Three Dimensional Contrast Enhanced Multi-Detector CT for Anatomic, Dynamic and Perfusion Characterization of Abnormal Myocardium to Guide VT Ablations

Running title: Tian at al.; Tian: 3D CE-MDCT Imaging Guide VT Ablation


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Abstract:

**Background:** Advances in contrast-enhanced multi-detector CT (CE-CT) enable detailed characterization of the left ventricular (LV) myocardium. Myocardial scar/border zone (BZ), as the target of ventricular tachycardia (VT) ablations, displays abnormal anatomic, dynamic and perfusion characteristics during first-pass CT. This study assesses how CE-CT can predict voltage-defined scar/BZ and integrate its scar reconstructions into clinical mapping systems to guide VT ablations.

**Methods and Results:** Eleven patients with ischemic cardiomyopathy underwent contrast enhanced computed tomography (CE-CT) before VT ablation. Segmental anatomic (end-systolic/diastolic wall thickness [ESWT/EDST]), dynamic (wall thickening [WT], wall motion) and perfusion (hypoenhancement) characteristics were evaluated. Receiver-operating characteristic curves assessed CT’s ability to determine voltage-defined scar and BZ segments. 3D epi- and endocardial surfaces and scar borders were reconstructed, co-registered and compared to voltages using a 17-segment model. Abnormal anatomic, dynamic and perfusion data correlated well with abnormal (<1.5mV) endocardial voltages (r=0.77). 3D reconstruction integrated into the clinical mapping system (registration accuracy 3.31±0.52 mm) allowed prediction of homogenous abnormal voltage (<1.5mV) in 81.7% of analyzed segments and correctly displayed transmural extent and intramural scar location. CT hypoperfusion correlated best with scar and BZ areas and encompassed curative ablations in 82% cases.

**Conclusions:** Anatomic, dynamic and perfusion imaging using CE-CT allows characterization of LV anatomy and 3D scar/BZ substrate. Integration of reconstructed 3D datasets into clinical mapping systems supplements information of voltage mapping and may enable new image approaches for substrate-guided VT ablation.

**Key words:** Ablation; 3D Imaging; Contrast Enhanced Multi-Detector Computer Tomography; Image Reconstruction; Ventricular Tachycardia
List of abbreviations:

VT – Ventricular Tachycardia
CE – Contrast Enhanced
CT – Computed Tomography
MDCT – Multi-detector CT
CE-MR – Contrast Enhanced Magnetic Resonance
3D – Three Dimensional
LV – Left Ventricle
VA – Visual Alignment
LM – Landmark point registration
SF – Surface shell registration
MV – Mitral Valve
ROC – Receiver Operating Characteristics
AUC – Areas under the curve
INTRODUCTION

An increasing number of patients with internal cardiac defibrillators (ICD) present with frequent and appropriate shocks for ventricular tachycardia (VT). As the use of antiarrhythmic medications is frequently limited by its side effects and decreasing long-term efficacy, radiofrequency ablation of VT is required in many of these patients. Multiple VT morphologies and hemodynamic intolerance require a “substrate guided” approach in 60-90% of patients.\(^1,2\) In these approaches, linear ablation lines are placed across and along the myocardial scar and its border zone to interrupt conducting channels of surviving myocardium.\(^1,3,4\) Endocardial voltage mapping is frequently used to define the scar substrate, but has a limited ability to detect intramyocardial or epicardial scar. Additionally, suboptimal catheter contact can result in falsely low voltage measurements.

The purpose of this study was to compare the CT-derived anatomic, dynamic, and perfusion characteristics of scar/ border zone with standard voltage mapping and to assess the feasibility of integrating 3D CT scar/border zone reconstructions into clinical mapping systems to guide VT ablations.
METHODS

Eleven consecutive ICD patients with an ischemic cardiomyopathy scheduled to undergo VT ablation were enrolled. Coronary artery disease was assessed as clinically indicated. Written informed consent was obtained from the patients. Protocols were reviewed and approved by the University of Maryland Institutional Review Board.

64-Multidetector CT (MDCT) Imaging

A 64-MDCT system (Brilliance 64, Philips Medical System) was used to obtain CT images during breath-hold. Total scan times were 10-15 secs. IV metoprolol (5-15mg) was used for heart rates above 75 beats per minute (bpm). The CT data from the initial series was retrospectively reconstructed into ten equally spaced phases of the R-R interval (from early systole to end diastole). The data were transferred to a dedicated Philips workstation (Extended Brilliance workspace, V3.5.0.2254). To minimize motion, end-systole/end-diastole CT images were reconstructed at 40% and 80% of the R-R interval phases based on literature review and validation in a recent cohort of VT patients.

Extraction of Anatomic, Dynamic, and Perfusion CT Parameters

Regional anatomic (end-systole wall thickness [ESWT], end-diastole wall thickness [EDWS]) and dynamic (wall thickening [WT], wall motion [WM]) datasets were extracted from end-systole (40%) and end-diastole (80%) CT images using pre-commercial Phillips software (Brilliance 3.5.0.2254) according to the AHA 17-segment model. Wall motion was defined as the change of the radial distance between the center position of the endocardium at ED and ES in the radial direction. Average ESWT, EDWT, WT, and WM in each segment were collected and compared between ED and ES
[Figures 1A, 1B, 2A]. Perfusion defects were visualized in 2D images and assigned a regional location according to the 17-segment AHA model by an experienced radiologist using the ED (80%) CT images. CT scar border was defined as transition from hypoperfused myocardium to normally perfused myocardium.

**Voltage Map Dataset**

All voltage maps were created using a clinical 3D-mapping system (CartoMERGE, Johnson & Johnson, NY, USA) and a 3.5mm Navistar cooled-tip catheter (filling threshold of ≤15mm). Bipolar electrograms were filtered at 10-30 to 400-500Hz. Standard clinical voltage criteria were used to define scar (<0.5mV), border zone (BZ, 0.5mV-1.5mV) and normal (>1.5mV) myocardium.1 In areas of abnormal voltage recordings (<1.5mV), fluoroscopy, electrogram characteristics and real-time intracardiac echocardiography (ICE) were used to confirm stable catheter contact. Mapping points with fractionated potentials, diastolic potentials within the scar areas and the location of successful ablation sites were documented.

For quantitative analysis, voltage maps were exported from the mapping system and were divided into segments according to the AHA 17-segment model [Figure 1D].9 The average, minimum, and maximum voltage measurements in each segment were calculated. Voltage segments containing only voltage measurements <0.5mV were defined as “scar”; segments with only voltage measurements between 0.5mV and 1.5mV were defined as “border zone” and segments containing voltage measurements from 0-1.5mV and no values greater than 1.5mV were defined as “mixed - scar and border zone”. Segments with all voltage measurements > 1.5mV were defined as “normal”. Myocardial segments belonging to any of these four groups were considered homogenous.
(all voltage measurements in the pre-specified range) and were compared with their corresponding CT parameters in order to characterize their anatomic and dynamic features. Homogenous scar, BZ, and mixed segments were summarized as “abnormal” segments when compared to normal (>1.5mV) areas. Segments with some voltage values <1.5 mV AND >1.5 mV were considered heterogeneous and were excluded from the further segmental analysis.

**Comparison of Anatomic, Dynamic and Perfusion Parameters between Voltage-Defined Abnormal and Normal Myocardium Segments**

Mean and standard error (SE) for anatomic (ESWT, EDWT) and dynamic (WT, WM) parameters in each segment were calculated and compared between abnormal and normal homogeneous voltage segments.

ROC curves were used to assess the ability of CT-derived parameters to determine myocardial abnormality. Bipolar voltages were used as the gold standard with 1.5mV as the cut off value to define abnormal myocardium. Generalized estimating equations (GEE) were used to identify the most significant CT-derived parameter(s) in predicting the voltage defined categories.

17-segment analyses were conducted for all of the voltage maps and 2D CE-CT slices with hypoperfusion areas. Hypoperfused segments were examined in both voltage maps and CE-CT images to calculate their location matching percentage.

**3D CE-CT Image Reconstruction**

CE-CT images were reconstructed into mesh files with Amira 5.2.1 (Visage Imaging, Inc., San Diego, CA) using ED (80%) images to best correlate with the Carto point map by a radiologist blinded to the voltage data. Epicardial/endocardial LV
surfaces, endocardial RV surfaces, hypocontractility segments and hypoperfusion areas were reconstructed separately. Mesh files were converted into the Carto XP readable mesh format using MATLAB R2009a (The Mathworks Inc., Natick, MA) and imported into the Carto XP mapping system. The reconstructed images were registered with voltage maps using pre-commercial CartoMERGE software.\textsuperscript{10} Primary registration was performed with Landmark points [LM] and visual alignments [VA] as previously published.\textsuperscript{10} Scar surface area and LV scar burden (scar surface area/total LV surface) were compared between the voltage map and 3D CE-CT reconstructions with the internal CartoMERGE measurement tools.

**VT Ablation**

The stimulation protocol was performed from the RV apex and outflow tract as well as up to two LV sites (if not inducible from RV) using three drive train cycle lengths (350ms, 400ms, 600ms) and 1, 2 and 3 extrastimuli with minimal coupling interval of 200ms. Substrate-modification was performed as clinically indicated using linear lesion sets along the scar border. Information about the LV anatomy, scar and BZ derived from CE-CT was used for supplementary characterization of the VT substrate and to assist in therapy planning. Clinical VTs (documented by 12 lead ECG) or presumed clinical VTs (defined by cycle lengths/morphology of the ICD recordings) were the target of the ablation procedures.

**Statistics**

SPSS for Windows 16.0 was used to perform the statistical analysis. Continuous variables are expressed as mean ±1 SD or median and range or otherwise specified. Comparisons were conducted with one-way ANOVA for different registration strategies.
GEE were used to compare the measurements for ESWT, EDWT, WT and WM and to identify the most significant CT-derived parameter(s) in predicting the voltage defined categories. Differences were considered significant at a level of $P<0.05$. ROC curves were created for each of the four CT-derived parameters in predicting voltage defined abnormal segments. Areas under the curve (AUC) for each parameter were reported and compared. Pearson correlations were computed to investigate the relationship between EP and CT-derived myocardial attributes. In particular, abnormal LV myocardium area was correlated between CE-CT reconstruction and voltage mapping, and also abnormal LV myocardium burden was correlated between CE-CT and voltage mapping.

The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

RESULTS

Patient Characteristics

All patients were men with an average age of $65\pm9$ years (Table 1). All 11 patients had ischemic cardiomyopathy and previous MI, evidenced by the documented ECG. Previous MIs were located at inferolateral (N=2), inferior/anterior (N=4), inferior (N=4) and anteroseptal wall (N=1), respectively. Heart rates during the CT scan were $62\pm6$ bpm. Radiation exposures were $12.35\pm3.67$ mSv.

Electrophysiological Mapping

Endocardial maps were created using a retrograde aortic approach in all patients with $275\pm109$ mapping points. All patients had scar, BZ, and/or mixed scar/BZ segments.
Comparison of Anatomic and Dynamic and Perfusion Parameters between Voltage-Defined Abnormal and Normal Myocardium Segments

40% of segments were homogenous (N=71) and used for further analysis, including 43 normal segments and 28 abnormal (5 scar, 3 BZ and 20 mixed scar and BZ) segments. There were $15 \pm 11$ (median 14, range 1-43) mapping points in each segment [Figure 1D].

Anatomic Parameters: ESWT and EDWT were $6.5 \pm 0.75$mm (SE) and $6.6 \pm 0.94$mm for abnormal segments, and were $10.1 \pm 1.8$mm and $12.9 \pm 1.7$mm for normal segments, respectively (P<0.001 for ESWT, P=0.01 for EDWT) [Figure 1A, 1B, 1C].

Dynamic Parameters: WT and WM were $0 \pm 4.4$% and $1.1 \pm 0.4$mm for abnormal segments, and were $32.7 \pm 5.5$% and $3.5 \pm 0.92$mm for normal segments, respectively. All parameters differ significantly between abnormal and normal segments (P<0.001 for WT, P=0.01 for WM) [Figure 2A, 2B].

Perfusion Parameters: Sixty-seven (38%) out of total one hundred and seventy six segments showed hypoperfusion. Fifty-two segments (78%) of the 3D CE-CT hypoperfusion segments matched with those abnormal voltage segments [Table 2].

Prediction of Abnormal Voltage Segments with Anatomic and Dynamic Parameters

AUC for parameters of ESWT, EDWT, WT and WM were $0.83 \pm 0.05$, $0.75 \pm 0.06$, $0.79 \pm 0.06$ and $0.68 \pm 0.06$ [Figure 3]. ESWT had a significantly better discrimination value than WM to identify the abnormal voltage segments when comparing the AUC (P=0.04). There was no significant difference between ESWT and WT in predicting the voltage categories (P=0.58).
GEE bivariate analyses demonstrated that ESWT, EDWT, WT and WM were all significantly correlated with the presence of the abnormal voltage segments (P=0.005, P=0.025, P=0.001, P=0.019 for each of the four variables). Using ESWT and WT jointly (AUC=0.85±0.05) has a better discrimination value than WM to identify the abnormal voltage segments (P= 0.03) [Figure 3]. This joint model allowed the correct classification of 81.7% of the segments.

**Integration of CT- Derived 3D Cardiac Anatomy with Embedded Anatomic, Dynamic, and Perfusion Scar/Border Zone into Clinical Mapping System**

The LV reconstruction allowed the 3D display of the complete LV endocardium/epicardium in the clinical mapping system[Figure 1C].

Mild-to-moderate ICD artifacts were seen in the septal wall in all cases extending to the inferior wall [Figure 4C]. Successful reconstruction of the LV anatomy and scar/BZ was achieved in all cases.

CE-CT derived 3D LV anatomy could be accurately registered and displayed. VA demonstrated a position error of 3.72±0.58mm [Table 3]. The addition of surface shell registration (SF) improved the registration accuracy to 3.41±0.79mm. While LM alone resulted in a position error of 3.68±1.53mm, the combination with SF enhanced the position error to 3.31±0.52 mm [Table 3]. Accuracy of the four registration strategies did not differ significantly (p=0.13).

3D CE-CT reconstructions of the LV wall and the segments with low wall thickening values demonstrated areas and segments with low contractility including ventricular aneurysms [Figure2A, 2B].
CE-CT perfusion imaging demonstrated location and transmurality of hypoperfused myocardium. This allowed the assessment of its transmural extent and the differentiation in endo-, mid- and epicardial scar components (Figure 4A, 4B). After 3D reconstruction of endo/epicardial borders and integration of 3D hypoperfused myocardium, this provided a detailed appreciation of the 3D scar anatomy during the VT ablation (Figure 5A).

All patients (100%) were found to have perfusion defects [Figure 4] and 3D CE-CT reconstructions from hypoperfused myocardium were successfully embedded in the LV myocardium [Figure 5A]. One patient (9%) was found to have calcifications and layered endocardial thrombus [Figure 4B]. The average voltage value in this area was 0.64±0.23mV [Figure 4].

**Comparison of Voltage Map with 3D DE-CT Hypoperfusion Reconstruction**

The size of the 3D CE-CT hypoperfusion reconstruction area correlated best with abnormal voltage area (scar + BZ) [Figure 5C, 5D].

Voltage mapping demonstrated an abnormal voltage area of 97.7±41.3cm² representing 37.4±11.4% of the LV. The 3D CE-CT hypoperfusion map demonstrated a myocardial hypoperfusion area of 88.2 ± 36.3 cm² with an abnormal LV myocardium burden of 33.3±8.5%. Abnormal LV myocardium area (r=0.77, P=0.006) and abnormal LV myocardium burden (r=0.63, P=0.04) correlated well between CE-CT hypoperfusion reconstruction and abnormal voltage area. Voltage measurements within the CE-CT hypoperfusion reconstruction area demonstrated a voltage amplitude of 1.14± 0.23 mV compared with 8.4±5.1mV in the area with normal myocardium perfusion (p<0.001).
VT Ablation:

An average of 2.2±1.5 VTs were inducible during programmed electrical stimulation. Based on 12-lead morphology of the clinical VT or rate/morphology of the ICD recordings, 16 VTs were deemed to represent clinical or presumed clinical VT and were targeted for ablation. 69% of the clinical VTs have RBBB morphology with right inferior (n=8), right superior (RS, n=2) and left superior (n=1) origin. The remaining 31% have LBBB morphology with RS (n=3), and left inferior (n=2) origin. Cycle lengths were 230-640ms. RF ablation rendered 15 of 16 (94%) clinical/presumptive clinic VTs non-inducible. One patient remained inducible. Five VTs of unclear significance from five patients were inducible after the ablation. One patient was not tested after ablation due to hemodynamic instability.

Activation/entrainment mapping was used in 2 VTs; and pace-mapping and limited activation mapping was used for the remaining 14 clinical VT due to hemodynamic disability. Successful ablation sites of 2 entrainable VTs were within 1cm of the CT-defined border. Pace-mapping points indentifying the exit site of clinical VT were located within 1cm from CT-defined border in 10 (91%) patients (Figure 5B).

No complications occurred in any of the eleven ablation procedures.

Patient Follow Up

The median follow-up time was 12 months (range: 4-37 months). Five (45%) patients received two recurrent ICD therapy (ATP and/or shock) at a median follow-up time of 10 months (range: 2 -18 months) after the first ablation. Of those, three patients (27%) underwent a second VT ablation at a median of 7 months (range: 2 -10 months)
after the first ablation. There was one (9%) death within 6 month of follow-up period due to chronic heart failure and there were 4 deaths (36%) within 12 months.

DISCUSSION

The novel findings of this study are that 1) anatomic, dynamic and perfusion parameters derived from a single CE-CT scan allow a comprehensive characterization of scar/border zone, 2) among anatomic and dynamic parameters, ESWT and WT jointly were the best predictors for the presence of the abnormal voltage segments 3) areas of CT hypoperfusion correlate best with areas of abnormal voltage (<1.5mV rather than scar (<0.5mV) alone and 4) 3D CT-defined abnormal myocardium can be accurately extracted and embedded in clinical mapping systems displaying areas of abnormal anatomic, dynamic and perfusion parameters to guide substrate-guided VT ablations.

Currently, image integration for VT ablation consists mostly of endocardial surface reconstruction. Additional information about the scar substrate available from cardiac imaging needs is currently not integrated into the mapping system and could provide better guidance for VT ablations. While the current VT ablation strategies based on voltage-defined criteria are well established, the 6 months success rate from recurrent VT is only 53%. Correlation of scar geometry obtained by cardiac imaging and correlating VT circuits was demonstrated in recent animal studies and has been shown in histological series in patients with ischemic VT. As such, an image-guided approach has the potential to facilitate and improve substrate-based ablations.

Echocardiography can provide anatomic and contractile parameters. However, they cannot provide currently relevant clinical information about transmural extent and intramyocardial location of the scar. Delayed-enhanced magnetic resonance imaging
(MRI) is able to accurately characterize the location and extent of myocardial scar.\textsuperscript{13, 14} However, the presence of an ICD is still considered as a contraindication in the majority of patients requiring a VT ablation.\textsuperscript{15} While PET (Positron Emission Tomography) provides a metabolic characterization of the myocardial scar and its BZ, the spatial resolution of $\geq 6$mm and its restricted availability in cardiology practices limits its applicability.

Cardiac CT, on the other hand, is a rapidly evolving technology with no such contra-indications and high spatial (up to $\leq 1$mm) and temporal resolution. CT enables a detailed and comprehensive evaluation of LV myocardium using three different and complementary pathophysiological characterizations of myocardial scar/ BZ, which can be derived from a single CT scan. Perfusion imaging from CT could indicate scar transmurality and intramyocardial scar location. CT visualization of alive epicardial myocardium that cannot be detected by endocardial voltage mapping may identify areas able to sustain reentrant VT and may suggest an epicardial ablation approach. This study used triple, multi-modality imaging based on anatomic, dynamic, and perfusion parameters to identify abnormal substrate, which is the target for the majority of VT ablation.

**Regional Anatomic, Dynamic Parameters and Perfusion Characterization for Ventricular Tachycardia Ablations**

Multidetector CT imaging has been used to assess the regional LV function,\textsuperscript{7,16,17} and permits accurate, non-invasive assessment of LV function and perfusion in patients with previous infarction.\textsuperscript{17} Previous research found a significant correlation between MDCT and MRI for anatomic LV parameters such as ESWT, EDWT and dynamic LV
parameter of WT and concluded that MDCT can accurately estimate regional LV wall thickness and wall thickening.18, 19

In this study, all the regional LV anatomic and dynamic CE-CT parameters differed significantly between abnormal and normal voltage segments. Multivariate stepwise logistic regressions demonstrated that the statistical prediction model including ESWT and WT resulted in an AUC of 0.85±0.05, which correctly classified 81.7% of the voltage segments into abnormal category. This was consistent with the previous research where 88.3% segments with decreased wall motion were correctly identified by MDCT.7

The current “gold standard” of voltage mapping has several limitations: a single endocardial voltage measurement only incompletely describes a complex intramural scar anatomy.12 Detailed voltage maps prolong the procedure time and falsely-low voltage measurements (due to suboptimal catheter contact) can lead to incorrect scar definition. Additionally, small areas of scar might not be detected given the spatial resolution of ≥5mm covered by the 3.5mm catheter tip/proximal ring distance.

Many of these limitations could be overcome by integrating scar imaging into the VT ablations. CE-CT imaging has been used to identify myocardial infarction in both animal and human studies.7, 17, 20-25 When compared with contrast enhanced magnetic resonance (CE-MR)13 absolute sizes of early hypoperfused and late hyperenhanced regions were similar on CE-CT and CE-MR.15, 26

As well-delineated delayed hyperenhancement was seen only in a minority of our patients hypoperfusion was used for scar extraction in this study. A good correlation between areas of abnormal myocardium with decreased voltage amplitude (<1.5mV) and
CE-CT hypoperfusion was found. 78% of the 3D CE-CT hypoperfusion segments matched with those abnormal voltage segments.

There are several possible explanations for the scar mismatches between MDCT and voltage mapping. MDCT could be more sensitive in demonstrating scar as a non-transmural, mid-myocardial or epicardial scar might influence the segmental wall thickness, contractility and myocardial perfusion. Surviving endocardial components or far-field from surviving mid/epicardium may be able to create normal or near-normal endocardial voltages. Indeed, about 4% of successful ablation sites are located within healthy myocardium defined by voltage mapping, which could possibly represent non-endocardial scar. Conversely, low voltage recordings in normal anatomic, dynamic and perfusion myocardium most likely represent falsely-low voltage recordings due to suboptimal catheter contact. Finally, registration errors may lead to the misrepresentation of the scar location in their adjacent segments.

Areas of hypoperfusion appeared to correlate best with areas of abnormal endocardial voltage (<1.5mV) rather than scar alone (0.5mV). A possible explanation is that BZs that demonstrate on histology a mixture of fibrosis and surviving myocardium may have already altered perfusion characteristics that can be detected with MDCT.

**Image Integration of 3D Scar Maps**

In this study we were able to display 3D LV anatomy with embedded hypoperfused myocardium indicating scar area based on the CE-CT imaging. LV scar area burden correlated well between the voltage-defined scar and 3D scar reconstruction from CE-CT. This is consistent with findings from both animal and human studies indicating the microvascular obstruction area characterized by hypoperfusion correlated well with the
scar area indicated by reduced flow regions as measured by microsphere blood flow. In this study integration resulted in an immediate pre-procedural anatomy/substrate display with sub-millimeter resolution otherwise only available after detailed endocardial voltage mapping. Using first pass MDCT a comprehensive scar characterization using anatomic, dynamic and perfusion data was possible.

Of special interest in the ability of CE-CT to characterize the transmural extent and intramyocardial location of scar tissue. The visualization of surviving mid-and epicardial myocardium at sites of endocardial scar could help identify areas involved in myocardial reentry representing appropriate ablation targets. This would help to overcome one of the significant limitations of endocardial voltage mapping. Additionally, the presence of an epicardial VT substrate may facilitate planning of VT ablations such as for a combined endocardial and epicardial approach. It could also supply information helpful in differentiating low epicardial voltages due to fat or scar formation. With improved understanding of scar geometry, a pre-procedural modeling of anatomically fixed circuits and likely successful ablation sites may become increasingly possible.11

LIMITATIONS

The presence of ICDs resulted in mild-to-moderate metal artifacts on CE-CT images. However, the artifacts were limited and mostly located in the septal wall with only occasional extension into the inferior wall. While no obvious effect on the extracted anatomic, dynamic and perfusion information was noted, it cannot be excluded.

Radiation was minimized and comparable to exposure in other studies. A further reduction could possibly result in an average dose around 4.3mSv with new reduction strategies.
Only 40% of all the segments were homogeneous and included in the analysis. Future software developments will allow an increase in the percentage of homogenous segments by decreasing the LV segment size.

Given the relatively large size of the individual segments when using the validated 17-segment myocardial analysis most segments contained voltage measurements of scar and BZ. The small number of exclusive scar or BZ segments did not allow a meaningful analysis for each individual single voltage category at the current time.

The relative small sample size limited the generalizability of this study, which will need to be confirmed in future randomized trials.

CONCLUSIONS

To our knowledge this is the first study to assess a comprehensive three-modality characterization (anatomic, dynamic and perfusion) of LV scar from a single CE-CT and demonstrate the ability to integrate this information into clinical mapping systems to guide VT ablation. Our results indicate that regional LV anatomic, dynamic and perfusion parameters can be utilized to correctly predict abnormal voltage locations in advance of the mapping procedure. This may allow the electrophysiologist to concentrate on areas of likely myocardial scar and identify false low voltage recordings in areas of normal perfusion due to suboptimal catheter contact. This novel approach has the potential to significantly facilitate substrate-based VT ablations.

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Conflict of Interest Disclosures: Dr. Dickfeld reports receiving research support from Biosense, Webster, Israel. Mr. Turgeman and Dr. Abbo are employed by Biosense Webster, Israel.

Reference:


TABLES:

Table 1. Patient Characteristics (N=11) at Time of Ventricular Tachycardia Ablation

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Table 2. Abnormal Segment Location according to 17 Segment AHA model

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Table 3. Position Error. Individual average position error for each patient after different registration algorithms (LM - landmark point registration, SF – surface registration, VA - visual alignment)

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<td>3.12</td>
</tr>
<tr>
<td>10</td>
<td>5.69</td>
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<tr>
<td>11</td>
<td>1.22</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>3.68±1.53</td>
</tr>
</tbody>
</table>
FIGURE LEGENDS:

**Figure 1.** Anatomic Information of the CE-CT Images. A. Short axis view of the CE-CT image. Green and the yellow lines delineate endocardial and epicardial contours. The anterior-septal wall (arrow) is thinner than the lateral/inferior wall, indicating anterior-septal scar. The bright areas in the septal and lateral wall are the ICD artifacts (arrows) B. 17-segment analysis for the End-Systole Wall Thickness (ESWT). Values for ESWT are presented in the 17-segment model format with color coding indicating anterior-septal scar with decreased ESWT values (purple segments, arrow). C. 3D Reconstruction for Anatomic Information. Short axis view of the 3D LV reconstruction of CE-CT image with epi-and endocardial surfaces. Arrows indicating anterior-septal wall thinning corresponding to areas seen in 1A. D. 17-segment analysis of voltage map. Voltage map indicates anterior-septal scar with small voltage values (red area, arrows). Consistent with the wall thinning areas demonstrated in 1A, 1B and 1C. Apical cap was excluded from analysis.

**Figure 2.** Dynamic Information of the CE-CT Images. A. 17-segment analysis of Wall Thickening (WT). Values for WT were extracted and presented in the 17-segment model format with color scale indicating anterior-septal scar with decreased WT values (purple segments, arrow). B. 3D Reconstruction for Dynamic Information. 3D LV reconstruction of CE-CT image with endocardial surface (turquoise) and myocardial display of low contractility segments (yellow) corresponding to wall thinning as shown in 2A.

**Figure 3.** Receiver-Operator-Characteristic (ROC) curves for Anatomic and Dynamic Parameters. Plots of ROC curves for ESWT (blue), EDWT (brown), WT (green) and WM (purple) to predict abnormal voltage segments as defined by <1.5mV for all the voltage points in the segment. Areas under curve (AUC) for each parameter were displayed by the figure.

**Figure 4.** First-Pass Contrast-Enhanced CT. A: Transmural perfusion defect located in the apical and apical-septal wall (arrow). B: Delineation of endocardial, non-transmural
perfusion defect of ~25% located in the anterior wall (black arrow). White solid arrows indicate epicardium and RV endocardium border. White dashed arrows pointing to the border of the endocardium perfusion defect (CT defined scar) C: Wall thinning with extensive intramural calcification (arrow) in the anterior apical wall.

**Figure 5.** A. Fusion of 3D LV Anatomy and 3D Hypoperfusion Reconstruction. The LV reconstruction is seen from cross-sectional view allowing visualization of endocardium (purple) and epicardium (blue) surfaces. Areas in solid brown color represent endocardial scar delineated by perfusion imaging from patient shown in Figure 4B. B. ≥11/12 pace mapping match. Ventricular tachycardia with corresponding ≥11/12 pace match (identical QRS morphology between paced beats and VT) This area denotes a potential site for successful ablation. C: Corresponding voltage map demonstrating anterior abnormal myocardium (scar and border zone, arrow) D: 3D reconstruction of non-transmural, hypoperfused scar (black mesh) superimposed on the voltage map. Pace-mapping (brown marker indicated by white arrow) is located at the border zone of the 3D reconstruction of the scar from perfusion defect in CT image in both 5C and 5D.
Three Dimensional Contrast Enhanced Multi-Detector CT for Anatomic, Dynamic and Perfusion Characterization of Abnormal Myocardium to Guide VT Ablations
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