Ventricular Structure and Function

Noninvasive Localization of Accessory Pathways in Wolff–Parkinson–White Syndrome by Three-Dimensional Speckle Tracking Echocardiography

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Background—We have developed a noninvasive isochrone activation imaging (AI) system with 3-dimensional (3D) speckle tracking echocardiography (STE), which allows visualization of the wavefront image of mechanical propagation of the accessory pathway (ACP) in Wolff–Parkinson–White syndrome.

Methods and Results—Patients with manifest Wolff–Parkinson–White syndrome were imaged in 3D-STE AI mode, which quantified the time from QRS onset to regional endocardial deformation. In 2 patients with left- and right-side ACP, we confirmed that intraoperative contact endocardial electric mapping and the 3D-STE AI system showed comparable images pre- and postablation. In normal heart assessment by 3D-echo AI, the earliest activation sites were found at the attachment of the papillary muscles in the left ventricle and midseptum in the right ventricle, and none showed earliest activation at the peri-atrioventricular valve annuli. An analyzer who was unaware of the clinical information assessed 39 ACP locations in 38 Wolff–Parkinson–White syndrome patients using 3D-STE. All showed abnormal perimital or tricuspid annular activations, and the location of 34 ACP (87%) showed agreement with the successful ablation sites within a 2-o’clock range. Especially for left free wall ACP, 17/18 (94%) showed consistency with the ablation site within a 2 o’clock range. Among 15 ACP at the ventricular septum, 9 (60%) showed early local activation in both right and left sides of the septum.

Conclusions—Isochrone AI with 3D-STE may be a promising noninvasive imaging tool to assess cardiac synchronized activation in normal hearts and detect abnormal breakthrough of mechanical activation from both atroventricular annuli in Wolff–Parkinson–White syndrome. (Circ Cardiovasc Imaging. 2016;9:e004532. DOI: 10.1161/CIRCIMAGING.116.004532.)

Key Words: echocardiography, 3-dimensional □ isochrone imaging □ speckle tracking echocardiography □ Wolff–Parkinson–White syndrome

Three-dimensional (3D) echocardiography provides noninvasive information of the real heart morphology throughout the cardiac cycle without any assumptions or through-plane effects, which is the intrinsic limitation of conventional 2-dimensional (2D) imaging of the beating heart. Speckle tracking echocardiography (STE), based on the 3D volume speckle data unit, is the ideal modality in comparison to 2D-based speckle tracking because 3D-STE is free from the loss of tracking of the target ultrasonic speckle within the 3D region of interest, whereas the 2D echo speckle may disappear from the ultrasonic 2D beam plane and result in noise. 3D-STE has been extensively validated in sheep experiments in both the left (LV)2 and right (RV) ventricles. Furthermore, 3D-STE can provide the temporal sequence of myocardial deformation (iscochrones) in addition to the magnitude of strength of the myocardial contraction (ie, strain or torsion).

See Editorial by Maréchaux

See Clinical Perspective

Activation imaging (AI) mapping has recently been proposed as a modality to noninvasively map isochrones based on 3D-STE,4 and it is reported to reveal LV and RV dysynchrony5,6 in patients who have undergone cardiac resynchronization therapy. To evaluate further the clinical utility of 3D-STE-derived AI mapping, we attempted to apply AI mapping to detect the preexcitation present in Wolff–Parkinson–White (WPW) syndrome. In WPW syndrome, accessory pathways (ACP) are fibers that connect the atrium to the ventricle outside the normal atrioventricular nodal–His-Purkinje conduction system. ACP result in abnormal preexcitation around the atroventricular annuli and produce a dysynchronous contraction of cardiac chambers. Importantly, ACP

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Ablation provides a model of abrupt altered activation changes in both ventricles.

**Methods**

**Subjects**

The study population consisted of 41 patients with manifest WPW syndrome (all with structurally normal hearts) who were referred to Tsukuba University Hospital for ablation between March 2012 and October 2015. They were all symptomatic and underwent successful ablation. One day or immediately before catheterization and ablation, each patient underwent 3D-STE. Patients who showed suboptimal 3D echocardiographic image quality were excluded. Twenty subjects with a normal heart were also included in this study as a control group to assess normal contraction sequence mapping by 3D-STE. The study was approved by the local institutional review committee, and informed consent was obtained from all subjects before the study.

**Electrocardiogram**

The 12-lead electrocardiogram (ECG) was assessed by a single physician analyzer who was unaware of the successful ablation site of the WPW ACP. The estimation of the ACP location was determined by the criteria of Arruda et al. Briefly, the initial 20 ms of the delta wave in leads I, II, aVf, and V6, classified as positive, negative, or isoelectric, and the ratio of the R- and S-wave amplitudes in leads III and V6 were assessed. According to the diagnostic flowchart, we localized the ACP to 1 of 10 sites around the tricuspid and mitral annuli or at subepicardial locations within the venous system of the heart.

**Echocardiography**

For 3D-STE, full-volume R waves of ECG-gated 3D data sets were acquired from apical positions using a matrix array 2.5-MHz transducer. To obtain these data sets, 6 sectors were scanned and automatically integrated into a wide-angle (max 90×90 degrees) pyramidal data image covering the entire ventricle. The volume rate of each image was set to at least >20 Hz to balance the image quality and time resolution. The data were stored and transferred to a computer for offline analysis. The images were analyzed with 3D Wall Motion Tracking software (Toshiba Medical Systems Co, Tochigi, Japan) in a manner reported previously. Each 3D echocardiographic data set was acquired for analysis of the LV and RV, respectively. In the RV 3D echo, the focus was the tricuspid annulus, and the pulmonary valve was excluded from the analysis. Data were transferred to the offline Ultrasound workstation (Toshiba Medical Systems Co) and then 3D-STE was performed, and the endocardial area change ratio to time curve was obtained. For the analysis of AI, the baseline strain value was set as zero just before the initiation of the delta wave.

To visualize the timing of early deformation with AI mapping, the time at which a local wall motion index reaches a threshold with respect to the peak value is displayed in different colors. We set the threshold as 25% of the maximum area change ratio value in each region, according to the previously determined protocol by comparison with the endocardial contact mapping data. The parameter representing the time to onset of regional activation was named AI time (ms). This process is shown schematically in Figure 1 and Movie 1 in the Data Supplement. In the polar map function, AI time is displayed in each segment (Figure 2B).

The physician analyzer was unaware of the clinical information of ACP location. According to the color map of AI, the initial site of contraction in the WPW syndrome patients was determined if early contraction was observed in the area surrounding the tricuspid or mitral annulus. For localization, each atrioventricular annulus was divided into 12 1-hour clock divisions. The anterior part of each annulus was classified as positive, negative, or isoelectric, and the ratio of the R- and S-wave amplitudes in leads III and V6 were assessed. According to the diagnostic flowchart, we localized the ACP to 1 of 10 sites around the tricuspid and mitral annuli or at subepicardial locations within the venous system of the heart.

**Statistical Analysis**

Numeric data are presented as mean±SD, with these measurements made over the entire patient population. The relationship between the 3D-STE endocardial activation maps and the ACP site determined by ablation was determined by Pearson’s correlation coefficient (r), and in the statistical calculations, the mitral annulus was represented as 1 to 12 o’clock and the tricuspid annulus as 13 to 24 o’clock. To compare between ≥2 groups, 1-way analysis of variance followed by Dunnett’s post hoc test was used for comparisons of the WPW groups with the control group.
initial contraction sites of activation and then moved in superior directions to activate the basal wall of the LV. For the RV, the breakthrough point was observed at the mid-anteroseptal wall in 16/20 subjects (80%; Figure 2B, left panel) and at the mid-anterior or apical segments in the others. Endocardial activation begins at the mid part of the RV and spreads from the apex to base. None of the control subjects showed contraction breakthrough in the perimital or tricuspid valve annuli segments.

**CARTO Mapping and 3D-STE Mapping**

The pre- and postablation ECG, CARTO activation map, AI mapping by echo, and fluoroscopy image in subject W1 are shown in Figure 3 and Movie III in the Data Supplement. According to the ECG (Figure 3K), the ACP is at the left lateral or left anterolateral location of the mitral annulus. After the ablation, the delta wave disappeared in the ECG (Figure 3L). For CARTO imaging, the earliest activation site was confirmed by the ablation. The endocardial electric activation propagates from the basal lateral wall to the apex. After the ACP ablation (Figure 3B and 3D), activation mapping shows subendocardial electric breakthrough at the apical anterior segment, which may correspond to the attachments of the anterior and posterior papillary muscles (Figure 3B). Then, electric activation travels from the apex.

![Figure 1. Schematics illustrating the concept of activation imaging. A, The time-dependent wall deformation curves in 9 segments. The wall deformation value on the Y axis is normalized by the magnitude of the peak deformation in each segment. When the segment corresponding to the brown-colored curve reaches the deformation threshold at time a, the plastic-bag model for the left ventricle (B) is colored with blue according to the color coding bar. Subsequently, adjacent segments reach deformation threshold at times b, c, and d, and the propagation of the deformation wave is demonstrated as the colored plastic bag images in B (Movie I in the Data Supplement).](image)

![Figure 2. A, Corresponding clock positions shown in the left anterior oblique (LAO) view of the mitral and tricuspid annuli. B, Corresponding clock positions shown around the polar map of the 16 left ventricular segments. The numbers on each segment indicate the activation imaging (AI) time (ms). Typical right and left ventricular endocardial activation mapping is presented. In this representative subject, mechanical activation was initiated in the midanterior septal segment of the right ventricle, with an AI time of 90 ms (left) and in the midanterior (AI time, 108 ms) and inferior segments (AI time, 119 ms) of the left ventricle (right). MA indicates mitral annulus; and TA, tricuspid annulus.](image)
to the base in the LV. Figure 3E and 3G and Movie IIIE and IIIG in the Data Supplement shows the LV activation map by preablation 3D-STE. A similar color-coding mode was used in the CARTO mapping. The mechanical activation propagation pattern by 3D-STE was equivalent to the pattern provided by CARTO imaging. For the ACP location,
Figure 3. Periprocedural imaging of Wolff–Parkinson–White (WPW) syndrome in case W1. A–D, The left ventricular activation map from the CARTO system. E–H, The corresponding activation map; I and J, the time-dependent strain curves by 3-dimensional (3D) speckle tracking echocardiography–derived isochrone activation imaging on a plastic-bag model of the left ventricle. A, C, E, G, and I are preablation, and B, D, F, H, and J are postablation endocardial activation sequences. The same color code, in which red indicates the earliest and violet the latest activation, is used for both imaging types. Before ablation (A, C, E, G), the activation wavefront sweeps from the left lateral mitral annulus (red arrows in C and G, yellow arrow in I) to the basal inferior segment. After the ablation (B, D, F, H), the apical anterior and base of the anterior papillary muscle activates first and then the wavefront propagates from the apex to base. K and L, Pre- and postablation 12-lead ECGs, respectively. M, The LAO fluoroscopic view during the successful ablation with the schema of the mitral annulus clock orientation overlaid. White arrowheads indicate the ablation catheter, and the yellow asterisk indicates the tip of the ablation catheter at the successful ablation site of 3 o’clock at the mitral annulus, which agrees with the site of the 3D echo-estimated accessory pathway (G, red arrow). ACP indicates the accessory pathway; LAO, left anterior oblique; and RAO, right anterior oblique.
3D-STE-derived AI mapping demonstrated the earliest activation site at 3 o’clock (Figure 3G, red arrow, and Figure 3I, yellow arrow), which perfectly matched with the X-ray and CARTO mapping. Comparison of pre- and postablation AI mapping in the right anterior oblique and left anterior oblique views shows that the pattern of the mechanical activation sequences changes dramatically, which also corresponds to that shown on the CARTO mapping.

The RV endocardial electric and mechanical propagation maps by CARTO (Figure 4, left panels A–D) and 3D-STE (Figure 4, right panels E–H; Movie 4E-H) are shown in Figure 4 for case W19 listed in Table. The preablation ECG

![Image of RV activation maps by CARTO and 3D-STE](https://example.com/image.png)

**Figure 4.** Periprocedural imaging of Wolff–Parkinson–White (WPW) syndrome in case W19. A to M are as in Figure 3. M shows the LAO fluoroscopic view during the successful ablation with the schema of the tricuspid annulus clock orientation overlaid. White arrowheads indicate the ablation catheter, and the yellow asterisk indicates the tip of the ablation catheter at the successful ablation site of 8 to 9 o’clock at the mitral annulus, which agrees with the site of the 3D echo-estimated accessory pathway (E, red arrow). ACP indicates the accessory pathway; LAO, left anterior oblique; and RAO, right anterior oblique.
(Figure 4K) indicated the right lateral tricuspid annulus ACP. Before ablation, preexcituation by the ACP was depicted as the earliest activation site at 9 o’clock on the tricuspid annulus. The endocardial activation of the basal lateral RV free wall initiated at the ACP site (Figure 4A and 4E; Movie IV in the Data Supplement) was followed by delayed septal activation (Figure 4C and 4G; Movie IVG in the Data Supplement). After the ACP ablation, peri-tricuspid annular contraction disappeared, and the opposite pattern of activation from the septum to free wall was observed (Figure 4B, 4D, 4F, 4H; Movie IVF and IVH in the Data Supplement).

3D Echocardiography-Derived Endocardial Activation Sequence in WPW Syndrome
Among the 38 patients with WPW syndrome, all showed at least one site of abnormal periannular excitation. Among the 14 patients with a septal ACP, 8 (57%) showed both right- and left-sided abnormal early contraction signals. There was a 1.25±0.89 o’clock (range, 0–2) gap of the right- and left-side endocardial breakthrough sites across the ventricular septum, based on the clock position association between the mitral and tricuspid annuli orientation in Figure 2A. After the abnormal preexcitation at early systole, the wavefront of the activation by the ACP and by the normal conduction system became confluent, and various activation patterns were observed in each individual.

Ablation Sites and 3D-STE Prediction Sites of ACP
One of the 38 patients with WPW syndrome (case W18 in Table) had dual ACP, and thus, 39 ACP localizations were assessed. The correlation between predicted ACP location by 3D-STE and the actual location based on ablation site is shown in Figure 5. Perfect agreement was achieved in 15/39 (38%) patients. If a 1 o’clock range on either side of the matched sites was defined as acceptable prediction, 27/39 (69%) ACP locations were accurately predicted. Furthermore, in the 2 o’clock range, 34/39 patients (87%) were accurately diagnosed. Among the mitral free wall ACP, accurate prediction of the ACP location within the 1 o’clock range occurred in 5/18 (83%) and in the 2 o’clock range in 17/18 (94%) patients. The ACP in the right anterior or anterolateral site in one patient could not be localized correctly. Among 15 subjects, 9 (60%) demonstrated early local mechanical activation at the ventricular septum on both the RV and LV endocardial sides (Table). Regression analysis showed significant correlation of the 3D-STE-estimated sites with the ablation sites ($R^2=0.92; P<0.001$).

ECG and 3D-STE Prediction of ACP and Ablation Sites
We assessed the agreement between ablation sites and the site of ACP predicted by ECG in 37 ACP, except for the subject with dual ACP (Table I in the Data Supplement). According to the Arruda ECG algorithm, perfect agreement was achieved in 17/37 (45%) patients, and agreement within a range on either side of the segment with perfect agreement or with the opposite side of the septum was observed in 26/37 (70%) patients. In the comparison between 3D-STE and ECG, 21/37 (56%) patients showed agreement in ACP location. Among them, 20 (95%) ablation sites were accurately identified except in 1 patient (case W24) who failed prediction by either ECG or 3D-STE. Among the remaining 16 patients in whom ACP prediction by 3D-STE and ECG disagreed, 3D-STE prediction was accurate in 12 (75%) patients and ECG prediction in 3 (19%), and both modes failed in 1 patient (case W35).

Figure 5. Correlation between predicted accessory pathway location (3D echomapping) and the actual location based on ablation site. Green cell indicates a perfect match between echo-predicted site and the ablation site; pale green cell, 1 o’clock position beside a perfect match; gray cell, corresponding position across the interventricular septum; and ( ), number of sites estimated by both left and right ventricular speckle tracking echocardiography.
Regional LV Area Strain in Controls and WPW Patients
Table II and Figure I in the Data Supplement show the segmental area strain. Even though there was no difference in global area strain, in the WPW patients with LV free wall ACP, the basal to mid-anterior wall strains were significantly impaired than those in the control group.

Reproducibility
The absolute difference between the first and second measurements was in the 0.7±0.6 o’clock range (−1, +2 o’clock) and 0.9±0.8 o’clock range (−2, +2 o’clock) for the intra- and interobserver measurements, respectively. Over the entire 12 o’clock range, the coefficient of variation was 5.2% for intraobserver and 6.5% for interobserver reproducibility.

3D Echocardiography-Derived Mechanical Activation in WPW Syndrome
3D-STE-derived AI mapping could approximate the location of the ACP within a 2 o’clock range. Precise estimation to assess which contraction is the earliest may be difficult by 3D-STE. One reason may be due to the spatial resolution of 3D-STE, in which the size of the 3D template of the voxel unit of tracking for pattern matching is at a several millimeter cubic scale. Other reasons may be because of the anatomic variation of the ACP width and length, which could be over 10 mm in some cases. The summation of atrioventricular delay of 80 to 100 ms plus the normal conduction time of 70 to 90 ms may give chance to obtain at least 3 to 6 volumetric 3D image frames, in the present study. Therefore, 3D-STE may have appropriate time resolution to identify the approximate location of the abnormal activation sequence in WPW syndrome. Several echocardiographic methods have been reported previously to locate the exact site of the ACP by identifying early deformation, including M-mode, tissue Doppler, 2D imaging, and 2D speckle tracking. These methods offer superior time and spatial resolution in comparison to 3D-STE. However, 3D echocardiography has the great advantage of allowing the assessment of complex 3D morphological objects of the nonplaner mitral and tricuspid annulus information. In addition, 3D-STE revealed segmental contraction abnormalities, which were consistent with a previously conducted 2D strain analysis concluding that preexcitation is associated with local hypokinesia. Therefore, comprehensive diagnostic strategies could be proposed to localize the ACP, first by grasping the overall activation propagation with 3D-STE and then following with tissue Doppler–based velocity imaging or M-mode echo to assess accurate site detection, including distinguishing the subendocardial or subepicardial location.

The right-sided ACP remains a diagnostic challenge by any echocardiographic procedure. This may be because of the 3D morphological complexity of the tricuspid annulus, which cannot be assessed in a single 2D echocardiographic plane. Furthermore, it is sometimes difficult to obtain an optimal echo image of the anterior part of the tricuspid annulus and RV infundibulum via the transthoracic approach. Case W24 (Table), with an 11 o’clock tricuspid annulus ACP, could not be diagnosed correctly. Although assessment of the tricuspid annulus near the RV infundibulum might be difficult even with this new software. A newly developed application of 3D-STE to the RV with prototype software has recently been reported by our laboratory,3 and further trials will be required.

Normal Activation Propagation in the Healthy Heart
Based on the 3D-STE analysis, 3 common earliest sites of mechanical activation of the normal ventricle were observed in the present study at the (1) mid-anterior and (2) mid-inferior LV free wall and (3) at the mid to apical septum of the RV. These findings are consistent with a previous extensive electric mapping study in isolated human hearts,12 which showed ≤3 LV endocardial breakthrough sites: (1) the anterior and (2) inferior wall and (3) the center of the left side of the septum. In comparison with this classic experimental report, our results from the 3D-STE-derived AI mapping could approximately match these physiological activation patterns based on the electric activation. Furthermore, there are individual variations in contraction pattern in the present study even in the normal subjects.

Limitations
There are several limitations in this study. First, the number of subjects is relatively small. Although the physician analyzing the echoes was unaware of the results of radiofrequency ablation, the investigators were not blinded to the patient groups (WPW and control). Nevertheless, the study sufficiently revealed the abnormal LV and RV activation sequences in conjunction with invasive CARTO mapping or radiofrequency catheter ablation. Only 2 subjects underwent both CARTO mapping and 3D-STE. Because of its invasive nature and the longer procedure time required to assess ACP with CARTO mapping, we limited use of the CARTO system to a minimum number of patients in the study protocol.

The second fundamental limitation is that 3D-STE deals with mechanical activation and not electric activation. We assume that electromechanical coupling is well preserved in the normal heart such that the mechanical activation pattern seems to reflect the electric phenomenon of the myocardium. Each electric activation is followed by an electromechanical one, that is, the depolarization of a cardiac muscle cell is followed by an uptake of calcium, which triggers contraction after a certain electromechanical delay of a few milliseconds. Finally, we did not focus on the cardiac memory phenomenon, which is defined as the occurrence of persistent repolarization abnormalities after successful ablation.22 Only 2 of the present patients underwent combined postablation echo and CARTO assessment, and neither had any abnormal contraction at 48 hours after ablation. Persistent contraction abnormalities...
48 hours post ablation have been reported in 42% of WPW syndrome patients,17 and our 2 patients may have just happened to experience resolution. Further detailed investigation should be performed to address the effect of cardiac memory on the 3D contraction sequence of the myocardium.

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Disclosures

None.

References


**Clinical Perspective**

Wolff–Parkinson–White syndrome is a preexcitation syndrome caused by an accessory atrioventricular pathway that results in dysynchronous ventricular contraction. Patients with tachyarrhythmia require treatment by radiofrequency catheter ablation. The effectiveness of ablation depends on the precise localization of the accessory pathway. Three-dimensional speckle tracking echocardiography can assess left and right ventricular deformation and has been successfully applied to the assessment of myocardial dyssynchrony. In the present study, noninvasive isochrone activation mapping generated on the basis of 3-dimensional speckle tracking echocardiography data demonstrated abnormal periannular excitation compatible with that shown on invasive electric contact mapping or in catheter ablation procedures in 38 patients with Wolff–Parkinson–White syndrome with an overall accuracy of 87% agreement within the 2 o’clock ring range around the valvular annulus. Therefore, isochrone activation mapping derived from noninvasive 3-dimensional speckle tracking echocardiography may be a promising imaging tool to attain a complete view of the biventricular dysynchronous contraction sequence to localize accessory pathway preexcitation in Wolff–Parkinson–White syndrome.
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Supplemental Table S1. Correlation Between Predicted Accessory Pathway Location (ECG Algorithm) and the Actual Location Based on Ablation Site

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Green cell indicates a perfect match between ECG-predicted site and the ablation site; pale green cell, 1 block position on either side of a perfect match; gray cell, corresponding position across the interventricular septum; ASTA, anteroseptal tricuspid annulus; CSOs, coronary sinus ostium; LAL, left anterolateral; LL, left lateral; LP, left posterior; LPL, left posterolateral; MSTA, mid-septal tricuspid annulus; MV, mitral valve; PSMA, posteroseptal mitral annulus; PSTA, posteroseptal tricuspid annulus; RA, right anterior; RAL, right anterolateral; RL, right lateral; RP, right posterior; RPL, right posterolateral; and TV, tricuspid valve. *Patient WPW 18 with dual ACP whose ACP was included at TV9.
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* p< 0.05 vs. control, † p< 0.01 vs. control. LVFW indicates left ventricular free wall; RVFW, right ventricular free wall; and WPW, Wolff-Parkinson-White.
**Movie legends**

**Movie 1.** Schematics movie for the concept of activation imaging.
Right lower panel shows the time-dependent wall deformation curves in 9 segments. The wall deformation value on the Y-axis is normalized by the magnitude of the peak deformation in each segment. When the segment deformation reaches the threshold, the plastic-bag model or polar map for the left ventricle is colored according to the color-coding bar.

**Movie 2.** Peri-procedural echo movies of WPW syndrome in case W1.
A-D show the activation map by three-dimensional speckle tracking echocardiography on a plastic-bag model of the left ventricle. Movie 2A correspond to Figure 3E, Movie 2B to Figure 3F, Movie 2C to Figure 3G, and Movie 2D to Figure 3H. Movie 2A is the pre-ablation left ventricular (LV) map from right anterior oblique (RAO) view, 2B is the post-ablation LV map from RAO view, Movie 2C is the pre-ablation LV map from left anterior oblique (LAO) view, 2D is the post-ablation LV map from LAO view.

**Movie 3.** Peri-procedural imaging of WPW syndrome in case W19.
A-D show the activation map by three-dimensional speckle tracking echocardiography on a plastic-bag model of the right ventricle. Movies 3A-D correspond to Figure 4E-H. Movie 3A is the pre-ablation right ventricular (RV) map from RAO view, 3B is the post-ablation RV map from RAO view, Movie 3C is the pre-ablation RV map from LAO view, 3D is the post-ablation RV map from LAO view.