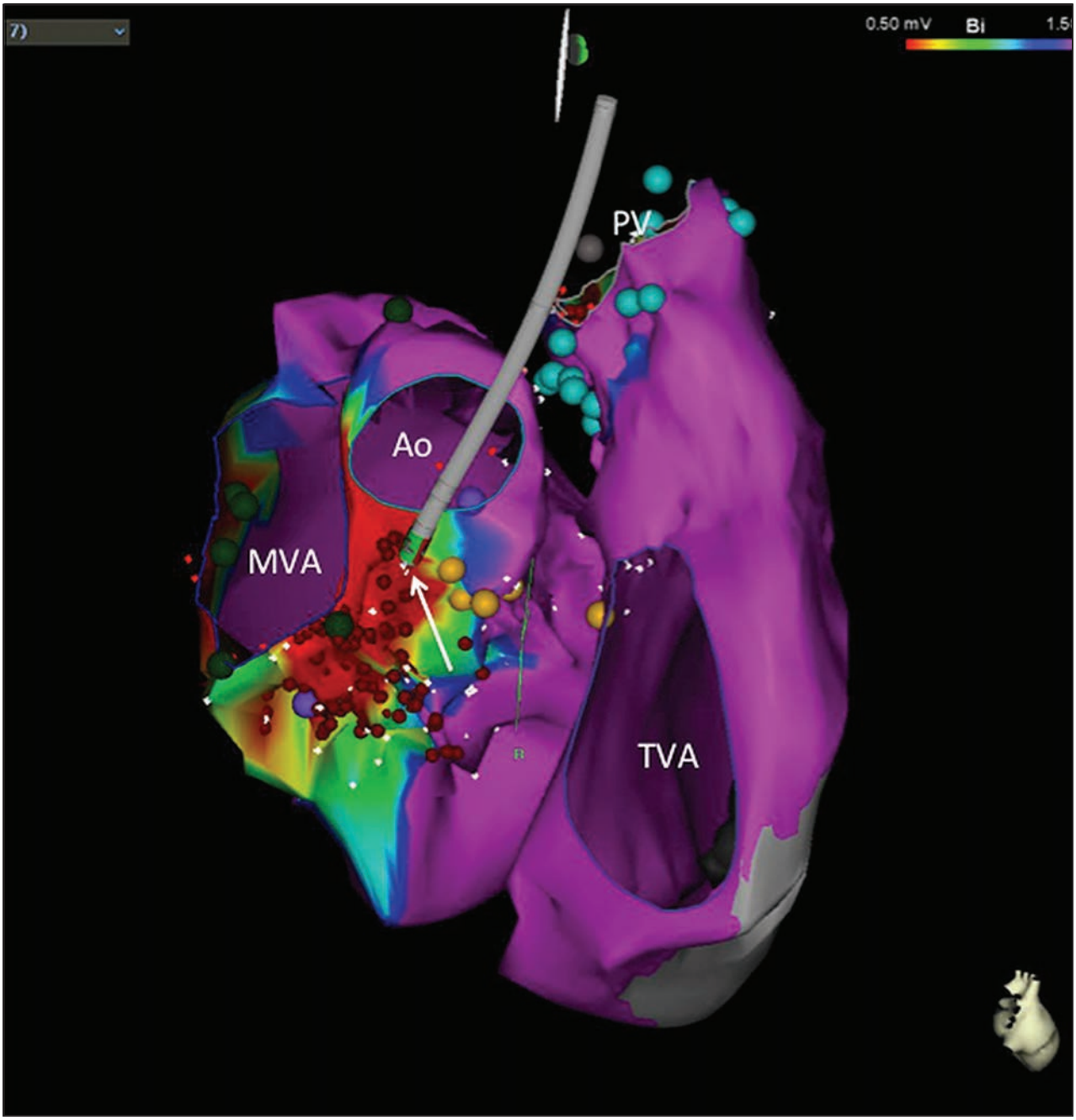

Figure 8-3 3D reconstruction of echocardiographic contours of the left ventricle from a posterior view showing the aortic valve (AV), the mitral valve annulus (MVA), and a superimposed voltage map. The catheter location with a matching pace map to the targeted VT (Figure 8-2) is indicated by an **arrow**. The pulmonic valve and tricuspid valve annulus (TVA) are also shown. **Orange tags** indicate the location of the bundle of His.

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2. Yokokawa M, Siontis KC, Kim HM, et al. Value of cardiac magnetic resonance imaging and programmed ventricular stimulation in patients with frequent premature ventricular complexes undergoing radiofrequency ablation. *Heart Rhythm*. 2017;14(11):1695–1701.

CASE 23

CLINICAL HISTORY

A 56-year-old man presented with a history of nonischemic cardiomyopathy (ejection fraction [EF] of 15%) and a history of hypertension, dyslipidemia, diabetes mellitus, and COPD. The patient had several failed prior VT ablation procedures. A CMR showed an intramural septal scar. The patient presented for a repeat ablation procedure.

Questions

Where does the VT shown in **Figure 23-1** originate? What is a possible ablation strategy for this VT?

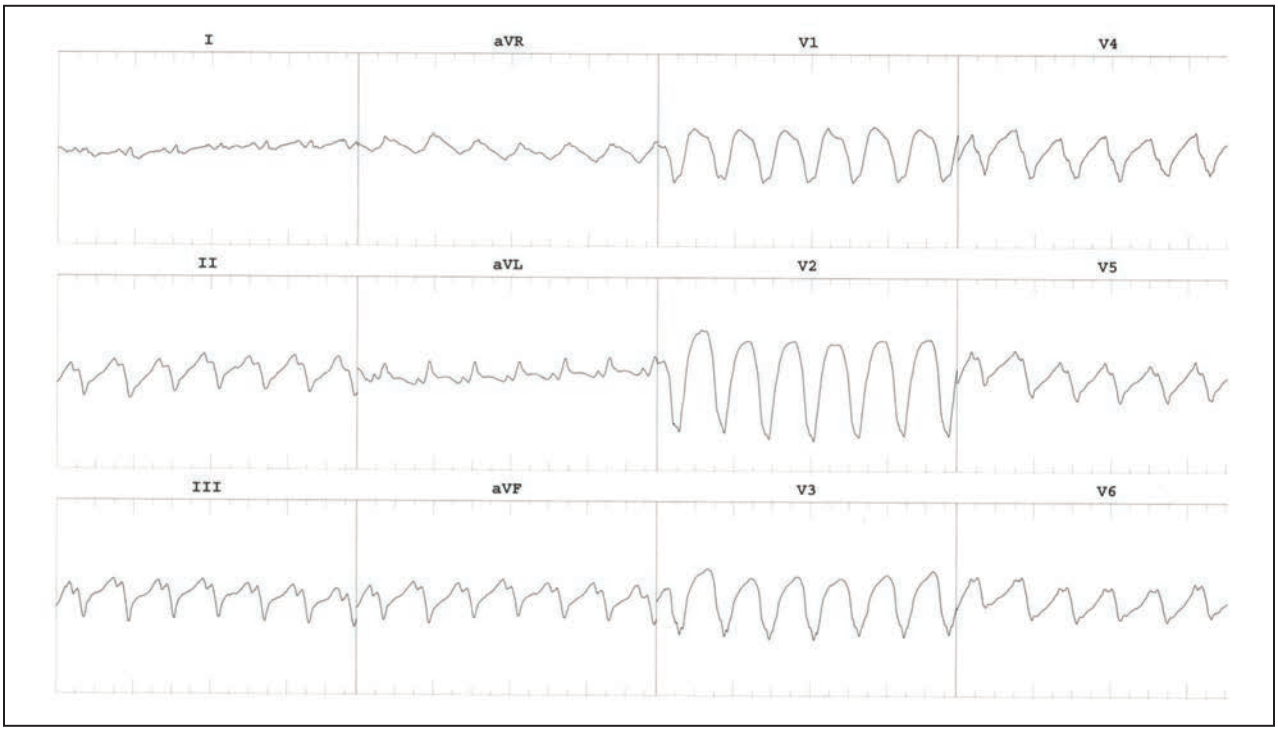

Figure 23-1 A 12-lead ECG of the patient's VT.

Answer

The origin is the intramural septum. There is a left bundle branch block (LBBB) inferior axis morphology, with a transition in V5/V6 and notching in the inferior leads. The scarring located in the high intramural septum makes localization of the origin difficult. In a patient without structural heart disease, based on the above-mentioned features, the origin would be more in the lateral RVOT free wall. Although the best pace maps were located in the high septum just below the pulmonary artery (**Figure 23-2**), the stimulus-QRS interval was 80 ms and the pace map was only about 10/12, i.e., the exit was likely intramural where the scar was located in the CMR (**Figure 23-3**). It is not uncommon in patients with septal scar to have a late transition even though the exit is located on the septum.[1] This is usually not seen in patients with idiopathic RVOT VTs, where the transition is earlier if the origin is on the septum. The case illustrates that in order to identify critical sites for intramural septal VTs, the entire RV septum needs to be mapped. With respect to an ablation strategy, we could demonstrate that the success rate of ablation of intramural VTs depends on the depth of the intramural scar as it projects to the closest endocardial surface. The larger the projected scar surface (cut-off is > 16%) at a depth of > 5 mm, the less likely an ablation will be successful when using a conventional irrigated tip catheter for ablation. In case a conventional approach (targeting sites with abnormal electrograms, matching pace maps, sites with early activation or sites with concealed entrainment) fails to eliminate VT, a more extensive approach has been shown to be effective when both aspects of the intramural scar are targeted.[2]

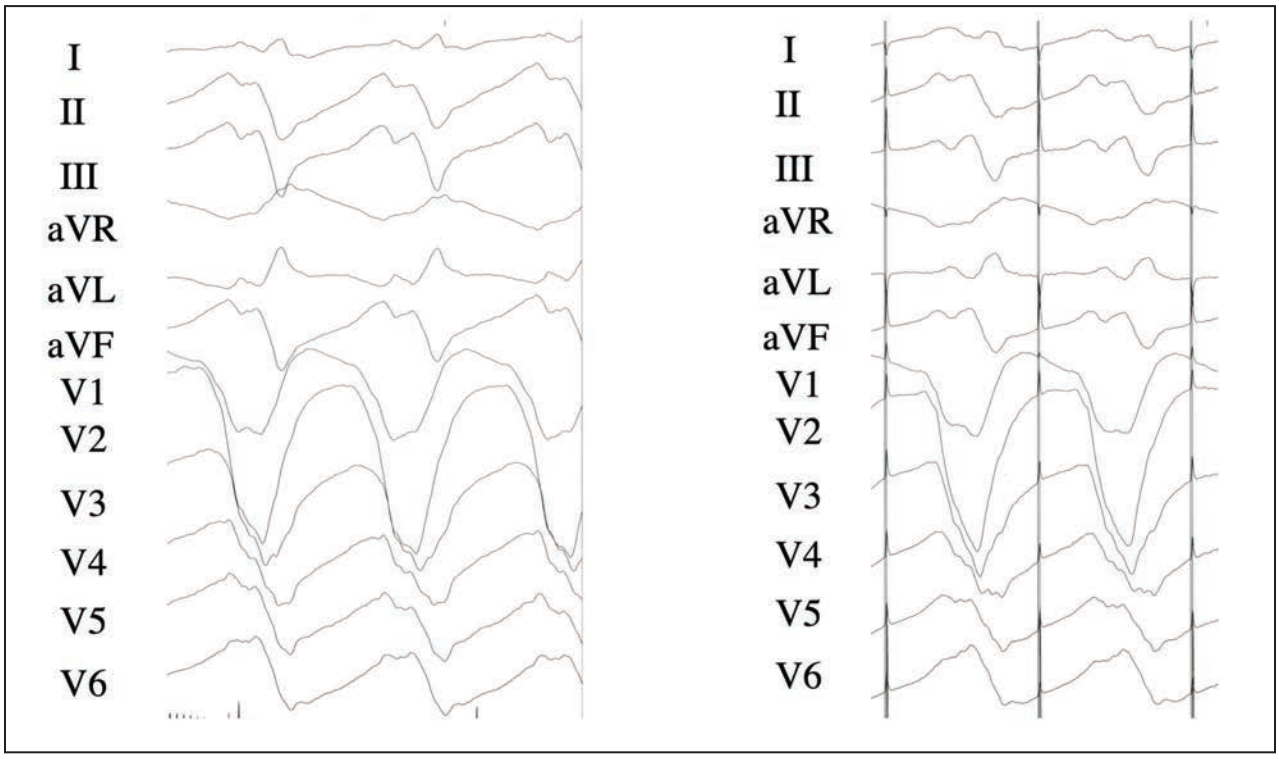

Figure 23-2 **Left panel:** A 12-lead ECG of the patient's VT. **Right panel:** Pace map from the high septal right ventricular outflow tract.

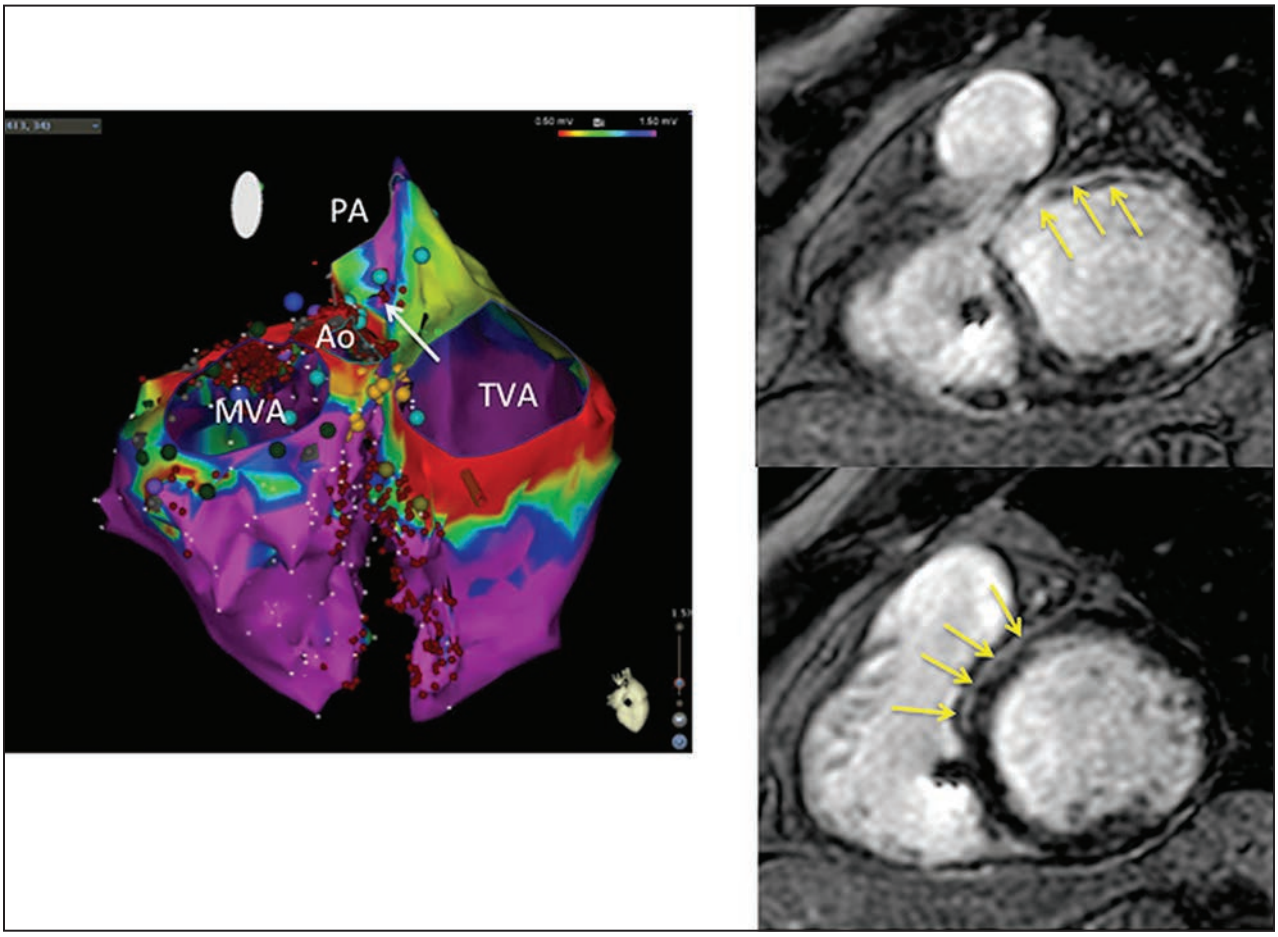

Figure 23-3 **Left panel:** 3D reconstruction of echocardiographic contours of right and left ventricles from a posterior view showing the mitral valve annulus (MVA), the aortic (Ao) valve, the tricuspid valve annulus (TVA), the pulmonary artery (PA), and a superimposed biventricular voltage map. A **white arrow** indicates the location of the site of pace mapping from Figure 23-2. **Right panel:** Biventricular short-axis view of CMR showing intramural scarring: The **top panel** indicates a basal and the **bottom panel** a more apical view. The intramural scar is indicated with **yellow arrows**.

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APPENDIX

Case 1: PVC and VT with an intramural anterobasal left ventricular origin
Case 2: PVCs originating from the intramural basal myocardium
Case 3: PVC originating from the tricuspid annulus
Case 4: Ventricular arrhythmias with an intramural septal origin
Case 5: VT originating from the lateral tricuspid annulus
Case 6: PVC originating from the inferior mitral annulus
Case 7: VT due to bundle branch reentry
Case 8: VT originating from the intramural LVOT
Case 9: VT originating from the septal bands in the right ventricle
Case 10: VT originating from the intramural myocardium between left sinus of Valsalva and the great cardiac vein
Case 11: PVC originating from the anterolateral papillary muscle
Case 12: PVC from the intramural septal outflow tract
Case 13: Post-infarction VT originating from the septum
Case 14: PVC originating from the parahisian area
Case 15: VTs originating from scar at the mitral valve annulus
Case 16: VT originating from the epicardial crux of the heart
Case 17: VT originating from the tricuspid valve annulus
Case 18: VT originating from scar close to the inferoseptal mitral annulus
Case 19: PVC originating from the epicardial inferobasal septum
Case 20: PVC originating from the intramural inferoseptum
Case 21: PVC originating from the posterior mitral annulus
Case 22: VT originating from scar at the mitral annulus
Case 23: VT originating from the intramural septum
Case 24: PVC originating from the Purkinje fiber system
Case 25: VT originating from the anterobasal left ventricular epicardium
Case 26: VT originating from the basal right ventricular free wall
Case 27: PVC originating from the anterobasal LVOT
Case 28: PVC originating from the anterobasal intramural LVOT
Case 29: VT originating from scar in the anterior left ventricular free wall
Case 30: VT originating from the subepicardial anterior left ventricle