A case of typical atrioventricular nodal reentrant tachycardia showing a rare potential of compact atrioventricular node

Soichiro Yamashita, MD, Makoto Takemoto, MD, Masamichi Iwasaki, MD, Masanori Okuda, MD

From the Department of Cardiology, Hyogo Prefectural Awaji Medical Center, Sumoto, Japan.

Introduction

The circuit of common atrioventricular nodal reentrant tachycardia (AVNRT) has several components, including the fast pathway (FP) and slow pathway (SP) in the atrioventricular node (AVN). Multiple atrioventricular (AV) nodal anterograde pathways are known; unidirectional block occurs at the interface between the AVN and its input pathways.[1–3] In vitro studies using optical maps can visualize various interactions of multiple pathways.[4] Otherwise, all potentials in the AVN are difficult to visualize via a clinical electrophysiological examination using an electrode catheter. This case report demonstrated rare AVN potentials with interesting electrophysiological dynamics in Koch's triangle.

Case report

A 41-year-old male patient complaining of palpitations, diagnosed with drug-refractory supraventricular tachycardia (SVT), was referred for catheter ablation. The patient had no structural heart disease or comorbidities. A 12-lead electrocardiogram revealed a short RP tachycardia with a cycle length of 240 ms. The electrophysiological study was performed under general anesthesia. We placed a 10-pole catheter (EPstar [5 mm spacing]; Japan Lifeline, Tokyo, Japan) in the His bundle (HB) region, a 20-pole catheter (BeeAT; Japan Lifeline, Tokyo, Japan) from the right atrium to the coronary sinus, and a 4-pole catheter in the right ventricular apex. During sinus rhythm, the baseline AH interval was 96 ms and the HV interval was 40 ms. The earliest atrial activation site of retrograde conduction was the HB region. The retrograde conduction was observed as a single sequence and had a decremental property without a jump-up phenomenon. A programmed atrial single extrastimulus also revealed a decremental property. Shortening the coupling interval resulted in (1) the fused potential splitting of the HB and X potentials, and (2) the sequence of X potential change from proximal-distal to distal-proximal direction on the mid septum (Figure 1A), while both the AX and XH intervals were prolonged upon further shortening. After the jump-up phenomenon in the AH interval, X and H returned to a single fused potential, and the sequence of the X potential returned to the initial state. The antegrade AV nodal conduction curve is shown in Figure 1B.

SVT was induced by atrial extra pacing at 500/270 and 260 ms. During tachycardia, the cycle length of the right atrium was alternated from 228 to 238 ms, although the X-X was stable at 230 ms. The sequence of SVT had an H-A-V pattern, and the earliest site of atrial activation was in the HB region. The His-refractory premature ventricular contraction (PVC) did not reset the tachycardia. There was ventricular overdrive pacing during tachycardia, followed by resumption of tachycardia with the initial V-A-V activation sequence, thereby excluding the diagnosis of atrial tachycardia. Thus, the SVT was diagnosed as a typical slow-fast AVNRT, which was sometimes sustained with a 2-to-1 AV

KEY TEACHING POINTS

- Compact atrioventricular node (AVN) potential is rarely visualized using a catheter electrode in clinical settings. In this study, low-frequency and small-magnitude potentials were identified in the superior middle area of Koch's triangle.
- Electrophysiological studies suggest that their potentials are independent of the atrium, His bundle, and lower common pathway.
- Dynamic changes of compact AVN potentials in various activation patterns can help us understand the interactions of multiple AVN pathways and anatomy of the atrioventricular nodal reentrant tachycardia.

KEYWORDS Atrioventricular nodal reentrant tachycardia; Koch's triangle; Compact atrioventricular node; Supraventricular tachycardia; Tachycardia with atrioventricular block

(Heart Rhythm Case Reports 2024;10:502–505)

Address reprint requests and correspondence: Dr Soichiro Yamashita, Department of Cardiology, Hyogo Prefectural Awaji Medical Center, Shioya 1-137, Sumoto, Hyogo, Japan. E-mail address: soyamst@gmail.com.

https://doi.org/10.1016/j.hrcr.2024.04.018

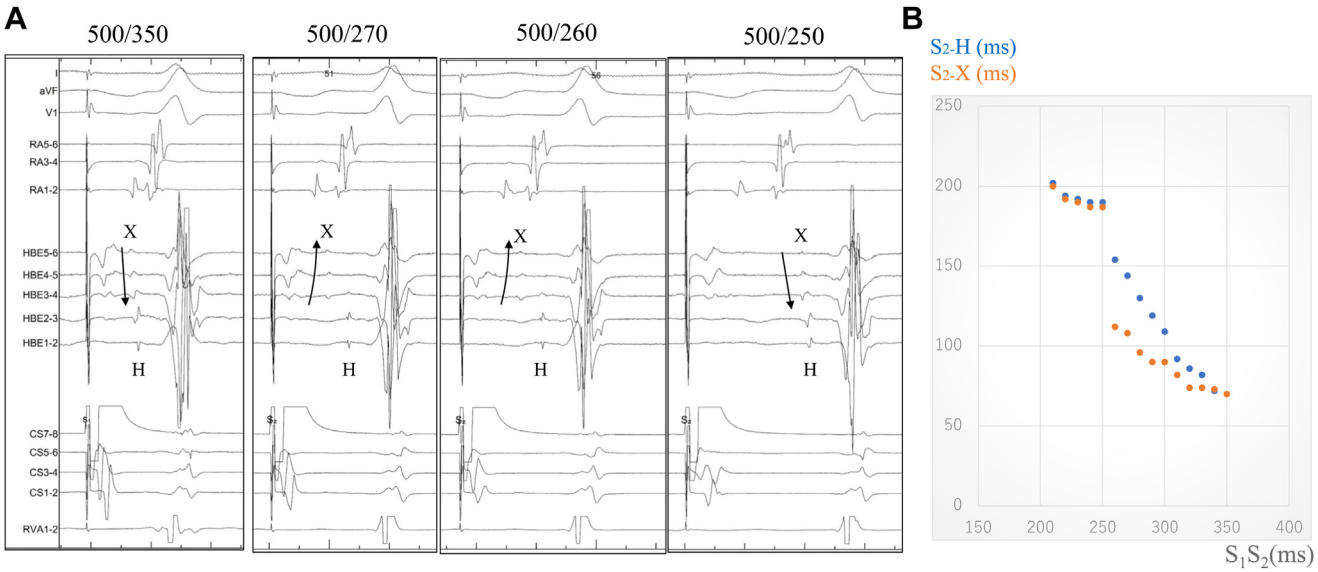

Figure 1 **A:** Programmed extra pacing from the atrium showed a dynamic change of X and H potentials. At coupling of 500/350 ms, X and H were fused together and were conducted in an antegrade fashion, but at 500/270 ms, X and H were split and the sequence of X potentials changed. When the coupling interval was shortened, St-X and St-H showed decremental properties. At 500/250 ms, St-H was suddenly prolonged, as explained by the jump-up phenomenon, and X returned to its initial sequence. Although the St-A interval was prolonged by a conduction delay in the atrium, the AH interval was clearly prolonged. **B:** Atrioventricular conduction curve. St-X (orange) and St-H (blue) each exhibited decremental properties. The split of X and H occurred at extra pacing of 500/300 ms. Tachycardia was inducible at 500/270 and 280 ms. A jump-up phenomenon was seen at 500/250 ms. H = His bundle; St = stimulus.

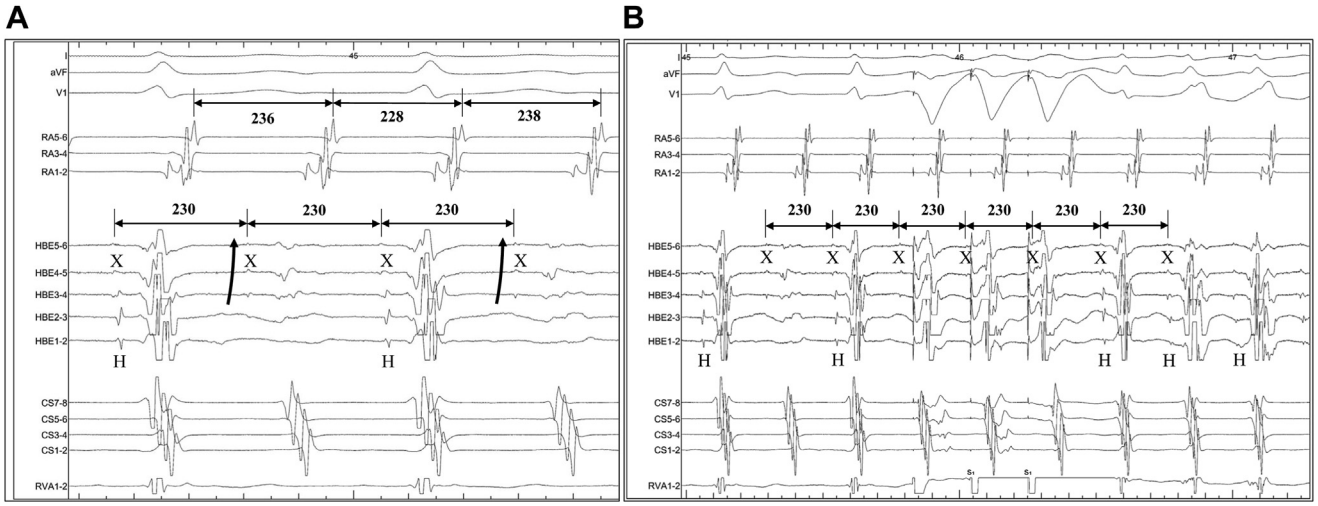

Figure 2 **A:** Atrioventricular nodal reentrant tachycardia (AVNRT) with 2:1 atrioventricular (AV) block. The sequence of X was clearly identifiable at the timing of AH block. During programmed atrial extra pacing, the sequence corresponded to that before the jump-up. **B:** Triplet premature ventricular contractions paced from right ventricle apex during AVNRT. Although they reset the His bundle and changed AV conduction, the X-X intervals remained unaffected and unchanged, suggesting that X is independent from the His bundle.

block. During tachycardia with AV block, the X potential continued with an antidromic sequence. Although the early triplet of PVCs during tachycardia changed AV conduction by resetting the HB potential, the cycle length of X-X was not affected, indicating that X was independent from HB (Figure 2).

We created a 3-dimensional activation mapping of Koch's triangle during atrial pacing at 500 ms (CARTO; Biosense Webster, Irvine, CA), using a multielectrode mapping catheter. X potentials were detected between the HB and the ostium of the coronary sinus, and these connected to the HB at the superior middle side of Koch's triangle (Figure 3). After ablation for antegrade SP, AVNRT became noninducible by programmed stimulation under isoproterenol infusion. After the ablation, a jump-up phenomenon disappeared, indicating the SP was eliminated. Although the X potential was identified even after the ablation, the sequence was not changed by a various coupling of atrial stimulation. The tachycardia did not recur over a follow-up period of 18 months.

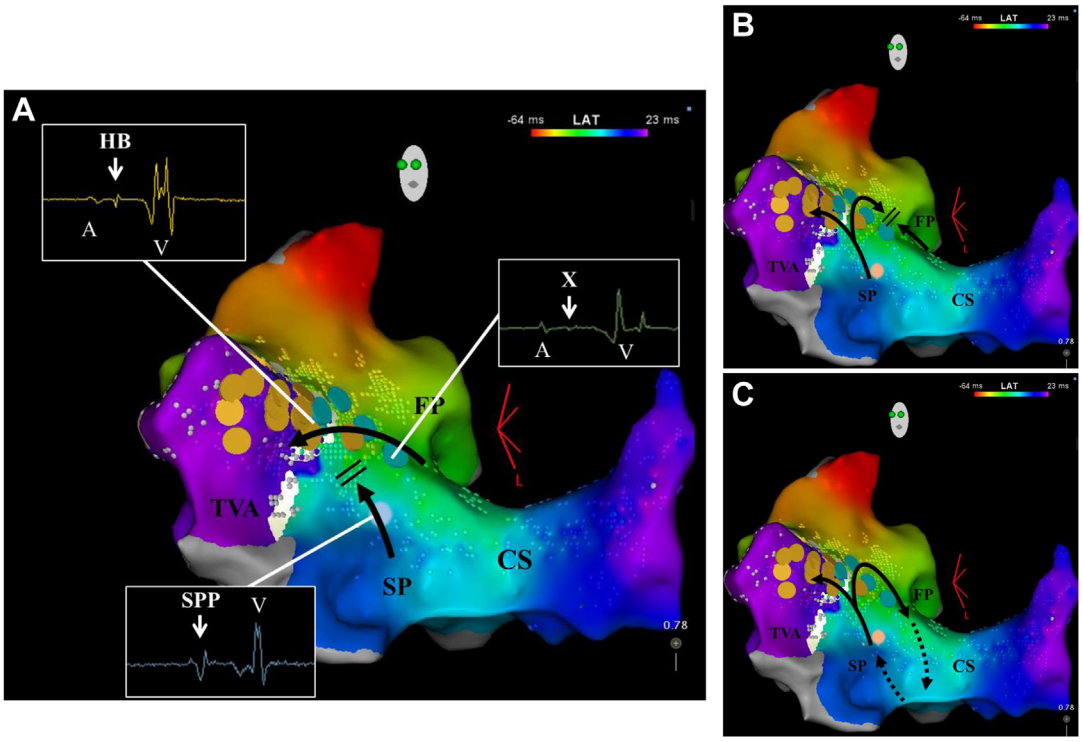

Figure 3 Electroanatomical mapping in Koch's triangle during atrial pacing at an interval of 500 ms created by multielectrode mapping catheter. A slow pathway (SP) potential was recognized at the site of the right inferior extension, and ablation at the location was successful. Blue dots represent the location of X potential, and orange dots represent the His bundle (HB). X was located in superior middle area of Koch's triangle and connected to the HB. Panels A–C illustrate the probable mechanism of sequence change of X. **A:** At longer A-A intervals, propagation through the fast pathway (FP) conducted to compact the atrioventricular node (AVN) antegradely. **B:** At shorter intervals, FP block occurred, and conduction via SP turned at the lower common pathway and reached the compact AVN retrogradely. **C:** The activation pattern of atrioventricular nodal reentrant tachycardia. CS = coronary sinus; CSO = coronary sinus ostium; SPP = slow pathway potential; TVA = tricuspid valve annulus.

Discussion

This report demonstrates a rare X potential with various conduction patterns within Koch's triangle at the interface between multiple pathways. The electrophysiological findings of the X potential suggest the following: (1) X exists in Koch's triangle and connects the atrium and HB; (2) X has a decremental property; and (3) at the atrial pacing with a longer coupling interval, X is antegrade. X changed its conduction to antidromic after shortening the interval. The sequence of X was also antidromic during AVNRT.

Kaneko and colleagues[5] reported a lower common potential recorded in Koch's triangle during slow-slow AVNRT. The X potential was small in magnitude and low in frequency, similar to the lower common potential. Nevertheless, the X potential's sequence had a dynamic change bidirectionally along a prolongation of the A-H interval. Therefore, X was not a simple connection such as a lower common pathway. During tachycardia, atrial cycle length was alternated, although the X-X was stable at 230 ms. This phenomenon supports the evidence that X was not part of the atrium. Moreover, if X was far-field atrial potential, the dynamic change of sequence in different coupling interval was difficult to explain. Longitudinal dissociation within the HB is often recognized in clinical settings.[6] In this case, although PVCs during SVT reset the HB, the X-X interval remained unchanged, indicating that X was independent of HB.

Anatomically, X potentials were located between the HB and the ostium of the coronary sinus, which is consistent with a compact AVN.[7] Clinically, it is unusual to see the potential of compact AVNs. Accordingly, Mazgalev and colleagues[8] demonstrated a surface potential and electrophysiological properties of compact AVNs in vitro using a rabbit heart. A compact AVN potential was visualized using microelectrodes, and had a decremental property with the jump-up phenomenon. These electrophysiological properties are similar to those of the X potentials. The AVN potential differs strongly from the atrial or His bundle electrograms. They have an average amplitude of 0.9 mV when it is recorded by bipolar electrodes spaced at 0.2 mm.[8] The maximum rate of rise of action potential in AVN is significantly smaller (4–6 V/s) than atrial muscles (80–100 V/s).[9,10] Moreover, AVN cells also have shorter action potential durations relative to atrium (113 ms relative to 155 ms).[11] For these reasons, AVN potential may be difficult to detect in clinical settings. Nevertheless, we could visualize AVN potentials

as X in the present case. A possible cause is that the AVN of this patient may have a special characteristic of high amplitude or high maximum derivative, although the characteristic of AV conduction curve was nonspecific. Or they may have been missed during routine studies because of their small potential. Another possible reason is that the X may have included a wide area of transitional cell (TC) layers. TC layers are suggested to be a component of the FP, and action potentials of TC cells have characteristics closer to that of the atrium than AVN.

Multiple preferential AV nodal input pathways have been demonstrated in optical mapping studies.[4] They showed a dynamic change of bidirectional conduction and interaction among fast, slow, and intermediate AVN pathways alongside various coupling intervals. A possible mechanism behind the sequence change of X could be that the conduction via SP turns at the lower common pathway and collides with the antegrade conduction via FP at the middle of the compact AVN (Figure 3B). At that time, conduction via the SP is transmitted downward to the HB. As shown in Figure 3C, the activation pattern during AVNRT at the level of compact AVN is the same as the antidromic sequence of X. Under a coupling interval shorter than 500/250 ms, the sequence of X returned to its initial state with jump-up. This suggests the existence of another pathway, other than FP and SP (ie, a very slow pathway [VSP]). Because the induction zone of AVNRT was between 270 and 260 ms, the VSP was a bystander and unrelated to the tachycardia. Although its detailed anatomy was unclear, the VSP passed through the compact AVN in an antegrade fashion, according to the X sequence.

Conclusion

This is the first case report of rare potentials of compact AVNs that demonstrated various patterns in the interaction of multiple AVN pathways.

Funding Sources: This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: The authors have no conflicts of interest to declare.

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