Comparison of Noncontact and Electroanatomic Mapping to Identify Scar and Arrhythmia Late after the Fontan Procedure

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Background—The right atrium late after the Fontan procedure is characterized by multiple complex arrhythmia circuits.

We performed simultaneous electroanatomic and noncontact mapping to assess the accuracy of both systems to identify scar and arrhythmia.

Methods and Results—Mapping was performed in 26 patients aged 26.8±8.9 years, 18.7±4.4 years after Fontan surgery. The area and site of abnormal endocardium defined by electroanatomic mapping (bipolar contact electrogram <0.5 mV) were compared with those defined by noncontact mapping during sinus rhythm and by dynamic substrate mapping. Contact and reconstructed unipolar electrograms at a known distance from the multielectrode array, recorded by the noncontact system simultaneously at 452 endocardial sites, were compared for morphological cross correlation, timing difference, and amplitude. Mapping of arrhythmias was performed with both systems when possible. The median patient abnormal endocardium as defined by electroanatomic mapping covered 38.0% (range 16.7% to 97.8%) of the right atrial surface area, as opposed to 60.9% (range 21.3% to 98.5%) defined by noncontact mapping during sinus rhythm and 11.9% (range 0.4% to 67.3%) by dynamic substrate mapping. A significant decrease in electrogram cross correlation (P=0.003), timing (P=0.012), and amplitude (P=0.003) of reconstructed electrograms, but not of contact electrograms (P=0.742), was seen at endocardial sites >40 mm from the multielectrode array. Successful arrhythmia mapping by electroanatomic versus noncontact mapping was superior in 15 patients (58%), the same in 6 (23%), and inferior in 5 (19%; P=0.044).

Conclusions—Electroanatomic mapping is the superior modality for arrhythmia mapping late after the Fontan procedure. Noncontact mapping is limited by a significant reduction in reconstructed electrogram correlation, timing, and amplitude >40 mm from the multielectrode array and cannot accurately define areas of scar and low-voltage endocardium.

Key Words: mapping | arrhythmia | electrophysiology | ablation | Fontan procedure

Arrhythmia late after the Fontan procedure is believed to result from significant right atrial (RA) dilatation and scarring, which facilitates multiple potential reentrant circuits. Electroanatomic mapping allows sequential acquisition of contact bipolar electrograms (EGMs) to produce accurate definition of cardiac activation and endocardial amplitude. Although this has been used with clinical success after the Fontan procedure and has been shown to improve freedom from arrhythmia in congenital heart disease, it may be limited by nonsustained arrhythmia and/or hemodynamic instability. Noncontact mapping uses >3000 simultaneously and continuously recorded EGMs to provide continuous assessment of global atrial activation and may therefore overcome the limitations of sequential contact mapping, but its efficacy is limited by increasing distance of the multielectrode array (MEA) from the endocardium and the inability of unipolar EGMs to accurately define areas of scar. Recent studies in both humans and animal models have assessed different techniques with the noncontact system to better identify scar and low-voltage endocardium, with mixed results. To date, no prospective direct comparison has

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of unipolar EGMs to accurately define areas of scar. Recent studies in both humans and animal models have assessed different techniques with the noncontact system to better identify scar and low-voltage endocardium, with mixed results. To date, no prospective direct comparison has
been undertaken of contact and noncontact mapping systems in the clinical arena. The purpose of the present study was to examine the ability of the noncontact mapping system to accurately reconstruct EGMs and to identify scar and low-voltage endocardium in the human RA late after the Fontan procedure by comparison with simultaneous electroanatomic mapping and to assess the ability of both systems to identify different arrhythmia mechanisms.

Methods

Patients
Twenty-six adult patients (median age 26.8 ± 8.9 years) with a diagnosis of tricuspid atresia (n = 16), double-inlet ventricle (n = 9), and congenitally corrected transposition (n = 1) were studied 18.7 ± 4.4 years after either the atriopulmonary (n = 23) or atrioventricular (n = 3) Fontan procedure. Patients were selected on the basis of documented atrial arrhythmia resistant to ≥1 antiarrhythmic medication, including amiodarone (n = 17). Accurate knowledge of the underlying anatomy and prior surgical intervention was confirmed by reference to operation notes. Anatomic obstruction and atrial thrombus were excluded by cardiac magnetic resonance and/or transesophageal echocardiography, and oral anticoagulation was used in all patients. Antiarrhythmic drugs were stopped ≥5 half-lives before the study (amiodarone ≥2 months) unless sustained arrhythmia had been documented on current medication. The local ethics committee approved the study, and patients gave written consent before the procedure.

Electrophysiology Study
Studies were performed with simultaneous noncontact (EnSite; St. Jude Medical, St. Paul, Minn) and electroanatomic (CARTO; Biosense Webster, Diamond Bar, Calif) mapping systems. Contact catheter data were recorded simultaneously on a conventional electrophysiology system (LabSystem, Bard Electrophysiology, Murray Hill, NJ). Heparin boluses were used to maintain an activated clotting time >300 seconds. Sinus rhythm (SR) was recorded by both noncontact and electroanatomic systems at the beginning of the case, before any intervention. In the event of sustained tachycardia at 200 mm/s. Local endocardial activation was taken as the first steep deflection away from the baseline.

Noncontact Mapping
The noncontact mapping system has been described in detail previously. Briefly, a noncontact MEA mounted onto a 7.5-mL balloon detects raw data from the endocardial surface, which are relayed to a computer workstation via an amplifier. The MEA was placed over a 0.035-in J-wire and advanced from the left femoral vein to either the superior vena cava or left pulmonary artery. The noncontact system was used to locate a Navistar mapping catheter (Biosense Webster) with respect to the MEA to create a computer model of the endocardium (“virtual endocardium”). A ring electrode positioned on the proximal shaft of the MEA was used both to record a contact unipolar EGM from the distal electrode of the mapping catheter and as a reference for the reconstructed unipolar EGM.

Electroanatomic Mapping
Electroanatomic mapping was performed with the quadrupolar Navistar mapping catheter, which enabled color-coded atrial activation referenced to a stable bipolar catheter to be superimposed on a 3D geometry. Bipolar signals were filtered at 30 to 400 Hz and displayed at 200 mm/s. Local endocardial activation was taken as the first steep deflection away from the baseline.

Offline Analysis

Chamber Size and Distance From MEA
The RA surface area was automatically calculated by the electroanatomic system and by the noncontact system with a customized Excel template (Microsoft Corp, Redmond, Wash). To determine the RA surface area >40 mm from the MEA, in each patient, we recorded the distance of 256 equally distributed endocardial points from the MEA.

Determination of Abnormal Endocardium
The identification of scar tissue and abnormal endocardium with the electroanatomic mapping system has been validated previously against pathological13,14 and echocardiographic14 findings in animal models. On the basis of the results of previous clinical studies,15–17 abnormal endocardium was defined as the sum of (1) scar (no identifiable EGM or bipolar amplitude ≤0.05 mV15,16) and (2) low-voltage endocardium with bipolar contact amplitude of ≤0.5 mV.16,17

Bipolar voltage maps were created in which the color scale indicated local peak-to-peak EGM amplitude; scar was depicted by gray, normal endocardium by purple, and low-voltage endocardium by colors red to purple (Figure 1). A surface-area calculation tool was used to determine the area of abnormal endocardium, expressed as a fraction of the RA surface area.

To identify abnormal endocardium with the noncontact mapping system, we used previously described techniques that suggest this is the area where local EGM amplitude is <30% of the peak negative (<30%PN) EGM on static isopotential maps.18 During SR, this area was termed SR <30%PN. In an attempt to minimize “far-field” oversensing, we used dynamic substrate mapping (DSM), a technique that involves additional sequential pacing at 800 ms from multiple sites within the RA, to identify the area consistently <30%PN (ie, the intersection of all areas <30%PN defined during SR and pacing). This area was termed DSM <30%PN (Figure 1). During pacing, a blanking period was applied to eliminate stimulus artifact. The areas of SR <30%PN and DSM <30%PN were defined with a surface-area measurement tool incorporated in the noncontact mapping system and expressed as a fraction of the total RA area. In addition, the areas encompassed by <10%PN, <20%PN, and <50%PN were also defined during both SR and DSM. Using anatomic landmarks such as the caval vein orifices, RA to pulmonary artery connection, and coronary sinus os, we were able to determine an approximate measure of geometric concordance of the location of abnormal endocardium determined by the 2 mapping systems, defined as follows: good (>75% concordance between the 2 areas), moderate (50% to 75%), and poor (<50%).

Reconstructed EGM Validation
To assess the efficacy of the noncontact mapping system, we compared reconstructed unipolar EGMs from the endocardial surface with a simultaneously acquired contact unipolar EGM sampled from the distal pole of the mapping catheter tip at the identical location. The 2 EGMs were compared as described below (Figure 2).

Morphological Cross Correlation

The morphology of the reconstructed and contact EGM was compared with a validated18–20 template-matching algorithm that produces a cross correlation [C(k)] that indicates the morphological similarity, where X and Y are the corresponding amplitudes of all points constituting the reconstructed and contact EGMs, respectively:

\[
C(k) = \frac{\sum_{i=0}^{n-1} (X_i - \bar{X}) \times (Y_{i+k} - \bar{Y})}{\sqrt{\sum_{i=0}^{n-1} (X_i - \bar{X})^2} \sqrt{\sum_{i=0}^{n-1} (Y_{i+k} - \bar{Y})^2}}
\]
interval of as double potentials separated by an isoelectric or low-amplitude arrhythmia mechanism was assessed. Conduction block was defined if a complete loop of atrial activation could be demonstrated on isopotential or activation maps, corroborated by corresponding noncontact map demonstrates a large area of dense scar (gray) occupying the lateral wall of the RA, surrounded by low-voltage endocardium. ii, Corresponding noncontact map demonstrates the area defined by DSM <30%PN on the inferior aspect of the lateral wall, demarcated by the green line. Poor concordance (<50%) is seen between the 2 mapping systems. SVC indicates superior vena cava; IVC, inferior vena cava.

Timing Difference
The time shift \( k \) over a window of \( \pm 50 \) ms required to create the best morphological correlation between the 2 EGMs was taken as the timing difference.

Amplitude
Because the morphological correlation may be insensitive to changes in amplitude, contact and reconstructed EGMs were additionally expressed as the root mean squared of all positive and negative components of the EGM. To compensate for the effect of multiple measurements per patient, a mean value was calculated for EGM morphology, timing, and amplitude in each patient for endocardial sites <40 and >40 mm from the MEA.

Detection of Arrhythmia
Electroanatomic mapping and noncontact mapping were used to map all encountered arrhythmias, and subsequent analysis of those arrhythmias recorded successfully was performed offline. Macroreentrant arrhythmias were defined as an organized atrial tachycardia with stable cycle length, EGM morphology, and consistent pattern of atrial activation. Macroreentrant arrhythmias were only accepted as successfully mapped if a complete loop of atrial activation could be demonstrated on isopotential or activation maps, corroborated by noncontact and contact EGMs, and >90% of the tachycardia cycle length could be accounted for within the RA. Where appropriate, the contribution of fixed and/or functional conduction block to arrhythmia mechanism was assessed. Conduction block was defined as double potentials separated by an isoelectric or low-amplitude interval of >50 ms, conduction delay of >50 ms in adjacent contact electrodes, or acute wavefront cessation. Fixed conduction block was present under all circumstances, whereas functional block was deemed to be a factor of atrial cycle length or direction of activation. Focal arrhythmia was defined as an organized atrial tachycardia with stable cycle length, ECG morphology, and consistent pattern of centrifugal activation from a focal source.

To compensate for the fact that multiple arrhythmias may be recorded in any 1 patient, the efficacy of the 2 mapping systems to identify arrhythmias was calculated for the total number of patients and by dividing patients into groups according to different mechanisms. In any given patient, if electroanatomic mapping identified a greater number of arrhythmias than noncontact mapping, this was deemed to be the superior technique irrespective of the number of arrhythmias, and vice versa. If both systems identified the same number of arrhythmias, they were deemed equally effective/ineffective. For each of the 5 different arrhythmia mechanisms, the superiority of either electroanatomic or noncontact mapping was determined in an identical fashion. Patients with more than 1 identified arrhythmia mechanism (eg, functional and focal) were included in each appropriate group.

Radiofrequency Ablation
Irrigated radiofrequency ablation (RFA; 30 mL/min; 45°C/50 W) was used in all cases, and pace mapping and/or entrainment pacing was used to identify candidate sites for RFA when necessary. Success was defined in each patient as termination during RFA in the absence of atrial ectopy and/or noninducibility by rapid burst atrial pacing or programmed atrial stimulation of all targeted arrhythmia mechanisms.

Statistical Analysis
Continuous variables were assessed for normal distribution by Kolmogorov-Smirnov analysis. Normally distributed data were expressed as mean±SD and nonnormally distributed data as median (range). Comparisons between noncontact and electroanatomic voltage characteristics were assessed by the Bland-Altman technique. The distribution of EGM data <40 and >40 mm from the MEA in each patient was compared with the Wilcoxon signed rank test. The McNemar test with a continuity correction was used to determine the superiority of either electroanatomic mapping or noncontact mapping to detect arrhythmia in each patient. Ablation success was assessed per patient with the Fisher exact test. Statistical significance was taken as \( P<0.05 \). Data were analyzed with StatView (SAS.
Figure 2. A, Method of reconstructed ECG validation. The RA is displayed on the left of the figure, as seen from the right and left anterior oblique views. The mapping catheter is seen on the anteromedial wall (green spot) at a known distance from the center of the MEA (yellow line). Two unipolar EGMs, 1 contact and 1 reconstructed, are recorded simultaneously from the catheter tip and by the MEA, respectively. See Methods for full details of comparison between the 2 EGMs. RMS indicates root mean squared. B, Results of reconstructed EGM validation with the noncontact mapping system. Four examples demonstrate the comparative analysis performed between the reconstructed and contact unipolar EGMs, both of which are shown with lead V1. The results are as follows: a) Morphological correlation (Xcorr) 0.99; timing difference +0.83 ms; contact amplitude (RMS) 1.0 mV; reconstructed amplitude (RMS) 0.92 mV. b) Xcorr 0.99; timing difference +1.67 ms; contact amplitude (RMS) 0.74 mV; reconstructed amplitude (RMS) 0.42 mV. c) Xcorr 0.48; timing difference +9.17 ms; contact amplitude (RMS) 0.63 mV; reconstructed amplitude (RMS) 0.31 mV. d) Xcorr 0.36; timing difference −50 ms; contact amplitude (RMS) 0.38 mV; reconstructed amplitude (RMS) 0.18 mV. Examples (a) and (b) demonstrate that amplitude differences between the 2 EGMs can be significant despite an excellent morphological correlation.
### Results

#### RA Surface Area

The median RA surface area as defined by electroanatomic and noncontact mapping systems was $235 \text{ cm}^2$ (range $136$ to $455 \text{ cm}^2$) and $233 \text{ cm}^2$ ($108$ to $518 \text{ cm}^2$), respectively. The distance of $6400$ endocardial sites ($256$ from each patient) from the MEA was $36.9$ mm ($10.0$ to $108.2$ mm), from which it could be calculated that $35.2\%$ ($11.7\%$ to $77.0\%$) of the RA surface area was $>40$ mm from the MEA.

#### Determination of Abnormal Endocardium

Electroanatomic mapping of SR was not possible in $2$ patients owing to sinotriarial node disease, and noncontact mapping was not possible in $1$. Comparisons between scar and noncontact mapping could therefore be made in $25$ patients, and comparisons between abnormal endocardium and noncontact mapping could be performed in $23$. DSM $<30\%\text{PN}$ was performed in $11$ patients at a mean of $3.8$ pacing points per patient. The median patient abnormal endocardium as defined by electroanatomic mapping covered $38.0\%$ ($16.7\%$ to $97.8\%$) of the RA surface area (scar $15.07\%$ [$1.89\%$ to $56.15\%$] and low-voltage endocardium $25.25\%$ [$7.38\%$ to $55.11\%$]) as opposed to $60.9\%$ ($21.3\%$ to $98.5\%$) defined by noncontact mapping during SR and $11.9\%$ ($0.4\%$ to $67.3\%$) by DSM. Comparative analysis between electroanatomic and noncontact mapping to define abnormal endocardium is depicted in Table 1 and Figure 3. For $<10\%\text{PN}$ and $<20\%\text{PN}$, either SR or $1$ or more pacing points failed to identify any region with reconstructed EGM voltage of $<10\%$ or $20\%$ of the peak negative voltage. Because of this, a valid comparison could not be made between the area of abnormal endocardium defined by DSM $<10\%\text{PN}$ and DSM $<20\%\text{PN}$ with that defined by DSM $<30\%\text{PN}$ in $9$ of $11$ and $4$ of $11$ patients, respectively. The degree of geometric concordance between the $2$ systems was assessed between the nearest comparison (electroanatomic scar versus DSM $<30\%\text{PN}$), which was good in only $2$ cases and poor in the remaining $9$ (Figure 1).

#### Detection of Arrhythmia

We encountered $43$ RA arrhythmias, $3$ left atrial arrhythmias, and $1$ of undetermined mechanism. Left atrial arrhythmias were identified by an inability to entrain the arrhythmia from the RA endocardium. Of the RA arrhythmias, $33$ were macroreentrant, $9$ were focal, and $1$ was atrioventricular nodal reentry tachycardia. For the purposes of analysis, additional sources of centrifugal RA activation (3 left atrial arrhythmias and 1 atrioventricular nodal reentry tachycardia) were classified as focal. Macoreentrant arrhythmias were further classified into $4$ groups as follows: (1) fixed—wavefront rotation perpendicular to the vertical axis of the RA, dependent on fixed conduction block on the posterolateral wall of the RA at the position of the crista terminalis ($n=7$); (2) functional—wavefront rotation perpendicular to the vertical axis of the RA, dependent on fixed and functional conduction block on the posterolateral wall of the RA at the position of the crista terminalis ($n=8$); (3) isthmus—pericardial reentry via the cavitricuspid isthmus ($n=5$); and (4) scar—scar-related reentry in which the wavefront rotates

### Table 1. Comparison Between Scar and Abnormal Endocardium as Defined by Electroanatomic Mapping and Noncontact Mapping Both During SR and With DSM

<table>
<thead>
<tr>
<th>Noncontact Mapping (%PN EGM)</th>
<th>No. of Patients Available for Analysis</th>
<th>Electroanatomic Mapping: Scar</th>
<th>No. of Patients Available for Analysis</th>
<th>Electroanatomic Mapping: Abnormal Endocardium</th>
</tr>
</thead>
<tbody>
<tr>
<td>SR $&lt;30%\text{PN}$</td>
<td>25</td>
<td>$-39.85\pm11.95%$</td>
<td>23</td>
<td>$-13.86\pm14.93%$</td>
</tr>
<tr>
<td>DSM $&lt;30%\text{PN}$</td>
<td>11</td>
<td>$-6.65\pm16.25%$</td>
<td>11</td>
<td>$18.80\pm18.45%$</td>
</tr>
<tr>
<td>SR $&lt;10%\text{PN}$</td>
<td>25</td>
<td>$8.03\pm7.87%$</td>
<td>23</td>
<td>$34.22\pm9.93%$</td>
</tr>
<tr>
<td>SR $&lt;20%\text{PN}$</td>
<td>25</td>
<td>$-19.80\pm13.41%$</td>
<td>23</td>
<td>$6.25\pm15.92%$</td>
</tr>
<tr>
<td>SR $&lt;50%\text{PN}$</td>
<td>25</td>
<td>$-59.08\pm9.08%$</td>
<td>23</td>
<td>$33.30\pm12.86%$</td>
</tr>
<tr>
<td>DSM $&lt;10%\text{PN}$</td>
<td>11</td>
<td>$13.55\pm6.15%$</td>
<td>11</td>
<td>$39.01\pm7.50%$</td>
</tr>
<tr>
<td>DSM $&lt;20%\text{PN}$</td>
<td>11</td>
<td>$6.98\pm9.22%$</td>
<td>11</td>
<td>$32.07\pm11.31%$</td>
</tr>
<tr>
<td>DSM $&lt;50%\text{PN}$</td>
<td>11</td>
<td>$-34.37\pm18.02%$</td>
<td>11</td>
<td>$-8.92\pm21.41%$</td>
</tr>
</tbody>
</table>

With the noncontact system, abnormal endocardium was determined with variable ranges of the peak negative electrogram (%PN EGM), from $<10\%$ to $<50\%$. The result is expressed as the bias (mean area of electroanatomic mapping minus mean area of noncontact mapping) $\pm 2\ SE$ (95% confidence interval). The results for electroanatomic mapping against SR $<30\%$ and DSM $<30\%$ are displayed in Figure 3.
around scar or sites of fixed conduction block remote from anatomic obstacles (n=13).

Electroanatomic mapping identified a significantly greater proportion of all arrhythmias (P=0.044), but by individual mechanisms, only scar-related macroreentry was identified significantly more often by electroanatomic mapping (P=0.044). Results are summarized in Table 2. In none of the 4 patients with functional conduction block supporting the arrhythmia in whom DSM was performed did DSM <30% identify the site of functional conduction block. All 9 arrhythmias for which noncontact mapping alone was successful were not sufficiently sustained to permit electroanatomic mapping.

Radiofrequency Ablation
RFA was performed in 24 patients, targeting 41 of 43 RA arrhythmias, 33 during tachycardia and 8 during SR. Ablation was not performed in 2 patients with clinical left atrial arrhythmias, 3 during tachycardia and 8 during SR. Ablation was not performed in 2 patients with clinical left atrial arrhythmias and 2 RA arrhythmias owing to focal His bundle origin and inability to reinduce a focal arrhythmia terminated

EGM characteristics versus distance from the MEA

- **Figure 3.** Comparison of electroanatomic and noncontact mapping in the identification of scar. Scar and abnormal endocardium (scar plus low-voltage endocardium [<0.5 mV bipolar amplitude]) identified by electroanatomic mapping were compared with the area defined by noncontact mapping where the local unipolar reconstructed EGM amplitude was <30% of the peak negative EGM both during SR (A and B) and DSM (C and D). The Bland-Altman plots depict the difference vs the mean of the 2 values. The mean difference (bias) and 95% levels of agreement (±2 SD) are depicted by solid and dashed lines, respectively. The wide spread of points around the bias suggests poor agreement between the 2 techniques.

- **Figure 4.** EGM characteristics <40 or >40 mm from the MEA. Line charts demonstrate the mean value for each patient of (A) EGM morphological correlation (Xcorr), (B) timing, (C) contact EGM, and (D) reconstructed EGM amplitude (root mean squared [RMS]) <40 and >40 mm from the MEA. A significant deterioration in the morphological correlation, timing, and amplitude of the reconstructed EGM can be seen >40 mm from the MEA. E, Similarly, the ratio of contact to reconstructed EGM amplitude increases significantly at >40 mm from the MEA, which indicates a fall in reconstructed EGM amplitude relative to its contact counterpart.
Determination of Abnormal Endocardium

Electroanatomic bipolar voltage maps using previously reported amplitude criteria demonstrated that after the Fontan procedure, a significant proportion of the RA surface area is covered by abnormal endocardium (scar or low-voltage endocardium \(<0.5\, \text{mV}\)). Although this has been documented previously, this is the first time it has been accurately quantified in patients with congenital heart disease. For all comparisons between electroanatomic mapping and noncontact mapping using the previously defined figure of \(<30\%\text{PN}\), the corroboration between the 2 systems was poor, as judged by the bias and wide levels of agreement (Table 1; Figure 3) and supported by the finding of poor geometric concordance between scar defined by electroanatomic mapping and DSM \(<30\%\text{PN}\) in 9 of 11 patients. Identification of abnormal endocardium with noncontact mapping therefore appears to be fundamentally limited by the decrease in reconstructed EGM amplitude with increasing distance of the MEA from the endocardium. Additionally, the use of both \(<10\%\text{PN}\) and \(<20\%\text{PN}\) is further limited by the finding that in some patients (with clear evidence of abnormal endocardium on electroanatomic contact maps), the area of abnormal endocardium is 0 cm².

Bipolar contact EGMs remain the most commonly used and clinically accepted method to define areas of abnormal endocardium and arrhythmia. However, we have shown that this method may be misleading if used to define low-voltage endocardium in patients with enlarged atria.

Two previous studies have assessed the ability of DSM to identify scar in experimental models of myocardial infarction, with conflicting results. In line with the findings of the
present study, Thigalingam et al. found neither reconstructed EGMs nor DSM reliably identified ventricular scar. Conversely, Jacobson et al. found DSM to be a reliable method for the localization of infarcted myocardium compared with pathological findings, although the technique used to create an infarct of predictable location produced a dense transmural lesion, which may have little resemblance to the substrate produced by myocardial ischemia in humans or by surgical intervention and chronic stretch, as in the Fontan procedure.

**Reconstructed EGM Validation**

The results of the present study support the findings of previous investigators that at distances >40 mm between the endocardium and MEA, the accuracy of reconstructed EGM correlation and timing falls significantly (Figure 4). That such a significant proportion of the RA endocardium is >40 mm from the MEA suggests that EGM reconstruction in large areas of endocardium will be poor in the majority of patients after the Fontan procedure.

**Detection of Arrhythmia and RFA**

Electroanatomic mapping was the superior modality in arrhythmia identification in a significantly greater number of patients in all arrhythmias encountered. Additionally, the electroanatomic system was superior in the identification of scar-based macroreentry arrhythmia, in keeping with our finding that defining endocardial that has <30% voltage of the peak negative voltage with noncontact mapping does not accurately detect either scar or abnormal endocardium. Although the difference was not statistically significant, electroanatomic mapping identified all periannular arrhythmias, as opposed to only 1 identified by the noncontact system. This is not surprising given the natural position adopted by the MEA at the posterolateral aspect of the chamber, which is anatomically distant from the cavitricuspid isthmus. Similarly, noncontact mapping identified more arrhythmias that were dependent on functional conduction block at the crista terminalis, many of which were not sufficiently sustained to permit sequential contact mapping. No difference existed in RFA success between arrhythmias identified by either mapping modality, which suggests that when either mapping system does define an arrhythmia mechanism, this is clinically relevant, and the use of these maps to guide RFA is effective.

**Limitations of the Study**

At present, no system exists that would allow direct comparison between the anatomic location of scar and DSM <30% PN, and the degree of concordance was based purely on visual impression with empirical values considered to be of relevance to the clinical electrophysiologist. The advent of image integration to both electroanatomic and noncontact mapping systems may facilitate this process. Contact between the catheter and endocardium was assessed only by fluoroscopy and operator opinion as opposed to catheter impedance, which may have overestimated the areas of abnormal endocardium defined by electroanatomic mapping. The parameters used to define scar and abnormal endocardium are based on previous clinical studies, and as far as we are aware, they have not been validated by histological findings.

**Conclusions**

Electroanatomic mapping is the superior modality for arrhythmia mapping late after the Fontan procedure. Conversely, because large areas of RA endocardium are >40 mm from the MEA, the utility of noncontact mapping is limited by a significant reduction in reconstructed EGM correlation, timing, and amplitude. Additionally, noncontact mapping cannot accurately define areas of scar and low-voltage endocardium.

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**Disclosures**

Drs Abrams and Schilling are members of the Speakers’ Bureau for St. Jude Medical. Dr Schilling is a member of the Scientific Advisory Board of Biosense Webster. The remaining authors report no conflicts.

**References**


In early modifications of the Fontan procedure, the right atrium (RA) acts as the subpulmonary chamber anastomosed either directly to the pulmonary arteries or via the diminutive right ventricle. Atrial arrhythmias are a major cause of morbidity late after the Fontan procedure, facilitated by a dual process of severe RA dilatation and the development of atrial scarring and areas of low-voltage endocardium. Both electroanatomic (CARTO) and noncontact (EnSite) mapping have been used with varying degrees of success to map atrial arrhythmia in patients with congenital heart disease, although to date, no direct comparison of the 2 systems has been performed. In 26 adult patients (aged 26.8 ± 8.9 years) late after the Fontan procedure, we used simultaneous electroanatomic and noncontact mapping to determine the relative ability of the 2 systems to identify atrial scarring and arrhythmia. Compared with the electroanatomic system, which uses bipolar contact electrograms, we found the noncontact system did not accurately define scar either in terms of RA surface area or anatomic location. Similarly, electroanatomic mapping was superior in the identification of arrhythmia in all patients (P=0.044) and in those with scar-based reentrant arrhythmia (P=0.044) but not for those with other arrhythmia mechanisms. This relates to the significant deterioration in noncontact unipolar electrogram morphology (P=0.003), timing (P=0.012), and amplitude (P=0.003) at distances >40 mm from the multielectrode array. The median area of RA endocardium located >40 mm from the multielectrode array was 35.2% (range 11.7% to 77.0%). Noncontact mapping is limited late after the Fontan procedure by severe RA dilatation that significantly reduces the accuracy of electrogram reconstruction, and it cannot accurately define atrial scar. Electroanatomic mapping is therefore the optimal modality to identify the electrophysiological substrate and arrhythmia mechanism late after the Fontan procedure.
Comparison of Noncontact and Electroanatomic Mapping to Identify Scar and Arrhythmia Late After the Fontan Procedure


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