

Cold collision: A novel cryothermal ablation technique for junctional ectopic tachycardia



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Introduction

Junctional ectopic tachycardia (JET) is a tachyarrhythmia that is felt to arise from the atrioventricular (AV) node and His bundle area. The idiopathic form of JET in a structurally normal heart is rare and often resistant to antiarrhythmic drug therapy.¹⁻⁴ Transcatheter radiofrequency ablation has been reported for use of treatment for JET but is associated with a high risk of inadvertent AV nodal block.³ As a result, utilization of 3-dimensional (3D) mapping, atrial overdrive pacing, and cryoablation have been developed for ectopic focus localization, arrhythmia identification, and ablation of the junctional ectopic foci, respectively.⁵⁻⁷ Based upon previous JET cryoablation studies showing successful ablations in the area of the slow pathway and recent advancements in the identification of the location of the "slow pathway" by voltage and propagation mapping,^{2,7} we present a case where wavefront propagation mapping was used to identify the junctional ectopic focus in a 9-year-old boy.

Case report

A 9-year-old white boy was admitted with complaints of low-grade fever, cough, shortness of breath, and chest pain and was diagnosed with viral myocarditis. Prior to admission, cardiac markers at the referring center revealed elevated B-type natriuretic peptides (BNP) and high-sensitivity troponins (>99th percentile). Upon his transfer to our facility, the troponin nearly normalized but the BNP remained elevated. During hospitalization, he was noted to be in JET and his echocardiogram demonstrated moderately decreased left ventricular (LV) systolic function. The diagnosis of

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KEY TEACHING POINTS

- The idiopathic form of junctional ectopic tachycardia (JET) in a structurally normal heart is rare and often resistant to antiarrhythmic drug therapy.
- Utilization of 3-dimensional mapping, atrial overdrive pacing, and cryoablation can be used for successful ectopic localization, arrhythmia identification, and ablation of the junctional ectopic foci.
- Atrial pacing at a cycle length faster than the JET allows for wavefront propagation mapping.
- Cryoablation at the wavefront collision site can result in safe and successful termination of JET.

myocarditis was confirmed with a cardiac magnetic resonance image, demonstrating a normal LV diastolic dimension (indexed z-score -0.9), dilated LV systolic dimension (indexed z-score 1.6), decreased LV ejection fraction of 45%, and late gadolinium enhancement.

His hospital course was relatively uncomplicated, and he was discharged home after 2 days following resolution of his chest pain, defervescence, and normalization of his BNP. During follow-up, a 24-hour ambulatory electrocardiogram (ECG) demonstrated persistence of the accelerated junctional rhythm with an average heart rate of 123 beats per minute. Review of a previous ECG, which was obtained during an evaluation for a heart murmur 4 years prior to the current presentation, demonstrated junctional tachycardia at that time as well. Given these findings, it was postulated that his decreased ventricular function may have been in part due to ongoing tachycardia-induced cardiomyopathy.

Owing to the decreased function and presumed tachycardia-induced cardiomyopathy exacerbating the acute myocarditis, the family was approached with treatment options of medical management vs electrophysiology study

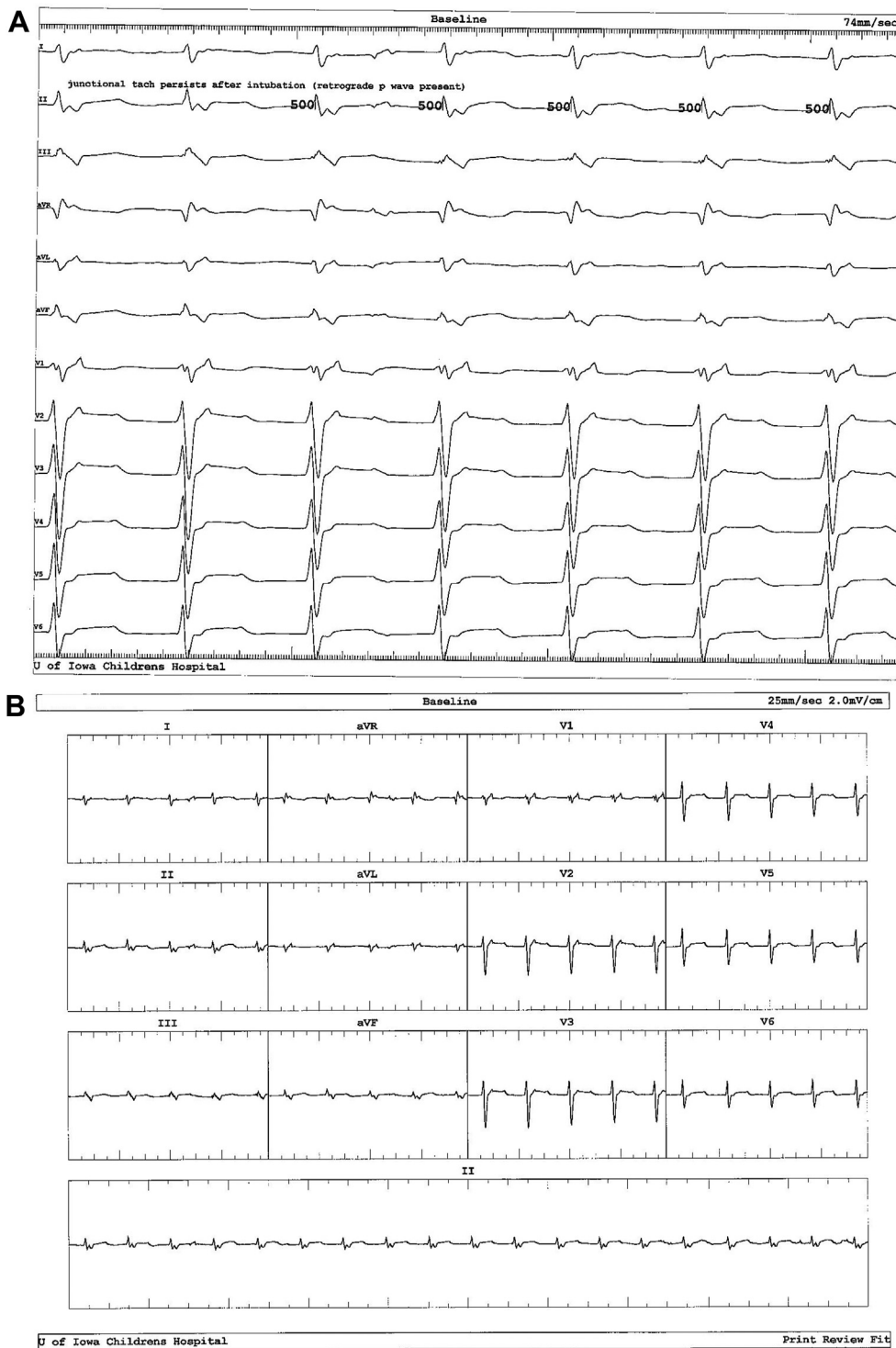


Figure 1 At baseline the patient was in a persistent junctional ectopic tachycardia (JET) (cycle length 500 ms) with evidence of retrograde P waves. The JET persisted after anesthesia induction. Demonstrated here in the surface electrocardiogram.

with possible ablation. The family elected to pursue an electrophysiology study with possible ablation procedure.

Following standard consent, the patient was taken to the pediatric electrophysiology lab, where he was induced and maintained under general anesthesia (propofol followed by sevoflurane) throughout the procedure. Three-dimensional mapping was performed using the Abbott's EnSite

3-Dimensional Precision Cardiac Mapping; no fluoroscopy was required (Abbott, St. Paul, MN).

At baseline the patient was in a persistent JET (cycle length 500 ms) with evidence of retrograde P waves. The JET persisted after anesthesia induction (Figure 1). Intermittently, there were periods of retrograde Wenckebach, consistent with a junctional tachycardia rather than an atypical AV

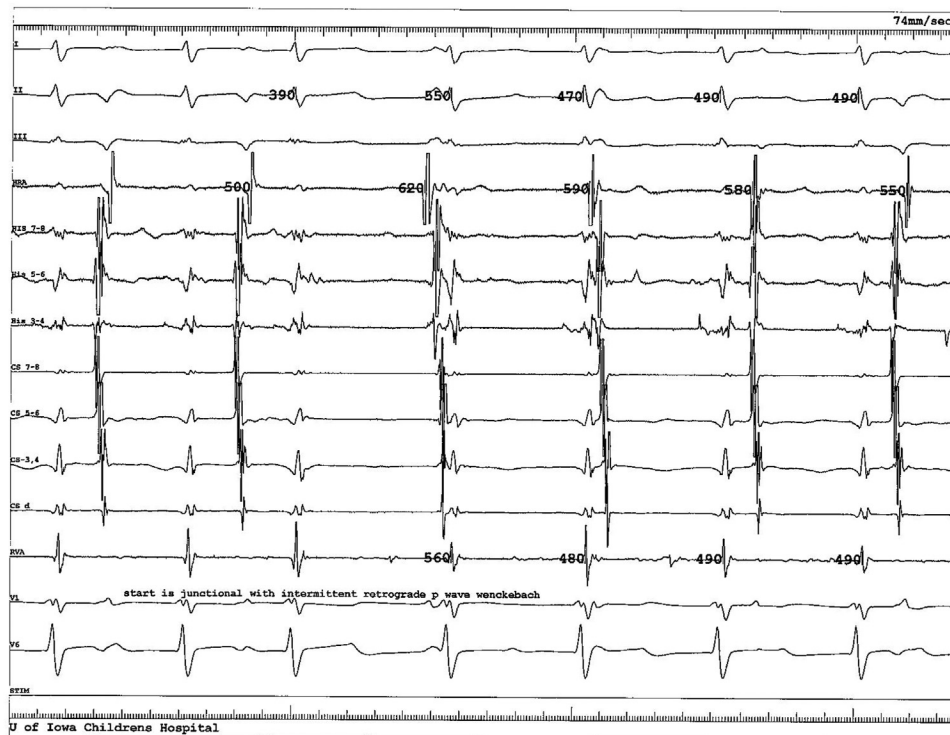


Figure 2 Intracardiac electrograms with the first 2 beats showing a junctional tachycardia with retrograde VA prolongation (VA Wenckebach). The third beat is likely an atrioventricular node echo beat with the subsequent beats showing fusion between a sinus-driven atrial activation and junctional tachycardia, transitioning back junctional tachycardia with retrograde VA Wenckebach, consistent with a junctional tachycardia.

nodal reentrant tachycardia (Figure 2). A wavefront propagation map in the area of the triangle of Koch was created during atrial pacing at a rate faster than the JET rhythm, using a steerable 6F octapolar catheter. The area of His potentials was identified. A collision point and a low-voltage bridge were identified during propagation mapping in the mid triangle of Koch (Supplemental Figure 1, Supplemental Video 1).

To reduce the risk of damage to the AV node and AV nodal artery, a cryothermal ablation catheter was selected (Freezor Xtra 3; Medtronic, Minneapolis, MN). The initial cryothermal lesion placed at the wavefront collision site resulted in slowing, then termination of the JET at 7.6 seconds (-10°C to -12°C), with transition to normal sinus rhythm (Figure 3). A full cryothermal lesion at -70°C was placed for 6 minutes, followed by a 4-minute lesion ("freeze-thaw-freeze" technique). Additional insurance lesions were placed around the successful site, extending toward the coronary sinus. The PR interval was monitored closely while all lesions were being placed. An isoproterenol bolus was delivered, which resulted in an expected increase in heart rate. Following the isoproterenol bolus, the patient developed a junctional rhythm at cycle length of about 500–520 ms, which gradually slowed and return to sinus rhythm. An intermittent slower junctional rhythm was noted after the cryoablation; no additional cryothermal applications were placed.

Postablation electrophysiologic testing, including atrial and ventricular burst and extrastimulus pacing, was

performed without evidence of JET on or off isoproterenol. No other arrhythmias or arrhythmia substrates were identified. The procedure was well tolerated, and the postprocedural recovery was without complication.

A follow-up ECG and Holter studies within the first 24 hours, 2 months, and 1 year postprocedure revealed sinus rhythm with no recurrence of JET. An echocardiogram performed at his 2-month follow-up visit demonstrated the return of normal ventricular function.

Discussion

JET is thought to be secondary to an automatic focus near the AV node and His bundle area. The idiopathic form of JET is often resistant to antiarrhythmic drug therapy and radiofrequency ablation is associated with a high risk of unintentional AV nodal block.^{1-4,8} We previously reported the largest study to date analyzing the location of the JET focus using 3D mapping and cryothermal ablation and thereafter described the procedural technique.^{2,7} In this case report we demonstrated that the ectopic focus may be located at the lower part of the triangle of Koch, close to the slow pathway area, and either cryoablation or a careful radiofrequency lesion may be successful. Cryothermal ablation may be preferable in smaller patients because of the smaller dimensions of the triangle of Koch and catheter stability.

Based on the theory that the JET automaticity arises from the slow pathway area, we hypothesized that identifying the

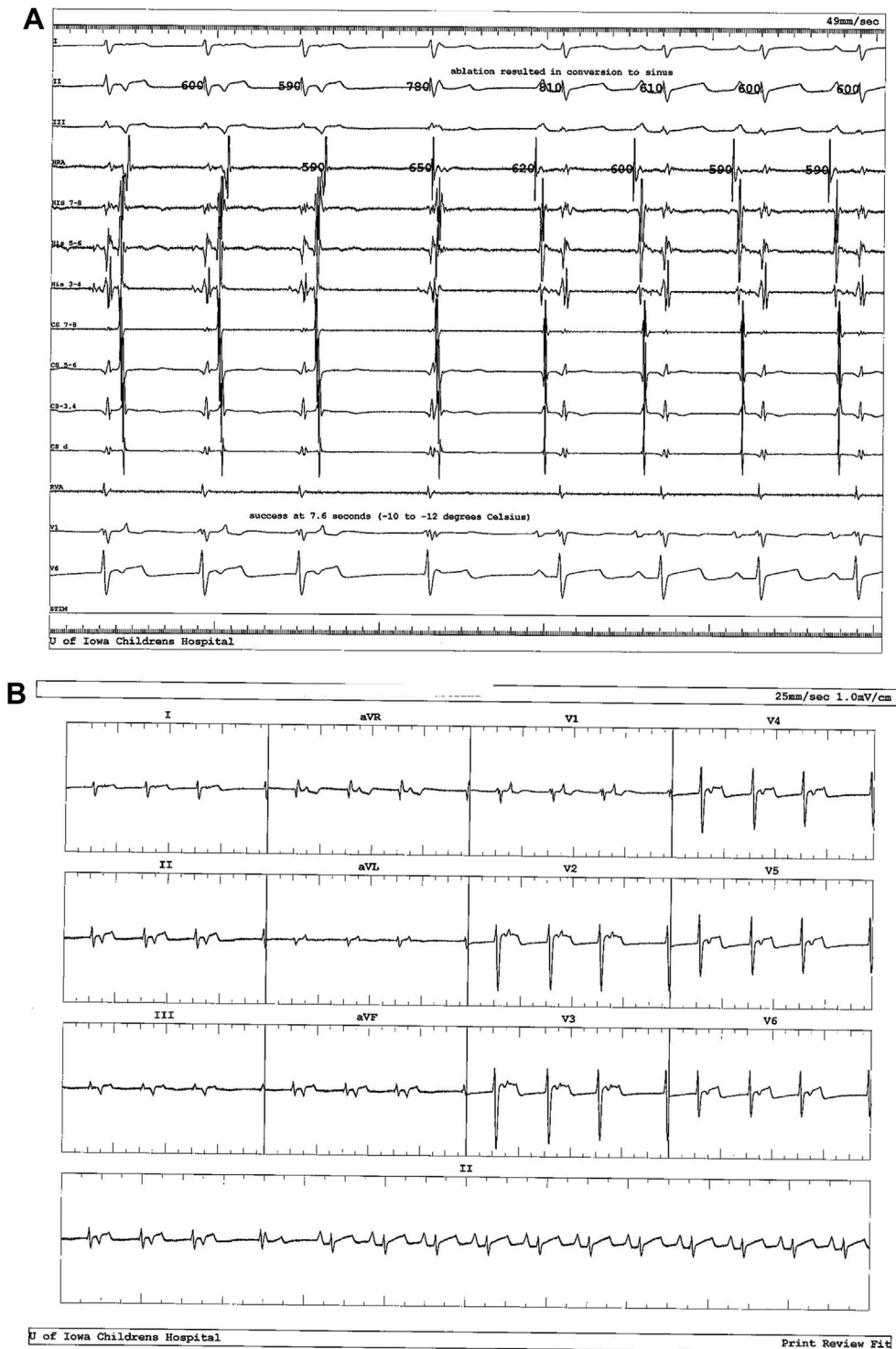


Figure 3 The initial cryothermal lesion placed at the wavefront collision site resulted in slowing and then termination of the junctional ectopic tachycardia at 7.6 seconds (-10°C to -12°C) with transition to normal sinus rhythm.

area of wavefront propagation collision, previously described for AV nodal reentrant tachycardia ablation, could help identify the ectopic focus. Atrial pacing at a cycle length faster than the JET cycle length allowed for 3D mapping of the wavefront collision site, which coincided with the successful

cryothermal ablation site. In this case, it is important to differentiate between atypical AV nodal reentrant tachycardia and JET. The diagnosis of JET was strongly supported by retrograde Wenckebach occurring during the spontaneous arrhythmia, as well as atrial pacing fusing in and out of the

JET. As previously described by Fan and colleagues,⁶ atrial overdrive pacing during junctional tachycardia resulted in transient suppression of the tachyarrhythmia. After cessation of pacing, the junctional tachycardia spontaneously resumed with a junctional beat, led by a His signal, which resulted in an Atrial-His-His-Atrial response. This was the expected response given the automatic mechanism of the junctional tachycardia and its site of origin in the AV junction. To our knowledge, this technique has not previously been documented. A possible limitation to this technique would be a faster junctional escape rate that would in turn require a very fast atrial overdrive pacing, making ablation in this delicate area more challenging.

Conclusion

We present a novel cryothermal ablation approach to the definitive treatment of JET, in which the automatic focus of JET was identified at the area of wavefront collision within the triangle of Koch. Atrial pacing at a cycle length faster than the JET allowed for wavefront propagation mapping. Although further studies will be required to confirm this hypothesis, the reported technique may be useful in the treatment of JET.

Appendix Supplementary Data

Supplementary data associated with this article can be found in the online version at [10.1016/j.hrct.2022.09.013](https://doi.org/10.1016/j.hrct.2022.09.013).

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