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Pediatric Electrocardiographic Imaging (ECGI) Applications

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Summary

Noninvasive electrocardiographic imaging (ECGI) has been used in pediatric and congenital heart patients to better understand their electrophysiologic substrates. In this article we focus on the 4 subjects related to pediatric ECGI: 1) ECGI in patients with congenital heart disease and Wolff-Parkinson-White syndrome, 2) ECGI in patients with hypertrophic cardiomyopathy and pre-excitation, 3) ECGI in pediatric patients with Wolff-Parkinson-White syndrome, and 4) ECGI for pediatric cardiac resynchronization therapy.

Keywords

Electrocardiographic Imaging; Pediatrics; Congenital Heart Disease; Wolff-Parkinson-White; Cardiac Resynchronization Therapy

Introduction

Electrocardiographic imaging (ECGI) is a novel noninvasive imaging modality used to study the electrophysiologic substrate in patients. This tool has also been successfully applied to pediatric patients, some as young as 18 months of age, to create patient-specific models of atrial and ventricular activation and repolarization. In this article, we review the pediatric applications of ECGI and what these studies have contributed the general knowledge of pediatric electrophysiology.

Manifest Accessory Pathway in a Patient with Ebstein's Anomaly

Introduction

Ebstein's anomaly is a rare congenital heart disease involving the septal and posterior leaflets of the tricuspid valve, where the leaflets may be deformed, displaced or adherent to the intraventricular septum [1]. Patients are prone to electrical problems as well, with up to 25% having concomitant pre-excitation on surface 12-lead electrocardiograms. Arrhythmic substrates in these patients include supraventricular tachycardia, atrial flutter and atrial

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fibrillation. Accessory pathways (APs) are typically single pathways localized to the right-sided Ebstenoid tricuspid valve.

ECGI was performed in this patient to construct ventricular activation maps pre- and post-ablation, as well as post-ablation potential maps.

Methods and Results

A 16-year-old with Ebstein's anomaly and ventricular pre-excitation underwent ECGI to construct ventricular activation maps during a single pre-excited beat prior to ablation [1]. Applying the Arruda algorithm [2] to the baseline pre-ablation pre-excited ECG (Figure 1), the delta wave mapped the AP to a subepicardial/right posteroseptal location.

ECGI was performed during 2 time points, pre and post ablation. Figure 2 demonstrates the pre-ablation activation isochronal map (panels A-B), post-ablation activation map (panels C-D) and the post-ablation potential maps (panels E-F) in the right anterior oblique (RAO) projection (panels A, C, and E) and the left lateral projections (panels B, D, and F). In the pre-ablation isochronal maps, the earliest site of ventricular activation (area of red) is localized to the right posterolateral location on the tricuspid valve annulus as marked by the white asterisk (Figure 2, panel A). Ventricular activation then propagates in a uniform manner with the LV activating late (areas of blue, Figure 2, panel B).

The following day, the patient underwent an electrophysiology study (EPS) and transcatheter ablation. A 3-dimensional electroanatomic map was created (CARTO, Biosense Webster) to localize the accessory pathway. Figure 3, panel A, shows the earliest site of activation identified in the right posterolateral region of the tricuspid valve annulus (TVA), similar to where ECGI had predicted the pathway location. A cryocatheter was positioned at the site of the AP (Figure 3, panel B) there was cessation of AP activity within 8 seconds of initiating cryotherapy.

Post-ablation ECGI images were obtained (Figure 2, panels C and D) and showed the activation in the right posterolateral region to now be late (area of blue; marked by the + sign). The earliest ventricular activation post-ablation is now in the anterior paraseptal region and inferior anteroapical region of the RV.

Figure 2, panels E and F show the ECGI epicardial potential map postablation (at 38 msec post-QRS). A local minimum potential (area of blue, marked by black asterisk) is seen on the anterior RV surface midway between the RV base and the LAD artery indicating RV breakthrough. This site of RV breakthrough occurs at 38msec after QRS onset and 13 msec after earliest LV epicardial breakthrough. Conduction throughout the RV is slow with the RVOT being the latest to activate. Activation of the LV occurs in the normal apex-to-base with the basal posterolateral area being the last to activate (area of blue, panel D).

Conclusions

During normal ventricular activation, the earliest area of epicardial activation occurs on the anterior RV (18-20msec after the QRS) indicating normal conduction and function of the right bundle branch. This is followed by additional RV epicardial breakthrough sites

resulting in a synchronized contraction. Epicardial LV activation occurs next, about 10 msec after RV epicardial breakthrough. In this case, however, RV breakthrough (RVBT) post-ablation lags behind the LV breakthrough, occurring 13 msec after LV breakthrough and 38msec post-QRS onset (Figure 2, panel C). On the post-ablation potential map (figure 2, panel E) we see a site of potential minimum (black asterisk) near the site of RV epicardial breakthrough (panel C).

However, the delayed appearance of the RVBT (38msec post-QRS, 12 msec post-LV breakthrough) suggests that the conduction through the right bundle branch is slower compared to normal. Additionally, the paraseptal and apical areas of the RV are late to activate (areas of blue, Figure 2, panel C) supporting slow RV bundle conduction. This is reflected in the surface ECG post-ablation (figure 1) where there are notched R waves in leads V1 and V2 with a QRS duration of 90 msec. ECGI demonstrated that this finding on the surface ECG represented true slow conduction through the RV bundle branch.

The patient underwent successful ablation of a single AP in the posterolateral aspect of the tricuspid valve annulus. ECGI successfully predicted the pathway location. Patients with congenital heart disease highlight the inaccuracies of standard noninvasive AP localization algorithms, such as the Arruda algorithm. ECGI, which incorporates patient-specific heart-torso geometry with electrophysiologic properties, provides more accuracy in AP localization.

Pediatric Hypertrophic Cardiomyopathy with Ventricular Pre-excitation

Introduction

Hypertrophic cardiomyopathy (HCM) accounts for 42% of all childhood cardiomyopathies with an incidence of 0.47/100,000 children [3, 4]. It is defined as the presence of ventricular hypertrophy without an identifiable cause.

This is the case of an 18-year-old male patient with hypertrophic cardiomyopathy with no history of arrhythmias [3]. The patient's echocardiogram revealed significant HCM (septal thickness of 2.17cm, Z-score 7.5, with LV posterior wall thickness of 1.43cm, z-score 3.6) with no evidence of LV outflow tract obstruction. Twelve-lead ECG demonstrated manifest pre-excitation with delta wave map (by Arruda algorithm [2]) prediction of a right anteroseptal AP.

Methods and Results

ECGI epicardial activation maps were created during pre-excited sinus rhythm (figure 4). Panel A, figure 4, demonstrates the earliest area of activation was localized to the right anterior/anteroseptal region (area of white) of the tricuspid valve annulus. Additionally, the midseptal region and right posteroseptal regions were also early (areas of pink). The entire LV basal area (area of red) was also activated early. Activation spread from base to apex (figure 4, panel C) in a uniform manner with an elongated area of early activation in the RV along the interventricular groove. In normal (non pre-excited) sinus rhythm, this area is typically the earliest area of epicardial activation; however in the face of this patient's pre-excitation syndrome, activation in this region was 26msec later than the right anteroseptal

location. (Figure 4, panels D-F demonstrate epicardial ventricular activation in a healthy, non pre-excited 21 year old male. Areas of early epicardial activation occur in the RV along the interventricular groove with uniform activation spreading throughout the ventricles, and the left posterobasal area being last to activate.)

This patient underwent EPS the following day. Initial intervals demonstrated a short HV interval at 12 msec. With atrial extrastimulus testing, there was no change in the QRS morphology despite AH decrement. The shortest pre-excited RR interval with atrial overdrive pacing was 320msec, after which Wenckebach occurred. Atrial pacing was performed from multiple sites in both the right and left atria with no change in QRS morphology. Intravenous adenosine was administered which showed PR prolongation with no augmentation of pre-excitation (no change in QRS). Ventricular pacing demonstrated no retrograde conduction. Activation mapping found a fragmented multiphasic His recording at the 2:30 position along the TVA with a local activation time of -18msec pre-delta wave (figure 5). No tachyarrhythmias were induced throughout the study. These findings were consistent with a fasciculoventricular pathway.

Conclusion

Despite the abnormal activation sequence generated by ECGI mapping, the EPS confirmed the presence of a fasciculoventricular pathway which did not mediate SVT. The septal pre-excitation without inducible tachycardia may be attributed to enhanced conduction via a “short” bundle branch, or alternatively, a fasciculoventricular pathway. The presence of asymptomatic pre-excitation in a patient with HCM should raise the suspicion of a fasciculoventricular pathway.

Cardiac Memory in Pediatric Wolff-Parkinson-White Patients

Introduction

Cardiac memory is the phenomenon of repolarization changes induced by longstanding altered cardiac activation (for example, pacing) which persist for a period of time after normal activation has been restored [5]. The mechanism of cardiac memory is a remodeling process that alters molecular determinants of the action potential duration, such as membrane density and kinetic properties of ion channels.

Wolff-Parkinson-White syndrome involves longstanding altered activation sequence due to ventricular pre-excitation and provides a “natural model” for studying cardiac memory. Ablation of the accessory pathway with the restoration of sinus rhythm provides a model for understanding the time course of repolarization changes.

Objectives of this study were to: 1) utilize ECGI for the localization of AP location in patients with WPW and 2) to image activation and repolarization pre and at several time points post-ablation to study the time course of activation and repolarization changes, or cardiac memory [5].

Methods and Results

14 pediatric patients with WPW and structurally normal hearts referred for electrophysiology study and transcatheter ablation were enrolled in this study. Patients underwent ECGI pre-ablation, 45 minutes post-ablation (in sinus rhythm), 1 week and 1 month post-ablation.

In figure 6, there are pre-ablation epicardial potential maps in the first column and ventricular activation maps in the second column. Patient W1 (top row) has the earliest epicardial breakthrough (panel A) indicated by a deep local potential minimum in the posterior septum on the left AV valve (AVV) annulus, at the 7 O'clock position (area of dark blue). This was seen 8 msec after the onset of the delta-wave on surface ECG. The epicardial potential map during repolarization, 181 msec after the onset of delta wave, (Figure 6, panel B) showed a similar albeit reversed pattern as activation with the minimum now replaced by a potential maximum. The pre-ablation ventricular activation map (Figure 6, panel C) shows early ventricular activation at the same location indicating the location of the accessory pathway. Post-ablation ECGI isochrones map shows late activation in the left posterior area of the mitral valve annulus with the latest area of activation on the right AVV annulus, in the posterolateral region.

Patient W2 in figure 6 illustrates a left lateral pathway. The epicardial potential map shows a deep potential minimum in the left lateral aspect of the mitral valve (Figure 6, panel E) with the repolarization potential map (Figure 6, panel F) showing the inverse, a positive potential at the same location. In figure 6, panel G, the pre-ablation ventricular activation map localizes the accessory pathway location to the left lateral AVV annulus. Post-ablation, the site of the former accessory pathway is now an area of late activation.

The epicardial activation potential map in patient W3, (figure 6, panel I) shows two discrete sites of epicardial potential minima (circled areas), one in a left lateral location, one in a more posterolateral location. The epicardial repolarization potential map (Figure 6, panel J) shows the same 2 discrete maxima at the same locations where we saw the minima during activation. Pre-ablation activation map (Figure 6, panel K) demonstrated a broad area of early activation encompassing the same region as denoted on the potential maps. During the ablation procedure, there were 2 confirmed AP targets located—one on the left lateral aspect and one on the left posterolateral aspect of the L-AVV. The presence of the 2 minima during activation, and the 2 maxima during repolarization suggested the presence of 2 accessory pathways. Post-ablation isochrones indicated late areas of activation at the left lateral and posterolateral locations.

In figure 7, patient W4 we see an example of a patient with a right posteroseptal AP. ECGI activation (Figure 7, panel A) and repolarization (Figure 7, panel B) potential maps, as well as pre-ablation activation map localized the AP site to near the mouth of the coronary sinus. The post-ablation activation map (Figure 7, panel D) showed an area of late activation in the right posterior septum.

Patient W5 in figure 7 has an AP that demonstrates potential breakthrough (Figure 7, panel E) and early activation (Figure 7, panel G) in the midseptal area. For this location, ECGI

was not able to determine laterality of the pathway (right versus left midseptal) since the reconstructions are limited to the epicardial surface.

Patient W6 in figure 7 had epicardial activation (Figure 7, panel I) and repolarization (Figure 7, panel J) potential maps in the left posteroseptal region. Pre and post ablation epicardial activation maps (Figure 7, panels K-L) also confirmed this finding.

For 5 patients, ECGI maps of retrograde atrial activation were generated when clear retrograde P waves were visible on the body surface ECG. Figure 8 shows 2 representative examples of these maps. For patient W1 (Figure 8, top row), the retrograde atrial activation patterns during sustained tachycardia are shown in panels A-B. The earliest area of activation, in white is localized to the left posterior atrial wall. Activation then spread in a uniform fashion from this location throughout the atria, with the low anterior right atria activating last (blue).

In figure 8, second row, we see the retrograde atrial activation for patient W5 who had a right sided midseptal AP. The area of earliest retrograde activation occurs in the lower right atrium, through the right atrium and to the posterior aspect of the atria with the lateral left atrium/left atrial appendage being the last to activate.

Antegrade ventricular activation is demonstrated in patients W1 and W5 in figure 8. There was early antegrade ventricular activation in the RV in the apical (W1) and paraseptal (W5) regions, with late activation in the posterolateral RV (W1) or left lateral basilar area (W5). ECGI ventricular activation maps during SVT were found to be similar to normal sinus (non pre-excited) rhythm maps, indicating that antegrade conduction involved the AVN/His-Purkinje system.

Figures 9 and 10 show ECGI epicardial activation-recovery interval (ARI) maps that were generated at 4 distinct time points: 1) pre-ablation, 2) 45 minutes post-ablation, 3) 1 week post-ablation, and 4) 1 month post-ablation. In patients W1-W5, the epicardial electrogram at the site of the AP demonstrated a rS morphology, indicating an endocardial insertion site of the AP. In patient W6, however, the epicardial electrogram at the AP site demonstrated a pure QS morphology, indicating the insertion site of the AP to be epicardial or subepicardial. Additionally, the T waves for patients W1-W6 pre-ablation were inverted (figures 9-10, panels A, E, and I).

The ARI maps at 45 minutes post-ablation (figures 9 and 10, panels B, F, and J) demonstrated a change in the epicardial electrogram morphology from a rS (or QS) to a qR pattern, with persistent inversion of the T waves. By 1 week post-ablation, the T waves remained inverted though to a lesser extent, and finally became more flattened and upright by 1 month post-ablation.

The basilar annular region had the highest ARI in pre-excited rhythm (figures 9 and 10, panels A, E and I) with the area of longest ARIs at the pre-excitation site. This ARI distribution was largely unchanged at 45 minutes post-ablation. By 1 week post-ablation, the ARI decreased but remained high. At 1 month post-ablation, the ARI has normalized.

In Table 1, the predicted site of the AP by ECGI is compared to the predicted site using the Arruda algorithm. The site of successful ablation is also shown. ECGI localization was fairly accurate to site of ablation success, within a 1 hour arc along the annulus.

Conclusions

ECGI is able to accurately and precisely predict the site of accessory pathways. Additionally, ECGI was able to predict epicardial versus endocardial location, as well as multiple APs thus distinguishing it from noninvasive algorithms created to predict AP location.

Abnormal repolarization in WPW patients results in altered epicardial apical-basal ARI dispersion. While activation patterns normalize immediately upon ablation of the manifest AP, repolarization patterns remain abnormal and unchanged. At 1 week post-ablation, we see that the recovery gradients are still abnormal but have gradually decreased and have normalized by 1 month post-ablation. This time course is consistent with the phenomenon of cardiac memory.

Cardiac Resynchronization Therapy in Pediatric Congenital Heart Disease Patients

Introduction

Cardiac resynchronization therapy (CRT) has been extensively studied in adult heart failure patients; it has been less thoroughly studied in children and congenital heart disease patients [6]. Patients with congenital heart disease have been living longer due to improvements in surgical management of these patients, and are evolving into young adults with heart failure. CRT has been shown to be advantageous for certain disease substrates.

In this study, there were three distinct hypotheses involving ECGI in pediatric CHD: 1) to identify those CHD patients with substantial electrical dyssynchrony in baseline rhythm who would therefore likely benefit from CRT; 2) to help guide the lead placement of the resynchronization lead by identifying the electrophysiologic substrate and the area of latest activation; and 3) to evaluate intraventricular dyssynchrony post-CRT [6].

Methods and Results

Patients were included if they were: 1) <21 years old, 2) had congenital heart disease, 3) had heart failure symptoms despite maximized medical therapy, 4) undergoing evaluation for CRT or having a CRT device in place. ECGI was performed in these patients in the baseline presenting rhythm. For pre-CRT patients who had a pacemaker in place and were not pacemaker-dependent, ECGI was also performed in non-paced rhythm. For post-CRT patients, testing was performed under the following conditions: 1) optimal CRT (CRT-OPT) settings (including the optimal intraventricular, or V-V, delay as determined by referring physician), 2) nominal CRT (CRT-NOM) settings (which refers to the baseline factory setting of LV ahead of RV by 4 msec), 3) single site ventricular pacing (either RV or LV) and 4) underlying rhythm (where applicable).

An electrical dyssynchrony index (ED, measured in msec) was calculated from activation isochrones as the standard deviation of activation times at 500 discrete epicardial sites of the systemic ventricle). This measures the degree of dyssynchrony of electrical activation across the systemic ventricle (table 2). Normal values were calculated from control patients.

Pre-CRT ECGI Patients—Four patients were evaluated with ECGI as part of their pre-CRT evaluation (figure 11). Patients #1 and 2 were found to have relatively normal ED indicies; CRT was deferred in these patients and they were optimized on medical +/- single site pacing (for Patient #2) therapy with clinical improvement. Patient #1 has an ED of 22.4msec with synchronous LV activation. The area of late (in dark blue) us seen near the RV base and is consistent with a right bundle branch block pattern. Patient #2 had ECGI performed in a dual chamber paced rhythm (ventricular lead in the RV lateral apical free wall) and demonstrated an ED on 20.5msec. Patient #3 had an ED of 28.4msec, consistent with moderate dyssynchrony. This patient was treated with medication and had resultant clinical improvement, so CRT was deferred. Patient #4 had a severely dyssynchronous systemic ventricle (ED=37msec). This patient was optimized on medical therapy and had a brief period of improvement followed by quick decompensation. The patient went on to be listed as a status 1A for heart transplant (the highest status) but unfortunately expired while awaiting transplant.

Pre and Post-CRT ECGI Patients—Patients #5 and 6 underwent ECGI activation isochronal mapping both pre and post-CRT. The pre-CRT images were obtained to determine patient candidacy for CRT and potentially for targeted lead placement. The post-CRT images were obtained to determine response to CRT.

Patient #5 is a 6 year old male with congenitally corrected transposition of the great arteries. Past surgical intervention included the placement of a 16mm homograft from the left ventricle to the pulmonary artery which resulted in high grade AV block, requiring the implantation of a dual chamber pacemaker by age 15 months. Pre-CRT ECGI was obtained in the patient (figure 12, top panel) which demonstrated that the patient had an ED of 36.4, indicative of severe dyssynchrony. Activation isochrones delineated an area of late activation in the posterolateral basilar region of the systemic right ventricle as a potential target for resynchronization lead. Post-operative CRT ECGI images were subsequently obtained under 5 conditions with the following ED indicies: 1) CRT-OPT—ED 21msec, 2) CRT-NOM—ED 29.3msec, 3) PV-P (subpulmonary ventricular pacing only)—ED 36.4msec, 4) SV-P (systemic ventricular pacing only)—ED 32.3, and 5) SR (at this visit, the patient had intact conduction)—ED 23.4msec. Interestingly, when the patient presented he had intact AV conduction that was able to be imaged by ECGI. This condition showed a normal ED index with a normal activation pattern. Given the intermittent nature of his AV conduction, he was mostly in a paced rhythm, and the condition CRT-OPT had a normal ED index as well, a significant improvement over single site ventricular pacing. Clinically, the patient had symptomatic improvement and was able to return to activities.

Patient #6 is an 8 year old male with hypoplastic left heart syndrome, mitral atresia, and double outlet right ventricle who had a dual chamber pacemaker implanted at 3 months of age for post-operative heart block (original ventricular pacing lead placed at the site of the

white asterisk, figure 13). By age 8, the patient had developed symptoms of heart failure and was maximized on medical therapy. Figure 13 shows his ventricular activation maps imaged by ECGI. His pre-CRT maps show an ED index of 50msec, which is severely abnormal. Additionally, the latest site of electrical activation was identified (left anterior and inferior basal aspects of the single ventricle), and was the target site for epicardial lead implantation. Three months post-operatively, the patient underwent repeat ECGI studies under the following four conditions with the following ED indices: 1) CRT-OPT—ED 27msec, 2) CRT-NOM—ED 29msec, 3) ANT-P (anterior aspect of single ventricle)—ED 31msec, and 4) POST-P (posterior aspect of the single ventricle)—ED 50msec. CRT-OPT conditions yielded the most improved ED index though it remained slightly abnormally prolonged. Single site ventricular pacing, from either the anterior or posterior aspects of the univentricle yielded moderate to severely abnormal ED indices, respectively. Clinically, the patient was a CRT responder and improved with medical and device therapy. This patient's poor clinical status pre-CRT may be partially explained by the severe dyssynchrony seen in his baseline paced rhythm.

Post-CRT ECGI Patients—Patients #7 and 8 underwent ECGI activation mapping post-CRT only. Interestingly, these patients had similar underlying intracardiac anatomy.

Patient #7 is a 17 year old female with Double Inlet Left Ventricle (DILV), L-transposition of the great arteries (L-TGA) s/p fenestrated Fontan and a dual chamber epicardial pacemaker for SA nodal dysfunction and second degree AVB. She underwent maximization of medical therapy as well as an upgrade from dual chamber to CRT pacing in 2006 for progressive heart failure symptoms. The second ventricular lead was empirically placed approximately 180 degrees apart from the original ventricular pacing lead. Clinically, she had an improvement in her heart failure symptoms with medical and device therapy. ECGI ventricular activation isochronal maps were performed (figure 14) under four conditions with the following ED indices: 1) CRT-OPT—ED 18.4msec, 2) CRT-NOM—ED 21.2msec, 3) ANT-P—ED 32.8msec, and 4) POST-P ED—34.7. Single site ventricular pacing from either the anterior or posterior lead yielded abnormal ED indices as compared to CRT pacing which nearly normalized the ED index.

Patient #8 is a 21 year old female with DILV and L-TGA s/p fenestrated Fontan procedure and a dual chamber epicardial pacemaker for post-operative heart block. She underwent upgrade from a dual chamber pacemaker to a CRT pacemaker with the second lead empirically implanted approximately 180 degrees from the original lead. ECGI ventricular activation maps were constructed (figure 15) under 3 conditions with the following ED indices: 1) CRT-OPT—ED 37.3msec, 2) ANT-P—ED 36.5msec, and 3) POST-P—ED 38.8msec. Interestingly, these maps clearly show regions of slow conduction (crowded isochrones) and lines of conduction block (thick black lines) located on the anterior and posterior surfaces of the heart encompassing the ventricular pacing leads. This left large swaths of the ventricular myocardium unpaced and therefore not resynchronized. This resulted in persistent ED that did not improve with CRT-OPT pacing. Clinically, this patient was not a CRT responder and continued to have worsening heart failure symptoms. She eventually was listed for a cardiac transplant and was successfully transplanted.

Conclusions

ECGI provides an objective measure of dyssynchrony. If we were to make the assumption that electrical dyssynchrony precedes mechanical dyssynchrony, then identification of patients pre-CRT who have electrical dyssynchrony may help to reduce the number of CRT non-responders by identifying those patients with normal ED.

The ED index is expected to be higher for those patients with regions of late ventricular activation rather than those patients with generalized slow conduction. For example, patient #2 had a prolonged QRS duration of 125msec reflecting generalized slow conduction through the ventricular myocardium. However, ECGI demonstrated that the slow conduction was generalized throughout the myocardial mass, yielding a rather normal ED index of 20.5 msec. Contrastingly, patient #4 had a QRS duration of 100msec though ECGI activation maps demonstrated large areas of very late activation (dark blue) and a resultant abnormal ED index of 37msec.

Site selection for the resynchronization lead. Currently, there are no objective criteria for resynchronization lead placement. Often, epicardial resynchronization leads are placed approximately 180 degrees apart on the epicardium. However, targeted site selection for the resynchronization lead is more likely to achieve resynchronization, as seen in patients #5 and #6. In these patients, the maps were shown to the operating surgeons to identify the general anatomic area for lead placement. ECGI provides an objective basis for the site selection.

ECGI helps to define the EP substrate pre-CRT implantation. For example, patient #8 had a significantly abnormal EP substrate with regions of slow conduction and lines of conduction block. CRT did not yield the desired clinical response in this patient. In this patient, one of the ventricular leads was placed adjacent to an area of slow conduction leaving half of the ventricular mass devoid of resynchronization pacing. Optimally, the CRT leads should be paced in areas to resynchronize large portions of the myocardium. Performing ECGI pre-CRT may help to better understand each patient's unique EP substrate (regions of slow conduction, lines of block) prior to resynchronization lead placement.

Improvement in clinical response may be accompanied by more synchronized ventricular contraction and a lower ED index, as demonstrated by patients #5-7.

Conclusion

ECGI can be performed safely and effectively in the pediatric population. There is much to be learned mechanistically given the heterogeneity of pediatric substrates, including congenital heart disease patients. In addition, there may be clinically relevant data that may alter the way we take care of these young, ill patients. Future pediatric studies are needed to further our knowledge of these patient substrates.

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Key Points

- Noninvasive electrocardiographic imaging (ECGI) provides clinically useful information in the pediatric and congenital heart disease populations.
- Pediatric ECGI can be performed safely in young patients.
- ECGI can be used to localize the site of accessory pathways in patients with structurally normal hearts, congenital heart disease and cardiomyopathy.
- ECGI may provide important data in patients pre, during and post-cardiac resynchronization therapy.

Figure 1.

These pre and post-ablation ECGs are from a patient with Ebstein's anomaly and WPW. On the upper panel, pre-ablation, note the positive delta wave in lead I and negative delta waves in leads II and III, indicating a subepicardial/right posteroseptal location of the AP per the Arruda algorithm. In the bottom panel, post-ablation, there are no longer delta waves present and there is a notched R wave in leads V1 and V2 with a QRS duration of 90 msec. From Ghosh S, Avari JN, Rhee RK, et al. Noninvasive electrocardiographic imaging (ECGI) of epicardial activation before and after catheter ablation of the accessory pathway in a patient with Ebstein anomaly. *Heart Rhythm* 2008; 5(6):857-60; with permission.

Figure 2.

The right anterior oblique and left lateral projections during 3 time points are shown in this patient with Ebstein's anomaly and WPW. Panels A and B are ventricular activation isochronal maps obtained pre-ablation. Panels C and D represent ventricular isochronal activation maps obtained post-ablation. Panels E and F demonstrate the post-ablation potential map obtained 38 msec after the onset of surface QRS complex. From Ghosh S, Avari JN, Rhee RK, et al. Noninvasive electrocardiographic imaging (ECGI) of epicardial activation before and after catheter ablation of the accessory pathway in a patient with Ebstein anomaly. *Heart Rhythm* 2008; 5(6):857-60; with permission.

Figure 3.

These images were obtained during the invasive EP study in a patient with Ebstein's anomaly and WPW. Panel A is a 3-dimensional electroanatomic map (CARTO map) of the tricuspid valve annulus that demonstrates the area of earliest activation along the right posterolateral region. Panel B demonstrates the ablation catheter (CryoCatheter) at the site of ablation success, in the RAO (top panel) and LAO (bottom panel) views. This catheter location correlates with the location predicted by ECGI in figure 1, panels A and B. Adapted from Ghosh S, Avari JN, Rhee RK, et al. Noninvasive electrocardiographic imaging (ECGI) of epicardial activation before and after catheter ablation of the accessory pathway in a patient with Ebstein anomaly. *Heart Rhythm* 2008; 5(6):857-60; with permission.

Figure 4.

ECGI generated epicardial activation maps obtained in a patient with hypertrophic cardiomyopathy and pre-excitation (top row), in a posterior, left lateral and anterior projection. The second row demonstrates the epicardial activation maps from a normal healthy male for comparison. From Ghosh S, Avari JN, Rhee EK, et al. Hypertrophic cardiomyopathy with preexcitation: insights from noninvasive electrocardiographic imaging (ECGI) and catheter mapping. *J Cardiovasc Electrophysiol* 2008; 19(11):1215-17; with permission.

Figure 5.

Intracardiac signals from an electrophysiology study performed in a patient with hypertrophic cardiomyopathy and pre-excitation. In this figure, the ablation catheter has been placed in the area of earliest ventricular activation and shows that there is septal pre-excitation (measuring 15 msec pre-QRS) but it comes after the His signal. From Ghosh S, Avari JN, Rhee EK, et al. Hypertrophic cardiomyopathy with preexcitation: insights from noninvasive electrocardiographic imaging (ECGI) and catheter mapping. *J Cardiovasc Electrophysiol* 2008; 19(11):1215-17; with permission.

Figure 6.

ECGI maps for patients W1-W3. Maps in the first column of are activation and repolarization potential maps pre-ablation. In the second column are ventricular activation maps pre and post-ablation. Initiation site marked by asterisk. From Ghosh S, Rhee EK, Avari JN, et al. Cardiac memory in patients with Wolff-Parkinson-White syndrome: noninvasive imaging of activation and repolarization before and after catheter ablation. *Circulation* 2008;26:118(9):907-15; with permission.

Figure 7.

ECGI maps for patients W4-W6. The first column shows activation and repolarization potential maps pre-ablation. In the second column are ventricular activation maps pre and post-ablation. From Ghosh S, Rhee EK, Avari JN, et al. Cardiac memory in patients with Wolff-Parkinson-White syndrome: noninvasive imaging of activation and repolarization before and after catheter ablation. *Circulation* 2008;26:118(9):907-15; with permission.

Figure 8.

ECGI-generated maps during sustained supraventricular tachycardia of retrograde atrial activation (first column) and anterograde ventricular activation (second column) in 2 patients, W1 and W5. From Ghosh S, Rhee EK, Avari JN, et al. Cardiac memory in patients with Wolff-Parkinson-White syndrome: noninvasive imaging of activation and repolarization before and after catheter ablation. *Circulation* 2008;26:118(9):907-15; with permission.

Figure 9.

Activation recovery interval maps of patients W1-W3 created pre-, post-, 1 week post-and 1 month post-ablation. Asterisk marks the pre-ablation site of pre-excitation. From Ghosh S, Rhee EK, Avari JN, et al. Cardiac memory in patients with Wolff-Parkinson-White syndrome: noninvasive imaging of activation and repolarization before and after catheter ablation. *Circulation* 2008;26:118(9):907-15; with permission.

Figure 10.

Activation-recovery interval maps of patients W4-W6 created pre-, post-, 1 week post- and 1 month post-ablation. From Ghosh S, Rhee EK, Avari JN, et al. Cardiac memory in patients with Wolff-Parkinson-White syndrome: noninvasive imaging of activation and repolarization before and after catheter ablation. *Circulation* 2008;26:118(9):907-15; with permission.

Figure 11.

ECGI activation isochronal maps from pre-CRT patients 1-4. The left column projects the heart in an anterior 4-chamber view, and the right column projects the heart in an inferior 4-chamber view. Activation maps were obtained in normal sinus rhythm in patients 1, 3 and 4, and during dual chamber AV sequential pacing in patient 2. The white asterisk on the map of patient 2 denotes the pacing lead site as derived from CT scan image. For each patient, the QRS duration (QRSd) is listed, followed by the calculated ED index. From Silva JNA, Ghosh S, Bowman TM, et al. Cardiac resynchronization therapy in pediatric congenital heart disease: Insights from noninvasive electrocardiographic imaging. *Heart Rhythm* 2009; 6(8): 1178-1185; with permission.

Pre- and Post-CRT ECGI Activation-Isochrones in Patient #5

Figure 12.

The pre- and post-CRT activation ECGI maps from patient 5, shown in anterior and inferior projections. For each condition, the QRS duration (QRSd) is listed, followed by the calculated ED index. The epicardial pacing sites are marked by white asterisks. From Silva JNA, Ghosh S, Bowman TM, et al. Cardiac resynchronization therapy in pediatric congenital heart disease: Insights from noninvasive electrocardiographic imaging. *Heart Rhythm* 2009; 6(8): 1178-1185; with permission.

Figure 13.

The pre- and post-CRT activation ECGI maps from patient 6, shown in anterior and inferior projections. For each condition, the QRS duration (QRSd) is listed, followed by the calculated ED index. White asterisks denote sites of pacing leads. From Silva JNA, Ghosh S, Bowman TM, et al. Cardiac resynchronization therapy in pediatric congenital heart disease: Insights from noninvasive electrocardiographic imaging. *Heart Rhythm* 2009; 6(8): 1178-1185; with permission.

Figure 14.

The post-CRT activation ECGI maps from patient 7 are shown in anterior and inferior projections. For each condition, the QRS duration (QRSd) is listed, followed by the calculated ED index. White asterisks denote epicardial leads. From Silva JNA, Ghosh S, Bowman TM, et al. Cardiac resynchronization therapy in pediatric congenital heart disease: Insights from noninvasive electrocardiographic imaging. *Heart Rhythm* 2009; 6(8): 1178-1185; with permission.

Post-CRT ECGI Activation-Isochrones in Patient #8

Figure 15.

The post-CRT activation ECGI maps from patient 8 are shown in anterior and inferior projections. For each condition, the QRS duration (QRSd) is listed, followed by the calculated ED index. White asterisks mark the location of epicardial pacing leads. From Silva JNA, Ghosh S, Bowman TM, et al. Cardiac resynchronization therapy in pediatric congenital heart disease: Insights from noninvasive electrocardiographic imaging. *Heart Rhythm* 2009; 6(8): 1178-1185; with permission.

Table 1

Patient characteristics in a study of cardiac memory in patients with Wolff-Parkinson-White syndrome

Patient	Age (yrs)	Gender	ECGI prediction of AP location	Arruda prediction of AP location	Site of successful ablation
W1	12	F	MA/7:00/endocardial	LAL/LL	MA/7:00/endocardial
W2	16	M	MA/4:00/endocardial	LAL/LL	MA/4:00/endocardial
W3	5	M	2 APs/MA/3:00 and 5:00/endocardial	LAL/LL	2 APs/MA/3:00 and 5:00/endocardial
W4	13	M	TA/5:00/endocardial	PSTA/PSMA	TA/5:00/endocardial
W5	12	F	Either TA 4:00 or MA 8:00/endocardial	PSTA/PSMA	TA/3:00/endocardial
W6	13	M	MA/7:00/epicardial	PSTA	MA/7:00/MCV, epicardial
W7	8	F	MA/7:00/endocardial	LPL/LP	MA/7:00/epicardial
W8	13	F	Either TA 3:00 or MA 9:00/endocardial	PSTA/PSMA	TA/3:00/endocardial
W9	10	M	MA/7:00/endocardial	LPL/LP	MA/7:00/endocardial
W10	15	M	MA/6:00/endocardial	LPL/LP	MA/6:00/endocardial
W11	13	F	TA/6:00/endocardial	RP/RPL	TA/6:00/endocardial
W12	12	F	MA/3:00/endocardial	LL/LAL	MA/3:00/endocardial
W13	12	F	MA/5:00/endocardial	LAL/LL	MA/5:00/endocardial
W14	10	F	TA/5:00/epicardial	MCV/venous anomaly	TA/5:00/CS diverticulum, epicardial

Adapted from Ghosh S, Rhee EK, Avari JN, et al. Cardiac memory in patients with Wolff-Parkinson-White syndrome: noninvasive imaging of activation and repolarization before and after catheter ablation. *Circulation* 2008;26:118(9):907-15

Table 2

Electrical dyssynchrony index

Electrical Dyssynchrony	Value
<24msec	Normal
24-28msec	Mild dyssynchrony
28-32	Moderate dyssynchrony
>32msec	Severe dyssynchrony

Data from Silva JNA, Ghosh S, Bowman TM, et al. Cardiac resynchronization therapy in pediatric congenital heart disease: Insights from noninvasive electrocardiographic imaging. *Heart Rhythm* 2009; 6(8): 1178-1185