Three-Dimensional Contrast-Enhanced Multidetector CT for Anatomic, Dynamic, and Perfusion Characterization of Abnormal Myocardium To Guide Ventricular Tachycardia Ablations

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

Methods and Results—Eleven patients with ischemic cardiomyopathy underwent contrast-enhanced CT before ventricular tachycardia ablation. Segmental anatomic (end-systolic and end-diastolic wall thickness), dynamic (wall thickening, wall motion), and perfusion (hypoenhancement) characteristics were evaluated. Receiver operating characteristic curves assessed the ability of CT to determine voltage-defined scar and BZ segments. Three-dimensional epi- and endocardial surfaces and scar borders were reconstructed, coregistered, and compared to voltages using a 17-segment model. Abnormal anatomic, dynamic, and perfusion data correlated well with abnormal (>1.5 mV) endocardial voltages (r=0.77). Three-dimensional reconstruction integrated into the clinical mapping system (registration accuracy, 3.31±0.52 mm) allowed prediction of homogenous abnormal voltage (<1.5 mV) 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 contrast-enhanced CT allows characterization of left ventricular anatomy and 3D scar and BZ substrate. Integration of reconstructed 3D data sets into clinical mapping systems supplements information of voltage mapping and may enable new image approaches for substrate-guided ventricular tachycardia ablation. (Circ Arrhythm Electrophysiol. 2010;3:496-504.)

Key Words: ablation techniques ■ imaging three-dimensional ■ computerized emission tomography ■ image processing computer assisted ■ tachycardia ventricular
3D CE-MDCT Imaging To Guide VT Ablation

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Regional myocardial function.5–7 Regional left ventricular (LV) wall motion (WM) expressed by dynamic changes in myocardial wall thickness has been used to reveal detailed information on the functional state and viability of ischemic and nonischemic myocardium.5–7 This study compared the CT-derived anatomic, dynamic, and perfusion characteristics of scar and BZ with standard voltage mapping and assessed the feasibility of integrating 3D CT scar and BZ reconstructions into clinical mapping systems to guide VT ablations.

Methods

Eleven consecutive patients with ischemic cardiomyopathy and an ICD 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 (Baltimore, Md) Institutional Review Board.

64-Multidetector CT Imaging

A 64-multidetector CT (MDCT) system was used to obtain CT images during breath hold. Total scan times were 10 to 15 seconds. IV metoprolol (5 to 15 mg) was used for heart rates above 75 bpm. The CT data from the initial series was retrospectively reconstructed into 10 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 version 3.5.0.2254). To minimize motion, end-systole and end-diastole CT images were reconstructed at 40% and 80% of the R-R interval phases based on a review of the literature8 and validation in a recent cohort of patients with VT.

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], WM) data sets were extracted from end-systole (40%) and end-diastole (80%) CT images using precommercial Philips software according to the American Heart Association 17-segment model.9 WM was defined as the change of the radial distance between the center position of the endocardium at end diastole and end systole in the radial direction. Average ESWT, EDWT, WT, and WM in each segment were collected and compared between end diastole and end systole (Figures 1A, 1B, and 2A). Perfusion defects were visualized in 2D images and assigned a regional location according to the 17-segment American Heart Association model by an experienced radiologist using the end-diastole (80%) CT images. CT scar border was defined as transition from hyperperfused myocardium to normally perfused myocardium.

Voltage Map Data Set

All voltage maps were created using a clinical 3D mapping system and a 3.5-mm Navistar cooled-tip catheter (filling threshold, ≤15 mm). Bipolar electrogroms were filtered at 10, 30, 400, and 500 Hz. Standard clinical voltage criteria were used to define scar (<0.5 mV), BZ (0.5 mV to 1.5 mV), and normal (>1.5 mV) myocardium.1

In areas of abnormal voltage recordings (<1.5 mV), fluoroscopy, electrogram characteristics, and real-time intracardiac echocardiography were used to confirm stable catheter contact. Mapping points with fractionated potentials, diastolic potentials within the scar areas, and location of successful ablation sites were documented.

For quantitative analysis, voltage maps were exported from the mapping system and divided into segments according to the American Heart Association 17-segment model (Figure 1D).9 The average, minimum, and maximum voltage measurements in each segment were calculated. Voltage segments containing only voltage measurements <0.5 mV were defined as scar; segments with only voltage measurements between 0.5 mV and 1.5 mV were defined as BZ, and segments containing voltage measurements from 0 to 1.5 mV and no values >1.5 mV were defined as mixed scar and BZ. Segments with all voltage measurements >1.5 mV were defined as normal. Myocardial segments belonging to any of these 4 groups were considered homogenous (all voltage measurements in the prespecified 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 compared to normal (>1.5 mV) areas. Segments with some voltage values <1.5 mV and >1.5 mV were considered heterogeneous and excluded from the further segmental analysis.

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

Mean±SE for anatomic (ESWT, EDWT) and dynamic (WT, WM) parameters in each segment were calculated and compared between abnormal and normal homogeneous voltage segments. Receiver operating characteristic curves were used to assess the ability of CT-derived parameters to determine myocardial abnormality. Bipolar voltages were used as the gold standard, with 1.5 mV as the cut-off value to define abnormal myocardium. Generalized estimating equations were used to identify the most significant CT-derived parameters in predicting the voltage-defined categories. The 17-segment analyses were conducted for all of the voltage maps and 2D CE-CT slices with hyperfusion areas. Hypoperfused segments were examined in both voltage maps and CE-CT images to calculate their location-matching percentage.

Three-Dimensional CE-CT Image Reconstruction

CE-CT images were reconstructed into mesh files with Amira 5.2.1 using end-diastole (80%) images to best correlate with the Carto point map by a radiologist blinded to the voltage data. Epicardial and endocardial LV surfaces, endocardial right ventricle surfaces, hypocontractility segments, and hyperperfusion areas were reconstructed separately. Mesh files were converted into the Carto XP-readable mesh format using MATLAB R2009a and imported into the Carto XP mapping system. The reconstructed images were registered with voltage maps using precommercial CartoMERGE software.9 Primary registration was performed with Landmark points and visual alignments as previously published.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 right ventricular apex and outflow tract as well as up to 2 LV sites (if not inducible from the right ventricle) using 3-drive train cycle lengths (350, 400, and 600 milliseconds) and 1, 2, and 3 extrastimuli with minimal coupling interval of 200 milliseconds. 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 and morphology of the ICD recordings) were the target of the ablation procedures.

Statistical Analysis

Statistical analysis was performed using SPSS for Windows version 16.0. Continuous variables are expressed as mean±1 SD or median and range, unless otherwise specified. Comparisons were conducted with 1-way ANOVA for different registration strategies. Generalized estimating equations were used to compare the measurements for ESWT, EDWT, WT, and WM and to identify the most significant CT-derived parameters in predicting the voltage-defined categories. Differences were considered significant at a level of P<0.05. Receiver operating characteristic curves were created for each of the...
4 CT-derived parameters in predicting voltage-defined abnormal segments. Areas under the curve (AUCs) for each parameter were reported and compared. Pearson correlations were computed to investigate the relationship between electrophysiological and CT-derived myocardial attributes. In particular, abnormal LV myocardium area was correlated between CE-CT reconstruction and voltage mapping, and abnormal LV myocardium burden was correlated between CE-CT and voltage mapping.

Figure 1. Anatomic information of the CE-CT images. A, Short-axis view of the CE-CT image. The green and yellow lines delineate endocardial and epicardial contours. The anterior-septal wall (arrows) 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, A 17-segment analysis for the 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, arrows). C, Three-dimensional reconstruction for anatomic information showing the short-axis view of the 3D LV reconstruction of CE-CT image with epicardial and endocardial surfaces. Arrows indicate anterior-septal wall thinning corresponding to areas seen in 1A. D, A 17-segment analysis of the voltage map indicating anterior-septal scar with small voltage values (red area, arrows) consistent with the wall thinning areas demonstrated in 1A through 1C. Apical cap was excluded from analysis.

Figure 2. Dynamic information of the CE-CT images. A, A 17-segment analysis of WT. Values for WT were extracted and presented in the 17-segment model format, with the color scale indicating anterior-septal scar with decreased WT values (purple segments, arrow). B, Three-dimensional reconstruction for dynamic information, showing 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.
The authors had full access to the data and take responsibility for its integrity. All authors have read and agreed to the manuscript as written.

Results

Patient Characteristics
All patients were men aged 65±9 years (Table 1). All 11 patients had ischemic cardiomyopathy and previous myocardial infarction as evidenced by the documented ECG. Previous myocardial infarctions were located at inferolateral (n = 2), inferior and anterior (n = 4), inferior (n = 4), and anteroseptal wall (n = 1). Heart rates during the CT scan were 62±6 bpm. Radiation exposures were 12.35±3.67 mSv.

Electrophysiological Mapping
Endocardial maps were created using a retrograde aortic approach in all patients with 275±109 mapping points. All patients had scar, BZ, or mixed scar and BZ segments.

Comparison of Anatomic, Dynamic, and Perfusion Parameters Between Voltage-Defined Abnormal and Normal Myocardium Segments
Forty percent (n = 71) of segments were homogenous and used for further analysis, including 43 normal segments and 28 abnormal segments (5 scar, 3 BZ, and 20 mixed scar and BZ). There were 15±11 (median, 14; range, 1 to 43) mapping points in each segment (Figure 1D).

Anatomic Parameters
ESWT and EDWT were 6.5±0.75 mm and 6.6±0.94 mm, respectively, for abnormal segments and 10.1±1.8 mm and 12.9±1.7 mm, respectively, for normal segments (ESWT, P <0.001; EDWT, P =0.01) (Figure 1A through 1C).

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

Perfusion Parameters
Sixty-seven (38%) out of 176 segments showed hypoperfusion. Fifty-two (78%) of the 3D CE-CT hypoperfusion segments matched with abnormal voltage segments (Table 2).

Table 1. Patient Characteristics (n=11) at Time of VT Ablation

<table>
<thead>
<tr>
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<th>Value</th>
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<td>Sotalol</td>
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Data are presented as mean±SD or %, unless otherwise indicated.

Table 2. Abnormal Segment Location According to 17-Segment American Heart Association Model

<table>
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<th>Abnormal Voltage Segment</th>
<th>CT Hypoperfusion Segment</th>
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</table>

Prediction of Abnormal Voltage Segments With Anatomic and Dynamic Parameters
AUCs the parameters of ESWT, EDWT, WT, and WM were 0.83±0.05, 0.75±0.06, 0.79±0.06, and 0.68±0.06, respectively (Figure 3). ESWT had a significantly better discrimination value than WM to identify the abnormal voltage segments when comparing the AUCs (P =0.04). There was no significant difference between ESWT and WT in predicting the voltage categories (P =0.58).

Generalized estimating equation bivariate analyses demonstrated that ESWT, EDWT, WT, and WM were all significantly correlated with the presence of the abnormal voltage segments (P <0.005, 0.025, 0.001, 0.019, respectively). 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.

Figure 3. Receiver operating characteristic curves for anatomic and dynamic parameters. Plots of the receiver operating characteristic curves for ESWT (blue), EDWT (brown), WT (green), and WM (purple) were used to predict abnormal voltage segments as defined by <1.5 mV for all the voltage points in the segment. AUC for each parameter are displayed.
Integration of CT-Derived 3D Cardiac Anatomy With Embedded Anatomic, Dynamic, and Perfusion Scar and BZ Into Clinical Mapping System

The LV reconstruction allowed the 3D display of the complete LV endocardium and 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, scar, and BZ was achieved in all cases.

CE-CT-derived 3D LV anatomy could be accurately registered and displayed. Visual alignment demonstrated a position error of 3.72±0.58 mm (Table 3). The addition of surface shell registration improved the registration accuracy to 3.41±0.79 mm. Although landmark point registration alone resulted in a position error of 3.68±1.53 mm, the combination with surface shell registration enhanced the position error to 3.31±0.52 mm (Table 3). Accuracy of the 4 registration strategies did not differ significantly (P=0.13).

Three-dimensional CE-CT reconstructions of the LV wall and the segments with low WT values demonstrated areas and segments with low contractility, including ventricular aneurysms (Figure 2A and 2B). CE-CT perfusion imaging demonstrated location and transmurality of hypoperfused myocardium, which allowed the assessment of its transmural extent and the differentiation in endo-, mid-, and epicardial scar components (Figure 4A and 4B). After 3D reconstruction of endo- and 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 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 (9%) patient was found to have calcifications and layered endocardial thrombus (Figure 4B). The average voltage value in this area was 0.64±0.23 mV (Figure 4).

Table 3. Position Error

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<tr>
<th>Patient</th>
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<th>VA, mm</th>
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<td>Mean±SD</td>
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Individual average position error for each patient after different registration algorithms. LM indicates landmark point registration; SF, surface registration; VA, visual alignment.
Comparison of Voltage Map With 3D CE-CT Hypoperfusion Reconstruction

The size of the 3D CE-CT hypoperfusion reconstruction area correlated best with abnormal voltage area (scar+BZ) (Figure 5C and 5D). Voltage mapping demonstrated an abnormal voltage area of $97.7 \pm 41.3$ cm$^2$, representing $37.4 \pm 11.4\%$ of the LV. The 3D CE-CT hypoperfusion map demonstrated amyo-cardial hypoperfusion area of $88.2 \pm 36.3$ cm$^2$, with an abnormal LV myocardium burden of $33.3 \pm 8.5\%$. 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 $1.14 \pm 0.23$ mV compared with $8.4 \pm 5.1$ mV in the area with normal myocardium perfusion ($P<0.001$).

VT Ablation

An average of $2.2 \pm 1.5$ VTs were inducible during programmed electrical stimulation. Based on 12-lead morphology of the clinical VT or rate and morphology of the ICD recordings, 16 VTs were deemed to represent clinical or presumed clinical VT and were targeted for ablation. Sixty-nine percent of the clinical VTs have right bundle branch block morphology with right inferior (n=8), right superior (n=2), and left superior (n=1) origin. The remaining 31% have left bundle branch block morphology with right superior (n=3) and left inferior (n=2) origin. Cycle lengths were 230 to 640 milliseconds. Radiofrequency ablation rendered 15 (94%) of 16 clinical and presumptive clinical VTs noninducible. One patient remained inducible. Five VTs of unclear significance from 5 patients were inducible after the ablation. One patient was not tested after ablation due to hemodynamic instability.

Activation and entrainment mapping was used in 2 VTs, and pace mapping and limited activation mapping were used for the remaining 14 clinical VTs due to hemodynamic disability. Successful ablation sites of 2 entrainable VTs were within 1 cm of the CT-defined border. Pace-mapping points identifying the exit site of clinical VT were located within 1 cm from CT-defined border in 10 (91%) patients (Figure 5B). No complications occurred in any of the 11 ablation procedures.

Patient Follow-Up

The median follow-up time was 12 months (range, 4 to 37 months). Five (45%) patients received 2 recurrent ICD therapies (ATP, shock, or both) at a median follow-up time of 10 months (range, 2 to 18 months) after the first ablation. Of those, 3 (27%) patients underwent a second VT ablation at a median of 7 months (range, 2 to 10 months) after the first ablation. There was 1 (9%) death within 6 months follow-up due to chronic heart failure, and there were 4 (36%) deaths 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 for a comprehensive characterization of scar and BZ; (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 (<1.5 mV) rather than scar alone (<0.5 mV); 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 for 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 currently is not integrated into the mapping system and could provide better guidance for VT ablations. Although the current VT ablation strategies based on voltage-defined criteria are well established, the 6-month success rate from recurrent VT is only 53%. Correlation of scar geometry obtained by cardiac imaging and of 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, but it cannot provide currently relevant clinical information about transmural extent and intramyocardial location of the scar. Delayed-enhanced MRI can assist in accurately characterizing the location and extent of myocardial scar, but the presence of an ICD still is considered a contraindication in the majority of patients requiring a VT ablation. Although PET scan provides a metabolic characterization of the myocardial scar and its BZ, the spatial resolution of 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 contraindications and high spatial (≤1 mm) and temporal resolution. CT enables a detailed and comprehensive evaluation of LV myocardium using 3 different and complementary pathophysiological characterizations of myocardial scar and 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. The current study used triple, multimodality imaging based on anatomic, dynamic, and perfusion parameters to identify abnormal substrate, which is the target for the majority of VT ablations.

Regional Anatomic, Dynamic Parameters, and Perfusion Characterization for VT Ablations
MDCT imaging has been used to assess regional LV function and permits accurate, noninvasive assessment of LV function and perfusion in patients with previous infarction. Previous research found a significant correlation between MDCT and MRI for anatomic LV parameters, such as ESWT, EDWT, and the dynamic LV parameter of WT, and concluded that MDCT can accurately estimate regional LV wall thickness and WT. In the present study, all 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 the abnormal category. This finding is consistent with previous research, where 88.3% segments with decreased WM were correctly identified by MDCT.

The current gold standard of voltage mapping has several limitations; for example, a single endocardial voltage measurement only incompletely describes a complex intramural scar anatomy. 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 5 mm covered by the 3.5-mm catheter tip and 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. When compared with CE magnetic resonance absolute sizes of early hypoperfused and late hyperenhanced regions were similar on CE-CT and CE magnetic resonance.

Because 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 (<1.5 mV) and CE-CT hypoperfusion was found. Seventy-eight percent 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 because a nontransmural, midmyocardial, or epicardial scar might influence the segmental WT, contractility, and myocardial perfusion. Surviving endocardial components or far field from surviving mid- and epicardium may be able to create normal or near-normal endocardial voltages. Indeed, approximately 4% of successful ablation sites are located within healthy myocardium defined by voltage mapping, which could possibly represent nonendocardial 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 (<1.5 mV) rather than with scar alone (0.5 mV). 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, which is consistent with findings from both animal and human studies, and showed that the microvascular obstruction area characterized by hypoperfusion correlates well with the scar area indicated by reduced flow regions as measured by microsphere blood flow. Integration resulted in an immediate preprocedural anatomy and substrate display with submillimeter resolution that is 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 is 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 to identify areas involved in myocardial reentry representing appropriate ablation targets and 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 also could supply information helpful in differentiating low endocardial voltages due to fat or scar formation. With improved understanding of scar geometry, a preprocedural modeling of anatomically fixed circuits and likely successful ablation sites may become increasingly possible.

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. Although 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 4.3 mSv 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 homogeneous 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 for a meaningful analysis of each individual single-voltage category at the present time. Finally, the relatively small sample size limits the generalizability of this study, which will need to be confirmed in future randomized trials.

Conclusions

To our knowledge, this study is the first to assess a comprehensive 3-modality characterization (anatomic, dynamic, and perfusion) of LV scar from a single CE-CT and to 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 used to correctly predict abnormal voltage locations in advance of the mapping procedure, which may allow the electrophysiologist to concentrate on areas of likely myocardial scar and identify falsely 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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Disclosures

Dr Dickfeld reports receiving research support from Biosense Webster, Israel. Mr Turgeon and Dr Abbo are employed by Biosense Webster, Israel.

References

Identification of areas of scar are useful to guide catheter ablation of ventricular tachycardia. Use of MRI for this purpose is limited by the presence of implantable cardioverter defibrillators in many patients. This study evaluated contrast-enhanced CT to assess left ventricular scar based on analysis of anatomy, motion, and perfusion. Images were integrated into a clinical mapping system to guide ventricular tachycardia ablation. The regional left ventricular anatomy, dynamic, and perfusion parameters agreed well with areas of abnormal voltage, allowing these regions to be identified before the mapping procedure. CE-CT also is able to characterize the transmural extent and intramyocardial location of scar tissue that could potentially help to identify intramural and epicardial arrhythmia substrate, overcoming a limitation of endocardial voltage mapping. Differentiating low epicardial voltage due to fat also may be possible. Further study may improve understanding of scar geometry as it relates to ventricular tachycardia and facilitate ablation.
Three-Dimensional Contrast-Enhanced Multidetector CT for Anatomic, Dynamic, and Perfusion Characterization of Abnormal Myocardium To Guide Ventricular Tachycardia Ablations

Jing Tian, Jean Jeudy, Mark F. Smith, Alejandro Jimenez, Xianghua Yin, Patricia A. Bruce, Peng Lei, Aharon Turgeman, Aharon Abbo, Raj Shekhar, Magdi Saba, Stephen Shorofsky and Timm Dickfeld

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