Direct visualization of the slow pathway using voltage gradient mapping: a novel approach for successful ablation of atrioventricular nodal reentry tachycardia

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Aims

Ablation of atrioventricular nodal reentry tachycardia (AVNRT) has become treatment of choice because of a high success and low complication rate. Most ablations are successful in utilizing an anatomic approach, but anatomic variance, unusual pathway locations, or multiple pathways may complicate the procedure. Visualization of the slow pathway could expedite ablation success and enhance safety. Our purpose is to determine whether voltage gradient mapping can directly image the slow pathway and aid successful ablation of AVNRT.

Methods and results

Three-dimensional voltage maps of the right atrial septum were constructed from intracardiac recordings obtained by contact mapping. Voltage values were adjusted until low-voltage bridging was observed within the Triangle of Koch. Forty-eight consecutive patients undergoing ablation for inducible AVNRT, underwent voltage gradient mapping. The slow pathway was identified in all 48 patients via its corresponding low-voltage bridge. Ablation of the slow pathway associated low-voltage bridges in 48 patients was successful in preventing reinduction following the first lesion in 43 of 48 patients. Five patients had multiple slow pathways and 1 lesion was required to prevent reinduction. Repeat mapping confirmed the absence of low-voltage connections previously observed in all 48 patients.

Conclusion

Voltage gradient mapping can assist in visualization of the slow pathway. Ablation of the associated low-voltage bridge results in loss of slow pathway function and significant changes in the post-ablation voltage map. We conclude that voltage gradient mapping offers the ability to target the slow pathway for successful ablation.

Keywords

Supraventricular tachycardia • Ablation • 3-D endocardial mapping • Voltage gradient mapping • Slow pathway • AV nodal reentry tachycardia

Introduction

Atrioventricular nodal reentry tachycardia (AVNRT) is the most common supraventricular tachycardia inducible in the electrophysiology lab.1,2 Since the early 1990s, ablative therapy proved to be the treatment of choice, beginning with radiofrequency (RF) application and more recently cryoablation.1,3 Although the procedure is highly successful with a relatively low complication rate, both complications and unsuccessful procedures remain. Successful ablations are seen in 91–99% of patients, with recurrence seen in 5–9%, and heart block 1%.1,4–7 There is a higher chance of recurrence in patients with single AV nodal echo vs. patients with complete elimination of the slow pathway at the end of the procedure.1,8–11 Additionally, multiple AV nodal pathways are observed in ~39% of patients with AVNRT.12,13 The slow pathway is located along the inferior aspect of Triangle of Koch (see Figure 1A), towards the tricuspid valve with an atrial voltage ratio 1/10 to 1/2 that of the ventricular voltage.3,14–18 Ablative therapy is applied in the region of the slow pathway until either a junctional response is produced (RF energy), or non-inducibility with, or without, AV nodal echo beats results (cryo ablation or RF energy).19–23 If the ablation lesion was

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The catheter is repositioned, superiorly towards the AV nodal region and the His catheter. Success is measured by the inability to induce AVNRT and/or a change in AV nodal properties (induction of a single echo beat) with an isuprel infusion. The objective of the present study was to determine if the slow pathway could be directly visualized and successfully ablated using voltage gradient mapping. With this technique, high-voltage regions are connected together by regions of low voltage that create low-voltage bridges. We report that low-voltage connections corresponding to the anatomic position of the slow pathway can be directly observed. As a result, successful slow pathway ablation may be accomplished easily and safely, even in patients with multiple slow pathways or challenging anatomy.

**Methods**

Forty-eight consecutive patients with inducible AVNRT were evaluated by voltage gradient mapping. After obtaining informed consent, the patients underwent a standard elective electrophysiological study that included mapping and ablation. Voltage gradient maps were created using the Ensite Navx™ (St Jude Medical, St Paul, MN, USA) system. The creation of voltage maps has been previously validated. A 3-D reconstruction of the atrial endocardial geometry was created by contact voltage mapping using either a 20-pole electrode catheter (St Jude Medical reflexion HD) or a quadrapolar ablation catheter. Peak-to-peak voltage data were collected during sinus rhythm using a Dx Landmark Map (St Jude Medical) of the right atrium, paying particular attention to collection of data from the atrial septum within the Triangle of Koch. Interpolation was set to 10 mm and the interior/exterior projection set to 7 mm. To create a voltage gradient map, the high-voltage slider was adjusted to 1.5 mV, and the low-voltage slider was adjusted dynamically to reveal low voltage bridges within the atrial septum. Voltage data below the low-voltage value is shown as grey, voltage data between the low-voltage and high-voltage values are displayed as red and yellow, while voltage data above the high-voltage value is displayed as purple. Data points were reviewed within each low-voltage bridge to confirm the validity of the recorded data to exclude pre-mature atrial, junctional, or ventricular beats. Ablation site was selected by low-voltage bridge position only, and not based on anatomic position or AV ratio. After confirmation of the data, ablation of the slow pathway associated low-voltage bridges was performed using either CryoMax Medtronic (44 patients) or Safire (St Jude Medical; four patients) ablation catheters. Ablation was performed in tachycardia when using cryoablation, and in sinus rhythm when using RF ablation. Following ablation, a repeat voltage gradient map was performed. A 1 h waiting period

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**Figure 1** (A) The anatomy of the Triangle of Koch is displayed. The triangle is formed by an area bounded by the Tendon of Todaro, the coronary sinus ostium, and the septal leaflet of the Tricuspid valve. The atrioventricular node and bundle of His are located at the apex of Koch’s triangle (Image courtesy of Robert H. Anderson). (B) The Triangle of Koch is outlined over a 3-D voltage gradient map. The region of interest containing the slow pathway associated low-voltage bridge is contained within this outline. (C) The 3-D map is projected over the anatomic preparation. The atrioventricular nodal region is projected over the apex of Koch’s triangle.
following successful ablation included stimulation with isuprel infusion. The study endpoint was the loss of antegrade slow pathway function and the absence of consistent AV nodal echo beats.

Results

The mean patient age was 39 years old, with a range of 11–79 years old. Thirty (63%) of the patients were female and 18 (37%) were male. Forty patients had typical AVNRT, six had atypical AVNRT, and two exhibited both forms of tachycardia. Acute success was achieved in all patients (48 of 48). Successful ablation was defined as the absence of antegrade slow pathway function, inability to re-induce AVNRT (with and without isuprel), and absence of consistent AV nodal echo beats.

First lesion success was 43 of 48 patients (90%). Complete slow pathway ablation was confirmed in 45 of 48 (94%) patients. Seven patients had consistent single echoes following initial mapping, which required remapping, and further ablation. Inconsistent echoes were found in three patients. Cryoablation was used in 44 patients. Radiofrequency ablation was used in four patients with coexisting atrial flutter (2), one patient with coexisting Wolff-Parkinson-White syndrome, and in one patient with inappropriate sinus tachycardia. The average number of lesions placed was 3.4 (range 2–10) per patient. Additional lesions were placed to insure complete coverage of the low-voltage bridge by overlapping lesions, to ablate any residual persistent AV nodal echoes, or to ablate multiple AV nodal slow pathway inputs. The average procedure time measured from the time the patient entered and exited the procedure room was 2 h and 41 min. The average total fluoroscopy time was 14.7 min. The procedure time and fluoroscopy time depended on several factors:

(i) we created a complete right atrial geometry and voltage map (n = 7) using the classic Ensite system, which required separate geometry building and voltage recording. The procedure time and fluoroscopy time was 3.26 h and 22.9 min;

(ii) by limiting geometry creation to Koch’s triangle (n = 28), procedure and fluoroscopy times were 2.34 h and 12.1 min;

(iii) converting to the velocity Ensite system which builds geometry and voltage measurement simultaneously (n = 9), the procedure and fluoroscopy times were reduced to 1.57 h and 9 min.

No complications were observed during this study. All patients were evaluated in clinic 4 months following ablation. No recurrences of AVNRT were noted during a final clinic visit (48 of 48 long-term success).

The average number of maps created was 2.8 per patient. The mean voltage low setting for visualizing the slow pathway associated low-voltage bridge in the study was 0.197 mV (range 0.06–0.387 mV). The mean surface area data point density was 3.9 data points per cm² (range 2.0–7.1 data points per cm²). Total data points collected was 214 per map with an average of 37.8 points that projected on the Triangle of Koch geometry.

The anatomic structure of the Triangle of Koch is displayed in Figure 1A (adopted from Anderson and Ho27). The anatomic relationships associated with the 3-D endocardial reconstruction created by voltage gradient mapping, is shown in Figure 1B. Figure 1C superimposes the 3-D endocardial voltage gradient map upon the anatomic structures of the right atrial septum.

Several morphologies of slow pathway associated low-voltage bridges were observed. The majority of patients (44 of 48 or 90%) had a discrete low-voltage bridge that connected the high-voltage region of the coronary sinus ostium (CS Os) to the high-voltage region of the AV node/His. We designated these discrete connections as Type I low-voltage bridge connections (see Figure 2A). In this group, an average of 3.3 (range 2–6) lesions were placed; however, all were rendered uninducible with the first lesion. Subsequent lesions were placed adjacent to the first lesion to overlap the width of the low-voltage bridge in order to prevent recurrence. These additional lesions were placed based on the voltage gradient map (VGM) and did not involve adjustment of the lesion site closer to the compact AV node. In four patients (8%), the slow pathway associated low-voltage bridge was a narrow region between adjacent high-voltage gradient regions. These were designated as Type II low-voltage bridge connections (see Figure 2B). Patients with a Type II low-voltage bridge required an average of 9.2 (range 7–10) lesions for successful ablation. All slow pathway associated low-voltage bridges were found within the boundaries of the Triangle of Koch.

A clinical example of a Type I slow pathway associated low-voltage bridge study is displayed in Figure 2A. Voltage gradient mapping demonstrates a clear visualization of the slow pathway associated low-voltage bridge before ablation (Figure 2A). Following ablation, significant changes within the Triangle of Koch’s are observed (Figure 2B). The absence of the low-voltage bridge correlated with successful slow pathway ablation. In Figure 2C, the slow pathway associated low-voltage bridge is seen as a narrow band separating regions of high voltage, and is an example of a Type II low-voltage bridge connection. Following ablation, the voltage gradient map changed and no viable connection is observed between the CS Os region and the AV nodal region (Figure 2D). This finding correlates with successful ablation of the slow pathway.

In some patients, the slow pathway was located in unusual positions, or multiple slow pathways were observed within the Triangle of Koch. An example is provided in Supplementary material online, Figure S4). As noted, in this patient, a second slow pathway associated low-voltage bridge was located just inferior and proximal to the AV node. Ablation in this region had an electrogram with A>V, and given proximity to the AV node, may have presented a challenge for successful ablation without voltage gradient map guidance.

Evidence low-voltage bridges located within the Triangle of Koch represent the slow pathway are supported by the following observations:

(i) The presence of low-voltage bridges correlates with electrophysiological evidence of slow pathway function. Slow pathway associated low-voltage bridge was identified in all patients with inducible AVNRT. We did not observe any qualitative difference in the patients with inducible non-sustained AVNRT, compared with patients with inducible sustained AVNRT.

(ii) In patients with dual AV nodal physiology, slow pathway refractoriness correlates with the disappearance of the slow
pathway associated low-voltage bridge. During atrial premature stimulation, mapping within Koch’s triangle demonstrates a loss of the slow pathway associated low-voltage bridge when the slow pathway refractory period is reached. (Figure 3A–C)

(iii) Ablation of the slow pathway associated low-voltage bridge results in termination of AVNRT and inability to re-induce AVNRT. The lack of inducibility correlated with absence of slow pathway associated low-voltage bridge following successful ablation. The high-voltage regions of the CS Os and the AV node are electrically altered and demonstrate an absence of low-voltage bridge connection following ablation.

(iv) A consistent isolated AV nodal echo is associated with an incomplete low-voltage bridge ablation. In such cases, a residual low-voltage bridge connection persists (see Supplementary material online, Figure S5). Following further voltage gradient map directed ablation, successful ablation demonstrated the absence of both the slow pathway associated low-voltage bridge and consistent AV nodal echoes. The presence of consistent single AV nodal echo following ablation may also represent the presence of a second, separate, and distinct slow pathway associated low-voltage bridge, which requires further voltage gradient map guided ablation.

(v) Slow pathway associated low-voltage bridge electrograms often agree with conventional electrophysiological criteria and may demonstrate complex atrial signals that have been previously associated with slow pathway potentials. By evaluating the electrograms within and immediately outside the low-voltage bridge, AV ratios can be assessed. In most cases, the AV ratio complies with the standard electrographic criteria; however, critical limbs of the slow pathway are also observed in regions not commonly associated with the slow pathway where the ‘A’ signal is larger than the ‘V’ signal. Additionally, complex atrial potentials are also frequently observed. (see Supplementary material online, Figure S6).

Discussion

In the present study we describe a method for direct visualization of the slow pathway by using voltage gradient mapping and low voltage bridge identification. The advantage of this approach is the ability to visualize and target the slow pathway within the Triangle of Koch, as well as, provide a definitive endpoint for ablation in patients where the tachycardia may not be inducible. While conventional approaches for AV nodal modification have proven to be successful, multiple slow pathways, anatomical challenges, and interprocedure recurrences may complicate and prolong such procedures. Additionally, there is an increased risk for damage to the AV node with a conventional step-wise approach to targeting the slow pathway. Commonly, if the initial lesion is unsuccessful, the ablation catheter is advanced towards the apex of the Triangle of Koch, increasing the risk of heart block associated with energy application. Definitive identification of the slow pathway location would minimize such a risk, since the catheter is positioned in response to the low-voltage bridge rather than empiric anatomic location or electrogram characteristics.
In the present study we were able to correctly identify the critical slow pathway connections within the Triangle of Koch in all patients. While a dedicated multipolar mapping catheter can be used, it is important to note, we also demonstrated this technique can be used with a standard quadrupolar ablation catheter. This would significantly decrease both the cost and the time required to create the voltage gradient maps. Therefore, the additional time required to create the voltage gradient map may be offset by the increased efficiency and safety of this technique for slow pathway ablation. Further studies will be needed to assess the value of routine voltage gradient mapping during AVNRT ablation.

To properly identify the slow pathway associated low-voltage bridge, the anatomic relationship to the AV node must be clearly understood. The AV node is found at the apex of the Triangle of Koch’s, formed by the CS Os at the floor, the septal leaflet of the tricuspid valve at the medial margin, and the tendon of Todaro at the lateral margin (Figure 1A). The AV node appears as a low-voltage bridge at the anterior superior tricuspid annulus in the expected anatomic position (Figure 5A). This low-voltage bridge must not be identified as a slow pathway associated low-voltage bridge. In contrast, the slow pathway associated low-voltage bridge is observed to connect the high-voltage region at the CS Os to the AV nodal region. There is usually a discrete low-voltage bridge just superior and anterior to the CS Os (Type I). However, in some patients, the slow pathway associated low-voltage bridge is a narrow band between adjacent high-voltage regions, rather than existing as an isolated low-voltage bridge (Type II).

Regardless of baseline voltage characteristics, following successful ablation, the connection between the region surrounding the CS Os and the AV node are significantly modified and demonstrate an absence of low-voltage bridging between the regions.

The fluoroscopic location of the slow pathway identified by voltage gradient mapping conforms to conventional ablation strategies previously described. The low-voltage bridges were observed within the Triangle of Koch and were consistent with location of the slow pathway previously described clinically. However, in some patients, the voltage gradient map directed location was outside the usual expected location, as previously noted (Figure 4). In such cases, conventional anatomical or electrophysiological criteria may not successfully identify the correct slow pathway location and result in treatment failure or complication.

In patients where there is a documented history of tachycardia, but an inability to induce AVNRT in the electrophysiology lab, conventional ablation techniques cannot accurately predict success unless there is absence of slow pathway function. However, as previously noted, the presence of dual physiology and isolated AV nodal echoes is also considered an endpoint for successful ablation, albeit, less certain long-term success. Because the underlying atrial substrate can be assessed, voltage gradient mapping can be used to objectively document the success of the ablation procedure and therefore, long-term success can be better achieved.

**Limitations**

The value of the voltage gradient mapping is greatly influenced by the ability to obtain sufficient sampling of electrograms within the areas of interest (Triangle of Koch). Errors can be introduced by inadequate voltage data sampling within the Triangle of Koch, which reduces the ability to identify the slow pathway associated low-voltage bridges. Also, the data should be reviewed in order to create the voltage gradient maps.
to validate the data sampled and eliminate any ventricular voltage recordings or artefact. Although accurate, creating a voltage gradient map may add additional time for successful ablation of AVNRT. However, when compared with published procedure times using conventional approaches, Steven et al. reported the mean procedure time was 115.1 ± 23.6 min in the group with and 88.9 ± 23.3 min in the group without waiting period (P = 0.009). The waiting period in that study was 30 min, and therefore, the procedure times reported in the present study using VGM does not appear to have adversely affected the total procedure time compared with conventional approaches.

The morphology of the slow pathway associated low-voltage bridge may be varied, as a result, the slow pathway-related low-voltage bridge might be challenging to identify. Review of the data points within the low-voltage bridge provides a means to identify low voltage bridges that should be targeted for ablation by the presence of slow pathway potentials.

We have observed dual AV nodal physiology in patients evaluated for other tachycardias in whom, the slow pathway function was incidental and not clinically relevant. Because of the small numbers of patients (n = 5), we have not included them in the analysis; however, a slow pathway associated low-voltage bridge is observed in such patients. Therefore, the characteristics of the low-voltage bridge cannot be used to distinguish clinically relevant slow pathways, i.e. those associated with clinical AVNRT, from those that are incidentally observed in isolated dual AV nodal physiology. The decision to ablate is dependent on clinical criteria such as documented tachycardia or a history consistent with recurrent reentry tachycardia.

Conclusions

Successful slow pathway ablation has been based primarily on anatomical and electrophysiological characteristics. Unfortunately in some patients, both anatomic and electrophysiological criteria may not be sufficient. In such patients, ablations may become prolonged and may have a greater potential for failure or complication. In the present report, we present a method for visualizing the slow pathway directly using voltage gradient mapping. By identification of the slow pathway associated low-voltage bridge, the slow pathway can be precisely targeted and successfully ablated. In challenging cases, voltage gradient mapping offers the ability to safely and accurately identify critical atrial substrate in AVNRT.

Supplementary material

Supplementary material is available at Europace online.

Conflict of interest: S.J.B. is a member of the Medical Advisory Board of St Jude Medical. M.A.K. and N.J.W. are employees of St Jude Medical. C.J.H. has no conflicts of interest.

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Arrhythmic manifestation of sarcoidosis
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A 52-year-old man with a history of pulmonary sarcoidosis presented an episode of sustained monomorphic ventricular tachycardia (MVT) with left bundle branch block morphology, inferior axis, and transition between V3 and V4. At cardiac magnetic resonance with delayed enhancement left and right ventricle (RV) had preserved function with RV mid-septal and free-wall subepicardial post-inflammatory fibrosis (short axis view, Panel A). Electroanatomical (EA) endocardial voltage map (Carto3 System, Biosense Webster, Diamod Bar, CA, USA) of the RV showed a scar zone (<0.5 mV) involving corresponding areas (Panel B). At electrophysiological study clinical non-sustained MVT was inducible. Radiofrequency ablation (RFA) was successfully applied in the scar zone by using both substrate and pace-mapping (12/12 leads). No recurrences have been reported at 6-month follow-up. Cardiac magnetic resonance with delayed enhancement combined with EA mapping may guide RFA in arrhythmic manifestation of sarcoidosis.

Conflict of interest: none declared.