The Electrophysiological Cardiac Ventricular Substrate in Patients After Myocardial Infarction

Noninvasive Characterization With Electrocardiographic Imaging

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Objectives
The aim of this study was to noninvasively image the electrophysiological (EP) substrate of human ventricles after myocardial infarction and define its characteristics.

Background
Ventricular infarct border zone is characterized by abnormal cellular electrophysiology and altered structural architecture and is a key contributor to arrhythmogenesis. The ability to noninvasively image its electrical characteristics could contribute to understanding of mechanisms and to risk-stratification for ventricular arrhythmia.

Methods
Electrocardiographic imaging, a noninvasive functional EP imaging modality, was performed during sinus rhythm (SR) in 24 subjects with infarct-related myocardial scar. The abnormal EP substrate on the epicardial aspect of the scar was identified, and its location, size, and morphology were compared with the anatomic scar imaged by other noninvasive modalities.

Results
Electrocardiographic imaging constructs epicardial electrograms that have characteristics of reduced amplitude (low voltage) and fractionation. Electrocardiographic imaging colocates the epicardial electrical scar to the anatomic scar with a high degree of accuracy (sensitivity 89%, specificity 85%). In nearly all subjects, SR activation patterns were affected by the presence of myocardial scar. Late potentials could be identified and were almost always within ventricular scar.

Conclusions
Electrocardiographic imaging accurately identifies areas of anatomic scar and complements standard anatomic imaging by providing scar-related EP characteristics of low voltages, altered SR activation, electrogram fragmentation, and presence of late potentials. (J Am Coll Cardiol 2011;58:1893–902) © 2011 by the American College of Cardiology Foundation

Delayed enhanced magnetic resonance imaging (DE-MRI) has been used to noninvasively identify anatomic myocardial scars in patients with prior myocardial infarction (MI) (1). DE-MRI has been used to improve prediction of ventricular arrhythmia inducibility at electrophysiology study (2,3), and of spontaneous ventricular arrhythmia and mortality (4).

Typically, the abnormal electrophysiological (EP) substrate extends beyond the dense anatomic scar, into regions of heterogeneous tissue containing excitable “islands” of myocardia (5). This “border zone” (BZ) is characterized by abnormal cellular electrophysiology and altered structural properties due to remodeling processes. The BZ has been recognized as a contributor to scar-related arrhythmias and a target for catheter ablation. Attempts have been made, in recognition of its important role in arrhythmogenesis, to identify the BZ as the “gray zone” in DE-MRI images. However, this is not a natural application of MRI, which images anatomy and not electrophysiology. This distinction is of utmost importance, because EP properties of the scar determine arrhythmogenesis.

Currently, scar electrophysiology after MI is studied with EP catheter mapping. However, this process is invasive, time-consuming, and carries a modest risk. Additionally, bipolar mapping incompletely represents the transmurality...
of the scar architecture (6,7). Although invasive unipolar endocardial mapping was recently reported to address some of the challenges related to identification of scar beyond the endocardial surface (8,9), the time constraints and patient risks prohibit it from becoming a routine cardiac test.

Experimental, theoretical, and clinical studies have shown that scar-related abnormal electrical properties are reflected in specific characteristics of cardiac potential distributions and electrograms (EGMs). Canine studies in a 5-day-old infarct showed low-level fractionated EGMs in the infarct, which correlated with slow, discontinuous conduction—a property that supports arrhythmogenicity (10). Low-amplitude potentials, broad fractionated EGMs, and delayed local activation are seen in invasive mapping during sinus rhythm (SR) in patients with history of MI (11–13). Our laboratory has demonstrated the ability of a new noninvasive EP imaging modality (electrocardiographic imaging [ECGI]) to reconstruct, from body surface potentials, such characteristics of epicardial potentials and EGMs over the scar region in canine experiments and human intraoperative studies (14–16). The purpose of this study is to explore the limits of ECGI to noninvasively identify areas of human infarct-related ventricular scar and to characterize the local EP properties in relation to its anatomic substrate.

Methods

All protocols were approved by the Institutional Review Board at Washington University, and informed consent was obtained from all patients. Subjects with history of MI were identified during inpatient and outpatient visits.

The ECGI methodology was described previously (Fig. 1) (17,18). Electrodes (n = 256) on strips were applied to the torso of the patient. Computed tomography (CT) markers were attached to each electrode. All strips were connected to a portable mapping system (BioSemi, Amsterdam, the Netherlands). After electrodes application, patients underwent thoracic noncontrast gated CT with axial resolution of 3 mm. Scans were gated at 70% of the R-R interval (ventricular diastole). Patient-specific ventricular epicardial surface geometry and body surface electrode positions were labeled and digitized from CT images.

The 256 channels of body surface potentials were sampled at 1-ms intervals from the start of the QRS complex through the ST segment (ventricular activation) during SR. The body surface potentials and geometrical information (torso-heart geometric relationship) were combined by ECGI algorithms to noninvasively construct epicardial potentials, unipolar epicardial EGMs, and epicardial activation sequences (isochrones). The ECGI is constructed on a beat-by-beat basis and does not require accumulating data from many identical beats. Fewer than 1% of body surface electrocardiograms were rejected before the ECGI reconstruction. After ECGI reconstruction, none of the EGMs were rejected.

**EGM magnitude (voltage).** For each subject, evenly distributed reconstructed unipolar epicardial EGMs were used for data analysis. The EGMs from valvular regions were excluded. The EGM magnitude (voltage) was measured peak-to-peak. Because absolute EGM magnitude is patient-specific, EGMs were indexed to the maximum value for each patient. Low-voltage regions and very low-voltage regions were defined by EGMs with amplitudes <30% or 15% of the maximum, respectively. Epicardial electrogram magnitude maps (EMMs) were created with patient-specific geometry.

**EGM characterization and localization.** Electrogram morphology was characterized by degree of fractionation (number of deflections; low-pass filtering at 120 Hz) and displayed as epicardial electrogram deflection maps (EDMs). Combining both voltage and fractionation of EGMs, the “electrical scar” is defined as the area with EGMs that demonstrated both low-voltage (<30% of maximum value) and multiple deflections. Electrical scar maps (ESMs) represent this combined criterion visually. **Comparison of ECGI-determined electrical scar with conventionally imaged anatomic scar.** Clinical noninvasive cardiac imaging was available for comparison, including DE-MRI for patients without cardiac devices, and myocardial perfusion imaging with single-photon emission computed tomography (SPECT) for patients with cardiac devices. For DE-MRI, coregistration of ECGI and MRI images was performed to demonstrate relationship between electrical and anatomic scar (Fig. 1B). The epicardial aspect of the DE-MRI scar was used for analysis. For SPECT comparison, determination of myocardial scar was performed in an automated fashion, with AutoQuant software (Phillips Medical Systems, Andover, Massachusetts). The ECGI maps were divided into a standard 17-segment classification and scored on the basis of presence or absence of electrical scar. Because ECGI reconstructs epicardial EGMs, septal segments were not scored. The anatomic scar imaging and ECGI scar imaging were analyzed independently to eliminate bias.

**Functional imaging: SR activation, late potentials, and electrical scar.** During SR, activation times were determined by the maximal negative slope of EGMs, and activation isochrone maps were created (17,18). Lines/regions of block (thick black lines) were inferred if adjacent activation times differed by more than 50 ms. Slow conduction is represented by crowded isochrones. Deflections in
EGMs above the ambient electrical noise level that occurred after the surface QRS were labeled as late potentials (LPs).

**Results**

Twenty-four patients with history of MI were included in the study (mean age 62 years, mean left ventricular [LV] ejection fraction 30%). The ECGI procedure was completed successfully in all subjects without adverse events. Patient demographic data are in Table 1. Representative examples are presented in the report; all data are in the Online Appendix.

For each subject, an average of 769 EGMs were used for analysis (range 620 to 885). The average maximum voltage was $8.6 \pm 2.8 \text{ mV}$ (range 2.3 mV to 14.9 mV), and average minimum was $0.17 \pm 0.14 \text{ mV}$ (range 0.02 mV to 0.58 mV). Average mean voltage was $2.0 \pm 0.83 \text{ mV}$ (range 0.51 mV to 3.9 mV).

Low-voltage EGMs comprised $55 \pm 9\%$ of all EGMs for each patient (range 36% to 73%), and very-low-voltage EGMs $19 \pm 9\%$ (range 6% to 43%). Fractionated EGMs (2 or more deflections) were observed in every patient. On average, they comprised $18 \pm 11\%$ of EGMs in each patient (range 5% to 48%), with 91% of fractionated EGMs...
### Table 1  Patient Characteristics

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AAD = antiarrhythmic drug; ACE = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor-blocker; BB = beta-blocker; LVEF = left ventricular ejection fraction; VT = ventricular tachycardia.

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(A) **Electrical Scar (red)** shown in the left anterior oblique view. **Top image** uses a reduced voltage criterion to identify scar. **Bottom image** uses reduced voltage and EGM fractionation to identify scar. (B) EGMs a, b, and f (red) from low-voltage regions demonstrate low amplitude alone. EGMs c to e (red) demonstrate both fractionation and low amplitude. EGMs g to i (blue) from neighboring regions outside the scar demonstrate considerably larger amplitude and single deflection. (C) EGMs c to e amplified to clearly demonstrate multiple deflections. Abbreviations as in Figure 1.
inside low-voltage regions and 51% inside very-low-voltage regions.

**EGM characterization and localization.** Examples of constructed epicardial EGMs from inside and around an electrical scar are shown in Figure 2. Panel A shows the electrical scar in red. Top image uses a conservative criterion of reduced amplitude (low voltage) alone. Bottom image uses a strict criterion combining low voltage with EGM fractionation to define electrical scar. Panel B shows representative EGMs from areas within the low-voltage regions (a to f; red). For comparison, EGMs from neighboring myocardium outside the scar (g to i) are shown in blue. Panel C shows amplified EGM signals from the electrical scar, demonstrating high degree of fractionation.

**Comparison of ECGI-determined electrical scar with anatomic scar from DE-MRI.** Anatomic scar from DE-MRI was compared with ECGI electrical scar for 5 subjects without a cardiac device in a blinded fashion. An example of anterior scar is shown in Figure 1B. Figure 3 shows 2 additional examples: one with a localized apical infarction, and the other with a complex morphology inferoapical infarction. The ECGI colocalizes the epicardial electrical scar to the anatomic scar with high accuracy. The 2-dimensional shape of the anatomic scar is accurately reproduced with electrical scar imaging, as highlighted by the serpiginous scar shape in Figure 3B. Representative EGMs from non-scar regions (blue) and electrical scar regions (red) are shown below each example, highlighting...
the low amplitude and fractionated EGM qualities seen in electrical scar.

Comparison of ECGI-determined electrical scar with anatomic scar from SPECT. Anatomic scar from SPECT was compared with ECGI electrical scar in 18 subjects in a blinded fashion. Segmental sensitivity and specificity for ECGI is shown in Figure 4. Overall, the sensitivity and specificity of ECGI to detect scar in each segment of myocardium, compared with SPECT imaging, was 89% and 85%, respectively. Apical and mid-cavitary segments had the highest sensitivity, whereas basal segments had the lowest sensitivity. This is most likely due to the purposeful exclusion of basal valvular regions from ECGI analysis. Inferior segments had the lowest specificity.

Figure 5 shows 2 examples comparing ECGI electrical scar with SPECT imaging for a subject with an inferoapical infarct and a subject with a large apical infarct/aneurysm. The ECGI co-localizes the epicardial electrical scar (red region, ESM) to the anatomic scar with high accuracy. The EGMs from the scar region (red) demonstrate fractionated, low-amplitude qualities of electrical scar.

Functional imaging: alteration in SR activation and LP. Normal epicardial activation patterns have been reported previously (18). In general, earliest epicardial activation is in the anterior right ventricle, and latest is along the basal lateral LV. Activation isochrone maps in Figures 3 and 5 show how the presence and location of electrical scar alters the epicardial activation pattern during SR. Figure 3A shows normal location for earliest epicardial breakthrough (asterisk) but a line of block along the inferior and apical aspect of the electrical scar (thick black line). This forces LV activation to progress in a base-to-apex pattern, with the area of latest activation near the apical scar. Figure 5A shows a similar pattern, with earliest activation in the right ventricle (white) and a line of block along the septal border of the electrical scar. Latest activation (dark blue) occurs at the LV side of the scar, nearly 200 ms after the first breakthrough. The activation pattern of unidirectional block at 1 border of a scar, followed by marked conduction delay and latest activation near the other border of a scar, is often associated with initiation of re-entry and re-entrant ventricular arrhythmia.

Of the 19 subjects imaged during SR, 16 (84%) demonstrated altered or delayed epicardial activation at the location of electrical scar. Ten (52%) had latest activation at the electrical scar location. Ten (52%) had a line of block at the scar border.

In addition to the functional relationship between electrical scar and SR activation patterns, EGMs with LP were observed. The LPs were present in an average of 8.4% of all EGMs for each patient (range 0% to 35%). Of 24 subjects, 17 had >3% LPs. Almost all LPs were found within the electrical scar (94%); 62% were found within the very-low-voltage region. Figure 6 shows 3 examples of LP EGMs within the electrical scar.

Discussion

Key findings of this study include: 1) within anatomic myocardial scar in post-MI patients, ECGI noninvasively reconstructs EGMs characterized by reduced amplitude (low voltage) and fractionation; 2) with combination of low voltage and fractionated EGMs, ECGI images the “electrical scar,” which colocalizes with the anatomic scar as determined by DE-MRI or SPECT; and 3) ECGI is a functional imaging modality that complements MRI and SPECT by imaging scar-related EP characteristics of altered SR activation, EGM fragmentation, and presence of LP. This study is a step toward noninvasively imaging the relationship between myocardial anatomic scar and its EP properties.

Fractionated, low-magnitude EGMs are commonly found in post-infarct myocardium, and it is well-accepted that they reflect slow, nonuniform and discontinuous conduction through the heterogeneous scar substrate.
Figure 5  Relationship Between ECGI-Derived Electric Scar and SPECT Anatomic Scar

Relationship between ECGI-derived electrical scar and single-photon emission computed tomography (SPECT) anatomic scar. Similar format to Figure 3, except SPECT images replace MRI scar maps. (A) Apical MI. Latest activation is in the anterior (ANT) apex (dark blue in AI map), which is abnormal. ESM demonstrates an electrical scar in the apex, extending anteriorly and inferiorly (red). Resting myocardial perfusion images (SPECT), shown in a standard “bullseye” configuration (left) and long-axis view (right), demonstrate large area of infarction in the ANT, apical, and inferior (INF) LV. (B) Apical aneurysm. ESM demonstrates a large electrical scar across the apex. SPECT imaging shows similar extensive apical distribution of scar. LAO = left anterior oblique; other abbreviations as in Figures 1 and 3.
In a finding consistent with invasive studies, the ECGI noninvasive EGMs from scar regions are fragmented, of small magnitude, and prolonged duration, reflecting similar substrate properties. Low-amplitude, high-frequency signals have been recorded from the body surface in post-MI patients at the end and after the QRS complex (20). These LPs are thought to originate from the infarct and correspond to late deflections on EGMs recorded directly from the heart (21,22); they are thought to reflect late activation via slow discontinuous conduction along viable myocardial fibers. Endocardial catheter mapping of patients with coronary artery disease identified LPs in 12% of the sampled EGMs, similar to the results of this study (23). The ECGI reconstructed LPs were localized to electrical scar regions.

The presence of scar provides EP substrate that supports asymmetrical electrical loading on a propagating wavefront, a property that favors formation of unidirectional block. With ECGI, lines of block and altered wavefront propagation were clearly seen and were associated with the borders of electrical scar. Similarly, scar-related regions of slow conduction were detected. The combination of unidirectional block and slow conduction is highly arrhythmogenic, providing conditions for re-entrant ventricular tachycardia (VT). “Patchiness” of the scar substrate (islands of viable myocardium within scar tissue) supports slow discontinuous conduction and conditions for block; it manifests electrophysiologically in fragmented EGMs and presence of LP. Typically, each feature of the electrical scar (location, size and shape, EGM fragmentation, LP, ventricular activa-
tion patterns) is assessed in isolation during a clinical EP study, and EGMs from different parts of the scar are recorded sequentially with a roving-catheter process that requires a long time to cover the entire scar. The ECGI provides the ability to gather this information from the entire epicardial scar in a single noninvasive study, during a single beat.

A noninvasive method for identifying post-MI patients at risk of arrhythmia would aid in making decisions with regard to preventative intervention, such as implantable cardioverter-defibrillator insertion. Also, with increasing numbers of catheter-based VT ablation procedures, there is great interest in defining scar architecture before a procedure. ECGI is still a novel research tool, and its limited availability precludes large-scale multicenter clinical studies. However, our results suggest a potential role for ECGI in arrhythmia risk stratification or identification of targets for ablation on the basis of the EP properties of the scar.

**Study limitations.** At this stage of development, ECGI is limited to imaging the epicardium and cannot image the ventricular septum. Intraoperative mapping suggests a purely epicardial location of the arrhythmia substrate in 33% of patients (24,25), although this number is increasing with epicardial mapping becoming more common. The ECGI reconstructs epicardial potentials and unipolar EGMs, which are more affected by far field influences than bipolar EGMs. However, evolution in time of the epicardial potential pattern and unipolar EGM properties provide information on the intramural depth of the VT circuit (8,9,26), which partially addresses the aforementioned limitation. Because of small signal amplitude in regions of scar and because ECGI cannot image active depolarization during a repolarization period (T wave), it is likely that ECGI does not image all LPs.

The extent of the ECGI-determined EP scar depends on the threshold chosen for scar EGM magnitude. With our choice of 30% of the maximum value for each patient, the EP scars and DE-MRI-imaged anatomic scars were highly correlated in our patient population, and ECGI achieved a reasonable level of specificity and sensitivity, compared with SPECT. As shown in Figure 2, compared with using EGM magnitude alone, the use of multiple criteria (EGM magnitude and fractionation) increases the specificity of scar determination and reduces the confounding influence of epicardial fat (27). It will require a larger-scale study to establish a universal criterion for ventricular electrical scar with high level of statistical significance.

**REFERENCES**


Key Words: border zone • ECGI • infarct • noninvasive imaging • ventricular substrate.

For supplementary figures, tables, and text, please see the online version of this article.