Three-dimensional endocardial impedance mapping: a new approach for myocardial infarction assessment

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Three-dimensional endocardial impedance mapping: a new approach for myocardial infarction assessment. Am J Physiol Heart Circ Physiol 280: H179–H188, 2001.—Precise identification of infarcted myocardial tissue is of importance in diagnostic and interventional cardiology. A three-dimensional, catheter-based endocardial electromechanical mapping technique was used to assess the ability of local endocardial impedance in delineating the exact location, size, and border of canine myocardial infarction. Electromechanical mapping of the left ventricle was performed in a control group (n = 10) and 4 wk after left anterior descending coronary artery ligation (n = 10). Impedance, bipolar electrogram amplitude, and endocardial local shortening (LS) were quantified. The infarcted area was compared with the corresponding regions in controls, revealing a significant reduction in impedance values [infarcted vs. controls: 168.8 ± 11.7 and 240.7 ± 22.3 Ω, respectively (means ± SE), P < 0.05] bipolar electrogram amplitude (1.8 ± 0.2 mV, 4.4 ± 0.7 mV, P < 0.05), and LS (−2.36 ± 1.6%, 11.9 ± 0.9%, P < 0.05). The accuracy of the impedance maps in delineating the location and extent of the infarcted region was demonstrated by the high correlation with the infarct area (Pearson’s correlation coefficient = 0.942) and the accurate identification of the infarct borders in pathology. By accurately defining myocardial infarction and its borders, endocardial impedance mapping may become a clinically useful tool in differentiating healthy from necrotic myocardial tissue.

myocardial viability assessment; electromechanical coupling; electrophysiology

ISCHEMIC HEART DISEASE is currently a major cause of morbidity and mortality and is predicted to be the primary cause of death within several decades (3). Evaluation of myocardial function in patients with ischemic heart disease is of importance and may possess varying implications concerning the therapeutic approach.

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endocardial impedance, could be used to enhance the accuracy of existing viability assessment tools.

METHODS

Animal Study

The study included 20 dogs, weighing 20–32 kg (10 controls and 10 dogs subjected to chronic coronary ligation), in which assessment was performed at both baseline and 4 wk after left anterior descending (LAD) coronary artery ligation. The experimental protocol was approved by the Animal Use and Care Committee of the Technion Faculty of Medicine. Anesthesia was induced using ketamine (10 mg/kg iv) and diazepam (1 mg/kg iv), with isoflurane (1%) and fentanyl (0.25 mg·kg⁻¹·min⁻¹ iv) used after intubation. Ventilation was maintained with a veterinary anesthesia ventilator (model 2000, Hallowell EMC). Left thoracotomy was performed, after which the LAD was ligated distally to the first diagonal branch. After surgery, we treated the animals with analgesics and antibiotics and allowed them to recover for a period of 4 wk.

Nonfluoroscopic Electromechanical Mapping System

The nonfluoroscopic electromechanical mapping system has been described previously (2, 12). Briefly, the system (NOGA, Biosense Webster) utilizes ultralow magnetic fields generated by three external magnetic field emitters that are placed beneath the operating table. Additional location sensors are integrated into 7-Fr standard electrophysiological deflectable tip catheters (NOGA-STAR, Biosense Webster); one serving as a reference catheter, and the other serving as the mapping catheter. The data acquired by the sensors is sent to a processing unit, enabling real-time accurate tracking of the catheter tip within the left ventricular (LV) cavity without the use of fluoroscopy.

Mapping Protocol

Electromechanical mapping of the LV was performed on each of the dogs 4 wk after LAD artery ligation in the chronic infarction group and acutely in the control group. In each case, a reference catheter was placed on the back of the animal, and a mapping catheter was introduced into the LV cavity under fluoroscopic guidance. Subsequently, fluoroscopy was turned off. The location of the navigated mapping catheter was recorded relative to the fixed position of the reference catheter, compensating for animal movement during the procedure. Accurate navigation of the catheter was enabled by real-time display of the location and orientation of the catheter tip on a Silicon Graphics workstation. A 3D reconstruction of the LV chamber was created based on location and local electrogram data acquired from multiple endocardial sites.

The quality of each sampled endocardial point is important for the accurate analysis of the acquired data and consequent 3D maps. Thus points were deleted from the map according to the following criteria: 1) detection of a premature beat or a beat after a premature beat; 2) location stability of >4 mm (location stability was defined as the difference in end-diastolic location of the catheter throughout two consecutive heart beats); 3) loop stability of >4 mm (loop stability was defined as the average distance between the location of the catheter at 2 consecutive heart beats at corresponding time intervals in the cardiac cycle); 4) cycle length deviation >15% from the median cycle length; 5) different local electrogram morphologies throughout two consecutive beats; 6) local activation time difference between two consecutive beats >3 ms; and 7) varying QRS morphologies of the body-surface electrocardiogram.

The catheter pressure on the endocardium, as indicated by marked ST segment elevation (>0.1 mV above baseline) in the local unipolar electrogram, was also evaluated.

Electrical Maps

Maps were obtained using a NOGA-STAR (Biosense Webster) catheter with a 2-mm tip electrode and closely spaced (0.5 mm) ring electrode (1 mm), enabling the recording of local unipolar and bipolar electrograms (filtered at 0.5–400 Hz and 30–400 Hz, respectively) at each sampled site. The electrical information, color-coded and superimposed on the geometrical reconstruction of the LV chamber, included local endocardial impedance, local activation time, and bipolar peak-to-peak amplitudes. All such data was observed online throughout the mapping procedure.

Local Endocardial Impedance Maps

Impedance was measured using a generator with a stabilized output amplitude, producing a sine signal of 1 μA with the frequency of 50 kHz. The output current source buffer with a high output impedance was connected to an intracardiac electrode and provided the constant alternating current (AC) through the cardiac tissue. A large return electrode was connected to the reference point of the circuit and placed beneath the animal's back. One input of a differential amplifier was connected to the intracardiac electrode, and the second input was connected to the return electrode. The output of the amplifier was then passed through a band-pass filter with a central frequency equal to the measuring signal frequency. A synchronous detector converts the AC voltage (proportional to the measured impedance) to the direct current.

The impedance meter was calibrated using known resistors. The coefficients for calculation of the measured impedance were subsequently entered into the software program for calculation. The personal computer dedicated to the NOGA system calculates the measured local endocardial impedance value at each of the acquired points using the data from the analog-to-digital converter and known coefficients.

Owing to the contractile nature of the heart and its motion during the cardiac cycle, variations in impedance values may occur due to the positioning of the catheter tip electrode relative to the endocardial surface; an increase of up to 20 Ω could be observed during systole, in accordance with previously published data (26). Thus impedance measurements were performed continuously and averaged throughout a time frame of 1,000 ms at each acquired point.

Mechanical Maps

Global and regional mechanical function of the myocardium was assessed using an algorithm that calculates the fractional shortening of regions of the endocardium at end systole. This calculation, termed local endocardial shortening (LS), has been described in detail elsewhere (11). Briefly, LS is calculated as the difference between the distance of each endocardial site and all its neighbors at end diastole and end systole (normalized to end diastole). A positive LS ratio is given to a site in which the distance between two neighboring sites decreases during systole (i.e., physiological contraction), whereas a negative ratio depicts a site in which the distance between two neighboring sites increases during systole (abnormal myocardial motion).
Regional Parameters

For data analysis, a fixed cylindrical polar reference coordinate system was defined. The center of mass of the reconstructed LV was calculated from the collection of sampled endocardial points. The line connecting the LV apex with the center of mass was defined as the long axis. The long axis was then divided into three parts (apex, midventricle, and base, consisting of 20, 40, and 40% of the long-axis length, respectively), and the longitudinal location of each endocardial site was determined on the basis of its projection on the axis. The midventricle and base were further divided into six different circumferential regions: anterior, septal, posterior, lateral, inferoseptal, and anteroseptal. In total, the endocardial surface was divided into 15 different regions for comparison.

Pathological Verification of Myocardial Infarction

At the end of each experiment, the animal was euthanized by administration of intravenous KCl, and the hearts were excised. The coronary arteries were perfused with 300 ml of 2,3,5-triphenyltetrazolium chloride (TTC; 5 g/250 ml of normal saline), and the hearts were fixed in 4% formaldehyde solution. On gross examination, infarcted areas were identified as those regions not stained by TTC, the presence of a fibrous scar, and myocardial thinning. The hearts were then sliced transversely into 5- to 7-mm-wide sections and scanned. The outlines of each slice (basal side facing upward) and the extent of the infarcted regions were traced and calculated using special morphometric software.

The endocardial surface area of each slice was calculated by multiplying its measured endocardial circumference by the slice width. Total endocardial area was then derived from the summation of the surface areas of all slices. The endocardial infarcted area (EIA) was calculated in the following manner: The inferior surface of each slice was taken to represent the superior surface of the subsequent slice. For each slice, the circumference of the endocardium overlaid with infarcted tissue was calculated. Subsequently, the EIA of each slice was calculated as a trapezoidal area (the bases with infarcted area was then calculated as EIA/total infarcted area \times 100.

Samples (6 \mu m) of healthy and infarcted tissue (lateral and midanterior walls, respectively) were obtained from the experimental group, and 6-\mu m samples of healthy tissue (midanterior wall) were obtained from the control group. These sections were then embedded in paraffin and stained with hematoxylin-eosin and Masson’s trichrome (22). Histological examination and verification of the infarcted tissue was performed using conventional light microscopy.

Correlation of Local Endocardial Impedance and Bipolar Voltage Maps with Pathology

Myocardial infarction is visualized in the local endocardial impedance maps as the region with low impedance values surrounded by the steepest impedance gradient. By adjusting the color-fill threshold of the reconstruction, the surface area of the infarct was visualized as the area in which impedance values were lower than the threshold value for each map. This threshold was determined for each LV as the sum of the minimal local endocardial impedance value and a percentage of the difference between the maximal and minimal endocardial impedance values. After experimenting with different values, a 40% difference was found to be superior for determining the steepest impedance gradient at the border of the infarcted region (Fig. 1). The accuracy of this parameter in detecting infarcted tissue was tested in nine animals. Guided by the impedance maps, radio-frequency ablation [500-kHz RF generator (RFG-3C; Radionics) in a temperature-controlled mode (70°C) for 60 s] was applied at three to four sites on the border of the infarcted areas. In addition to the assessment of impedance accuracy, quantification of the infarct size was performed using peak-to-peak bipolar voltage values, as described elsewhere (11).

Statistical Analysis

Values are given as means ± SE. Because of minor changes performed in the impedance measuring circuit, comparison of the endocardial impedance values between normal and infarcted myocardial tissue was performed using two-way ANOVA. Multiple comparisons of bipolar voltage and LS values were performed between infarct and control groups (corresponding regions) and within the same hearts (infarcted vs. noninfarcted regions) using one-way ANOVA. Statistical significance was achieved for P values < 0.05. Comparison between the infarcted area derived from the impedance and bipolar voltage maps, and the infarced area, as calculated from the pathological specimens themselves (TTC stained), was performed using linear regression and Pearson’s correlation.

RESULTS

Electrical and Mechanical Maps

The electromechanical maps of all animals in the infarcted group (n = 10) displayed similar characteristics. A total of 141 ± 18 points were acquired, with 118 ± 13 remaining after the editing procedure. Typical maps of endocardial impedance, bipolar voltage, and linear local shortening of the healthy and infarcted LV chambers, as observed in real time, are shown in Figs. 2-4 (LAO projection, all parameters shown are from a single healthy or infarcted LV). Note that the infarcted region in the midanterior wall is characterized by reduced impedance values (red area) compared with both noninfarcted regions of the same heart and to the corresponding region of the control heart (Fig. 2, A and B). The simultaneous reduction in both bipolar voltage and LS values in the infarcted region is shown.
in Figs. 3B and 4B (red area). However, no such changes were observed in the noninfarcted hearts (Figs. 3A and 4A). Looking at Figs. 2–4, the border of the infarcted region can be visualized and assessed as the steepest gradient in either LS, bipolar voltage, or endocardial impedance values.

Table 1 summarizes the changes in regional values of endocardial impedance, bipolar electrogram amplitude, and LS in the healthy and infarcted animal groups.

Endocardial impedance was found to be significantly reduced in the midanterior wall \(168.8 \pm 11.7 \, \Omega\), (means ± SE) compared with all other noninfarcted regions of the same hearts \((198.1 \pm 16.7\) to \(218 \pm 15.1 \, \Omega, \, P < 0.01\)) and to the corresponding region in the healthy hearts \((240.7 \pm 22.3 \, \Omega, \, P < 0.05\)) regardless of the type of circuit used.

Similarly, both LS and bipolar voltage values were found to be significantly reduced in the midanterior wall \((-2.36 \pm 1.6\% \) and \(1.8 \pm 0.2 \, \text{mV, respectively}\) compared with both noninfarcted regions of the same hearts \((8.3 \pm 2.3 \) to \(11.1 \pm 0.7\% \) and \(2.7 \pm 0.6 \) to \(6.2 \pm 0.4 \, \text{mV, respectively, } P < 0.01\)) and to the corresponding region in the healthy hearts \((11.9 \pm 0.9\% \) and \(4.4 \pm 0.7 \, \text{mV, respectively, } P < 0.05\)).

Fig. 2. Endocardial impedance map of a healthy (A) and an infarcted (B) canine left ventricle (LV) (LAO view). Red areas: severely reduced impedance values (<147 \, \Omega); purple areas: normal impedance values (>192 \, \Omega).

Fig. 3. Bipolar voltage map of a healthy (A) and an infarcted (B) canine LV (LAO view). Red areas: severely reduced bipolar voltage (<0.7 \, \text{mV}); purple areas: normal bipolar voltage (>7 \, \text{mV}).
Correlation with Pathology

Gross pathological examination revealed the infarcted region to be a pale, unstained zone. Histological examination of samples obtained from the infarcted regions demonstrated typical changes of chronic infarct, namely, loss of myocytes and myocyte degeneration, domination of collagen deposits, inflammatory cells, macrophages (containing pigment, i.e., hemosiderin), and few capillaries. All healthy samples demonstrated normal myocyte morphology.

Local endocardial impedance. Local endocardial impedance measurement was found to be an accurate tool...
in defining the presence, extent, location, and border of myocardial infarction.

1) The percentage of the infarcted area as derived from the local endocardial impedance maps was calculated as the area in which impedance values were lower than a predefined threshold value (179.7 ± 13.4 Ω (means ± SE)) at the margin of the infarct in nine of the chronically infarcted dogs.

The percentage of EIA was then determined by pathological examination. Subsequently, high correlation (Pearson’s correlation coefficient = 0.942) was demonstrated between the percentage of endocardial infarcted area as determined from pathology [21.5 ± 2.7% (means ± SE)] and from the local endocardial impedance maps [23.6 ± 3.0% (means ± SE)] [Fig. 5; regression equation: EIA pathology (in %) = 0.843 × EIA impedance (in %) + 1.596].

2) In nine animals, after completion of the mapping procedure, the catheter was guided back to the periphery of the infarct (observed as the steepest impedance gradient), and three to four ablation points were applied (Fig. 6A). In all cases (n = 32), pathological examination of the LV revealed that the lesions were located precisely at the margins of the infarct (Fig. 6B).

Bipolar voltage. The threshold value for quantification of the percentage of infarct area in the bipolar voltage maps ranged from 2.5 to 3.7 mV and averaged 2.9 ± 0.4 mV. Pearson’s correlation coefficient was 0.797 for bipolar voltage [P < 0.05, regression equation: EIA pathology (in %) = 0.6764 × EIA bipolar voltage (in %) + 4.844].

DISCUSSION
Measurement of Local Endocardial Impedance

Myocardial tissue structure can be considered to consist of cells separated from the conducting medium surrounding them by an electrically insulating membrane. Hence, the components of the electrical model to be considered are the resistance of the intercellular and intracellular mediums and the cell membrane capacitance. When charged with AC, a high-resistivity cell membrane will act as a resistor or capacitor, and the low-resistance extracellular fluid will act as a conductor.

The results of the present study illustrate significant differences in endocardial impedance values of healthy versus necrotic myocardial tissue. Endocardial impedance values were reduced in the infarcted region when compared with both healthy regions in the same heart and with the corresponding region in healthy hearts. Furthermore, the present study demonstrates that endocardial impedance mapping can be used to delineate the presence, location, and extent of chronic canine myocardial infarction. This was evident by the high correlation between the infarcted area as depicted by both pathological examination and endocardial impedance maps and by the precise identification of the infarct borders.

The decrease in endocardial impedance values after scar formation may be explained by the increase in the extracellular-to-intracellular volume ratio. This increase, due to the reduced number of cardiac myocytes and low volume resulting from necrosis and scar formation (as observed in the histological examination), is associated with an increase in the extracellular pathways of electrical signal conduction. Cinca et al. (6) hypothesized that the reduced resistivity of scar tissue may be due to the biochemical composition of the extracellular matrix (various fibrous proteins immersed in an amorphous substance: mainly water and glycoproteins), which allows ionic diffusion. Subsequently, tissue impedance values decrease. Another hypothesis is that the improved conductance may be caused due to thinning of the LV wall and loss of cardiac tissue mass.

Impedance values have been shown to change with the progression of myocardial pathologies (5, 6, 9, 10, 17, 26) and therefore may be of clinical importance as a diagnostic index. However, due to methodological constraints, prior work has focused mainly on cell preparations, isolated papillary muscles, isolated hearts, and a limited number of in situ preparations in which measurement of impedance was performed intramyocardially (via an epicardial approach). These studies revealed the existence of a correlation between such measurements and cardiac wall motion (26) and showed a significant increase in impedance values during ischemia (6, 9, 10). This increase could be explained by ischemically induced cell swelling, resulting in a decrease in extracellular space. Because the majority of the measured current passes within the extracellular space, any reduction in this volume will result in increased impedance.

The results of this study are in agreement with the work of Fallert et al. (10) and Cinca et al. (6), whose observations led them to hypothesize that such changes may indeed be attributed to alterations in the extracellular space volume. With the use of the four-electrode method and an epicardial mapping technique, they observed significant changes in intramyo-
cardial impedance values subsequent to acute ischemia (an increase in impedance values) and aneurysm formation (a decrease in impedance values) in both sheep and pig models, respectively. However, to date, there is little information regarding utilization of impedance in infarct assessment (29).

Limitations of Present Study

Our study incorporates several theoretical limitations. Tissue contact. Endocardial impedance values may be strongly influenced by the degree of tissue contact. There are three possibilities when considering tip electrode-tissue contact: In scenario 1, the tip electrode is positioned in the LV cavity, surrounded only by blood; therefore, the impedance measured is solely that of the blood (which is lower than tissue impedance). In scenario 2, the tip electrode is positioned adjacent to the endocardium but not pressing against it. In scenario 3, the tip electrode is pressed against the endocardial surface so that part of it is engulfed by endocardial tissue.

In scenarios 2 and 3, the measured cardiac impedance is dependant on the degree of tissue contact, i.e., as the tip electrode is pressed more and more into the endocardium, it encounters less blood and more tissue. This has the effect of raising the measured impedance values, because blood is a highly conductive medium. Therefore, the more pressure exerted, the higher the measured impedance.

To compensate for this limitation, several criteria defining optimal catheter-tissue contact were used.
These included local activation time, electrogram morphology, and location and loop stability, which examined the motion and electrical repeatability of each endocardial site. When contact is not optimal, each of the aforementioned criteria is considered to have poor stability. In addition, tissue contact was assured by a sudden increase in impedance values on initiation of catheter-tissue contact.

Our experience has shown that contact pressure results in immediate ST segment elevation in the local intracardiac electrogram. Thus, whenever ST segment elevation was observed (>0.1 mV above baseline), the catheter was withdrawn slightly until the ST segment returned to baseline. Furthermore, on completion of the mapping procedure, any point in which ST segment elevation was observed was deleted from the map. An attempt was made to try to examine the effect of catheter pressure exertion on viable tissue and nonviable scar tissue. Our qualitative observation was that while catheter pressure led to increased endocardial impedance values in viable areas, such pressure did not have an effect on infarcted regions, i.e., no increase in impedance values was observed. This indicates that catheter pressure would affect the gradient of impedance values between infarcted and noninfarcted regions.

**Depth of field.** Any catheter-based method is limited to the endocardial surface and may therefore reflect the passive electrical properties of only the endocardium. For example, in scenario 2 above, this might be due to current passing from the tip electrode through the endocardium adjacent to it. The current then prefers the path of lowest resistance, i.e., blood, instead of the relatively high-resistance myocardial tissue on its way back to the reference electrode.

Furthermore, current density is calculated by dividing the current amplitude by the area onto which it spreads. Therefore, current density is strongest at the place of electrode insertion and decays as a function of its distance. Hence, one may understand that a major conceptual limitation of our study of endocardial impedance concerns the depth of field demonstrated.

These constraints were not applicable to previous studies that performed impedance measurements using the four-electrode technique, in which measuring probes were inserted into the myocardial tissue (e.g., 3 mm in the study by Fallert et al. [10]).

**Measurement of impedance values.** Previously, impedance measurement using the four-electrode technique was devoid of the electrode-tissue impedance and, hence, produced a “true” absolute value of tissue impedance. Conversely, while using a two-electrode system, as with this study, the measured absolute tissue impedance incorporates the series electrode-tissue impedance as well as the “true” tissue impedance. Nonetheless, the aim of the current study was to distinguish between infarcted and healthy myocardium. Hence, whereas this limitation is significant when assessing absolute values, the importance of our findings is in the relative difference between the impedance measured in healthy and necrotic tissue.

Impedance values may change throughout the cardiac cycle and may display minute beat-to-beat variability. During systole, when the catheter was in stricter contact with the adjacent endocardium, an increase in endocardial impedance values of up to 20 Ω was observed (relative to diastole). This observation is in accordance with a previous study by Sasaki et al. (26), who reported synchronization of intramyocardial impedance measurements with cardiac contraction and changes in wall thickness (the observed change ranged from 5 to 20 Ω in the normal myocardium). To “average out” this effect, endocardial impedance values were averaged over a period of 1,000 ms at each site.

It is noteworthy that measurements of end-diastolic gated impedance, in a small subset of animals, revealed similar results to the averaged impedance values. Specifically, a reduction of values in infarcted as opposed to healthy myocardium. It would be interesting to define the contribution of decreased systolic wall motion to the impedance recorded. Indeed, because endocardial measurements have been shown to depend on the degree of contact between the electrode and the tissue, endocardial impedance measured at end diastole alone would not enable precise assessment of the tissue changes during myocardial infarction.

**Clinical Significance and Future Research Implications**

Because dysfunctional but viable myocardium can recover its function after revascularization, its identification has become an important objective of clinical cardiology. Therefore, various physiological markers have been developed in an attempt to recognize dysfunctional but viable areas.

Recent studies (4, 11, 18) have proposed a new concept of viability assessment by examining the electromechanical properties of the endocardium. In these studies, the reduction in bipolar signal amplitudes has proved useful in denoting the presence of necrotic tissue. However, the directional sensitivity of such recordings is well established (16), and we have occasionally encountered low bipolar voltage amplitudes in portions of the LV in the healthy canine, namely, the apical, midventricle, and anterior regions (unpublished results, Wolf et al.). In the current study, the midanterior, midanteroseptal, and apical regions within the healthy LV demonstrated average bipolar voltage amplitude values greater than those in the infarcted group. However, 102 of 304 points (34%) obtained within these regions had bipolar voltage amplitudes below the minimal bipolar threshold for infarct quantification (2.5 mV). Such false positive recordings in healthy myocardial tissue may contribute to inaccurate interpretation of the mapping results while performing comparison of bipolar voltage values between healthy and infarcted regions. Because unipolar recordings remained elevated in these regions, our hypothesis, which remains to be proven, is that these false positive recordings are due to the mapping method. Hence, whereas it is clear both from the
present study and from previous ones that bipolar voltage amplitude accurately depicts infarcted tissue, other parameters may be of additive value.

Despite the limitations discussed above, local endocardial impedance is capable of delineating the presence and extent of myocardial infarction. Moreover, the importance of endocardial impedance may be as a diagnostic utility in cases where bipolar recordings give a false positive impression or possibly as a joint index, which would enhance the precision of existing electrical parameters. Furthermore, the limitation relating to depth of field during endocardial impedance measurements might serve as an advantage when trying to assess and differentiate subendocardial from transmural infarcts, in which both voltage and LS values may not enable differentiation.

The results of the present study should be pursued in further research in an attempt to describe the characteristic endocardial impedance values of the different myocardial pathologies. A major goal of this study was the creation of a standardized procedure in which simple impedance measurements could be obtained in a minimally invasive manner. Hence, on the basis of previous reports and on the concept of endocardial impedance measurement presented in this study, measurements in human cardiac tissue could be performed. Subsequently, both human data and data from animal experiments pertaining to different cardiac pathologies, such as acute ischemia and the hibernating and stunned myocardium, could be carried out with relative ease and compared. In addition, optimization of this method may be achieved by localization of impedance measurements, such as using bipolar measurements as opposed to the current unipolar method. In addition, measurement of impedance at discrete time points throughout the cardiac cycle, namely, end systole and end diastole, may provide information on the effect of wall motion on impedance measurements.

The current study utilized a precedent frequency of 50 kHz for impedance measurements (26). Further evaluation of various myocardial pathologies may be performed by testing different current frequencies aimed at assessing the varying contribution of tissue capacitance and resistance to the endocardial impedance values.

The ability to accurately identify the border of the necrotic tissue may enhance the sensitivity of existing therapeutic tools. For example, in the field of clinical electrophysiology, ablation paradigms aimed at treatment of various ventricular arrhythmias may rely on accurate identification of the infarct border.

In conclusion, this study examined the passive electrical properties of the endocardium using local impedance measurements. We conclude that such measurements can be used to differentiate necrotic from healthy myocardial tissue. The ability to accurately determine the presence and extent of myocardial infarction may enhance the efficacy of real-time viability assessment in the catheterization laboratory.

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