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The low specificity of low voltage bridges associating atrioventricular nodal reentry in pediatric patients

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Abstract

Purpose Patients with atrioventricular nodal reentry tachycardia (AVNRT) often are managed successfully by ablation of the slow pathway with success rates reported as high as 99%. Low voltage bridges (LVBs) have been demonstrated to be helpful in guiding AVNRT ablation. Patients may present to the electrophysiology lab without evidence of inducible arrhythmia. In these scenarios, the demonstration of LVBs may be diagnostic and guide catheter ablation treatment. The purpose of our study was to prospectively investigate the specificity of LVBs as a diagnostic marker of AVNRT.

Methods Patients aged < 19 years with narrow complex tachycardia prospectively underwent electrophysiology study with intention to perform catheter ablation. In each patient, the primary objective was the collection of right atrial voltage data that was then used to identify LVBs.

Results Twenty-four patients were included after exclusion criteria were applied. Final diagnosis was 11 AVNRT and 13 non-AVNRT (nAVNRT). LVBs were identified in 11/11 AVNRT patients and 9/13 non-AVNRT patients ($p = 0.09$).

Conclusions LVBs are not specific to patients with AVNRT and cannot solely be used for diagnosis. However, in patients with documented AVNRT, the LVB can be used to identify the location of the slow pathway.

Keywords AVNRT · Low voltage bridges · Slow pathway · Pediatric

1 Introduction

Atrioventricular nodal reentry tachycardia (AVNRT) is a form of supraventricular tachycardia resulting from a reentry circuit incorporating both a fast and a slow conducting AV node pathway [1]. Because definitive treatment is achieved by disruption of the slow pathway using catheter ablation, knowledge of the slow pathway location is a key to success. Beginning in the 1990s, 3D electroanatomic mapping proved a reliable means to identify the structural anatomy of the triangle of Koch and to locate slow pathway potentials (SPPs) [2]. SPPs are also known to be present in adults without AVNRT [3]. Mani et al. reported dual AV node physiology to exist in up to 35% of the normal population [4]. More recently, the voltage gradient mapping technique has been developed to guide ablation of the slow pathway and has been demonstrated to improve success [5–9]. The technique identifies areas of low voltage connecting areas of relative high voltage, termed low voltage bridges (LVBs), which are associated with the slow pathway. (Fig. 1) Ablation in the area of LVBs has resulted in successful treatment of AVNRT in both pediatric and adult patients [5–8] with disappearance of the LVB following ablation [5]. Although LVBs appear as markers of the slow pathway in AVNRT, the association of LVBs with the slow pathway has not been studied in pediatric patients without AVNRT (nAVNRT). As most pediatric centers utilize general anesthesia or deep sedation for their electrophysiology studies, the ability to induce tachycardia is hampered. Patients with documented supraventricular tachycardia may have altered arrhythmogenic substrates secondary to changes in the clinical milieu under general anesthesia [10]. Subsequently, the ability to rely on the presence of LVBs to diagnose AVNRT without inducible tachycardia is called into question. The use of LVBs to identify patients without inducible tachycardia is contingent on the specificity of LVBs for AVNRT. The purpose of our study was to prospectively investigate the specificity of LVBs as a diagnostic marker of AVNRT.

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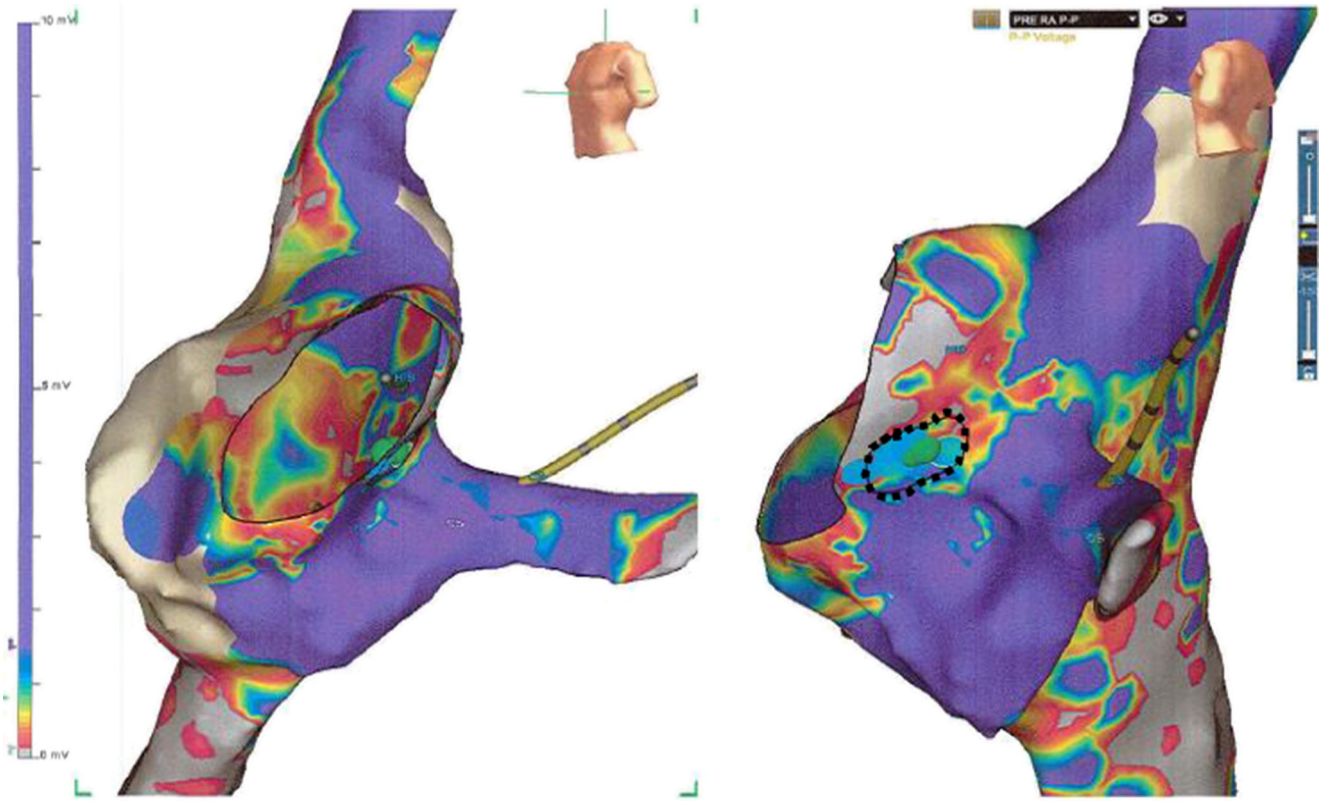

Fig. 1 Depiction of low voltage area (red) covered by cryothermal ablation lesions (blue/green) which resulted in elimination of slow pathway conduction

2 Methods

Study design was approved by the University of Nebraska Medical Center Institution Review Board. All pediatric patients (age < 19 years) with documented normal QRS tachycardia who underwent electrophysiology study for potential ablation were eligible to enroll. Exclusion criteria included those with congenital heart disease, non-sinus rhythm at time of data acquisition, previous cardiac catheterization or EP study, or lack of ablation attempt during the procedure. Additionally, any AVNRT patient who did not demonstrate inducible tachycardia was also excluded.

Data collection After standard pacing protocols were completed, the patients were divided into AVNRT and nAVNRT groups. AVNRT was diagnosed by demonstrating inducible tachycardia consistent with AVNRT: narrow complex tachycardia with short VA time, H-A-V signal on His catheter, His-refractory PVC resulting in no change in the A-A cycle length, and/or para-Hisian pacing excluding concealed accessory pathway. Patients diagnosed with AVNRT as their tachycardia mechanism underwent voltage mapping as per our institutional standard [8]. The nAVNRT patients underwent ablation catheterization therapy based on their tachycardia mechanism, and if ablation was attempted, voltage mapping was performed during our institution's standard 30-min post ablation waiting period. An atrio-His (AH) jump was defined as AH lengthening of at least 40 ms achieved with 10-ms decrements during atrial pacing. His-atrial (HA) jump was defined as HA lengthening of at least 40 ms achieved with 10-ms decrements during ventricular pacing.

Voltage data were collected with a 20-pole catheter (Inquiry II, Abbott, Inc., Abbott Park, IL). An Abbott representative assisted the electrophysiologists in the creation of a 3D map of the right atrium using the EnSite Velocity system (Abbott, Inc., Abbott Park, IL). Atrial voltage points were then collected. After the electrophysiologists were satisfied that sufficient points were collected, a voltage map was created and voltage high and low ranges were adjusted in order to visualize the LVB within the region using a previously published protocol [5]. As a starting point, our electrophysiologists place the upper voltage limit at 1.5 mV and the lower voltage limit at 0.1 mV before adjusting in order to create an idealized map. The Velocity software also enabled a propagation map to be developed from the acquired voltage points and a collision point, if present, was noted. The collision point was defined as the area at which the atrial wavefront during sinus rhythm collided on the propagation map indicating the area of slow pathway conduction. Presence of slow pathway potentials, AH or HA jumps and echo beats as well as the use of cryothermal or RF energy for the ablation were also documented.

Statistical calculations Fisher's exact test was used to compare the incidence of LVBs, SPP, echo beats, and AH/HA jumps between AVNRT and nAVNRT groups. One sample proportion testing against a population was used to compare the found incidence of LVBs in the nAVNRT group with previously reported literature estimates. Sensitivity and specificity were calculated for the above markers. Voltages used to idealize LVBs were compared by unpaired Student's *t* test.

3 Results

Thirty-two patients were prospectively enrolled in the study. There were no differences in age, weight, gender distribution, or average number of data points collected during voltage mapping between the two groups (Table 1). A total of eight patients met exclusion criteria after enrollment; four did not have an ablation attempted during their study, two did not have data collected secondary to either machine or human error, one was in junctional rhythm at the time of data collection, and one patient was diagnosed with atypical AVNRT based on retrograde H-A jump and single atypical AV nodal echo beat, but did not have inducible tachycardia during the EP study making it difficult to confirm the diagnosis. Of the 24 included patients, 11 were found to have AVNRT and 13 were found to have other diagnoses which included eight patients with a concealed accessory pathway, four patients with manifest accessory pathway, and one patient with atrial ectopic tachycardia. Tachycardia was inducible in all AVNRT patients and 9/13 nAVNRT. Isoproterenol was utilized in 7/11 and 5/13 of the AVNRT and nAVNRT groups, respectively. Table 1 summarizes the presence of slow pathway identifiers between the two groups. Of the nine nAVNRT patients with LVBs, all were accessory pathway-mediated tachycardia with five concealed and four manifest. SPPs were documented in 9/11 patients with AVNRT and 3/13 patients with nAVNRT ($p < 0.05$). Of note, the three with SPP in the nAVNRT group also had LVBs. Table 2 indicates the specificity and sensitivity of LVBs, SPP, AH jumps, and echo beats for the identification of AVNRT. Of the AVNRT patients, a true A-H jump was noted in three patients. Sustained slow pathway conduction was noted in an additional four AVNRT patients. Among patients in the nAVNRT group, one had an A-H jump, three had sustained slow pathway conduction, and no patient had a typical AV nodal echo beat. Among the three patients that had sustained slow pathway conduction with an A-H jump, all three had presence of a voltage bridge at the time of mapping.

Patients with AVNRT had the low voltage bridges targeted with an endpoint of no inducible tachycardia as the primary endpoint. All patients had ablation lesions confined solely to the area of the low voltage bridge. Nine of the 11 patients underwent cryothermal ablation utilizing a 6-mm-tip ablation catheter. In one patient, radiofrequency was used in combination with cryothermal ablation and one patient had only radiofrequency ablation. All patients had acute success at the time of procedure. One patient was noted to have recurrence during follow-up. In the nAVNRT group, one patient was noted to have recurrence of concealed accessory pathway conduction.

Table 1 Group demographics, parameters, and data findings. (*) Denotes comparison of AVNRT vs nAVNRT. (**) Marks statistical significance. Atrial-his (AH); collision point (CP); his-atrial (HA); low voltage bridges (LVBs); non-AVNRT (nAVNRT); slow pathway potentials (SPPs)

	AVNRT $n = 11$	nAVNRT $n = 13$	p value*
Mean age (years) Median	15.49 15.8	14.79 15.0	0.49
Mean weight (kg)	62.4	66.6	0.58
Gender (male/female)	3/8	5/8	0.68
Points collected			
Mean Range	1115 503–1568	1181 682–1678	0.65
Max voltage (mV) Range (median)	0.5–2.96 (1.83)	0.68–2.99 (1.4)	0.33
Minimum voltage (mV) Range (median)	0.05–0.68 (0.169)	0.048–0.24 (0.182)	0.2
LVBs	11	9	0.1
SPP	8	3	0.04**
Collision point	11	9	0.1
Echo beat	3	2	0.63
CP+LVB	8	5	0.12
AH or HA jump	3	1	0.3

4 Discussion

Our study found no difference in the incidence of LVBs between AVNRT and nAVNRT groups, showing diagnostic specificity of LVB for the discrimination of AVNRT from nAVNRT is low. In our study, LVBs were found in 69% of nAVNRT patients further challenging LVBs as specific

Table 2 Sensitivity and specificity of identifiers for AVNRT. Atrial-his (AH); collision point (CP); his-atrial (HA); low voltage bridges (LVBs); non-AVNRT (nAVNRT); slow pathway potentials (SPPs)

	Sensitivity	Specificity
LVBs	1	0.31
SPP	0.73	0.77
Collision point	1	0.31
Echo beat	0.27	0.84
CP+LVB	0.73	0.38
AH or HA jump	0.27	0.92

markers for a slow pathway outside the context of inducible AVNRT [4]. Although these data do not support LVBs as a specific diagnostic marker of AVNRT, once AVNRT is induced, ablation is successful when the LVB is targeted [5–8].

We speculate that not all slow pathways are capable of supporting clinical tachycardia. If this is true, then some patients with LVBs but without clinical tachycardia are expected. Furthermore, the historic estimate of up to 35% of the normal population with dual nodal physiology may be low. LVBs were found to be highly sensitive for AVNRT in this study. This would suggest that LVBs can be used to identify the location of the slow pathway in patients with *documented* or inducible AVNRT, but are not adequate to diagnose patients with AVNRT in absence of supporting data. These findings do contrast with the findings of *Drago* et al. [6] where low voltage bridges were found to be specific for AVNRT. These differences may be explained by methodology. When creating the voltage map, our study utilized a 20-pole catheter and collected an average of 1100 points accepted with over 4000 points collected. This is in contrast to the *Drago* et al. study that reports approximately 110 points accepted and 200–300 points collected. Although there is some inherent subjectivity to the interpretation and identification of low voltage bridges, particularly with the adjustment of scales, it should be noted that the ranges of low voltage values between the two studies are similar. However, our median low voltage value of 0.182 mV in our control group is less than that of the *Drago* et al. study which reports 0.33 mV. Indeed, the collection of more voltage points and a lower threshold for minimum voltage may explain our finding of more low voltage bridges in nAVNRT patients.

This study also prospectively identified SPPs, echo beats, and AH or HA jumps in both populations, affording the comparison of LVBs to more traditional markers of AVNRT (Table 2). In four patients with AVNRT, sustained slow pathway conduction was noted despite no evidence for a true AH jump. The presence of an AH or HA jump was insensitive, but most specific for AVNRT, while the identification of an LVB was highly sensitive. Overall, the identification of slow pathway potentials offered the highest combination of both sensitivity and specificity. Although slow pathway potentials were noted within the low voltage bridge, other signals not suggestive of the typical "hump and spike" pattern were also noted. No single diagnostic marker of AVNRT has adequate specificity and sensitivity to substitute for the diagnostic standard, which remains inducible tachycardia with demonstrated characteristics of AVNRT. Therefore, in the absence of inducible tachycardia, all available data should be utilized to make the diagnosis of AVNRT.

There are noted limitations to the study. Voltage mapping is dependent on the acquisition of data points by catheter manipulated within the right atrium. Previous literature has reported that successful identification of LVB is dependent on an adequate point acquisition, but no studies to date have established the number of points or their density required to establish the presence of absence of LVB. To avoid the error of under sensitivity born of collecting too few data points, this study was done according to institutional preference, utilizing a 20-pole catheter for data acquisition. After the data points have been acquired, the voltage bridge is visualized by adjusting both upper and lower voltage limits. We acknowledge the subjectivity of both the development and interpretation of the voltage map but draw attention to the technique's successful use at four separate institutions [5–8]. Additionally, the current standard of therapy, SPP identification, is not without subjectivity. We acknowledge that all patients had their studies performed under general anesthesia which can have electrophysiology effects, particularly on AH or HA jumps, presence of echo beats, and inducible tachycardia. Isoproterenol was not utilized in every patient, only those in whom the primary operator felt it was needed for completion of the case. Operators were not blinded to the tachycardia mechanism during collection of voltage points and this may result in bias for the identification of LVBs. The time to generate the voltage map was attempted to be minimized to avoid added anesthesia and procedure time as a patient safety issue. This study did not specifically evaluate the ablation results with regard to the presence of SPP or LVBs. The patients were categorized by diagnoses at time of EP study and do not take into account any patients that may develop symptomatic and clinically apparent AVNRT later in life.

LVBs are sensitive but not specific for AVNRT, and therefore other supporting data is required to assure accurate diagnosis of AVNRT in patients without inducible tachycardia. Once AVNRT has been diagnosed using traditional methods, the targeting of low voltage bridges may be helpful in ablation of the slow pathway.

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