Confirmation of Novel Noninvasive High Density Electrocardiographic Mapping with Electrophysiology Study: Implications for Therapy

Running title: Cakulev et al.; Electrocardiographic Mapping

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Abstract

**Background** - Twelve lead electrocardiograms (ECG’s) have limited value in precisely identifying atrial and ventricular activation during arrhythmias, including accessory atrio-ventricular (AV) conduction activation. The aim of this study was to report a single center’s clinical experience validating a novel, noninvasive, whole heart, beat-by-beat, 3D mapping technology with invasive electrophysiological studies (EPS), including ablation, where applicable.

**Methods and Results** - Using an Electrocardiographic Mapping (ECM) System (CardioInsight-USA) in 27 patients, 3D epicardial activation maps were generated from over 250 body surface ECG’s using heart-torso geometry obtained from CT images. ECM activation maps were compared to clinical diagnoses, and confirmed with standard invasive EPS mapping. 1) In 6 cases of WPW ECM accurately identified the ventricular insertion site of an accessory AV connection. 2) In 10 patients with premature ventricular complexes (PVCs), ECM accurately identified their ventricular site of origin in 8/10. In 2/10 transient PVC suppression was observed during ablation at the site predicted by ECM as the earliest. 3) In 10 cases of atrial tachycardia/atrial flutter (AFL), ECM accurately identified the chamber of origin in 10/10; and distinguished isthmus from non-isthmus dependent AFL. 4) In 1 patient with sustained exercise induced VT, ECM accurately identified the focal origin in the left ventricular outflow tract.

**Conclusions** - ECM successfully provided valid activation sequence maps obtained noninvasively in a variety of rhythm disorders that correlated well with invasive EPS.

**Key words:** ablation; catheter ablation; electrophysiology mapping; imaging; mapping
Introduction

Although widely used as the only noninvasive modality to record the electrical activity of the heart, the diagnostic capability of the 12-lead electrocardiogram (ECG) has acknowledged limits. The surface ECG does not provide precise information on the localization of regional electrical activity in the heart, nor does it provide the activation sequence during atrial and ventricular rhythm disorders.

Because of the above limitations, efforts have been made for more than 30 years to reconstruct epicardial activation sequences from body surface measurements obtained noninvasively, that is, to solve the so-called inverse problem of electrocardiography. This technique of Electrocardiographic Mapping (ECM) has been studied extensively, and has been tested and validated experimentally. (1-3) Our group has participated in this process as well. (4-7) The aim of the present study was to test the hypothesis that noninvasive epicardial activation mapping is a valid method to provide activation sequences in patients with atrial and ventricular rhythm disorders.

Methods

We performed noninvasive mapping of various patient arrhythmias, and the results were validated in a standard fashion with an invasive electrophysiological study (EPS), including subsequent ablation, where applicable.

A total of 27 patients were included in this study. Six patients had Wolff-Parkinson-White syndrome (WPW), 10 had symptomatic premature ventricular complexes (PVCs), one had sustained ventricular tachycardia and 10 had atrial tachycardia or atrial flutter. We excluded atrial tachycardia patients post pulmonary vein isolation or other ablations for atrial fibrillation. All patients completing the ECM protocol described below underwent subsequent
electrophysiologic study (EPS), and, in cases where appropriate, conventional catheter based
activation mapping and ablation. A correlation was made between the locations depicted by the
ECM activation map and the earliest activation and 3 D maps obtained during the EPS, and
further confirmed by an ablative procedure where applicable. To better quantify accuracy, the
correlation between the ECM activation maps and the results obtained during standard EPS was
made by segmenting the atria and ventricles according to the schematics included in the data
supplements.

The study was approved by the University Hospitals Case Medical Center Institutional
Review Board. Written informed consent was obtained from all patients or their legal guardian.

The ECM protocol was performed using the ECVUE system (CardioInsight
Technologies, Cleveland, USA), which noninvasively generated biatrial or biventricular
epicardial atrial or ventricular electrograms and constructed isochrone and isopotential activation
3D maps. The key component of the ECVUE system is a vest embedded with 252 electrodes that
is easily fitted to a patient’s torso. With the vest in position, all patients underwent a thoracic CT
scan to obtain the precise anatomical relationship between the cardiac geometry and the torso
electrodes, which were used to reconstruct 1,500 unipolar electrograms on the epicardial surface
of the heart. (1, 2, 8, 9) The system also computed 3D, color-coded, isopotential, isochronal and
voltage maps, and the dynamic propagation of activation wave fronts. ECM maps were
constructed on a single, beat-by-beat basis, and did not require the accumulation of data from
multiple beats.

The maximal negative slope of the epicardial electrograms determined activation times,
which were used to construct 3-D epicardial isochrone maps. Activation movies for several
consecutive beats were constructed by animating the activation wave front on the patient-specific
CT-derived epicardial surface. On the basis of the isochrone map, where applicable, lines or regions of block were inferred if activation times in adjacent areas differed by more than 50 ms. Slow conduction was represented by crowding of isochrones. A reentrant mechanism of the tachycardia was deduced if at least 90% of the tachycardia cycle length was mapped and head-tail interaction was present. In a tachycardia with a focal mechanism, the earliest and the latest points of electrical activation were anatomically well separated in the chamber being mapped. In addition, the timing from the earliest to the latest activation was less than 60% of the tachycardia cycle length. The site of the earliest ventricular septal activation (left vs. right) in cases where it was not readily apparent on the ECM map was inferred from the initial ventricular activation.

The earliest site of epicardial activation was determined from the isochrone map and from the earliest localized potential minimum in the epicardial potential map. Pure epicardial origin of the arrhythmia or premature beat was defined by a QS complex morphology pattern on the noninvasive ECM electrogram at the site of earliest activation. An endocardial origin was confirmed if the electrogram at the earliest site exhibited an rS morphology. When constructing isopotential maps, propagation of depolarization is shown. The whole process is depicted in Figure 1.

Results
The summary of our studies and outcomes are presented in table 1. ECM accurately predicted the location of interest in 25 out of 27 patients. In the two patients with PVCs in whom ECM was not confirmed successfully during EPS, only transient suppression of the PVCs was noted during ablation at the site where ECM identified the earliest activation.

Patients with WPW syndrome: Of six patients with WPW syndrome, four had ECGs suggestive
of a septal ventricular insertion of the accessory AV connection per published 12 lead ECG based algorithms. (10) In all of these patients, the ECG was indeterminate as far as whether the ventricular insertion was on the left or right side of the ventricular septum. In these patients, ECM accurately predicted whether the ventricular insertion of the accessory AV connection was in the left or the right ventricle, which was confirmed by the EPS. One of these four patients had an epicardial insertion of the pathway that was accurately identified by ECM. A representative example is shown in Figure 2.

Patients with PVCs: Of 10 patients with symptomatic PVCs, six originated from the right or left ventricular outflow tract (RVOT/LVOT) area. ECM accurately localized the ventricular chamber of origin and the earliest site of activation in four out of the six patients. A representative example is shown in Figure 3. Of the two patients with PVCs in whom ECM was not successfully confirmed with EPS, one had PVCs that were transiently suppressed with radiofrequency ablation of the posteroseptal area of the RVOT at the site of a previously repaired ventricular septal defect. In the other, again, only transient suppression of the PVCs was seen during delivery of radiofrequency energy in the LVOT. In both patients, transient suppression of the PVCs during ablation was seen at the sites of earliest onset of electrical activation predicted by ECM. This may not be indicative of ECM inaccuracy, but rather could represent a problem with delivering adequate radiofrequency energy at the site correctly identified by invasive EPS and ECM. Alternatively, both the invasive EPS and ECM maps, although concordant, failed to identify the correct location. In one patient who had an epicardial origin of PVCs, ECM was able to identify the epicardial origin, confirmed by the distinct QS complex morphology of the electrogram at earliest site of activation. (Figure 4).

Interestingly, the ECM was also able to deduce the origin of the PVCs in one patient who
had two simultaneous epicardial breakthroughs. In this patient, two simultaneous, but disparate epicardial breakthroughs were observed on both ventricles. Some of the breakthroughs consistently coincided with the same location of normal epicardial breakthroughs during sinus rhythm. This supported the diagnosis that the PVCs originated from the His- Purkinje system, and that both the left and right ventricles were involved. A representative figure of this example with the detailed explanation is shown in Figure 5. This type of diagnosis would have been much more difficult to deduce with conventional endocardial activation mapping.

One patient presented with sustained ventricular tachycardia. His tachycardia was reproduced during exercise. Because of multiple episodes of non-sustained ventricular tachycardia, ECM was performed. It accurately identified the focal origin of his tachycardia in the LVOT, where the arrhythmia was ultimately successfully ablated between the right and left coronary cusps. It also accurately identified the focal mechanism of this tachycardia.

Two patients had PVCs originating from the LV free wall and ECM accurately predicted the site of the earliest activation.

Patients with atrial tachycardia or atrial flutter: In all 10 patients with an atrial tachyarrhythmia, ECM was able to identify whether the arrhythmia was coming from the left or right atrium, and whether the arrhythmia was focal or reentrant. Five patients in this group had atrial tachycardia involving the left atrium. Representative examples are shown in Figure 6 and 7.

Discussion

Main Findings:

To our knowledge, this is the first report describing the use of ECM technology in a wide range of patients undergoing EPS with or without ablation, in both atria and the ventricles. Our study
showed that ECM precisely identified atrial and ventricular activation during the respective arrhythmias, or during sinus rhythm in the presence of conduction via an accessory AV connection. It also identified the mechanism of the tachyarrhythmia in patients who had atrial tachycardia and in a single (our only) patient with ventricular tachycardia. These findings correlated well with the data obtained from subsequent standard electrode catheter mapping techniques used during electrophysiological studies, further confirmed by successful ablation.

Previously, ECM was extensively validated in canine experiments, which compared reconstructed electrograms to recorded epicardial electrograms.\(^{(2, 4, 5, 11)}\) In addition, ECM has been evaluated by comparison with intraoperative mapping in patients undergoing cardiac surgery, and has demonstrated accuracy within 10 mm \((6.3\pm3.9\text{mm})\) in locating focal activity initiated by pacing. \(^{(12, 13)}\) But, to date, ECM has remained almost entirely experimental, and has not been used as a primary clinical tool for arrhythmia diagnosis and management. The current study is the first account validating its potential application as a primary clinical tool for arrhythmia diagnosis and management in variety of rhythm disorders. We have previously reported a clinical experience in a single patient with ventricular tachycardia.\(^{(7)}\) The present experience shows that this noninvasive technique can be used successfully and consistently in a wide variety of rhythm disorders in patients.

**Specific Findings and Comments:**

ECM accurately identified the site of initial electrical activation and/or the mechanism in rhythm disorders in which this information was critical. This may be beneficial when the findings on the 12 lead ECG are ambiguous. This is especially the case when initial activation of the left or right atrial or ventricular septum is desired. In our study in five patients with an accessory AV connection thought to have a septal ventricular insertion, ECM accurately identified the
ventricular chamber of insertion. Similarly, for PVCs originating in the septal area of the ventricular outflow tract, ECM accurately identified the chamber of origin. Knowledge of the location of the earliest site of activation prior to an invasive procedure in cases were ambiguity exists as to whether the left vs. right heart chamber is involved is very valuable. This can help plan the procedure, and can potentially result in shorter procedure times by focusing the mapping efforts on the heart chamber of interest.

In addition to accurately locating the origin of electrical activity in the heart and identifying the reentrant or focal mechanism of an arrhythmia, ECM provided valuable information as to whether the electrical activation originates in the ventricular epicardium or endocardium. ECM analyzes the unipolar recordings from the epicardial surface of the heart. A pure QS ventricular electrogram morphology on unipolar recordings is indicative of an epicardial origin. We have previously reported a clinical case with an epicardial origin of ventricular tachycardia. In a recent extensive report of this technology in human ventricular arrhythmias, epicardial origin was accurately detected in 100% of the patients with a ventricular tachycardia that originates in the epicardium. In the same report, an intramural initiation site of the ventricular tachycardia was accurately predicted in 88% of the patients, and was supported by an rS morphology of the electrogram at the earliest epicardial site of breakthrough. In our study, only one patient had an epicardial origin of PVCs, and ECM accurately identified it.

In our group of patients with atrial tachycardia, ECM readily identified the putative mechanisms of the arrhythmia (focal vs. reentry). We only had one patient with non-sustained VT and ECM accurately identified focal origin of the tachycardia. Importantly, concerning the identification of the arrhythmia mechanisms, ECM only needs one beat to obtain the full
activation sequence for the relevant beat. This unique feature allows mapping of irregular, and non-sustained rhythms. A report of mapping atrial fibrillation has already been made. (12)

Clinical Implications:
A major limitation of the standard 12-lead ECG is its limited spatial resolution. Multiple existing algorithms attempt to predict the earliest activation site in various arrhythmias, but none is 100% accurate. (14, 15) Significant alterations of the heart’s anatomy and its orientation in the chest, with variable precordial lead placement can yield confusing patterns. With accessory AV connections, the degree of ventricular pre-excitation and presence of multiple antegrade conducting pathways influences the validity of the algorithms. Therefore, definitive diagnosis presently requires an invasive approach.

Invasive EPS is an important clinical tool, and has provided enormous advances in understanding and treating arrhythmias. Recent technological advances have made the creation of three-dimensional electroanatomic and activation maps relatively routine in most of the electrophysiological laboratories. Nevertheless, this approach also has well recognized limitations.

ECM appears to overcome many of these limitations. It can be used noninvasively, relies on the patient’s exact anatomy derived by CT scan instead of a constructed anatomical shell, offers a very precise anatomical activation sequence, elucidates the mechanisms of arrhythmia, and needs only one beat to complete the analysis. This allows for mapping of non-sustained rhythms. The system can also define areas in the heart with respect to the voltage amplitude of the signals it has acquired. In a recent report in human subjects, ECM provided accurate identification of anatomic scar and scar-related electrophysiological characteristics of low voltage, altered sinus rhythm activation, electrogram fractionation, and presence of ventricular
late potentials.(16) All of the above indicates that ECM can be a useful primary diagnostic approach to obtain the desired activation data and identify the mechanism of abnormal rhythms.

**Limitations of ECM technology:**

A potential weakness of the ECM system is that it derives its diagnostic information from the reconstructed electrograms on the epicardial surface of the heart. The endocardial sequence of activation will not always be identical to the epicardial activation sequence, although a recent report showed close correlation between invasive endocardial mapping and noninvasive ECM epicardial imaging. (3) Detectable electrical activation is needed from a corresponding cardiac chamber in order for the analysis to be performed successfully. This may be important in cases where atrial activity or the P wave is “buried” within the inscription of the ventricular activation and is not clearly discernible. In some cases, just by looking at the electrograms of the earliest electrical activation, it is difficult to tell whether earliest activation is on the right or left side of the ventricular septum. Nevertheless, in every case we studied, it was possible to tell whether the activation was on the right or left side of the septum, as the initial propagation of activation was always in the chamber with the origin of the earliest activation. Also, very importantly, ECM requires a CT scan of the chest prior to the analysis, exposing patients to radiation, though quite minimal. Future technological advances likely will allow cardiac MRI to replace the CT scan for anatomic correlation. Finally, the use of ECM in patients with atrial dysrhythmias post pulmonary vein isolation needs to be evaluated. The data collection is in progress and is currently being analyzed.

**Conclusion:**

Our experience described in this report suggests that the use of ECM is not only feasible, but is also highly accurate in describing abnormal cardiac activation. In addition to being noninvasive,
we found that ECM does have some comparative advantages over the other systems currently in use. We have demonstrated that ECM may be quite clinically helpful in planning management strategies, including EPS. Our intention with this report was not to directly compare the ECM system with the conventional approach to plan, map and ablate abnormal rhythms. Results of a randomized study will be necessary to establish precisely the advantages and disadvantages of the ECM system as compared to conventional mapping systems. Nevertheless, our data demonstrate that ECM noninvasively provides very useful clinical information that can potentially be used to help plan invasive electrophysiological studies in patients.

Conflict of Interest Disclosures: Charu Ramanathan, PhD is a paid employee of CardioInsight Technologies. As a paid employee of CardioInsight Technologies she is also a stockholder of CardioInsight Technologies. Ping Jia, PhD is a paid consultant for CardioInsight Technologies. As a former employee of CardioInsight Technologies she is also a stockholder of CardioInsight Technologies. Maria Strom, PhD is a paid employee of CardioInsight Technologies. As a paid employee of CardioInsight Technologies she is also a stockholder of CardioInsight Technologies. Albert Waldo, MD is a consultant for CardioInsight Technologie. The remaining authors have nothing to disclose.

References:


**Table 1.** Summary of patient rhythm abnormalities and accuracy of ECM based on comparison with invasive EPS. In two patients with PVCs, only transient suppression of the PVCs during ablation was seen at the sites of earliest onset of electrical activation predicted by both the ECM and invasive EPS.

<table>
<thead>
<tr>
<th>Rhythm Abnormality</th>
<th>Accuracy of ECM</th>
<th>Case Specifics</th>
</tr>
</thead>
<tbody>
<tr>
<td>WPW</td>
<td>6/6</td>
<td>4 patients had septal insertion of a pathway; 1 patient with left and 3 patients with right ventricular insertion</td>
</tr>
<tr>
<td>PVCs and VT</td>
<td>9/11 (2 patients with PVCs originating from the right outflow tract had only transient suppression during ablation)</td>
<td>6 septal PVC were present. 4 of these 6 PVC originated from the outflow tract. Out of the 4 PVC from the outflow tract, 3 originated in the right and 1 in the left outflow tract.</td>
</tr>
<tr>
<td>Atrial Tachycardia or Atrial Flutter</td>
<td>10/10</td>
<td>3 focal originating in the left atrium</td>
</tr>
</tbody>
</table>

**Figure Legends:**

**Figure 1.** The key component of the mapping system is the 252 electrodes that are embedded in a vest that can easily be placed on a patient torso. With the vest on, a CT scan of the chest
obtains the precise anatomical relationship between the 252 electrodes on the vest and the epicardial surface of the heart. Once this anatomical relation was defined, the ECM algorithm was applied to solve the inverse problem in order to reconstruct 1,500 unipolar electrograms from the 252 electrodes. Out of these 1,500 unipolar electrograms, isopotential, isochronal and voltage maps can than be reconstructed.

**Figure 2:** A) 12 lead ECG of a 35-year-old male with ventricular pre-excitation. Two features are worth mentioning. The first is that the transition in the precordial leads is between V1 and V2. The insertion could be either on the right side or the left side of the ventricular septum although a larger R wave than S wave in lead I may suggest a right-sided pathway. The second feature is that the ECG is not fully pre-excited which again often adds to the ambiguity in determining the ventricular insertion site from the 12 lead ECG. B) ECM isopotential map shows that the site of earliest activation or the ventricular insertion site of the accessory pathway is approximately 1.1 cm off the ventricular septum on the right side. The recording of the electrogram from this earliest site of activation in the ventricles is also shown. This was also the site of successful ablation.

**Figure 3:** A) 12 lead ECG of a 44 year old male with a previously failed right ventricular outflow tract ablation in the right ventricle. The PVC morphology shows a late transition between V3 and V4 (3A). The limb leads display an inferior axis, with the QRS complex being positive in lead I and negative in lead AVL. This would likely place the origin of the PVC in the right ventricular outflow tract, but off the ventricular septum or in the posterior ventricular septum. Because of the late transition on the ECG, the PVC would not be expected to originate
on the left ventricular side. B) Two isopotential maps of the ECM are shown to show the propagation of the depolarizing wavefront. The first panel on the left shows the location of the earliest activation of the PVC in the vicinity of the proximal left anterior descending coronary artery, but on the left side of the ventricular septum. The site of the earliest depolarization is in white color. In both panels of Figure 3B, on the right side of the map, the electograms with a QS complex morphology are shown indicating epicardial origin. The electrograms around, but not at, the focus, lack the sharp negative deflection of the focal electrogram, and exhibit more slurring of the downslope. The verticle lines across the electrograms show the time point at which the potential was measured. The PVC was accurately mapped to this location during the invasive EPS.

**Figure 4.** Activation sequence obtained by ECM of a premature ventricular complex originating from the epicardial surface of the left ventricle. A color-coded scale with a range from 0 - 110 ms is provided for comparison. Left anterior oblique (LAO), left lateral (LL) and postero-anterior (PA) view of the cardiac activation sequence is represented. The earliest site of activation is represented with a yellow dot. The electrogram recording from that site shows a typical QS complex morphology identified by arrows indicating the earliest initial activation in the epicardium. MA – Mitral Annulus, TA – Tricuspid Annulus.

**Figure 5:** A) 12 lead ECG of a 22 year old female with multiple, symptomatic PVCs. She had a previously failed ablation in the right ventricular free wall. In the blue highlighted beats the normal sinus rhythm beat with a right bundle branch block (RBBB) morphology and a normal axis is shown. In the red highlighted beats shown is the morphology of the PVC. The morphology is very similar to that of the intrinsic normal sinus beat, as it also has an RBBB
morphology, an almost identical QRS complex width, but a slightly different axis. B)

Interestingly, the ECM map shows that the PVC has two simultaneous breakthroughs. The yellow and pink dots in the right and left ventricle correspondingly, represent the areas with the most negative potential measured by ECM, and are, hence, the sites of the earliest depolarization. The interrupted white dashed line represents the ventricular septum. At the same time the right and the left ventricles were activated. The right ventricular breakthrough preceded the left by 5 ms. A correlation with the ECM map during the sinus rhythm map was made, and shows that on the right side, the site of the PVC breakthrough was almost identical to the site of the native sinus rhythm breakthrough, while on the left side, they were 7.5 cm apart. Two simultaneous breakthroughs with similar activation patterns as during sinus rhythm suggested an origin of the PVC that invades the His-Purkinje system and activates the right and the left ventricles simultaneously. This would also explain the similar PVC morphology to the intrinsic QRS complex. The recordings of the electrograms from the earliest sites in the right and left ventricles are also shown. Mapping confirmed the earliest PVC activation to be high in the septum. RV – Right Ventricle, LV – Left ventricle

**Figure 6:** A) The ECG of a 72-year-old male after a single lung transplant for idiopathic pulmonary fibrosis. He had incessant atrial tachycardia that failed to be controlled with multiple antiarrhythmic medications. The ECG shows an atrial tachycardia with a P wave morphology that does not suggested any particular site of origin. Note the lack of an isoelectric interval between the P waves, which may suggest a reentrant mechanism of the arrhythmia. B) On the left side of the figure, the ECM map demonstrated focal activation of the left atrium, with the earliest site of activation on the left atrial roof. This area was close to the atrial incision that was
made during the left lung transplant. On the right side of the figure, the CARTO map with the successful site of ablation that terminated the tachycardia and rendered it non-inducible is shown. As can be appreciated, the ECM site of earliest activation matches the site of successful ablation. The activation pattern demonstrates focal activation of the left atrium. LAA- Left Atrial Appendage, SVC – Superior Vena Cava, RSPV, LSPV, LIPV, RIPV – right superior pulmonary vein, left superior pulmonary vein, right inferior pulmonary vein respectively.

**Figure 7:** A) ECG of a 67-year-old male, with a previous left atrial myxoma surgery. The ECG shows an atrial tachycardia with P waves in lead V1 resembling normal sinus rhythm morphology with positive component preceding the negative component. There are also positive P waves in inferior leads. This arrhythmia can be either from the left, or the right atrium. It could be either focal or reentry. B) The ECM isochronal map identified a reentrant circuit around the tricuspid annulus that represented typical clockwise atrial flutter. About 90% of the CL of the tachycardia is mapped. The recordings of the electrograms from the earliest and latest site in the circuit (head-meets-tail) are also shown. The color of the electrograms corresponds to the color of the dots in the map (purple and yellow) representing the sites where these two recordings were made. The tachycardia was confirmed during EPS to be a cavo-tricuspid-isthmus dependent, and was successfully ablated. T- Tricuspid Valve Area, M – Mitral Valve Area, R –Right Atria.
ECM Isopotentials Map
PA View

RVOT
RV
LV
MA
TA
Septum
Apex

Ventricular Insertion site ~ 1.1 cm from the posterior septum

-0.8 mV  0.8 mV

0mV
-4mV
ECM Isopotential Map - AP View

2.0mV

-2.0mV

RV breakthrough
(precedes LV by <5ms)

LV breakthrough

RV

LAD

LV

Apex

Circulation
Arrhythmia and Electrophysiology
ECM Isochrone - AP

RA

LAA

MV

TV

IVC

0 ms

270 ms

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SUPPLEMENTAL MATERIAL:

Figure 1A

1. Superior vena cava
2. Right atrial appendage
3. Anterior superior right atrium
4. Lateral tricuspid annulus
5. Anterior inferior right atrial free wall
6. Lateral superior right atrial free wall
7. Lateral inferior right atrial free wall
8. Posterior superior right atrium
9. Posterior inferior right atrium
10. Left atrial roof
11. Posterior left atrium
12. Left atrium floor
13. Septum
14. Superior mitral annulus
15. Lateral mitral annulus
16. Left atrial appendage
17. Left superior pulmonary vein
18. Left inferior pulmonary vein
19. Right superior pulmonary vein
20. Right inferior pulmonary vein
Figure 1B

Figure 1A and 1B: To better quantify accuracy we divided the atria into 20 segments and the ventricles into 32 segments. We used these segments to correlate our findings obtained during electrophysiology study and standard invasive 3D mapping with the maps created by the ECM system.