



Initial validation of a novel ECGI system for localization of premature ventricular contractions and ventricular tachycardia in structurally normal and abnormal hearts

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ABSTRACT

Background: View into Ventricular Onset (VIVO) is a novel ECGI system that uses 3D body surface imaging, myocardial CT/MRI, and 12 lead ECG to localize earliest ventricular activation through analysis of simulated and clinical vector cardiograms.

Objective: To evaluate the accuracy of VIVO for the localization of ventricular arrhythmias (VA).

Methods: In twenty patients presenting for catheter ablation of VT [8] or PVC [12], VIVO was used to predict the site earliest activation using 12 lead ECG of the VA. Results were compared to invasive electroanatomic mapping (EAM).

Results: A total of 22 PVC/VT morphologies were analyzed using VIVO. VIVO accurately predicted the location of the VA in 11/13 PVC cases and 8/9 VT cases. VIVO correctly predicted right vs left ventricular foci in 20/22 cases.

Conclusion: View into Ventricular Onset (VIVO) can accurately predict earliest activation of VA, which could aid in catheter ablation, and should be studied further.

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Introduction

Catheter ablation is an increasingly utilized treatment strategy for ventricular arrhythmias [1–4]. Localization of the arrhythmia based on the surface electrocardiogram is an important step in procedural decision making. A variety of algorithms have been proposed for localization of premature ventricular complexes (PVC) and exit sites of ventricular tachycardia (VT) with varying degrees of accuracy [5–9].

Body surface mapping has been investigated as a tool for anatomic localization of ventricular arrhythmias. Prior studies have shown that body surface mapping can provide accurate non-invasive activation mapping as well as substrate characterization [10–14]. Body surface mapping technology is, however, limited by the requirement for specialized electrode vests and other equipment for data acquisition.

In this report we describe our initial experience with “View into Ventricular Onset” (VIVO), a non-invasive tool designed to identify the site of earliest ventricular activation based on the 12 lead electrocardiogram. We conducted a pilot evaluation to describe the accuracy of VIVO in patients undergoing catheter ablation of PVC and VT, both with structurally normal and abnormal hearts.

Methods

Patient population

Patients presenting to a tertiary care electrophysiology lab for ablation of ventricular arrhythmias with pre-procedural cardiac imaging, either contrast enhanced cardiac computed tomography (CT) or cardiac magnetic resonance imaging (MRI), were considered eligible for the study. This study was reviewed and approved by the Johns Hopkins Institutional Review Board (IRB).

Prediction of PVC/VT location using VIVO

VIVO is an ECG imaging (ECGI) system that uses a patient specific myocardial model from MRI or CT, a 3D photo to define ECG electrode position, and the 12 lead ECG to localize the origin of the VA (Fig. 1). The methods used by VIVO have been described elsewhere [15–17]. VIVO assumes homogeneous propagation velocities within the myocardium.

VIVO uses semi-automated morphing of a reference model to generate a patient specific model of the heart and torso that includes the septum as well as accurate wall thickness¹⁵. This 3D heart model is used to determine the simulated activation sequences originating from discrete

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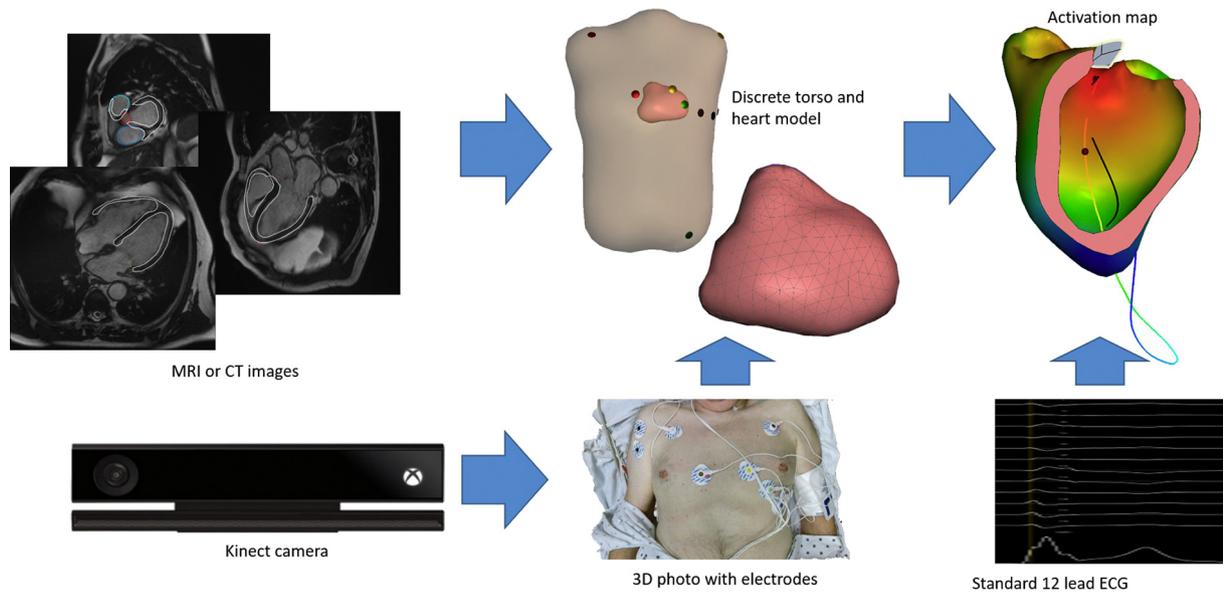


Fig. 1. Schematic diagram of VIVO. Myocardial and torso contours are generated from cardiac CT or MRI (upper panel). A 3D image of the patient torso with ECG electrodes applied (lower panel) is then merged with the torso model to allow for accurate positioning of the ECG electrodes relative to the heart model. The 12-lead ECG is then loaded and a model of ventricular activation is generated, with site of earliest activation marked.

nodes in the heart. The objective is to localize the node that is closest to the PVC or VT origin.

Traditional ECGI systems match the measured ECG on the thorax with simulated myocardial potentials. VIVO, however, matches the measured vector cardiogram (VCG) with the simulated vector cardiogram (“anatomic vector”). Within VIVO, a patient specific VCG is reconstructed from the ECG taking into account the electrode positions on the thorax relative to the heart¹⁷. The direction of activation can be directly derived from the simulated activation isochrones and is defined as the average activation direction at a certain isochrone. Matching in VCG terms means now that the VCG derived from the ECG and the anatomical VCG point in the same direction.

To limit computation, three isochrones are taken into account and the initial, mid, and terminal QRS vector directions are computed [17]. The combination of the mid and terminal QRS vectors identify the region from which the PVC or VT is originating, while the ventricular node for which the angle between both initial vectors (anatomic vector cardiogram and measured vector cardiogram) is minimal identifies the PVC/VT origin in that region.

Electroanatomic mapping and ablation

Informed written consent was obtained for all procedures. Cardiac imaging studies were performed according to institutional protocols. Procedural strategies were left to the discretion of the attending electrophysiologist. In all cases, CARTO 3© (Biosense-Webster, Diamond Bar, California, USA) was utilized for electroanatomic mapping. CARTOMerge© was utilized for cardiac model generation and registration with electroanatomic maps at the discretion of the electrophysiologist.

For PVC ablations, activation mapping and pace mapping were performed to localize the PVC focus. Radiofrequency (RF) ablation was performed using an irrigated, force-sensing catheter (Thermocool SmartTouch©, Biosense-Webster) targeting areas of earliest, pre-systolic ventricular activation and 12/12 pace matches.

For VT ablations, patients first underwent non-invasive programmed stimulation (NIPS) under sedation to establish the target clinical VT. Left ventricular mapping was performed either via a trans-septal approach or retrograde aortic approach at operator discretion. Epicardial access was obtained via an anterior approach that has been describe elsewhere [18]. A detailed substrate map was generated during sinus

rhythm using either a PentaRay© or DecaNav© catheter (Biosense-Webster). Late potentials were marked on the electroanatomic map. Pace mapping was performed. Activation and entrainment mapping during ventricular tachycardia were also performed as hemodynamically tolerated. Critical isthmus and exit sites were identified based on the following criteria at sites where concealed entrainment during VT was demonstrated:

- (a) Critical isthmus: (1) S-QRS 30–70% of the TCL (2) S-QRS = EG-QRS (3) PPI = TCL
- (b) Exit site: (1) S-QRS < 30% of the TCL (2) S-QRS = EG-QRS (3) PPI = TCL.

RF ablation was performed targeting the critical isthmus and areas of late potentials. Substrate modification was then performed at the discretion of the operator.

Analysis

Localization of PVC or VT based on VIVO and invasive electroanatomic mapping was performed using a segmental model of the right and left ventricle (Fig. 2). Analysis using VIVO was performed after the invasive electroanatomic mapping to facilitate data acquisition and avoid influencing the procedure. The site of earliest activation predicted by VIVO in both the PVC and VT cohorts was defined by one investigator who was blinded to the results of the procedure (PVD). For septal sites, the ventricle of origin was defined based on the direction of the initial activation vector.

For PVC ablations, the location of the PVC foci based on EAM was defined by the primary operator who was blinded to the results of VIVO prediction. In cases with ambiguous localization definition, a second investigator blinded to the results of VIVO prediction reviewed procedural data and defined localization (HT). All VT ablations were performed by a single operator (HT), blinded to the results of VIVO prediction, who identified the critical isthmus or exit site location based on the above specified criteria. If no exit site or critical isthmus was identified during the procedure, the region of successful ablation was recorded.

In addition, two attending electrophysiologists, blinded to the results of both VIVO analysis and electroanatomic mapping localization,

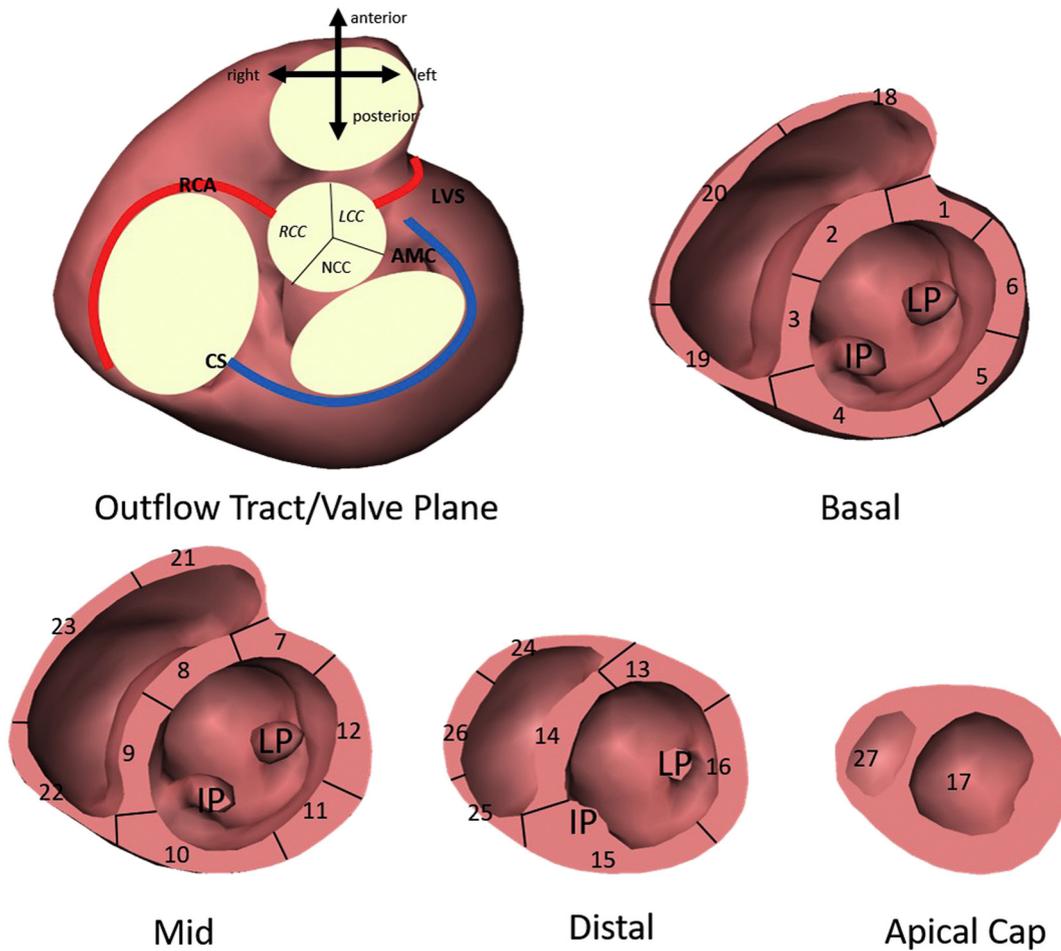


Fig. 2. Myocardial Segmental Model Used for Localization of PVC and VT. Schematic diagram used to localize PVC and VT sites for VIVO prediction model and during EPS. The RVOT was divided into four quadrants as shown above – rightward/anterior, rightward/posterior, leftward/anterior, leftward/posterior. Abbreviations: RCC – left coronary cusp; LCC – left coronary cusp; NCC – noncoronary cusp; AMC – Aortomitral continuity.

reviewed the 12 lead ECG of each analyzed PVC/VT and localized the VA using the segmental model.

For comparison purposes, a “near match” was defined as a predicted location within one contiguous anatomic segment of the actual location defined by electroanatomic mapping. These cases were then reviewed by two authors (SM, HT) to verify that the position of the predicted location within the segment was towards the segment containing the actual location. A perfect match was defined as a predicted location within the same anatomic segment of the actual location defined by electroanatomic mapping. Both near and perfect matches were considered in the final analyses as a positive match for the VIVO algorithm.

Results

Patient recruitment

A total of 29 patients were enrolled in the study. Nine patients were excluded from analysis. Seven were excluded due to absence of PVC or inducible VT at the time of procedure [4], incorrect image acquisition timing [2], or device artifact [1]. In two additional patients, software related factors precluded analysis including software error and inability to segment accurately due to complex congenital heart disease (VT patients). Twenty patients were included in the final analysis cohort.

In this cohort, two patients, one undergoing PVC ablation and one undergoing VT ablation, had two arrhythmia morphologies at baseline that were separately analyzed resulting in a total of 22 analyzed PVC and VT cases.

Cohort and procedural characteristics

Among the 20 patients enrolled, 8 patients underwent VT ablation and 12 patients underwent PVC ablation (Table 1). In the VT cohort, 4 patients had arrhythmogenic right ventricular dysplasia (ARVD), 2 patients had non-ischemic cardiomyopathy, and 2 patients had ischemic cardiomyopathy. All patients in this cohort underwent pre-procedural CT. Seven patients were determined to have scar-mediated re-entrant VT while one patient was identified as having triggered VT. Six patients underwent epicardial mapping as part of their procedure.

In the PVC cohort, most patients had structurally normal hearts [9]. Three patients underwent pre-procedural MRI while the remainder had pre-procedural CT. Seven patients underwent endocardial RV and RVOT mapping only. Two patients underwent LVOT mapping via a

Table 1
Baseline characteristics of PVC and VT patient cohorts.

	VT	PVC
Number	8	12
Age (range)	51 (24–75)	52 (17–77)
Female (%)	38%	58%
Substrate		
Ischemic cardiomyopathy	2	1
ARVD	4	0
Non-ischemic cardiomyopathy	2	2
Other cardiomyopathy	0	0
No structural heart disease	0	9

retrograde aortic approach and one patient underwent LV mapping via a trans-septal approach. The remaining patients underwent a combination of right and left sided mapping.

Intraprocedural image registration was performed in all patients in the VT cohort. In the PVC cohort, 4 patients did not have intraprocedural image registration performed including one patient who had two PVC morphologies included in this study.

Procedural outcomes

Ten of twelve patients had successful PVC ablation with no observed clinical PVC recurrence after a post-procedural waiting period. Among patients with successful ablation, four patients had foci in the RVOT, one in the RV moderator band, one in the basal septal RV near the His bundle, three in the LVOT, and one in the basal LV (Table 2). In one patient, ablation was not attempted due to PVC focus that was inferior to the right coronary cusp near the His bundle. In this patient, localization was defined as the site with best pace map match and pre-systolic local activation during PVC. In another patient, activation mapping localized the PVC focus to the mid-lateral RV wall where ablation transiently increased PVC frequency followed by transient suppression. In this patient, localization was defined by the site of best pace map match. Follow up was available in 7 of 10 patients with acutely successfully procedures, with recurrence reported in 1 patient (median follow up 50 days, range 13–414 days).

Of the 8 patients who had VT ablations, 7 had acute success with non-inducibility of VT at the end of the procedure. In the remaining patient, the clinical VT, which was included in this study, was non-inducible post-ablation but a second VT was induced. Follow up was available in 5 of the 7 patients with acutely successful procedures, with recurrence reported in 2 patients (median follow up 343 days, range 191–431 days). VT localization is summarized in Table 3. In the four patients with ARVD/C, the exit sites and sites of successful ablation were localized to the right ventricle; epicardial ablation was performed in three patients. In the remaining four patients, the exit sites and sites of successful ablation were localized to the left ventricle and LVOT. In patient 32, a mid-septal outflow tract focus of triggered VT was identified with successful ablation from the right coronary cusp and septal RVOT.

Comparison of predicted location to actual location

In the PVC cohort, there were a total of 13 analyzed PVC morphologies from 12 patients (illustrative cases shown in Fig. 3). The VIVO

predicted focus was a near match or perfect match to the focus identified through invasive electroanatomic mapping in 11/13 (85%) cases, with three cases achieving a perfect match and eight cases achieving a near match (Fig. 4). In the two remaining cases, one patient had a focus in the moderator band that was successfully ablated at both the lateral and septal insertions while the other patient had a focus in the posteromedial papillary muscle that was successfully ablated. In the first case, the predicted focus was in the basal anterior right ventricle. In the second case, the predicted focus was in the mid-inferoseptal left ventricle. Of note, the papillary muscle and moderator band are structures that are not currently segmented in VIVO.

In identifying left vs. right sided foci, VIVO accurately predicted PVC foci in 11/13 cases. There were two cases in which the side predicted was incorrect, both with septal sites of origin. In the first case, the PVC was localized to a right para-hisian focus in the basal right ventricular septum whereas VIVO predicted PVC focus in the left ventricle mid-septal region. In the second case, the PVC was localized to a mid-myocardial focus between the anterior interventricular vein and leftward/posterior RVOT; ablation at both sites was required for PVC elimination. In this case, VIVO predicted a PVC focus in the left ventricle, basal anteroseptal region. Among patients in whom intraprocedural imaging registration was performed, 50% of patients had a perfect match excluding the two patients in whom PVC foci were located in non-segmented structures.

In the VT cohort, there were 9 analyzed VT morphologies from 8 patients (illustrative case shown in Fig. 3). The predicted focus was a near match or perfect match to the EAM-identified focus in 8/9 (88%) cases, with 6 perfect matches and two near matches (Fig. 4). Including the patients in whom a software limitation precluded analysis, the accuracy rate was 8/11 (73%). In the remaining case, the VT focus was localized to the distal lateral right ventricle whereas VIVO predicted a left-sided apical focus. This was also the only case in this cohort in which VIVO incorrectly predicted chamber of origin.

Comparison to manual ECG analysis

For PVC localization, the accuracy was fair with 65.3% near or perfect matches (Reader 1 9/13 near or perfect match, Reader 2 8/13 near or perfect match; 1/12 perfect match for both readers). For VT localization, the accuracy was more limited with 38.9% near or perfect match (Reader 1 4/9 near or perfect match, Reader 2 3/9 near or perfect match; 2/9 perfect match for Reader 1, 1/9 perfect match

Table 2
PVC cohort procedural characteristics and match to predicted sites.

Patient ID	VT/PVC characteristics ^a	Substrate ^b	Predicted segment	Actual segment	Segment match ^d	Side match ^e	Sites mapped ^c	Sites ablated ^c	Procedural outcome
2	LB/Superior Axis/V5/Positive	Normal	20	23	2	1	Endo RV	Endo RV	Unsuccessful
8	LB/Inferior axis/V2/Negative	Normal	33	33	1	1	Endo RV/Endo LV via Ao	Endo LV via Ao	Successful
10	LB/Inferior Axis/V4/Negative	Normal	31	31	1	1	Endo RV	Endo RV	Successful
12	RB/Inferior Axis/Positive/Negative	Normal	5	6	2	1	Endo LV via Ao	Endo LV via Ao	Successful
12	RB/Superior Axis/Positive/Positive	Normal	4	5	2	1	Endo LV via Ao	Endo LV via Ao	Successful
15 ^e	LB/Superior Axis/V6/Positive	Normal	18	8 and 23	0	1	Endo RV	Endo RV	Successful
17	RB/Superior Axis/V3/Positive	Normal	4	9	0	1	Endo LV via Ao	Endo LV via Ao	Successful
20	LB/Inferior Axis/V4/Negative	Normal	30	31	2	1	Endo RV	Endo RV	Successful
23	RB/Indeterminate Axis/Positive/Positive	NICM	32	32	1	1	Endo LV via TS	Endo LV via TS	Unsuccessful
25	LB/Inferior Axis/V4/Indeterminate	NICM	8	2	2	0	Endo RV	Endo RV	Successful
26	LB/Inferior Axis/V4/Indeterminate	Normal	30	31	2	1	Endo RV	Endo RV	Successful
29	RB/Inferior Axis/Positive/Indeterminate	ICM	2	31	2	0	Endo RV/distal CS/Endo LV via Ao	Endo RV/distal CS/Endo LV via Ao	Successful
30	LB/Inferior Axis/V4/Negative	Normal	30	31	2	1	Endo RV	Endo RV	Successful

^a Description of PVC/VT morphology: V1 morphology/axis based on inferior leads/precordial transition/dominant vector in I/aVL. LB and RB refer to right bundle and left bundle.

^b ICM: Ischemic cardiomyopathy; NICM: Non-ischemic cardiomyopathy.

^c Endo RV: endocardial right ventricular including RVOT; Endo LV via Ao: LV/LVOT mapping via retrograde aortic approach; Endo LV via TS: endocardial LV mapping via trans-septal approach; Distal CS: mapping within distal coronary sinus.

^d Segment Match: 0 = no match, 1 = perfect patch, 2 = near match.

^e Side Match: 0 = no match, 1 = match #PVC focus in moderator band with ablation of both the septal and free wall aspects.

Table 3
VT cohort procedural characteristics and match to predicted sites.

Patient ID	VT/PVC characteristics ^a	Substrate ^b	Predicted segment	Actual segment	Segment match ^e	Side match ^f	Sites mapped ^c	Sites ablated ^c	Procedural outcome
5	LB/Superior Axis/Negative/Positive	ARVD	22	19	2	1	Endo RV	Endo RV	Successful
11	LB/Superior Axis/V4/Positive	ARVD	20	20	1	1	Endo RV/Epi	Endo RV/Epi	Successful
11	LB/Superior Axis/Negative/Positive	ARVD	17	22	0	0	Endo RV/Epi	Endo RV/Epi	Successful
14	RB/Inferior Axis/Positive/Negative	NICM	5	6	2	1	Epi	Epi	Unsuccessful
16	LB/Superior Axis/V2/Positive	NICM	9	9	1	1	Endo LV via TS/Epi	Endo LV via TS/Epi	Successful
19	LB/Inferior Axis/V5/Indeterminate	ARVD	20	18	1	1	Endo RV/Epi	Epi	Successful
21	LB/Inferior Axis/V6/Positive	ARVD	20	20	1	1	Endo RV/Epi	Endo RV/Epi	Successful
27	RB/Inferior Axis/Positive/Negative	ICM	6	6	1	1	Endo LV via TS	Endo LV via TS	Successful
31 ^d	LB/Inferior Axis/V4/Indeterminate	ICM	30	32 and 30	1	1	Endo RV/Endo LV via Ao	Endo RV/Endo LV via Ao	Successful

^a Description of PVC/VT morphology: V1 morphology/axis based on inferior leads/precordial transition/dominant vector in I/aVL. LB and RB refer to right bundle and left bundle.
^b ICM: Ischemic cardiomyopathy; NICM: Non-ischemic cardiomyopathy
^c Endo RV: endocardial right ventricular including RVOT; Endo LV via Ao: LV/LVOT mapping via retrograde aortic approach; Endo LV via TS: endocardial LV mapping via trans-septal approach; Distal CS: mapping within distal coronary sinus.
^d Suspected mid-myocardial focus ablated successful from right posterior RVOT and right coronary cusp.
^e Segment Match: 0 = no match, 1 = perfect patch, 2 = near match.
^f Side Match: 0 = no match, 1 = match.

for reader 2). These results are summarized in Table 4. The interobserver agreement for localization of VA was fair for PVC localization ($k = 0.498, p < 0.001$) but poor for VT localization ($k = 0.299, p < 0.001$).

Discussion

In this study, we performed an initial pilot evaluation of the accuracy of VIVO, a non-invasive ECGI tool that localizes the site of earliest

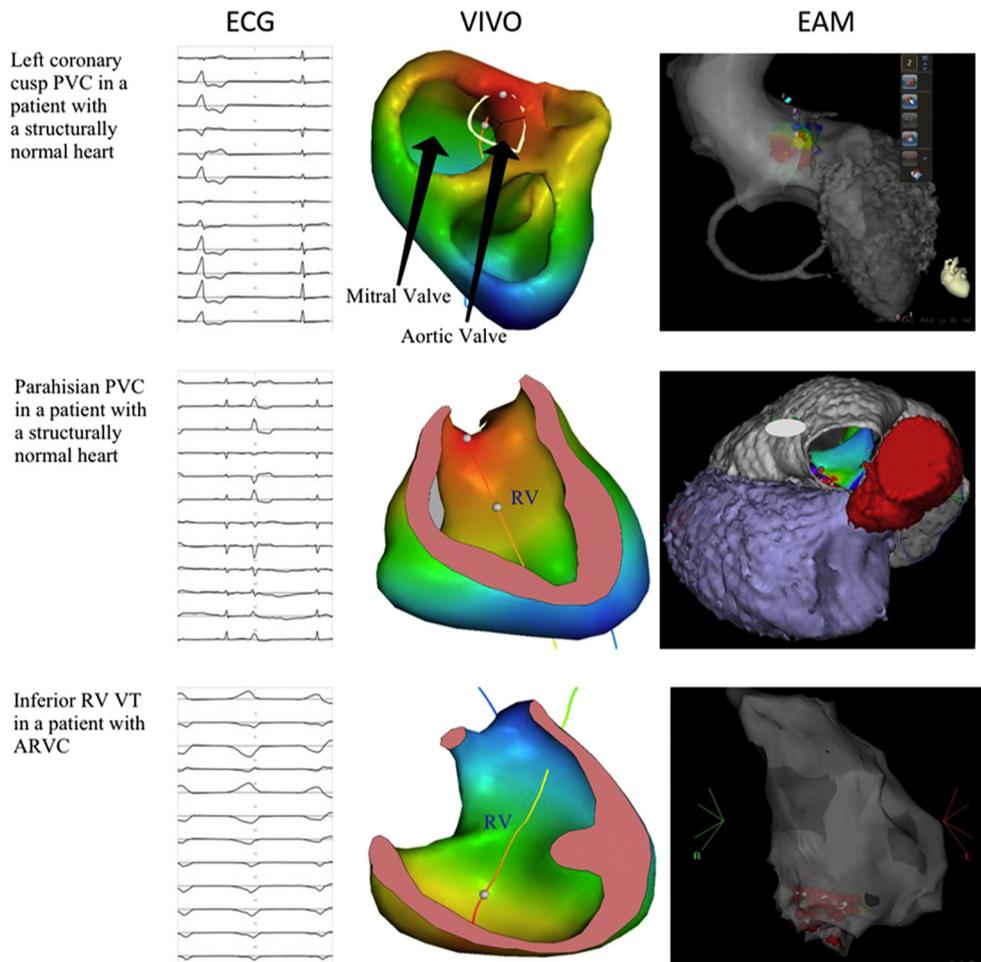


Fig. 3. Illustrative examples of ECG, VIVO predicted location, and electroanatomic mapping in 3 patients. Case examples. Left panel shows 12-lead ECG. Middle panel shows VIVO prediction model with ventricular activation map (red to blue reflecting early to late) and vector cardiogram with earliest activation at the start of vector loop (shown by red coloration of line). Right panel shows electroanatomic map with region of ablation marked by red tags. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

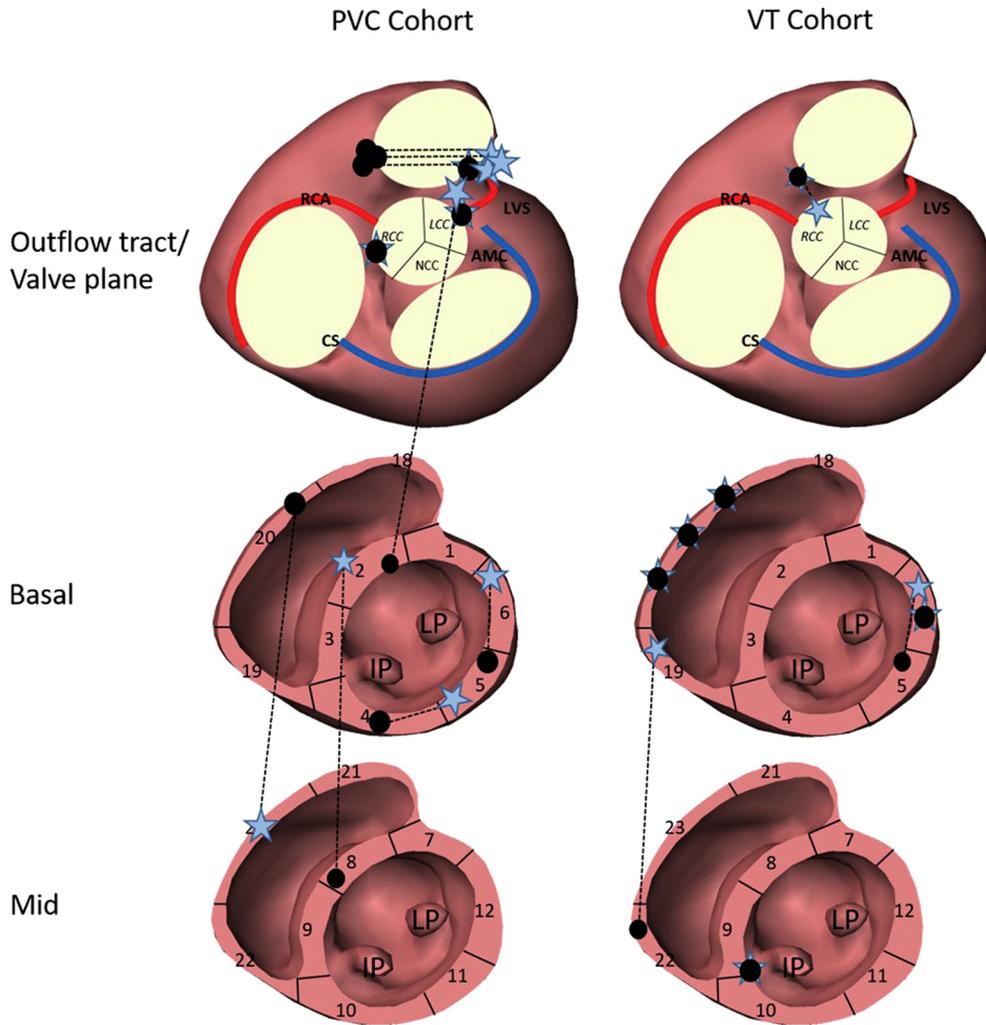


Fig. 4. PVC and VT localization by VIVO and electroanatomic mapping. Black circles represent predicted localization of PVC and VT. Stars represent localization based on invasive electroanatomic mapping. Perfect matches are represented by overlaid black circle and star. Dotted lines link near matches. Non-matches [3] are not included in this schematic.

ventricular activation by using a 3D image of the patient's torso to register electrode position to an imaging-derived, patient specific myocardial model. Using an inverse solution approach, VIVO models ventricular activation and corresponding surface QRS characteristics from nodes throughout the myocardium to identify the best match to a clinical PVC or VT template. In this initial study, we sought to characterize the general accuracy of VIVO for the localization of PVC and VT in patients with both structurally normal and abnormal hearts.

Main findings

The View into Ventricular Onset (VIVO) platform accurately predicted the location of PVC and VT focus in 85% and 88% of patients enrolled respectively. In this study, a near match was considered a match to allow for differences in myocardial models generated within VIVO and the EAM, which led to some variability in specific definition of segments. For predicted and actual PVC and VT foci that fell near the border of a particular segment, for example, these differences may have led to assignment to different adjacent segments despite proximity. Limiting matches to only perfect matches, the accuracy rate fell to 23% and 67% respectively. Excluding cases in which intraprocedural image registration was not performed or the PVC localized to non-segmented structures, the perfect match rate in the PVC cohort was 50%. The accuracy rate of VIVO, however, was substantially better than manual review and localization by electrophysiologists based on the 12 lead ECG, which was 63% and 39% for PVC and VT respectively.

Overall, these findings support further development and investigation of the VIVO platform as a novel clinical tool for ventricular arrhythmia management.

Prediction of earliest activation in ventricular tachycardia

Interestingly, the accuracy of the system was best in the VT cohort which is counterintuitive given the underlying complex myocardial scar in this cohort which was enriched for ARVD patients. Some of the difference in accuracy is likely related to procedural factors in the PVC cohort, in particular the suboptimal segmentation of the thin-walled outflow tracts in VIVO as well as limited use of intraprocedural image registration. However, one advantage of VIVO is the weighting of the initial QRS vector in determining site of earliest activation. For patients with scar related ventricular tachycardia, the initial ventricular depolarization is most likely reflective of the exit site of the re-entrant circuit into normal myocardium and the associated vector cardiogram may be less affected by scar. Additionally, integration of a detailed myocardial model as well as the relative positioning of the heart to ECG electrodes may partially account for scar in that the impacts of the associated anatomic distortions are integrated into the prediction model. This inclusion may also help explain the striking difference observed in accuracy when compared to manual ECG review, which is often based on rules derived from studies of patients with structurally normal hearts.

Table 4
Comparison of VIVO accuracy to manual ECG review.

Patient ID	Actual segment	Predicted segment	Segment match ^a	Reader 1	Reader 1 match	Reader 2	Reader 2 match
VT localization							
5	19	22	2	25	0	27	0
11	20	20	1	22	0	22	0
11	22	17	0	25	2	17	0
14	6	5	2	6	1	5	2
16	9	9	1	4	0	4	0
19	18	20	1	30	0	21	0
21	20	20	1	14	0	26	0
27	6	6	1	6	1	6	1
31	32 and 30	30	1	29	2	18	2
PVC localization							
2	23	20	2	22	2	22	2
8	33	33	1	33	1	1	2
10	31	31	1	30	2	28	2
12	6	5	2	1	2	1	2
12	5	4	2	3	0	3	0
15	8 and 23	18	0	26	2	23	1
17	9	4	0	15	0	15	0
20	31	30	2	30	2	29	0
23	32	32	1	3	0	3	0
25	2	8	2	31	2	31	2
26	31	30	2	30	2	30	2
29	31	2	2	6	0	1	0
30	30	31	2	30	2	28	2

Match: 0 = no match, 1 = perfect patch, 2 = near match.

^a Side Match: 0 = no match, 1 = match.

Strengths of VIVO platform

There are several strategies currently utilized for pre-procedural localization of ventricular arrhythmias to help guide ablation strategy. For both PVC and VT localization, there have been numerous criteria and algorithms proposed that have varying degrees of accuracy [5–9,19]. However, previous studies have shown that this approach can be affected by variability in lead positioning at the time of ECG acquisition [20,21]. Non-invasive mapping using 200+ surface electrodes and some form of myocardial imaging has also been evaluated a tool to identify VT and PVC foci. While studies involving these systems have shown impressive accuracy, the requirement for additional specialized equipment, specifically a multi-electrode vest, is a limitation [10–14].

The novel noninvasive mapping system in this study has some advantages worth noting. First, by using the 12 lead ECG, this platform uses data that is already being acquired. As a result, the only additional equipment requirement is a low-cost 3D camera for acquisition of electrode position on the torso. Second, by using contrast-enhanced pre-procedural imaging, a more accurate myocardial model with definition of endocardial and epicardial surfaces, including the septum, can be generated for analysis. Finally, analysis of ventricular activation vectors, rather than simulated surface potentials, may offer an advantage in identifying the low voltage pre-systolic signals that are frequently targeted during ablation of ventricular arrhythmias.

Opportunities for further development and accuracy improvement

Further work remains to be done to improve prediction accuracy particularly for PVC localization, which are generally very small foci of triggered activity. One limitation of the VIVO platform was that myocardial models generated were observed to be least accurate at the superior and inferior axial limits of the heart. These segmentation errors, which notably affected the outflow tract segmentation in several patients, may have contributed to some of the prediction errors in the PVC cohort. Improvement of the semi-automated segmentation tool would

likely aid in improving prediction accuracy, particularly for VT and PVC originating in the outflow tracts. Of note, predicted sites were on the correct side in nearly all cases. This finding is important as accurate prediction of whether a VA origin is right or left sided would help inform procedural strategies.

In the VT cohort, prediction accuracy was better despite complex ventricular anatomy and scar patterns. This result may be related to more accurate segmentation of the ventricles as opposed to the outflow tract. It is likely that the predicted VT focus would localize best to the exit site in scar mediated VT, particularly given the lack of scar data within the prediction model. This localization could provide useful procedural guidance in terms of a starting point to begin mapping the VT circuit and critical isthmus within the scar. Further studies incorporating scar localization based on imaging could further help refine the predicted VT exit site.

Study limitations

There are several limitations associated with this study. First, several patients had incomplete torso border definition due to radiation dose reduction strategies which limited the field of view during CT acquisition. As such, ECG positioning relative to the myocardial model may have been affected which could have further impacted accuracy. Second, procedural strategies were left to the discretion of the primary operator which introduces some variability in imaging registration. These limitations can be addressed through further software modifications to improve myocardial segmentation and registration of surface electrodes to myocardial models.

Finally, this study utilized a visual assessment of PVC and VT sites in both the predicted model and EAM to define segment localization and therefore matches. However, as this study is the first evaluating the VIVO platform, it was felt to be important to characterize general accuracy as well as opportunities for further refining modeling to optimize accuracy. Based on the promising results of this study, direct co-registration of EAM maps to the VIVO models with distance measurements between predicted and actual foci will be pursued in subsequent studies to further characterize accuracy of the VIVO platform.

Conclusion

The View into Ventricular Onset (VIVO) platform for localization for ventricular arrhythmias offers a unique approach to localization of ventricular arrhythmias. In its current iteration, localization accuracy for PVC and VT sites is promising and warrants further development and investigation.

Disclosures

Peter van Dam is the owner of PEACS BV (Anhem, Netherlands) and is a consultant to Catheter Precision Inc. on the development of the VIVO platform. Catheter Precision Inc. loaned the VIVO platform used in this study.

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