Significant respiratory-related fluctuations in atrioventricular nodal conduction time in a case of atrioventricular nodal reentrant tachycardia

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Introduction

Respiratory-related alterations in autonomic nervous activity have a substantial impact on the heart conduction system.[1] While the influence of respiration on the sinus node, known as heart rate variability, is widely recognized, there are limited reports on the effects of respiration on atrioventricular nodal function. This case report highlights significant respiratory-related fluctuations in AH intervals observed during atrial burst pacing.

Case report

A 65-year-old male patient presented to our institution experiencing frequent palpitations. A 12-lead surface electrocardiogram recorded during the palpitations revealed a narrow QRS regular tachycardia with indistinct retrograde P waves. The tachycardia was successfully terminated through intravenous administration of verapamil. No delta wave was observed during sinus rhythm. Following informed consent, we conducted an electrophysiological study under deep sedation with dexmedetomidine and propofol, using noninvasive positive pressure ventilation during the procedure. Baseline measurements showed a sinus cycle length of 1260 ms, an AH interval of 95 ms, and an HV interval of 55 ms. During right ventricular pacing, retrograde conduction occurred only during a long pacing cycle length (PCL), with the shortest VA interval being 320 ms. Electroanatomical mapping was carried out using an ultra-high-density mapping system (RHYTHMIA HDx™; Boston Scientific, Marlborough, MA). Sequential contact mapping was performed using a multipolar basket catheter (Intellamap Orion™; Boston Scientific). Notably, during ventricular pacing, the earliest atrial activation site was the His bundle lesion, demonstrating retrograde conduction via the fast atrioventricular nodal pathway.

Atrial extrastimulation revealed dual atrioventricular nodal pathway physiology. Wenckebach conduction was evident during atrial burst pacing at a PCL of 430 ms, and 2:1 conduction was observed at a PCL of 330 ms. Notably, significant fluctuations in AH intervals were observed during atrial burst pacing with a PCL ranging from 440 to 530 ms (Figure 1). Careful monitoring of the respiratory detection sensor in the mapping system revealed synchronized fluctuations in the AH interval with respiration; the AH interval lengthened during expiration and shortened during inspiration (Figure 2 and Supplemental Video 1). AH intervals ranged from 141 to 292 ms. Similar variations in AH intervals were not observed during atrial burst pacing with a PCL of >540 ms or during sinus rhythm. Furthermore, AH intervals were shortened following the infusion of isoproterenol, and respiratory-related fluctuations in AH intervals were no longer observed at any PCL.

Although clinical tachycardia was not induced during the procedure, we suspected the clinical tachycardia was slow-fast atrioventricular nodal reentrant tachycardia, based on the

KEY TEACHING POINTS

- The impact of respiration on the atrioventricular nodal function has not been fully elucidated.
- In this case, monitoring of the respiratory detection sensor in the mapping system revealed synchronized fluctuations in the AH interval with respiration; the AH interval lengthened during expiration and shortened during inspiration.
- The fluctuation of the AH interval might be modulated by the autonomic nervous activity with respiration.

KEYWORDS Respiration; Autonomic nervous system; Atrioventricular node; Atrioventricular nodal reentrant tachycardia; Catheter ablation

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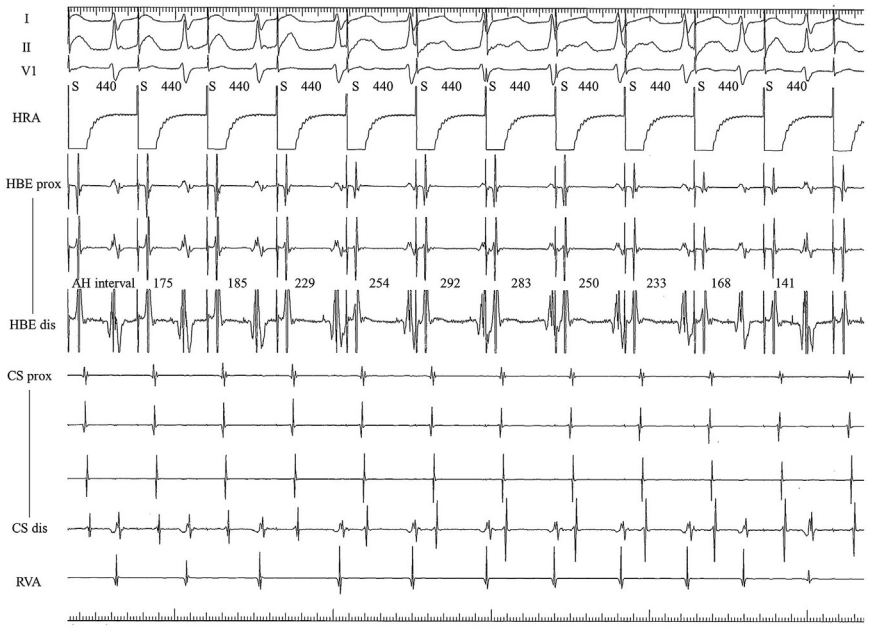

Figure 1 Intracardiac electrograms during atrial burst pacing. Atrial burst pacing was performed with a pacing cycle length of 440 ms, leading to notable fluctuations in AH intervals. CS = coronary sinus; HBE = His bundle electrogram; HRA = high right atrium; RVA = right ventricular apex.

presence of dual atrioventricular nodal physiology, retrograde conduction via the fast atrioventricular nodal pathway, and the absence of accessory pathway conduction. Owing to the severity of the patient's symptoms, we opted for ablation of the slow pathway. We chose the elimination of the slow pathway as the ablation endpoint to ensure the elimination of slow-fast atrioventricular nodal reentrant tachycardia. Using an irrigated catheter (INTELLANAV STABLEPOINT™; Boston Scientific), we performed slow pathway ablation, leading to elicitation of junctional beats during radiofrequency application. No AH jump-up was observed postablation, and no tachycardias were induced. Atrial burst pacing at any PCL failed to induce fluctuations in the AH interval. The patient remained symptom free during the 6 months of follow-up.

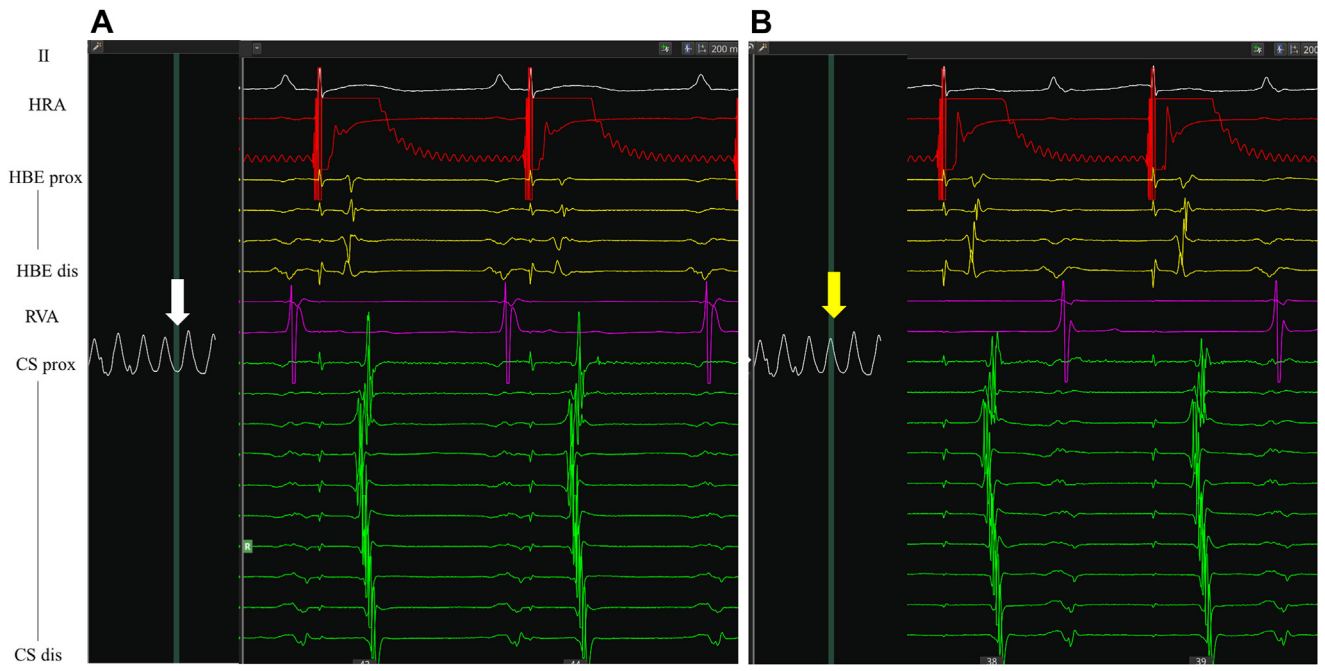

Figure 2 Intracardiac electrograms and respiratory detection monitor during atrial burst pacing. **A:** Recorded intracardiac electrograms during the expiration phase. Downward curves in the respiratory detection monitor indicate the expiration phase (highlighted by a *white arrow*). AH intervals were prolonged during this phase. **B:** Tracing recorded during the inspiration phase. Upward curves in the respiratory detection monitor indicate the inspiration phase (highlighted by a *yellow arrow*). AH intervals were shortened during this phase. Also, refer to Supplemental Video 1. Abbreviations are consistent with those in Figure 1.

Discussion

Heart rate variability is a result of respiratory-related alterations in autonomic nervous activity. Typically, heart rate decreases during expiration and increases during inspiration, correlating with increased parasympathetic nerve activation during expiration and its suppression during inspiration.[2] This respiratory-related alterations in autonomic nervous activity is thought to occur as inspiration reduces efferent vagal tone through the stimulation of pulmonary stretch receptors, while expiration enhances efferent vagal tone.[3]

Respiratory-related alterations in the autonomic nervous system also impact atrioventricular nodal function. For instance, the Valsalva maneuver, commonly used to terminate supraventricular tachycardia involving the atrioventricular node in its circuit, underscores this influence. However, the effects of respiration on atrioventricular nodal conduction, as reflected in AH intervals, remain incompletely understood. In the presented case, baseline observations revealed a prolongation of the AH interval during expiration and a shortening during inspiration. However, AH intervals exhibited no fluctuations after the administration of isoproterenol. This dissociation supports the hypothesis that autonomic activity modulates the fluctuation in the AH interval.

In contrast to normal respiration,[4] parasympathetic nerve activity increases during inspiration under positive pressure ventilation. Despite the administration of noninvasive positive pressure ventilation in this patient, a normal response to respiratory variability in autonomic activity was observed, characterized by AH interval prolongation during expiration and shortening during inspiration. This observation may be attributed to the time required for autonomic nervous activity to exert its influence on atrioventricular nodal function.

Notably, while respiratory-related fluctuations in AH intervals were evident during atrial pacing at a higher rate, such fluctuations were absent during atrial pacing at a lower rate or in sinus rhythm. This implies that respiratory-related fluctuations in AH intervals result from alterations in the refractory period of the atrioventricular node owing to respiration. An enhancement of parasympathetic nervous activity owing to expiration might have lengthened the refractory period of the fast atrioventricular nodal pathway, leading to a transition to the slow atrioventricular pathway during atrial pacing at a higher rate. Furthermore, the substantial fluctuation of the AH interval owing to respiration ceased after slow pathway ablation, indicating a significant role played by the interaction between the fast and slow pathways in this fluctuation. The underlying mechanism responsible for the marked respiratory-induced changes in the AH interval observed in this case remains unclear. However, it is hypothesized that an enhanced sensitivity of the pulmonary stretch receptors might play a significant role.

To our knowledge, this case report represents the first instance where careful monitoring of the respiratory detection sensor demonstrated significant respiratory-related fluctuations in AH intervals.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.04.008.

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